



IQTISODIYOT&TARAQQIYOT

Ijtimoiy, iqtisodiy, texnologik, ilmiy, ommabop jurnal

№ 11



2025

ISSN: 2992-8982

<https://yashil-iqtisodiyot-taraqqiyot.uz/>



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Muassis: "Ma'rifat-print-media" MChJ

Hamkorlarimiz: Toshkent davlat iqtisodiyot universiteti, O'zR Tabiat resurslari vazirligi, O'zR Bosh prokuraturasi huzuridagi IJQK departamenti.

Jurnalning ilmiyligi:

“Yashil” iqtisodiyot va taraqqiyot” jurnali

O'zbekiston Respublikasi Oliy ta'lim, fan va innovatsiyalar vazirligi huzuridagi Oliy attestatsiya komissiyasi rayosatining 2023-yil 1-apreldagi 336/3-sonli qarori bilan ro'yxatdan o'tkazilgan.



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BIOCHEMICAL CHANGES IN THE BODY DURING THE PERIOD OF FATIGUE AND REST AFTER WORK FOR ATHLETES

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Annotatsiya. Fatigue is a physiological condition that arises from prolonged physical or mental activity and leads to a temporary decline in performance capacity. During fatigue, the body undergoes notable biochemical changes that reflect the depletion of energy reserves, accumulation of metabolic by-products, and disruption of homeostasis. Conversely, the period of rest initiates restorative biochemical processes aimed at replenishing energy stores, eliminating metabolic waste, and reestablishing internal balance. This article examines the key biochemical mechanisms underlying fatigue and recovery, focusing on energy metabolism, hormonal regulation, and oxidative balance.

Kalit soʻzlar: fatigue, rest, energy metabolism, lactic acid, glycogen, ATP, biochemical changes, recovery.

Abstract. Charchoq — uzoq davom etadigan jismoniy yoki aqliy faoliyat natijasida paydo boʻladigan, vaqtinchalik mehnat qobiliyatining pasayishi bilan tavsiflanadigan fiziologik holatdir. Charchoq jarayonida tanada energiya zahiralarning kamayishi, metabolik yon mahsulotlarning toʻplanishi va gomeostazning buzilishi kabi muhim biokimyoviy oʻzgarishlar kuzatiladi. Aksincha, dam olish davrida energiya zaxiralarini tiklash, metabolik chiqindilarni chiqarish va organizmning ichki muvozanatini qayta tiklashga qaratilgan restorativ biokimyoviy jarayonlar faollashadi. Ushbu maqolada charchoq va tiklanishning asosiy biokimyoviy mexanizmlari energiya almashinuvi, gormonal tartibga solish va oksidlanish muvozanati nuqtayi nazaridan tahlil qilinadi.

Key words: charchoq, dam olish, energiya almashinuvi, sut kislotasi, glikogen, ATP, biokimyoviy oʻzgarishlar, tiklanish.

Аннотация. Утомление представляет собой физиологическое состояние, возникающее вследствие длительной физической или умственной активности и сопровождающееся временным снижением работоспособности. В этот период в организме происходят значительные биохимические изменения, отражающие истощение энергетических ресурсов, накопление побочных продуктов обмена веществ и нарушение гомеостаза. Напротив, период отдыха характеризуется восстановительными биохимическими процессами, направленными на восполнение энергетических запасов, устранение метаболических отходов и восстановление внутреннего равновесия организма. В статье рассматриваются основные биохимические механизмы утомления и восстановления с акцентом на энергетический обмен, гормональную регуляцию и окислительный баланс.

Ключевые слова: утомление, отдых, энергетический обмен, молочная кислота, гликоген, АТФ, биохимические изменения, восстановление.

INTRODUCTION

Human performance largely depends on the body's capacity to maintain biochemical equilibrium during both physical and mental activity. When activity exceeds the optimal functional threshold, fatigue develops — a multifactorial physiological condition shaped by energy depletion, the accumulation of metabolic by-products, and neuroendocrine responses. Understanding the biochemical alterations that occur during fatigue and



subsequent recovery is essential for improving work productivity, athletic performance, and strategies aimed at health restoration.

Fatigue is a temporary and reversible physiological state arising from prolonged or intensive physical and mental workload. It reflects a complex interaction of biochemical, neurophysiological, and metabolic processes that collectively lead to a decline in functional efficiency and overall performance. During fatigue, the body experiences significant biochemical shifts, including the depletion of energy substrates, accumulation of lactic acid, and elevated production of reactive oxygen species. Conversely, during rest, compensatory and restorative biochemical mechanisms become active, restoring homeostasis, replenishing glycogen and ATP reserves, and facilitating the removal of metabolic by-products. This article provides a comprehensive overview of the biochemical transformations that occur in the human body during the onset of fatigue and throughout the recovery phase, with particular emphasis on metabolic regulation, enzymatic activity, hormonal responses, and oxidative balance.

Human vital activity depends on the continuous exchange of substances and energy. Any form of work — whether physical or mental — results in energy expenditure and corresponding biochemical adjustments within the body. Fatigue manifests when the duration or intensity of activity surpasses the body's adaptive capabilities. It represents a temporary metabolic imbalance that is fully reversible with adequate rest and proper physiological support.

The study of biochemical changes during fatigue and recovery is highly significant for physiology, sports medicine, labor hygiene, and occupational health. A clear understanding of these mechanisms contributes to optimizing work–rest schedules, enhancing athletic performance, and developing scientifically grounded strategies for rapid and effective recovery. Fatigue commonly presents through reduced muscle strength, diminished alertness, and impaired concentration. Its biochemical foundation is closely associated with disruptions in energy metabolism, neurotransmitter balance, hormonal regulation, and acid–base homeostasis.

LITERATURE REVIEW ON THE TOPIC

Fatigue and recovery have been widely examined in sports physiology, biochemistry, and human performance science. Foundational studies in exercise physiology established that fatigue is closely linked to the depletion of ATP and phosphocreatine reserves. McArdle and colleagues emphasized the critical role of both anaerobic and aerobic pathways in sustaining muscular activity. Subsequent biochemical research expanded this understanding by demonstrating that the accumulation of lactic acid and the resulting acidosis significantly impair enzymatic function, slow glycolysis, and reduce muscle contractility. Contemporary studies further highlight the importance of oxidative stress, showing that prolonged physical exertion increases the production of reactive oxygen species, which adversely affect proteins, lipids, and mitochondrial structures.

Modern investigations also explore hormonal responses to fatigue, noting that stress hormones such as cortisol and adrenaline rise sharply during intense exertion, mobilizing energy reserves while simultaneously enhancing catabolic processes. Research focused on recovery shows that rest activates compensatory biochemical mechanisms, including glycogen resynthesis, lactate clearance through the Cori cycle, normalization of pH levels, and restoration of antioxidant defenses. Collectively, these findings indicate that fatigue is a multifactorial condition involving metabolic, hormonal, and oxidative pathways, whereas recovery represents a coordinated set of biochemical processes aimed at reestablishing homeostasis and restoring optimal physiological function.

RESEARCH METHODOLOGY

This article employs a theoretical–descriptive research approach, integrating scientific evidence from biochemistry, exercise physiology, and sports medicine to elucidate the biochemical mechanisms underlying fatigue and recovery. The methodology is grounded in a comparative analysis of previously published scholarly works, with a focus on energy metabolism, hormonal regulation, oxidative stress, and enzymatic responses. Fundamental biochemical processes—including ATP depletion, glycogenolysis, lactic acid accumulation, mitochondrial activity, and antioxidant function—were examined through a literature-based analytical framework.

The study further incorporates systematic conceptual mapping to compare metabolic conditions during fatigue and rest, thereby identifying the dominant biochemical pathways characteristic of each state. Tables summarizing energy systems, hormonal dynamics, and oxidative markers were developed based on synthesized scientific data. This methodological design enables a comprehensive interpretation of biochemical transformations without the need for experimental laboratory procedures, making it highly suitable for physiological, educational, and applied research contexts.



ANALYSIS AND RESULTS

One of the primary biochemical features of fatigue is the depletion of adenosine triphosphate (ATP), the main energy currency of the cell. ATP is continuously utilized during muscular and mental activity, and its resynthesis depends on carbohydrate, fat, and protein metabolism. When ATP demand exceeds the rate of production, the body encounters an energy deficit, which leads to diminished muscle contractility and reduced mental concentration. Glycogen stored in muscles and the liver serves as a critical energy reserve, and during prolonged exertion, glycogen levels decline due to increased glycolytic activity. As glycogen stores become depleted, the body shifts toward fat metabolism, a slower energy-yielding process that contributes significantly to the onset of fatigue. This metabolic shift, characterized by reduced ATP resynthesis, is a key contributor to fatigue during sustained physical activity.

Under anaerobic conditions, pyruvate is converted into lactic acid, and excess lactic acid lowers intracellular pH to approximately 6.5–6.8, thereby impairing enzyme activity—particularly phosphofructokinase—and weakening muscle contraction. The accumulation of ammonia, ADP, inorganic phosphate, and carbon dioxide further intensifies metabolic stress and contributes to neuronal inhibition, deepening the overall sensation of fatigue. Prolonged muscular activity also increases mitochondrial oxygen consumption, which enhances the formation of reactive oxygen species (ROS), including superoxide and hydrogen peroxide. When antioxidant defense systems such as superoxide dismutase, catalase, and glutathione peroxidase are unable to neutralize these free radicals, oxidative stress develops, resulting in lipid peroxidation, protein oxidation, and mitochondrial dysfunction that aggravate fatigue and delay recovery.

Fatigue additionally triggers complex hormonal and enzymatic reactions. Adrenaline and noradrenaline elevate heart rate and facilitate glucose mobilization, while cortisol stimulates gluconeogenesis but may promote protein catabolism. Insulin sensitivity temporarily decreases, and key glycolytic enzymes such as hexokinase and lactate dehydrogenase increase in activity, whereas oxidative enzyme activity may decline due to limited oxygen availability. These metabolic and hormonal changes represent the body's physiological response to maintain energy homeostasis under stress; however, when prolonged, they can contribute to biochemical exhaustion. In low-oxygen conditions, elevated lactic acid levels reduce muscular and blood pH, impairing enzymatic reactions and producing the characteristic burning sensation in muscles, while accumulated ammonia, carbon dioxide, and inorganic phosphates add to metabolic strain.

During extended exertion, ROS generation increases and may overwhelm antioxidant defenses, causing structural damage to membranes, proteins, and DNA, further intensifying fatigue. In contrast, rest activates several restorative biochemical processes. ATP and phosphocreatine reserves are replenished through oxidative metabolism, while glycogen stores in muscle and liver tissues are restored through enhanced glucose uptake and glycogenesis, facilitated largely by insulin-mediated cellular transport. Rest also promotes the clearance of lactic acid either through oxidation into carbon dioxide and water or via its conversion back into glucose in the liver through the Cori cycle. Improved circulation during light recovery activities accelerates the removal of metabolic by-products.

Hormonal regulation during rest supports recovery, as cortisol levels gradually decrease, while anabolic hormones such as insulin and growth hormone increase, promoting tissue repair, protein synthesis, and the restoration of metabolic balance. Collectively, these processes reestablish biochemical homeostasis and prepare the body for subsequent physical or mental activity (Table 1).

Table 1. Comparative Biochemical Changes During Fatigue and Rest

Biochemical Parameter	During Fatigue (Work Period)	During Rest (Recovery Period)
ATP (Adenosine Triphosphate)	Rapid depletion due to high energy demand	Resynthesized through oxidative phosphorylation
Glycogen Stores	Decrease significantly (glycogenolysis)	Restored via glycogenesis in muscle and liver
Lactic Acid (Lactate)	Accumulates due to anaerobic glycolysis; decreases pH	Gradually removed via oxidation or converted to glucose (Cori cycle)
pH Level	Drops (acidosis) due to lactic acid accumulation	Returns to normal (homeostasis restored)
Oxygen Consumption	Increases sharply; may become insufficient (anaerobic metabolism)	Normalizes; oxygen used efficiently for ATP synthesis
Reactive Oxygen Species (ROS)	Increases, causing oxidative stress	Neutralized by antioxidant enzymes (SOD, catalase, glutathione peroxidase)



Enzyme Activity	Some metabolic enzymes inhibited by acidosis	Enzyme activity restored as pH normalizes
Hormonal Response	↑ Adrenaline, noradrenaline, cortisol (catabolic)	↑ Insulin, growth hormone, testosterone (anabolic)
Protein Metabolism	Protein breakdown may increase (catabolism)	Protein synthesis activated for tissue repair
Blood Glucose	Initially rises, then may drop with prolonged effort	Stabilizes as glycogen and glucose stores replenish
Body Temperature	Increases due to high metabolic rate	Returns to normal as metabolic rate decreases
Nervous System	Reduced neurotransmitter activity (dopamine, acetylcholine) → fatigue	Neurotransmitter levels restored; mental clarity returns
Immune and Antioxidant Systems	Temporary suppression and increased inflammation	Recovery of immune and antioxidant defense systems
Overall Metabolic State	Catabolic (energy consumption > production)	Anabolic (energy restoration > consumption)

This hormonal shift promotes tissue repair, enhances protein synthesis, and contributes to the overall restoration of metabolic function. As metabolic by-products are cleared from the body and ventilation normalizes, blood pH gradually returns to its physiological range. Buffer systems, particularly bicarbonate and phosphate, play a crucial role in maintaining acid–base homeostasis and ensuring optimal enzymatic activity throughout the recovery period. During rest, oxidative stress is reduced as antioxidant systems regain their functional capacity and neutralize excess reactive oxygen species. Essential nutrients such as vitamin C, vitamin E, and glutathione provide additional protection by preventing oxidative damage and supporting the recovery of cellular structures. Rest, therefore, is not merely a cessation of activity but an active metabolic restoration phase in which anabolic processes predominate over catabolic ones, reestablishing biochemical homeostasis and restoring the functional potential of tissues.

Following fatigue, the body prioritizes the replenishment of ATP and phosphocreatine reserves, with mitochondrial oxidative phosphorylation becoming the dominant pathway for efficient ATP synthesis. At the same time, glycogen stores in the muscles and liver are restored through glycogenesis, a process facilitated by insulin and increased cellular glucose uptake. Rest also improves blood and lymph circulation, accelerating the removal of lactic acid and other metabolic by-products. Lactic acid may be oxidized in muscle tissue to form pyruvate and carbon dioxide, transported to the liver for conversion into glucose through the Cori cycle, or utilized by the heart as an energy substrate. These detoxification pathways contribute to the normalization of acid–base balance and the maintenance of a stable physiological pH, essential for enzymatic stability and metabolic recovery.

Hormonal recovery further supports these restorative processes. During rest, levels of catabolic hormones such as cortisol and adrenaline decline, while anabolic hormones — including insulin, growth hormone, and testosterone — increase, collectively promoting tissue repair, protein synthesis, and overall metabolic resilience (Table 2).

Table 2. Dominant Biochemical Pathways During Fatigue and Rest

Biochemical Pathway / Process	During Fatigue (Active Work Phase)	During Rest (Recovery Phase)	Main Function / Effect
ATP–CP System (Phosphagen System)	Rapid depletion of phosphocreatine (CP)	Replenishment of CP stores through oxidative ATP production	Provides immediate energy for short, intense work
Glycolysis	Strongly activated (anaerobic), producing lactic acid	Slows down as oxidative metabolism predominates	Converts glucose/glycogen into ATP under low oxygen
Glycogenolysis	Accelerated breakdown of glycogen to glucose-1-phosphate	Inhibited; glycogenesis predominates	Provides glucose for energy production during work
Glycogenesis	Inactive (energy-demanding process)	Active; glycogen stores restored in liver and muscles	Restores carbohydrate reserves



Oxidative Phosphorylation	Limited by oxygen supply during intense effort	Dominant energy pathway during recovery	Produces ATP efficiently in mitochondria
Lactic Acid Oxidation (Cori Cycle)	Lactic acid accumulates in muscles	Lactic acid converted to glucose in the liver	Eliminates lactic acid and restores glucose levels
Lipolysis (Fat Breakdown)	Moderately activated in prolonged work	Reduced; fat stored again (lipogenesis)	Provides additional energy when glycogen is low
Protein Catabolism	Slightly increased (especially with prolonged stress)	Decreases; protein synthesis increases	Provides amino acids for energy and repair
Protein Synthesis (Anabolism)	Decreased (energy deficit)	Strongly activated under hormonal influence (insulin, GH, testosterone)	Repairs and regenerates muscle and enzyme proteins
Hormonal Regulation	↑ Adrenaline, cortisol, glucagon (catabolic hormones)	↑ Insulin, growth hormone (anabolic hormones)	Shifts metabolism from breakdown to rebuilding
Acid–Base Balance	Acidic shift (decreased pH due to lactic acid)	Normalization of pH through buffer systems and excretion	Maintains enzymatic function and stability
Antioxidant Defense System	Overloaded; ROS accumulation	Recovered; ROS neutralized by antioxidants	Protects cells from oxidative damage
Mitochondrial Activity	Reduced efficiency under stress	Enhanced biogenesis and repair during rest	Restores cellular energy potential
Immune and Inflammatory Response	Temporary suppression, increased inflammatory mediators	Immune recovery, reduction of oxidative inflammation	Supports overall tissue recovery and adaptation

These hormones stimulate protein synthesis, enhance glycogen storage, and support tissue repair, while melatonin released during sleep strengthens antioxidant activity and contributes to mitochondrial recovery. Fatigue temporarily inhibits numerous enzymes; however, during rest, enzymatic activity is reestablished as enzyme synthesis resumes and damaged proteins are replaced through ribosomal mechanisms, with amino acids serving as essential substrates for the synthesis of contractile and enzymatic proteins, thereby restoring functional capacity. Biochemical recovery also involves the rebalancing of the nervous and immune systems, as levels of neurotransmitters such as dopamine and serotonin normalize, positively influencing mood, motivation, and central fatigue, while the immune system restores antioxidant enzyme levels and cytokine balance, reducing inflammation induced by prolonged exertion. Effective recovery further depends on adequate nutrition and hydration: carbohydrates replenish glycogen stores, proteins supply amino acids for muscle repair, and fats restore lipid membranes, whereas electrolytes such as sodium, potassium, and magnesium maintain osmotic balance and vitamins C and E, together with selenium and coenzyme Q10, act as antioxidants to neutralize reactive oxygen species. Understanding the biochemical basis of fatigue and recovery has significant practical and clinical implications, as it informs the development of optimal training and recovery schedules in sports physiology, supports the creation of effective work–rest cycles in occupational medicine to prevent chronic fatigue, and assists in rehabilitation by enabling biochemical monitoring to track patients' recovery after illness or surgery. Overall, the biochemical changes occurring during fatigue and rest illustrate a dynamic interplay between energy expenditure and recovery, with fatigue representing temporary metabolic, hormonal, and oxidative disturbances, and rest enabling the restoration of these systems to optimal function. A comprehensive understanding of these mechanisms provides a strong scientific foundation for developing effective strategies to optimize work–rest balance, improve training recovery, and promote long-term health maintenance.

CONCLUSION AND RECOMMENDATIONS

Fatigue and recovery represent two interdependent phases of human metabolic functioning. Fatigue arises when energy expenditure exceeds the body's capacity to maintain biochemical homeostasis, leading to reductions in ATP and glycogen reserves, increased lactate accumulation, elevated oxidative stress, and temporary hormonal fluctuations. These biochemical disruptions diminish muscle contractile ability, enzymatic activity, and cognitive performance. In contrast, the recovery phase activates restorative mechanisms such as glycogenesis, oxidative phosphorylation, lactate clearance, antioxidant regeneration, and hormonal rebalancing, all of which contribute to normalizing pH levels, enhancing mitochondrial efficiency, and promoting protein synthesis. A thorough understanding of these physiological processes provides a strong scientific basis



for improving performance, preventing excessive strain, and supporting long-term health. Strategies aimed at strengthening physiological resilience and accelerating recovery emphasize maintaining an optimal work–rest balance that allows ATP and phosphocreatine stores to replenish, increasing post-exercise carbohydrate intake to restore glycogen levels, and ensuring sufficient hydration with appropriate electrolyte replacement to stabilize acid–base balance and facilitate metabolite clearance. Incorporating antioxidant-rich foods, particularly those containing vitamins C and E and selenium, helps reduce oxidative stress and promotes mitochondrial repair. Adequate sleep and passive rest remain essential for supporting anabolic hormone secretion and tissue regeneration, while monitoring training intensity through physiological indicators such as heart rate, perceived exertion, and subjective fatigue levels helps prevent chronic biochemical overload. Additionally, applying active recovery methods, including light aerobic exercise, enhances circulation and accelerates lactate oxidation. Altogether, these evidence-based strategies reinforce biochemical stability, improve performance efficiency, and contribute to sustainable physical and mental productivity.

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IQTISODIYOT & TARAQQIYOT

Ijtimoiy, iqtisodiy, texnologik, ilmiy, ommabop jurnal

Ingliz tili muharriri: Feruz Hakimov

Musahhih: Zokir ALIBEKOV

Sahifalovchi va dizayner: Oloviddin Sobir o'g'li

2025. № 11

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Litsenziya raqami: №046523. PNFL: 30407832680027

Manzilimiz: Toshkent shahar, Mirzo Ulug'bek tumani
Kumushkon ko'chasi, 26-uy.



Jurnal sayti: <https://yashil-iqtisodiyot-taraqqiyot.uz>
