Academe *Journal of Education & Psychology* *ISSN: 2249-040X*
Volume-16, (Special Issue: March 2026) *Peer Reviewed & Refereed Journal (IF: 6.25)*
PP: 257-263 *Journal Website:* www.academejournal.in

EXERCISE PHYSIOLOGY AND MOLECULAR ADAPTATION TO TRAINING IN SPORTS- A STUDY

Vishwanathaiah.S

College Director of Physical Education, Govt First Grade College For Womens Ramanagara

ABSTRACT

Exercise training in sports induces complex molecular signals and physiological changes, moving the body from an untrained to a trained state characterized by adaptations such as increased mitochondrial density and muscle hypertrophy. While master regulators like PGC-1α and mTORC1 are key, training specificity dictates the outcome, offering benefits beyond performance, including improved metabolic and cardiovascular health.

Keywords: Exercise; physical activity, cardiovascular disease, cardiorespiratory fitness, resistance exercise training, aerobic exercise training**.**

INTRODUCTION

Regular exercise training has widespread health benefits by positively affecting nearly all organ systems of the body. The mysteries of human physiology and the adaptive response to acute and chronic exercise training have largely been elucidated through exercise science. The field has a rich history of uncovering some of the limits of exercise performance in both health and disease. Exercise physiologists have studied physiological response to physical activity, exercise, sport, and athletic competition, whereas clinical exercise physiologists use exercise training/prescription in the prevention and rehabilitation of acute and chronic disease. For many, the notion that ‘‘exercise works’’ to improve functional work capacity and metabolic health is self-evident. Exercise training is a clinically proven, cost-effective, primary intervention that can delay, and in many cases prevent, the health burdens associated with metabolic disorders.

APPLICATION IN SPORTS PERFORMANCE

In elite sports, the "holy grail" is bridging the gap between the lab and the field. Coaches and sport scientists apply these molecular principles to manipulate training loads, recovery, and nutrition for a competitive edge.

- **Sequential Loading:** To maximize **mTOR** activation (growth), coaches often schedule resistance training in the morning and endurance training in the afternoon (or on separate days) to allow the molecular signals to peak without competing with **AMPK** (energy sensing).
- **The "Low-Glycogen" Strategy:** Endurance athletes sometimes utilize "train-low, compete-high" protocols. By training with low glycogen stores, they amplify the **AMPK** and **PGC-1α** signals, forcing the body to become more efficient at burning fat as fuel.
- **T:C Ratio:** Monitoring the ratio of **Testosterone to Cortisol** via blood or saliva. A significant drop in this ratio suggests the body is in a chronically catabolic state, prompting a "deload" week to allow hormonal recovery.
- **CK Levels:** Measuring **Creatine Kinase** provides a proxy for mechanical muscle damage, helping staff decide if an athlete is physically ready for another high-intensity session.

- **Leucine Threshold:** Athletes consume high-leucine protein (like whey) immediately post-resistance training to maximize **mTORC1** signaling for Muscle Protein Synthesis (MPS).
- **Nitric Oxide (NO) Boosters:** Using dietary nitrates (e.g., beetroot juice) to improve mitochondrial efficiency and blood flow, targeting the **HIF-1α** pathway for better oxygen economy.
- **ACE & ACTN3:** The "sprinter gene" (**ACTN3**) influences the production of α-actinin-3, a protein found in fast-twitch fibers. Knowledge of an athlete's molecular makeup can help "slot" them into the most compatible sporting discipline (e.g., power vs. endurance).

ACUTE PHYSIOLOGY RESPONSES TO EXERCISE

Acute physiological responses are the immediate, short-term changes that occur in the body to meet the increased metabolic and energy demands of a single bout of exercise. These responses last only for the duration of the activity and a short recovery period afterward.

1. Cardiovascular Responses

The heart and blood vessels work to deliver more oxygen and nutrients to active muscles while removing waste products like carbon dioxide.

- **Heart Rate (HR):** Increases linearly with exercise intensity up to its maximum (HRmaxcap H cap R sub m a x end-sub $HRma$), often estimated as 220−age220 minus age 220−age
- **Stroke Volume (SV):** The volume of blood ejected per beat increases, typically plateauing at 40–60% of VO2cap V cap O sub 2 $VO2$ max in untrained individuals.
- **Cardiac Output (Q):** The total blood pumped per minute (HR×SVcap H cap R cross cap S cap V $HR×SV$) increases significantly, rising from ~5 L/min at rest to 20–40 L/min during maximal effort.
- **Blood Pressure:** Systolic blood pressure (SBP) increases linearly with intensity, while diastolic blood pressure (DBP) remains stable or may slightly decrease.
- **Redistribution of Blood Flow:** Blood is re-directed from non-essential organs (kidneys, digestive system) to active skeletal muscles.

2. Metabolic and Muscular Responses

Muscles adapt to sustain force and produce the necessary energy (ATP).

- **Motor Unit Recruitment:** Additional motor units are activated to increase force production.
- **Energy Substrate Utilization:** Increased use of ATP, phosphocreatine (PC), glycogen, and fats, depending on intensity.
- **Body Temperature:** Rises due to heat production from muscle contractions; dissipation occurs via sweating and skin vasodilation.
- **Lactate Production:** Increases during high-intensity exercise when anaerobic pathways are heavily taxed.

CHRONIC TRAINING ADAPTATION

Chronic training adaptations are long-term physiological and molecular changes that occur after consistent exercise over at least **6 to 12 weeks**. Unlike acute responses, which are immediate and transient, chronic adaptations represent a "new steady state" that improves the body's efficiency and performance.

a. **Mitochondrial Biogenesis**: Regular aerobic exercise induces the transcription of **PGC-1α**, the "master regulator" of mitochondrial growth, leading to increased mitochondrial density and size.

b. **Protein Proteostasis**: Resistance training primarily activates the **mTOR** pathway, shifting muscle metabolism toward protein synthesis and fiber hypertrophy.

c. **Epigenetic Priming**: Chronic training leaves stable epigenetic marks (such as DNA methylation changes) that may contribute to "muscle memory," allowing for faster retraining after periods of inactivity.

d. **Metabolic Flexibility**: Long-term training increases the expression of **GLUT4** transporters for better glucose uptake and enhances enzymes for fat oxidation, such as those in the TCA cycle.

e. **Cardiac Hypertrophy**: Endurance training causes **eccentric hypertrophy** (increased chamber volume), while resistance training often leads to **concentric hypertrophy** (thicker walls).

f. **Increased Stroke Volume**: The heart pumps more blood per beat, which typically results in a **lower resting heart rate** (bradycardia).

g. **Angiogenesis**: Increased capillary density in both the heart and skeletal muscle improves oxygen delivery and waste removal.

h. **Respiratory**: Improved efficiency of respiratory muscles (diaphragm and intercostals) allows for greater **tidal volume** and enhanced **pulmonary diffusion** during maximal work.

i. **Neuromuscular**: Early strength gains (first 2–4 weeks) are largely due to **neural adaptations**, such as improved motor unit recruitment and synchronization, before significant muscle mass is added.

MOLECULAR BASIS TRAINING ADAPTATION

Training adaptation is a process of **hormesis**, where repeated disruptions of cellular homeostasis by acute exercise bouts trigger a programmed remodeling of skeletal muscle. This process is driven by complex molecular signaling cascades that translate mechanical and metabolic stress into long-term structural and functional changes.

1. Key Molecular Signaling Pathways

Exercise-induced signals activate specific "sensor" proteins that initiate downstream adaptation:

- **AMPK (Metabolic Sensor):** Activated by an increased AMP/ATP ratio during energy-demanding exercise (primarily endurance). It promotes ATP-generating pathways (e.g., fatty acid oxidation) and inhibits energy-consuming anabolic processes.

- **mTORC1 (Growth Sensor):** Primarily activated by mechanical load and amino acids during resistance training. It is the central regulator of muscle protein synthesis and hypertrophy.
- **PGC-1α (The Master Regulator):** A transcriptional coactivator that orchestrates mitochondrial biogenesis and fiber-type switching. It is activated by both AMPK and CaMKII (calcium-dependent) pathways.
- **CaMKII & Calcineurin (Calcium Sensors):** Triggered by the rise in intracellular calcium during muscle contraction. These pathways influence fiber-type transformation (slow-twitch/oxidative).

2. Epigenetic and Transcriptional Regulation

Long-term training is increasingly recognized as an "epigenetic rewiring" process:

- **DNA Methylation:** Acute exercise induces rapid, transient hypomethylation of promoters for genes like *PGC-1α* and *PDK4*, facilitating their increased expression.
- **Histone Modifications:** Training remodels the activity of gene enhancers. For example, 6 weeks of endurance training can remodel over 7,000 enhancers in human muscle, many of which are linked to improved cardiovascular and metabolic health.
- **Muscle Memory:** Epigenetic marks (like DNA hypomethylation) can persist even during periods of detraining, allowing for faster "retraining" and hypertrophy upon resuming exercise.

3. Qualitative and Quantitative Adaptation Patterns

- **Submaximal vs. Exhaustive Response:** In trained individuals, the transcriptional response to a *submaximal* exercise bout is often attenuated (diminished) because the muscle has already adapted to that specific stress. However, exercise to *exhaustion* can still trigger robust signaling, sometimes with a faster onset in trained muscle.
- **Proteome Remodeling:** Endurance training causes a more extensive qualitative shift in the muscle proteome (e.g., hundreds of new mitochondrial proteins) compared to resistance training, which primarily induces quantitative increases in existing contractile proteins.
- **Specificity & Interference:** While the "AMPK-Akt master switch" once suggested that endurance training (AMPK) and resistance training (mTOR) were mutually exclusive, recent evidence shows both pathways can be activated simultaneously, explaining why concurrent training often works effectively.

MOLECULAR ADAPTATION IN DIFFERENT TRAINING TYPES IN SPORTS

Molecular adaptations in sports are highly specific to the training modality, as different types of exercise activate distinct cellular signaling pathways to remodel skeletal muscle and the cardiovascular system.

1. Endurance Training (Aerobic)

Endurance training focuses on improving metabolic efficiency and fatigue resistance without significant increases in muscle mass.

- **Key Molecular Pathway:** Primarily activates the AMPK-PGC-1α axis.

- Core Adaptations: Mitochondrial Biogenesis: Increased density and function of mitochondria via PGC-1α, the "master regulator".
- **Angiogenesis:** Improved capillarization to enhance oxygen delivery.
- **Fiber Type Shift:** A transformation from fast-twitch (Type IIx) toward more oxidative, slow-twitch-like (Type IIa) fibers.
- **Cardiac Remodeling:** Induces eccentric left ventricular hypertrophy (larger cavity size) to handle volume overload.

2. Resistance Training (Strength/Power)

- Resistance training disrupts muscle homeostasis through high-tension mechanical loads to drive structural growth.
- **Key Molecular Pathway:** Centered on the Akt-mTORC1 pathway, which stimulates myofibrillar protein synthesis.
- **Muscle Hypertrophy:** Significant increase in the cross-sectional area of muscle fibers, particularly Type II fibers, through the addition of contractile proteins (actin and myosin).
- **Structural Strengthening:** Increased connective tissue stiffness and bone mineral density.
- **Cardiac Remodeling:** Induces concentric left ventricular hypertrophy (thicker walls) to manage high-pressure loads.

3. High-Intensity Interval Training (HIIT)

HIIT serves as a "hybrid" stimulus, often triggering adaptations typically associated with endurance training but in a much shorter timeframe.

- **Key Molecular Pathway:** Potently activates both AMPK and p38 MAPK, which converge on PGC-1α.
- **Rapid Mitochondrial Gain:** HIIT can induce similar increases in mitochondrial content as long-duration endurance training due to the high intensity of muscle contractions.
- **Glycolytic Capacity:** Enhances anaerobic enzyme activity and lactate buffering capacity.
- **Metabolic Flexibility:** Promotes both glucose transport (GLUT-4) and fatty acid oxidation enzymes.

4. Concurrent Training (Strength + Endurance)

Athletes who combine training modes may face the "interference effect," where one pathway inhibits another.

- **Mechanism:** High activation of AMPK from endurance work can potentially inhibit mTORC1 activity via the TSC1/2 complex, theoretically blunting maximal muscle growth.
- **Outcome:** While metabolic adaptations usually persist, peak strength and hypertrophy gains may be slightly attenuated compared to pure resistance training.

ROLE OF HORMONES IN MOLECULAR ADAPTION

Hormones serve as chemical messengers that translate the stress of exercise into specific molecular instructions for tissue remodeling. They operate across three primary scales: **acute surges** (immediate response), **chronic conditioning** (long-term basal shifts), and **intracellular signaling** (activation of growth or metabolic genes).

1. Key Hormonal Players & Their Molecular Targets

Hormones regulate adaptation by binding to receptors that trigger specific intracellular "sensor" pathways:

- **Testosterone & Androgens:** The primary anabolic steroids for muscle hypertrophy. Testosterone binds to **Androgen Receptors (AR)**, translocates to the nucleus, and directly stimulates the transcription of genes for contractile proteins. It also activates the **Akt-mTOR** pathway to increase protein synthesis efficiency and inhibits catabolic signals from cortisol.
- **Growth Hormone (GH) & IGF-1 Axis:** GH, released from the pituitary, stimulates the liver and local muscle to produce **Insulin-like Growth Factor-1 (IGF-1)**. IGF-1 is a potent activator of the **PI3K-Akt-mTOR** cascade, which is essential for muscle cell growth, satellite cell proliferation (muscle repair), and structural Maturation.
- **Cortisol (Glucocorticoids):** Often misunderstood as purely "bad," cortisol is essential for energy mobilization. It stimulates **gluconeogenesis** (creating glucose from non-carb sources) and lipolysis. While it can inhibit protein synthesis at high, chronic levels, its acute spike during exercise is vital for managing metabolic stress and inflammation.
- **Catecholamines (Epinephrine/Norepinephrine):** These "fight-or-flight" hormones initiate rapid adaptations like increased heart rate and glycogen breakdown. At the molecular level, they bind to **βbeta β -adrenergic receptors**, increasing **cAMP** levels and activating **PGC-1 αalph** , which drives mitochondrial biogenesis and fat oxidation.

2. Training-Specific Hormonal Adaptations

The endocrine response varies significantly based on the training stimulus:

Training Type	Primary Hormonal Drive	Molecular Outcome
Resistance	Testosterone, GH, IGF-1	Activation of **mTORC1**; increased protein synthesis; muscle hypertrophy.
Endurance	Catecholamines, Cortisol, Glucagon	Activation of **AMPK** and **PGC-1** **αalpha** α ; mitochondrial biogenesis; increased oxidative capacity.
HIIT	Potent GH & Catecholamine spikes	Rapid metabolic shifts; improved glucose transport (**GLUT4**) and insulin sensitivity.

3. The Concept of "Muscle as an Endocrine Organ"

Skeletal muscle itself secretes hormone-like signaling peptides called myokines (a subset of "exerkines") during contraction:

- **Interleukin-6 (IL-6):** Acts as an energy sensor that increases during exercise to stimulate glucose uptake and fat breakdown in other tissues.
- **Irisin:** Promotes the "browning" of white adipose tissue, increasing overall metabolic rate and thermogenesis.

CONCLUSION

Exercise training in sports involves a highly coordinated process of molecular signaling and physiological remodeling that enhances athletic performance and health. At its core, exercise acts as a powerful physiological stressor that disrupts cellular homeostasis, triggering acute responses such as increased gene expression for metabolic substrates and activation of key signaling networks like AMPK (energy sensing) and mTOR (protein synthesis). When these acute bouts are repeated over time, the cumulative effect leads to chronic adaptations, including mitochondrial biogenesis, muscle fiber hypertrophy, and improved cardiovascular efficiency, such as increased VO_2max and cardiac remodeling.

Contemporary research in molecular exercise physiology emphasizes that while many signaling pathways (e.g., calcium flux, oxidative stress, and mechanosensing) are shared across various types of training, the specific outcome—be it the oxidative capacity of a marathon runner or the maximal strength of a powerlifter—is determined by the intensity, volume, and type of training stimulus. Furthermore, emerging fields like epigenetics and multi-omics reveal that training leaves stable molecular "marks" that may contribute to muscle memory and individualized responses to exercise, shifting the field toward personalized training protocols designed to maximize both elite performance and the prevention of chronic diseases.

REFERENCES

1. Fox, E.L., and Mathews, D.K. (1981). The Physiological Basis of Physical Education and Athletics. Philadelphia: Sanders College Publishing.
2. Guyton, A.C. (1976). Textbook of Medical Physiology. Philadelphia: W.B. Sanders co. - Richard, W. Bowers. (l989). Sports Physiology. WMC: Brown Publishers. SandhyaTiwaji. (1999). Exercise Physiology. Sports Publishers.
3. Shaver, L (1981). Essentials of the exercise Physiology. New Delhi: Subject Publications.
4. Vincent, T. Murche. (2007). Elementary Physiology. Hyderabad: Sports Publication.
5. William, D. Mc. Aradle. (1996). Exercise Physiology, Energy, Nutrition and Human