Volume 3, Issue 10, October - 2025 ISSN (E): 2938-3811

THE STATE OF THE BLOOD AND IMMUNITY SYSTEM IN ATHLETES DURING THE PROCESS OF ADAPTATION TO PHYSICAL ACTIVITIES

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Abstract
Intense physical activity during sports training can negatively impact an athlete's immune system, contributing to the development of secondary immunodeficiency states. These states are characterized by a decrease in the number of immunocompetent cells in the peripheral blood, suppression of the functional activity of T-lymphocytes, decreased levels of interleukins and interferons, disruption of cooperative cellular interactions, and the emergence of autoaggressive clones of B-lymphocytes, leading to autoimmunization. Imbalances in the athlete's immune system can be a pathogenetic mechanism for the development of infectious, tumor, autoimmune, and allergic diseases. Therefore, immune dysfunction is considered a critical factor limiting an athlete's performance. It is known that the intensity and focus of sports training cause biochemical changes not only in muscles and internal organs but also have a significant impact on blood composition and the volume of circulating blood volume. The bone marrow responds to changes in the body's needs under environmental conditions, thereby ensuring the body's adaptation to hypoxia, infection, and blood loss. In response to specific stimuli for each hematopoietic cell line, the proliferative and functional activity of the corresponding colony-forming cells (CFCs) increases. In adulthood, the compensatory responses of all hematopoietic cell lines and the secretion of regulatory hematopoietic cytokines are most appropriate to the current stimulus: hypoxia, infection, and blood loss. Strong stimuli nonspecific to hematopoietic tissue also trigger a response in the blood system, enhancing the body's resistance to environmental factors.

Introduction

The aim of the study was to identify the characteristics of adaptive changes in the activity of the circulatory and immune systems in athletes depending on seasonal environmental conditions during adaptation to aerobic physical activity.

Objectives of the Study

To compare indicators of the blood and immune systems in athletes with different aerobic exercise patterns throughout the year. To study patterns of seasonal

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changes in blood and immune system indicators in qualified athletes with different aerobic exercise patterns throughout the year.

Study Results

Significant differences in blood system parameters are known to exist among residents of different climatic and geographical regions. Thus, with increasing latitude, the average Hb level in the population increases, while the Er content in the blood decreases. Furthermore, a decrease in temperature and insolation in a geographic area is accompanied by a decrease in the average Er content in the population's blood, an increase in hemoglobin (Hb) content, as well as an increase in the average Er volume and the Hb content within them. With decreasing environmental temperature, adaptive changes in the erythron system are aimed at increasing the oxygen-carrying capacity of the blood.

The level of the minute volume of blood circulation (MOC) is affected by the size of the body surface, therefore, for its comparative assessment, the cardiac index is determined. During light work, the MOC increases to 10-15 liters and increases due to an increase in systolic volume and an increase in heart rate. A further increase in the MOC with increasing work intensity is due to an increase in heart rate. An increase in heart rate during physical work above 180-200 beats per minute is not accompanied by an increase in the MOC. In trained people, during intense muscular activity, the MOC reaches 30-35 liters or more. In endurance athletes, the MOC at rest is -5.68 l/min, at maximum load - 33.41 l/min, varying from 25 to 42 l/min. The resting heart rate of endurance athletes is 56-71 bpm, sometimes decreasing to 29-34 bpm. During intense physical exertion, the heart rate of qualified athletes increases to 185-200 bpm. In race walkers, the heart rate during physical exertion is 150-180 bpm. Cross-country skiers exhibit pronounced bradycardia at rest, reaching 32-45 bpm in men. Resting bradycardia in qualified athletes is a consequence of increased vagal influence on the myocardium. During a race, the heart rate of cross-country skiers can vary from 140-200 bpm depending on the terrain and speed.

Blood pressure (BP) is an important integral indicator of the functional state of the cardiovascular system. Most athletes have normal BP levels. During the recovery period after intense physical activity, some athletes may experience elevated BP, while others may experience a decrease. A persistent increase in BP in athletes is a symptom of overfatigue or overexertion due to inadequate individual physical activity dosage and an improperly organized training process, and may also indicate the early signs of a developing disease. A persistent decrease in BP in athletes may also be a sign of pathology. According to A.G. Dembo, in the vast majority of athletes examined, low BP is associated with the presence of foci of chronic infection or overfatigue. The incidence of hypotension in athletes is related to their sport specialization. During physical activity, arterial blood pressure rises to 150-200 mmHg, depending on the intensity of the work and the individual's characteristics. Under the influence of physical activity, the functional state of the artery changes. In particular, arterial impedance and vascular elasticity increase, while peripheral resistance decreases almost threefold. Total peripheral vascular resistance (TPVR) at rest averages 1698 dyn. sec / cm"5, and under load - 590 dyn. sec / cm"5. Such changes in TVR optimize the work of the cardiovascular system during physical activity. At rest, TVR in athletes averages 1400-2500 dyn. sec / cm"5. During physical work, peripheral vascular resistance decreases: during light work it is 1000 dyn. sec / cm"5 and lower; during heavy work - 800 dyn. sec / cm"5, and during very heavy work from 600 to 800 dyn. sec / cm"5. A decrease in total peripheral vascular resistance (TVR) during physical exercise is due to the dilation of arterioles and capillaries in skeletal muscles. Increased systemic hemodynamics during physical exertion can be associated with a physiological increase in cardiac filling, which is accompanied by an increase in systolic blood volume. Due to this, an increase in cardiac output (IOC) during physical exertion ensures a more optimal ratio of cardiac output to heart rate. Trained athletes experience an increase in IOC due to the mechanism of sinus arrhythmia. An increase in cardiac volume during intense and prolonged muscular activity indirectly reflects an increase in cardiac functional reserve; therefore, the highest cardiac volumes are observed in endurance athletes. Their peak cardiac volumes are observed during the competitive period at a higher level of training. Within a certain range of physiological values, a direct correlation is observed between cardiac volume and the level of athletic performance in endurance sports.

Myocardial hypertrophy can be either physiological or pathological. In pathological myocardial hypertrophy, there is a disproportion between the increase in cardiorespiratory performance and the dynamics of athletic performance. In physiological cardiac enlargement, there is a direct correlation:

the larger the cardiac volume, the higher the athletic performance and aerobic capacity.

Greater functional reserve and circulatory system performance ensure increased aerobic performance, as evidenced by increased maximum oxygen consumption in athletes. In trained athletes, heart volume is largely determined by sport specialization. For example, the largest hearts are observed in endurance athletes. Structural changes in the heart in endurance athletes are achieved through prolonged exertion, which requires a large volume of circulating blood. The strength and rate of heart contractions (HR) in athletes are regulated by biologically active substances, as well as the ionic composition of the interstitial fluid. Biologically active substances—hormones, peptides, mediators, and metabolites—have diverse effects on the strength and rate of heart contractions. Increased sympathetic activity during physical exertion is mediated by increased catecholamine secretion, while decreased vagal tone is accompanied by a decrease in acetylcholine levels. Thus, norepinephrine promotes an increase in heart rate by increasing the permeability of the sinoatrial node cell membrane to ions, promoting membrane depolarization, increasing the rate of diastolic depolarization, and decreasing the atrioventricular delay time. Furthermore, norepinephrine increases the force of cardiac contraction by increasing the permeability of the cardiomyocyte membrane to Ca^{2+} ions, enhancing its penetration into the cell, and increasing the amplitude of the action potential during the plateau phase. Furthermore, by increasing membrane permeability to ions, norepinephrine promotes increased myocardial excitability and conductivity.

The parasympathetic influence of the autonomic nervous system (ANS) is enhanced by increased secretion of acetylcholine (ACh). Unlike norepinephrine, ACh exerts a local and short-term effect on cardiac function, being rapidly broken down by acetylcholinesterase ACh reduces heart rate by increasing the permeability of the sinoatrial node membrane to K^{+} ions, resulting in hyperpolarization of the membrane , a decrease in the rate of diastolic depolarization, and an increase in atrioventricular delay time. ACh-induced reduction in myocardial contractility is associated with decreased cardiomyocyte membrane permeability to Ca^{2+} ions, which leads to a decrease in the action potential duration due to a shortened plateau phase. Increased cardiomyocyte membrane permeability to K^{+} ions under the influence of ACh leads to an increase in the stimulation threshold and a decrease in excitability and conductivity. This effect of ACh on myocardial function is most pronounced in athletes at rest. An increase in physical work intensity to a certain limit is accompanied by a synchronous increase in heart rate and contractility (chronoinotropic effect). This phenomenon is based on an increase in $Ca2+$ ion levels in the interfibrillar space due to the fact that, with an increase in heart rate, some of these ions fail to reenter the cardiomyocyte membrane, while a new influx of $Ca2+$ ions from the endoplasmic reticulum enters the intercellular space. This results in higher levels of extracellular $Ca2+$ than with a slow heart rate.

Humoral regulation of cardiac activity is mediated by hormones, peptides, mediators, and metabolites, as well as interstitial fluid ions, which exert varying effects on the strength and rate of heart contractions. Activation of the sympathoadrenal system during emotional arousal and physical exertion leads to an increase in catecholamine levels in the blood, resulting in increased heart rate and strength. Catecholamines enhance myocardial activity through β-adrenergic receptors by increasing the permeability of cell membranes for Ca ions, which promotes their mobilization from intracellular depots and an increase in the flow of ions from the intercellular space. Adrenaline, acting on β-adrenergic receptors of cardiomyocytes, leads to the activation of the adenylate cyclase mechanism, accelerating the formation of cyclic adenosine monophosphate (cAMP), which activates phosphorylase. Phosphorylase provides the myocardium with energy by breaking down intracellular glycogen. Corticosteroids, angiotensin, and vasopressin increase the force of heart contractions. Thyroxine increases heart rate and the sensitivity of the heart to sympathetic influences. The mechanism of action of hormones on the heart is realized through the activation of adenylate cyclase, which accelerates the formation of cAMP.

Metabolites stimulate cardiac function. Prostaglandins increase the force and rate of heart contractions by increasing coronary blood flow and enhancing myocardial metabolism, while kinins enhance cardiac activity by releasing coronary arteries.

$Ca2+$ ions enhance cardiac contraction by improving electromechanical coupling, activating phosphorylase, and increasing ATP breakdown. With an increase in $K+$ ion levels in the blood and interstitial fluid by 1.5-2 times, the excitability and conductivity of cardiomyocytes increases, as their resting potential decreases due to a decrease in the intracellular and extracellular $K+$ ion concentration gradient.

Volume 3, Issue 10, October - 2025 ISSN (E): 2938-3811

With an increase in extracellular K+ levels by 2 times or more, the excitability and conductivity of cardiomyocytes decreases due to a significant increase in membrane potential and the development of hyperpolarization. With a decrease in extracellular potassium ion levels below normal (4 mmol/L), pacemaker activity increases, heterotropic excitation foci are activated, which leads to cardiac arrhythmia.

The excitability of myocardial cells has the same bioelectrical nature as that of striated muscle. The presence of a charge on the membrane here is also ensured by the difference in K+ ion concentrations near the inner and outer surfaces and the membrane's selective permeability to them. At rest, K+ ions diffuse out of the cell, creating a positive charge on the surface. The inner side of the membrane becomes electronegative relative to the outer side. In pacemaker cells, the membrane potential, decreasing to a critical level, leads to the generation of an action potential. Normally, the heart rhythm is set by pacemaker cells of the sinoatrial node, whose membrane potential, having reached its maximum value during diastole, begins to gradually decline (the phase of slow spontaneous diastolic depolarization). This depolarization continues until the membrane potential reaches a critical level, at which point an action potential is generated. The slow depolarization phase is associated with a decrease in potassium and an increase in sodium and calcium membrane conductance during diastole, accompanied by a simultaneous decrease in the activity of the electrogenic sodium pump. At the beginning of diastole, membrane permeability to K+ ions increases, and the resting potential reaches its maximum diastolic value. Then, membrane permeability to potassium decreases, causing the membrane potential to decrease to a critical level. The simultaneous decrease in membrane permeability to sodium and calcium leads to the entry of these ions into the cell and the generation of an action potential. A decrease in electrogenic pump activity further reduces sodium efflux from the cell, facilitating membrane depolarization and the onset of excitation.

A decrease in pH, a reduction in blood oxygen levels, and an increase in carbon dioxide stimulate cardiac activity. An increase in body temperature increases heart rate, while a decrease in temperature decreases it. Acidification of the environment leads to stimulation of sympathetic centers and increased myocardial activity. Vascular tone is regulated by local and central mechanisms. The local mechanism regulates blood flow in a specific organ or tissue area, while

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the central mechanism maintains blood pressure and systemic circulation. Sympathetic innervation of blood vessels causes vasoconstriction through the interaction of norepinephrine with alpha- adrenergic receptors. The presence of cholinergic endings secreting ACh in sympathetic fibers ensures dilation of the blood vessels of the heart and skeletal muscles. Hormones, metabolites, and neuropeptides participate in the regulation of vascular health; they accumulate in the interstitial space, diffuse to the vascular wall, and have a direct effect on smooth muscle tone. Metabolites cause a weakening of the tone of the smooth muscle cells and vasodilation, which is accompanied by hyperemia and increased oxygen and glucose delivery. Vasodilation of skeletal muscle vessels is provided by CO2, lactate, pyruvic acid, ATP, adenosine diphosphate (ADP), adenosine monophosphate (AMP), nitric oxide, potassium and sodium ions, etc. Low concentrations of K+ ions in the blood cause vasodilation, and at higher levels, vessels constrict. Ca2+ ions induce arterial vasoconstriction, and ions are dilators. In addition, bicarbonates, lactate, nitrates, sulfates, chlorides, bisphosphates, and hydrochloric and nitric acid ions have a vasodilatory effect. It is generally accepted that humoral vasodilation in the body exceeds sympathetic vasoconstriction it causes vasodilation upon contact with adrenergic receptors, while at high concentrations, it causes vasoconstriction. In physiological concentrations, adrenaline excites predominantly (adrenergic receptors) and causes relaxation of vascular smooth muscles, especially those where (adrenergic receptors) predominate:skeletal muscles, brain, heart. This is due to the fact that the sensitivity of (adrenergic receptors) is higher than that of alpha- adrenergic receptors, therefore, adrenaline in physiological concentrations activates only (adrenergic receptors), which leads to vasodilation. Usually, endogenous adrenaline causes dilation of blood vessels, and a sharp increase in the concentration of adrenaline under stressful influences leads to vasoconstriction through the simultaneous activation of alpha- and (adrenergic receptors). Under extreme influences on the body, the concentration of adrenaline in the blood increases tens of times and its interaction with alpha- adrenergic receptors of vessels with a predominance of vasoconstrictor reactions is possible, especially in the skin, digestive organs and lungs, which have a large number of alpha-adrenergic receptors.

At the same time, adrenaline increases stroke volume and heart rate, resulting in systemic arterial pressure (at rest, during moderate physical exertion, and during emotional arousal) not changing significantly under the influence of adrenaline. Under these conditions, the main circulatory effect of adrenaline is the redistribution of cardiac output and the maintenance of blood flow intensity in skeletal muscles, the heart, and the brain. Norepinephrine also has a vasoconstrictor effect; when exposed to extreme factors, it interacts with α-adrenergic receptors, causing an increase in vascular tone, total peripheral vascular resistance, and blood pressure. The opposing effects of adrenaline and norepinephrine on vascular smooth muscle are due to the presence of α- and β-adrenergic receptors. Norepinephrine primarily interacts with α- adrenergic receptors, and adrenaline with β-adrenergic receptors. Atrial natriuretic hormone, synthesized by the secretory cells of the right atrium and ventricles when the heart rate increases, exerts a vasodilatory effect. Upon entering the bloodstream, it causes vasodilation by relaxing smooth muscle cells via cAMP, leading to a decrease in blood pressure and an increase in the excretion of ions and water through diuresis.

Hemodynamics largely depends on the condition of the smooth muscle cells of the vascular wall and the secretory activity of endothelial cells. The vascular endothelium has the ability to synthesize and secrete factors that cause relaxation and contraction of vascular smooth muscles in response to various stimuli. The involvement of the endothelium in the regulation of vascular tone is generally recognized. According, the endothelium plays a key role in ensuring optimal functioning of the body, as well as in ensuring adequate adaptive processes to the action of various external factors, including extreme ones (systematic physical activity). Under the influence of physical activity, optimization of the functional state of the vascular endothelium is observed, which predetermines a reliable active reaction, in particular, due to a pronounced vasodilatation effect. The endothelium affects the lumen of blood vessels, regulating arterial pressure in the vessels and blood flow. Endothelial cells' sensitivity to blood flow velocity is expressed by their release of a vascular smooth muscle relaxant. This relaxant causes arterial dilation. The signal transmission from endothelial cells to vascular smooth muscle structures during arterial dilation in response to increased blood flow is chemical in nature. Thus, arteries continuously regulate their lumen in response to blood flow velocity, stabilizing arterial pressure. During hypoxia, the response of smooth muscle cells to catecholamines is significantly reduced, which is associated with decreased membrane excitability due to decreased

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activity of ionic mechanisms. During hyperoxia and stimulation of lipid peroxidation (LPO) formation in the vessel wall, metabolic disturbances occur, with collagen and hyaluronic acid being destroyed.

According to modern concepts, heart rate variability (HRV) is one of the most important indicators of the body's functional state. Therefore, studies on the clinical diagnostics of myocardial sinus rhythm wave variability analysis have gained widespread popularity thanks to the non-invasive, highly dynamic, informative assessment of cardiovascular function. Electrocardiogram (ECG) interpulse interval variability is one of the most important markers of autonomic nervous system (ANS) activity and represents changes in the duration of intervals of successive heartbeat cycles over a period of time. High-frequency respiratory waves in the HF range have been shown to be markers of trophotropic parasympathetic regulatory mechanisms, low-frequency (LF) waves are markers of sympathetic influences, and very low-frequency (VLF) waves are markers of cerebral ergotropic mechanisms. When studying the autonomic regulation of myocardial activity, normalized wave power is often used as a percentage ratio of the power of a specific wave spectrum to the sum of the powers of fast, slow, and very low frequency waves. Normally, in the supine position, the proportion of high-frequency waves (HF) is 15-25%, the proportion of vasomotor waves is 15-40%, and the proportion of very low frequency waves is 15-30%. A state of autonomic regulation of myocardial activity characterized by a decrease in the proportion of the (HF) component and an increase in the proportion of (LF) waves indicates increased sympathetic influences.

Thus, physical activity leads to changes in hemodynamic parameters in athletes. For example, during standard physical activity, athletes experience a less pronounced increase in cardiac output, cardiac output, heart rate, and linear blood flow velocity compared to non-athletes, while during maximal physical activity, the increase is more pronounced.

Previously, a study of the dynamics of circulatory parameters in cross-country skiers revealed a decrease in average cardiac index values in spring and summer compared to autumn and winter. In the former case, this was due to an increase in heart rate, and in the latter, to an increase in SV. Characteristically, in wrestlers with increased anaerobic physical activity in spring and summer, average cardiac index values significantly decreased in summer due to a decrease in SV and HR. At the same time, their UPSS level significantly increased. In addition to physical activity, environmental conditions influence hemodynamics. In spring and autumn, vascular tone has a greater impact on cardiac function than in other seasons. In summer, glycolysis provides energy for cardiac contractility, while in winter, lipolysis provides energy. Exposure to strong irritants in spring and autumn, coupled with seasonal morphofunctional myocardial hyperfunction, leads to desynchronization with a predominance of irreversible processes, destruction of the mitochondria, and a decrease in cardiac contractility. The organism's existence in changing environmental conditions is determined by the interconnections between cells, tissues, organs, and functional systems, mediated by signaling molecules of various types. Effector cells perceive the effects of mediators, hormones, and growth factors through specific structures called receptors, which quantitatively and qualitatively transform the received information into an appropriate response. Therefore, studying the functional properties of receptors, their number, and their sensitivity to biological regulators is of interest to physiologists, biochemists, and pharmacologists. Prolonged or repeated exposure to high or low temperatures in humans and animals leads to adaptive restructuring of all regulatory systems. Adaptation to cold causes fundamentally different changes in the main parameters of α- and β-adrenergic reactions. It is known that the earliest reaction to cold is the activation of the sympathoadrenal system, which is one of the components of the stress response. The concentration of norepinephrine and adrenaline in the blood increases, which enhance thermogenesis and promote contraction of peripheral vessels, thereby reducing heat loss. At the same time, CA, acting on postsynaptic α1- and ββ-adrenergic receptors of the smooth muscle cells of the arterial wall, cause vasoconstriction of blood vessels, and through β2-adrenergic receptors ensure vasodilation of blood vessels. Studying the effect of natural factors, including cold, on the regulation of receptors of the cardiovascular system is of fundamental importance, since this may possibly allow for preventive changes in the number of receptors using cold hardening.

Conclusions

Thus, in athletes, resting blood and immune system parameters may deviate significantly from physiological norms. Er and Hb levels are higher in athletes than in non-athletes ESR values in athletes may also briefly increase above normal without clinical signs of pathological development. Lc levels in athletes are within the lower limit of normal due to a decrease in Lf levels under the influence of stressful physical activity. Athletes with predominantly aerobic muscle energy production may experience increased Mn levels in the blood. Granulocyte functional activity parameters may be higher or lower than normal. T-lymphocyte levels are typically decreased, while B-lymphocyte levels are increased. Secretory and serum levels decrease significantly under the influence of inappropriate physical activity.

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