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Effects of Physical Exercise on Hematological Parameters and Immune Function in Young Adults: A Comprehensive Review

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Abstract

Physical exercise is widely recognized an important determinant of health, contributing to cardiovascular fitness, metabolic regulation, and immune function. Emerging research suggest that physical activity (exercise) has been linked to hematological alterations, which have been reported to affect leucocytes, red blood cells, and thrombocytes depending on factors such exercise type, duration, and intensity. Additionally, a number of factors have an important role, such as the popularity of training, inflammation associated with exercise, age, gender, ambient conditions, and the individuals' nutritional state. Most hematological studies seem to focus on older people with pre-existing conditions or professional athletes, ignoring the healthy young adult population whose exercise routines are often self-directed and not under medical supervision. This review

establishes the association between exercise frequency and blood cell count in young adult population. This review further highlights the physiological characteristics of young adults and lifestyle factors affecting health in young adults. The growing responsibilities associated with adulthood being the reasons why young individuals might not have as much time to maintain a healthy lifestyle. Over time, less healthy lifestyle practices define health trajectories into adulthood, raising the chances of morbidities and chronic disease in the future. Evidence states that consistent moderate exercise enhances immune function and overtraining can lead to detrimental alterations in the immune system. Understanding these relationships is essential for building a healthy lifestyle habit, perform moderate physical exercise and disease prevention strategies among young adults.

Keywords: Exercise, Physical Activity, Hematological Parameters, Young Adults, Immune Function

Introduction

Exercise is a skeletal muscle voluntary action for recreational, sporting, or occupational activities. It is a planned, structured, and repetitive physical activity aimed at improving or maintaining one or more components of physical fitness ^[1]. Physical exercise is defined as organized physical activities aimed at improving mental and physical health. Extensive evidence indicates that

physical activity directly enhances psychological well-being, particularly among college students [2]. Resistance training increases muscular strength and arterial stiffness, aerobic exercise increases cardiac output and endothelial function, and combination training has synergistic advantages. Together, these physiological changes enhance lipid metabolism, blood pressure control, and general cardiovascular health. World Health Organization (WHO, 2024) defines physical activity as any bodily movement produced by skeletal muscles that requires energy expenditure [3]. This encompasses all movement including during leisure time, for transport to get to and from places, or as part of a person's work or domestic activities. Both moderate- and vigorous-intensity physical activity improve health. Popular ways to be active include walking, cycling, wheeling, sports, active recreation and play, and can be done at any level of skill and for enjoyment by everybody. A physical exercise is a designated activity undertaken to attain physical exertion or activity. To that end, the World Health Organization's (WHO, 2024) [3] public health guidance recommends that all adults engage in a minimum of: 150 minutes per week of moderate-intensity (or 75 minutes of vigorous-intensity) aerobic physical activity (e.g., walking, running); and two days per week of muscle-strengthening (MS) activities (for example, resistance training). Physical activity has been known to be helpful in enhancing the overall wellbeing of an individual. Exercise enhances cardiovascular health and is frequently associated with changes in an individual's homeostatic and biochemical conditions. Recent studies indicate that exercise affects an individual's haematological status, with these changes dependent on the type, duration, and intensity of the exercise undertaken [4]. Physical exercise induces many functional changes in the body, the magnitude and nature of which are contingent upon factors such as training level, exercise type, intensity, and duration [5]. Exercise physiology has undergone an expansion of its body of knowledge over the past several decades in tandem with new technology, which has facilitated breakthroughs in exercise study methods, data tracking, and analysis. Previously, the beginnings of exercise physiology began with a focus on energy production during activity. This work led to the formation of aerobic and anaerobic descriptions of physical activity based on the oxygen dependence of metabolic pathways required to generate ATP/energy. Aerobic workouts such as running, rowing, walking, swimming, and dancing use oxygen to create energy, while anaerobic exercises, such as weightlifting, sprinting, plyometrics, and high intensity interval training (HIIT), are primarily independent of oxygen. While this fundamental understanding of human physiology has guided exercise studies since the 1980s, this category does not effectively capture and characterise all the extensive advantages that exercise has on organ systems inside the body [6]. Repeated endurance training induces several adaptive modifications at the cellular level. This mostly pertains to a rise in mitochondrial quantity, leading to enhanced adenosine triphosphate (ATP) production via augmented oxidative phosphorylation. The ATP stores in muscles are minimal at rest, although an augmented and sustained supply of ATP is essential during exercise, irrespective of whether the duration is seconds or hours [5]. Exercise significantly raises the body's energy demand as compared to its resting state. At rest, the autonomic nervous system favours a parasympathetic tone, which lowers the respiratory and cardiac rates. During

exercise, the sympathetic nervous system is stimulated, resulting in an integrated response that helps maintain homeostasis to meet the increased demand in cellular metabolism. More recent research and literature has focused on changes outside of the cardiopulmonary system and benefitted from breakthroughs in analysing gene expression and molecular biology [7]. Connections between physical exercise and the central neurological, endocrine, musculoskeletal, gastrointestinal, and immunological systems are now better recognised, although certain doubts and conflicts exist.

Physiological effects of exercise on the body

The cardiovascular system quickly adapts during exercise to guarantee sufficient oxygen delivery to active tissues and the elimination of metabolic byproducts, and each component's function is impacted by the actions of the others. Changes in heart rate (HR), stroke volume (SV), cardiac output (CO), and peripheral vascular resistance are the main components of these acute adaptations [8].

An increase in heart rate is one of the first reactions to exercise. The typical heart rate ranges from 60 to 100 beats per minute [9]. Running, walking, and cycling are examples of dynamic submaximal activity that results in a reaction known as a steady state in English literature. The duration of the physical effort causes the heart rate to increase throughout this reaction. The sympathetic nervous system, which releases catecholamines like norepinephrine and adrenaline, is what causes this rise. These hormones cause depolarisation and an increased firing rate of the sinoatrial (SA) node by binding to beta-adrenergic receptors on cardiac pacemaker cells [8]. The heart beats more quickly as a result, enabling quicker blood flow throughout the body. One of the thermoregulatory processes is the constant rise in heart rate that occurs during exercise in high temperatures.

During exercise, stroke volume increases concurrently with an increase in heart rate. Adults typically have a stroke volume of 80 millilitres when standing or sitting and 110 millilitres when lying down. The interaction between the peripheral pump, which promotes venous return and guarantees diastolic filling of the heart, and the cardiac pump, which controls blood flow through working muscles, affects the stroke volume value. This is especially significant when exercising upright. The Frank-Starling mechanism and increased myocardial contractility are the main causes of this. Cardiac muscle fibres stretch as venous return to the heart rises, resulting in a stronger contraction during systole and increased blood ejection into the aorta [10]. Furthermore, sympathetic activation increases the amount of calcium that enters cardiac myocytes, which increases contractility even more. The cardiovascular system's adaptability to exercise is mostly dependent on the peripheral pump. The stroke volume first gradually rises with increasing exercise intensity before levelling out [9].

Heart rate (HR) multiplied by stroke volume (SV) is the heart's cardiac output. During exercise, there is a significant increase in cardiac output due to the combined effects of elevated heart rate and stroke volume. Exercise causes an increase in heart rate and stroke volume, which in turn causes an increase in the heart's cardiac output [11]. The amount of blood the heart expels in a given amount of time is known as cardiac output. This increased cardiac output makes it possible for the working muscles to receive more oxygen-rich blood, which improves their endurance and performance.

Until an exercise intensity level that corresponds to 40–60% of maximal oxygen consumption (VO₂max) is attained, this process is repeated^[9]. From this point on, the acceleration of cardiac activity is primarily responsible for the accumulation of cardiac output.

Blood flow is redirected from non-essential organs like the digestive system and kidneys to active skeletal muscles in order to meet the metabolic demands of working muscles. This redistribution is accomplished by sympathetic vasoconstriction in inactive areas and local vasodilation in the skeletal muscle vasculature, which is regulated by metabolites like adenosine and potassium^[8]. As a result, there is a significant increase in blood flow to the muscles, which facilitates the delivery of nutrients and oxygen as well as the elimination of metabolic waste products like lactate and carbon dioxide. Skeletal muscles receive 80–85% of the heart's minute volume during physical exercise, while cerebral flow increases by 30% and coronary flow increases by four to five times^[9]. Sustained physical activity and overall cardiovascular health are made possible by these acute cardiovascular adaptations to exercise, which guarantee effective oxygen utilisation and metabolic support for the working muscles. Changes in muscle fibre structure and function brought on by the particular demands of physical activity are known as muscular adaptations to exercise. Optimising training methods, improving athletic performance, and fostering long-term musculoskeletal health all depend on these adaptations as well as skeletal responses like increased bone mineral density.

Types of exercise intervention and their effects on physical fitness

Exercise interventions focus on aerobic, balance, flexibility, and resistance (strength and power) (Table 1)^[12]. Although they can be recommended as a multicomponent intervention based on need, each type enhances distinct areas of physical functioning. Exercise intervention programs should therefore be tailored to the resulting reaction and prescribed depending on an individual's physical functioning.

The American College of Sports Medicine (ACSM) defines aerobic exercise as any activity that engages large muscle groups, can be sustained continuously, is rhythmic in character, and causes the body to require more oxygen than while resting (Abeer *et al.*, 2024). The purpose of aerobic exercise is to improve cardiovascular endurance. Aerobic activity includes running, cycling, swimming, brisk walking, skipping rope, rowing, hiking, dance, tennis, continuous training, and long-distance running^[13]. Aerobic exercise is any sort of physical activity that primarily uses aerobic metabolism to generate adenosine triphosphate (ATP) from oxidative phosphorylation (e.g. brisk walking, jogging, swimming, cycling).

Strength is the greatest force or tension that a muscle or muscle group can produce in a single contraction^[14]. Reduced muscle volume and poor neuromuscular activation determine how much muscle strength is lost with ageing, inactivity, injury, and immobilization^[12]. Following an injury, arthrogenic muscle inhibition and decreased strength are linked to pain, joint effusion, and angle of immobilisation. Strength training helps preserve or increase bone density, lean muscle mass, and muscle strength by using machines, free weights, resistance bands, body weight, or water resistance. Flexibility is the range of motion (ROM) of a joint or group of joints^[15]. It is determined by the length of muscles,

tendons, and bones, and is defined as the extent to which muscle length enables movement over the joint where it functions. A range of stretches that help preserve or increase range of motion around joints are part of flexibility training, which may help prevent injuries, lessen joint pain, and enhance posture. The musculotendinous unit (MTU) plays a major role in ROM and is directly related to stiffness and tension provided by passive and dynamic components^[16].

Joint stability an important component of balance is the state of a joint that either remains in proper alignment or promptly returns to it through the equalisation of forces. Bones, joint capsules, ligaments, muscles, tendons, and sensory receptors must work together to accomplish this. Joint stability is attained through the integration of static and dynamic elements. Static stability denotes the structural stability attained "passively" by entities such as bones, capsules, ligaments, friction, and the articulation's bony shape. Dynamic stability pertains to the neuromuscular regulation of skeletal muscles influencing a joint to preserve its center of rotation in reaction to disturbances. Balance training test a person's capacity to maintain posture and stabilise their body through methods such as shrinking bases of support, unstable surfaces, weight changes and the elimination of upper body support^[16].

Table 1: Types of physical activities and their effects on physical fitness. Adapted from^[4]

Types of physical activities	Effect on physical fitness
Aerobic activity	Improves body composition and cardiorespiratory fitness
Muscle-strengthening activity	Improves muscular fitness such as muscular strength and endurance
Stretching activity	Improves flexibility such as range of motion
Neuromuscular activity	Improves neuromuscular fitness such as balance, agility and proprioception

Blood and its components

Overview of blood

Blood is made up of an intracellular liquid (plasma) which has a vital role of maintaining homeostasis and red blood cells, white blood cells, and platelets that are suspended in this liquid. The circulating blood volume constitutes roughly 7% of the total body weight^[17]. The normal density of blood, which makes up 7% of an adult's body weight, is 1060 kg/m³, which is quite comparable to the density of pure water, which is 1000 kg/m³. The blood volume of a typical adult is roughly 5 litres (11 US points), or 1.3 gallons, and is made up of plasma and formed components^[18]. Approximately 55% of the blood is plasma and the protein content is 7 g/dl (appx. 4 g/dl albumin and 3 g/dl plasma globulins)^[17]. The main purpose of the circulating blood is to supply O₂ and nutrients to the tissues, and eliminate the carbon dioxide and waste products. The blood volume and hemoglobin quantity rise during activity. As the blood volume increases concurrently with the hemoglobin content, the hemoglobin content remains steady, or can even slightly decrease. Regular exercise can change the blood parameters. Thus the hematological parameters can change, depending on the type, intensity and duration of the exercise. The hematological parameters can change during and after severe activity, which can vary according to gender, age, environment or nutrition^[19]. Certain haematological parameters have been found to increase following regular exercise, while other researchers have found no change^[4].

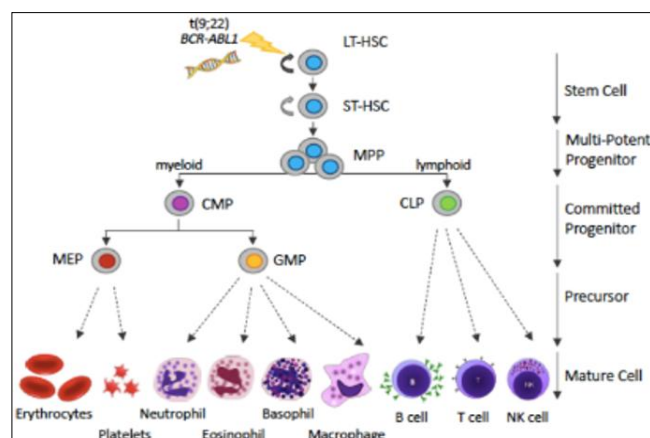
Table 2: Morphology and functions of blood cells. Adapted from [20]

Blood cell	Progenitor	Morphology	Main function
Red Blood Cells	MEP	Biconcave, discoid, and lacking nucleus. Contain hemoglobin	Transport of respiratory gases (oxygen and carbon dioxide) via hemoglobin
Platelets	MEP	Small and lacking nucleus. Develop from fragments of megakaryocytic cytoplasm	Essential for hemostasis via secretion of clotting cofactors, as well as adhesion, aggregation, and provision of a platform for coagulation reactions
Neutrophils	CMP	Multilobed nucleus (two to five lobes). Contain azurophilic cytoplasmic granules	Members of the innate immune system, especially important against bacterial infections
Eosinophils	CMP	Contain acidophilic cytoplasmic granules	Immunity against parasites and play a role in allergy
Basophils	CMP	Contain basophilic cytoplasmic granules	Role in inflammatory reactions and allergy
Monocytes	GMP	Large, with blue-gray cytoplasm and fine lilac granules (ground-glass appearance)	Can differentiate into macrophages or dendritic cells. Involved in innate immunity
macrophages	GMP	Thought to derive from tissue-resident monocytes. Large cells with abundant clear and vacuolated cytoplasm	Phagocytosis and antigen presentation. Wound healing and iron hemostasis
T Lymphocytes	CLP	Contain scant cytoplasm, dense chromatin in a round nucleus	Cell-mediated immunity. Critical role in cancer immunity and viral infections
B Lymphocytes	CLP	Contain scant cytoplasm, dense chromatin in a round nucleus	Humoral immunity via production of antibodies after differentiation into plasma cells, as well as antigen presentation
NK Cells	CLP	Large granular lymphocytes	Innate immune system. Play a role in immunity against viruses and malignant cells

Hematopoiesis (Blood Cell Formation)

Haematopoiesis is the perpetual process of blood cell creation and turnover, essential for fulfilling daily requirements and responding to heightened demands, such as those arising from damage or illness. On average, adult humans generate roughly one trillion blood cells daily, comprising 200 billion erythrocytes and 70 billion neutrophils [21]. Haematopoietic stem cells reside in the medulla of the bone (bone marrow) and have the unique potential to give rise to all of the numerous mature blood cell types and tissues. HSCs are self-renewing cells: As they differentiate, at least part of their daughter cells stay as HSCs so the pool of stem cells is not depleted [22]. The haematopoietic stem cell (HSC) occupies the pinnacle of haematopoiesis and is characterised by two essential traits: The capacity for self-renewal, a division that produces two HSCs, and the potential for multipotent differentiation into all mature blood lineages, namely erythrocytes, platelets, lymphocytes, monocytes/macrophages, and granulocytes. Hematopoiesis follows a hierarchical arrangement with hematopoietic stem cells (HSCs) on top and adult blood cells at the bottom Fig 1. Haematopoietic stem cells (HSCs) are further categorised into long-term (LT-HSCs) and short-term (ST-HSCs) subtypes. The primary function of LT-HSCs is self-renewal, with the majority of their progeny adhering to this pathway, while a smaller proportion differentiate into ST-HSC [20]. ST-HSCs have decreased self-renewal potential and their preferred fate is to develop into multipotent progenitors (MPPs). These then develop into lymphoid primed multipotent progenitors (LMPPs) and common myeloid progenitors (CMPs). CMPs and LMPPs then continue their differentiation into lineage-committed progenitors, concluding with the distinct mature blood cells [20]. Hematopoietic progenitors can be categorised into multilineage progenitors and single-lineage progenitors according to their differentiation capacity. The two primary multilineage progenitors identified are the CMP and the common lymphoid progenitor (CLP; the immediate progeny of LMPPs), as shown in Fig 1. The CMPs give rise to erythrocytes, platelets (via megakaryocytes), basophils, eosinophils, neutrophils, and macrophages following a succession of intermediate forms, which are commonly

dubbed burst-forming units (BFUs) or colony-forming units (CFUs) [23]. This nomenclature is derived from *in vitro* studies performed by cultivating cells on methylcellulose, a semi-solid medium, combined with different cytokines designed to stimulate differentiation into several lineages [20]. The time of appearance, the morphology, the cellular components, and the cytokine needs of the colonies formed in these assays describe each of the intermediate forms, with the practice that a cell population identified by a particular assay is referred to by the assay name. CLPs ultimately develop into B, T, and NK cells. While myeloid and lymphoid lineages are assumed to emerge independently, several investigations have revealed that the granulocyte–macrophage progenitor (GMP) can also be produced from LMP [24].



Abbreviations: LT-HSC: Long-term hematopoietic stem cell; ST-HSC: Short-term hematopoietic stem cell; MPP, multi-potent progenitor; CMP, common myeloid progenitor; CLP, common lymphoid progenitor; MEP, megakaryocyte-erythrocyte progenitor; GMP, granulocyte-monocyte progenitor

Fig 1: Hierarchy of hematopoietic stem cell. Adopted from [23]

Structure and function of Red blood cell

Approximately 94% of the cells in blood are red blood cells, also known as erythrocytes. This amounts to a cell count of $4.6\text{--}6.1 \times 10^{12}/\text{l}$ in men and $4.2\text{--}5.4 \times 10^{12}/\text{l}$ in women [25]. They are biconcave discs in shape that, by the time they enter the circulation, have no nucleus nor organelles. They are 6.5–

8.5 mm in diameter and 1.5-2.5 mm thick^[25]. This distinctive shape gives them a vast surface area to assist gas exchange and also reversibly deforms to allow passage via the capillary beds. The average life span of the red blood cell in the circulation is 120 days after which they get absorbed and phagocytosed by cells of the Reticuloendothelial system (RES) primarily in the spleen, bone marrow and liver^[26]. The red cell membrane is a lipid bilayer made of 50% protein, 40% lipid and 10% carbohydrates. Glycoproteins and glycolipids, the carbohydrates on the membrane's outer surface, play a part in ABO compatibility and are in charge of the cells' antigenic identity. An erythrocyte requires energy to maintain its form and to preserve the iron stored inside it in its reduced (ferrous) state. ATP is created within the cell by the Embden-Meyerhof pathway and NADPH by the hexose monophosphate shunt (details of which are outside the scope of this article)^[25]. Red blood cells (RBCs) (erythrocytes) serve as a carrier of haemoglobin. Haemoglobin combines with oxygen in the blood to produce oxyhaemoglobin during respiration. RBCs in mammals are non-nucleated biconcave shaped cells, very flexible and without intracellular organelles. They are flattened and depressed in the center. Pronormoblasts, the precursors of erythrocytes, develop in the bone marrow through a process known as erythropoiesis. These precursor cells are closely linked to a macrophage and produce haemoglobin until it makes up about 90% of the cell's dry weight. As the cell matures, the nucleus is forced out of the cell and consumed by the macrophage. In addition the no-longer-needed proteins are ejected from the cell in vesicles called exosomes.

Packed cell volume, haemoglobin, and mean corpuscular haemoglobin are critical metrics for assessing circulating erythrocytes, playing a vital role in the diagnosis of anaemia and serving as valuable indicators of the bone marrow's capacity to manufacture red blood cells^[19]. According to Çiçek, (2018) red blood cell is involved in the transport of oxygen and carbon dioxide in the body^[17]. Thus, a reduced red blood cell count implies a reduction in the level of oxygen that would be carried to the tissues as well as the level of carbon dioxide returned to the lungs.

The primary functions of erythrocytes are to transport haemoglobin (Hb), which transports oxygen from the lungs to the tissues. Over 99% of the oxygen carried from the lungs to the tissues is carried by haemoglobin (Hb), a metalloprotein found in erythrocytes that makes up 33% of red cell mass. Haemoglobin is present in blood at quantities of 13.5 18.0 g/dL in men and 11.5 16.0 g/dL in women. Each erythrocyte contains roughly 200 to 300 million molecules of haemoglobin^[27]. It comprises of four subunits each having a polypeptide globin chain and an iron-containing porphyrin termed haem. The haem, which is made from succinic acid and glycine, has one iron atom in its reduced, ferrous state (Fe^{2+}). One molecule of Hb has four atoms of iron and binds four molecules of oxygen. There are two pairs of polypeptide globin chains in each Hb molecule. Normal adult haemoglobin (HbA) comprises two α and two β chains^[28]. Another regular form (HbA₂) contributes for up to 2.5% of Hb and comprises two α and two δ chains. Fetal Hb (HbF) includes two α and two γ chains. The change in structure of the γ chains affords HbF a stronger affinity for oxygen so permitting the transfer of oxygen from the maternal to the fetal circulation^[28]. The major job of Hb is to transport oxygen (O_2) from the lungs to tissues, but they also specifically interact with the 3 other gases, carbon dioxide

(CO_2), carbon monoxide (CO), and nitric oxide (NO), that have vital biological roles. Haemoglobin combines with oxygen when the blood goes through the lungs and delivers it to the tissues as the blood passes them. It strictly limits the amount of O_2 available to the tissues through its oxygen buffering function^[27].

Hb is an excellent acid-base buffer^[25]. The erythrocyte has a role in the transport of carbon dioxide (CO_2) from the tissues to the lungs primarily in the form of bicarbonate, to facilitate acid-base balance^[17]. Although CO_2 is 20 times more soluble than O_2 , only up to 7% of CO_2 is carried dissolved in the plasma. The great majority of CO_2 undergoes a chemical reaction within the erythrocyte either by a hydration reaction catalysed by carbonic anhydrase, which converts CO_2 to bicarbonate ions (70%) or by binding directly to haemoglobin to produce a carbaminohaemoglobin complex^[28].

Erythrocyte indices and their clinical significance

Erythrocyte indices, such as mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), and mean corpuscular haemoglobin concentration (MCHC), are important markers of the health and function of red blood cells^[27]. Important information about the physiological state of red blood cells is provided by each of these indices. The average volume of red blood cells, or MCV, is used to categorise anaemia as either macrocytic (bigger than normal), normocytic (normal size), or microcytic (smaller than normal)^[29]. Variations in MCV can indicate chronic illnesses that affect RBC growth and longevity or dietary shortages such iron, folic acid, or vitamin B12. MCH and MCHC offer additional information regarding the concentration and content of haemoglobin in erythrocytes, respectively. MCH quantifies the average concentration of haemoglobin per red blood cell, whereas MCHC evaluates the concentration of haemoglobin in a specific volume of compressed erythrocytes. According to Park & Chang, (2025)^[29], these metrics are helpful in recognising various forms of anaemia and assessing oxygen carrying capacity, which is crucial for preserving cellular energy and health throughout the body. A healthy erythrocyte index indicates adequate oxygen transport to tissues, whereas abnormalities may point to diseases like iron deficiency anaemia (typically microcytic), folate or B12 deficiency anaemia (typically macrocytic), and inherited or acquired disorders that affect the morphology of red blood cells^[27].

White Blood Cells

White blood cells (leucocytes) are significantly less abundant in the blood than erythrocytes, with a cell count of $4 \times 10^9/\text{l}$ under normal conditions, accounting for less than 1% of total blood volume^[25]. There are various different varieties of leucocyte, differing in size, structure and function. They contain nuclei and other organelles but do not contain haemoglobin. Collectively, they comprise the body's principal defense against disease; defending against damage from infections and eliminating damaged cells, poisons and waste products. Leucocytes can be classed by numerous different techniques, but a typical differentiation is between granulocytes and agranulocytes. Granulocytes are neutrophils, eosinophils and basophils. They are all about spherical in form and feature lobar nuclei. They contain cytoplasmic granules that are easily stained and observed on microscopy. They are non-specific as they can be activated by a multitude of different stimuli^[28]. Agranulocytes consist

of lymphocytes and monocytes (precursors of macrophages). While they do still contain secretory vesicles these are hard to discern under light microscopy, hence the term agranulocytes^[25]. They are relatively dissimilar from one other functionally yet are structurally comparable.

The primary roles of white blood cells and their subtypes are to combat infections, protect the body through phagocytosis against foreign invaders, and to manufacture or facilitate the transport and distribution of antibodies during immune responses. Consequently, animals with diminished white blood cell counts face an elevated risk of disease infection, whereas those with elevated counts can produce antibodies during phagocytosis and exhibit a heightened resistance to diseases, thereby improving adaptability to local environmental and disease condition.

Platelets

Platelets are small, non-nucleated blood cells that represent a minor fraction of the circulating blood cells under normal circumstances. The normal platelet count in humans ranges from $150 \times 10^9/L$ to $400 \times 10^9/L$ ^[30]. Approximately 100×10^9 of these tiny anucleate cells must be released from mature megakaryocytes into the circulation each day to maintain a normal platelet count because platelets have a circulating lifespan of about 8-12 days and about one-third of platelets are sequestered in the spleen at any given time^[30]. A continual balance is, therefore, required between thrombopoiesis, and platelet consumption and senescence.

Thrombopoietin is the principal humoral regulator of megakaryocyte development and platelet number under steady state settings. It is generated in the liver and kidney and exerts its actions through its receptor c-Mpl which is found on megakaryocyte and platelet membranes^[31]. Levels of thrombopoietin are controlled via binding to, and internalization into, cells expressing the receptor. Less thrombopoietin is extracted from plasma and the thrombopoietin level rises when platelets and megakaryocytes are reduced; conversely, when platelet counts rise, more thrombopoietin is extracted from the plasma and the thrombopoietin level decreases once more^[30]. The functions of platelets in the human body are altered by physical training and regular exercise habit^[32], and platelets also play a critical role in haemostasis and function in beginning WBC recruitment^[4]. Because platelets prevent blood loss following vascular injury, they are essential for maintaining normal haemostasis^[33]. Primary haemostasis is achieved by adhering to damage sites, attracting more platelets and blood cells to the forming clot, and initiating the plasma coagulation cascade. A more stable clot rapidly forms in tandem with the coagulation cascade's final products, which are primarily crosslinked fibrin. However, platelets also directly contribute to pathologic vascular thrombosis through these same processes^[34]. A deeper comprehension of platelet structure and function is crucial in the continuous quest for ways to reduce or prevent arterial thrombosis while maintaining physiologic haemostasis. In humans especially those with chronic diseases like diabetes mellitus and hypertension, exercise influences on platelet function are still up for more scientific investigations^[32]. Blood platelets and certain of the proteins that are dissolved in plasma interact to generate clots sealing the burst sites of blood arteries when the vessels get damaged, resulting to bleeding. Hence, the body is safeguarded from any additional loss of blood.

Exercise and Immune Function

Exercise induced hematological changes

Blood volume and its components are modified in different health or illness states. Diverse forms and categories of exercise, including acute or chronic and low or high intensity, may induce these alterations. Research indicates that persons engaged in exercise can experience an increase in blood volume of up to 10–12% within a 24-hour period. This augmentation may attain a plateau between 10 and 14 days of training^[4]. Furthermore, exercise has been documented to enhance blood volume and cardiac output within a period of less than six weeks of training^[35]. There is no consensus regarding the specific type of exercise that induces alterations in haematological markers and cardiometabolic enzymes linked to physical activity. However, contradicting results showed that short-term activity induced a rise in WBC count and a lower RBC count. A decrease in WBC count and an increase in RBC count have been recorded, although no significant changes in red blood cell characteristics were noted post-exercise. However, a recent study, has demonstrated that short-term exercise can improve participants iron status and raise RBC count in healthy guys^[36]. These and other haematological parameters can be adjusted depending on the nature, time and intensity of the physical activity in addition to gender, age, environment or nutrition based on the experimental design of the investigations.

Acute respiratory response to exercise

Pulmonary ventilation is the amount of air that enters and leaves the lungs in one minute^[37]. It is the result of multiplying respiratory rate with tidal volume. With a typical tidal volume of 500 ml and a respiratory rate of 12 litres per minute, it is roughly 6 litres per minute. Pulmonary ventilation rises to approximately 60 litres per minute during moderate exercise and up to 100 litres per minute during severe exercise due to an increase in respiratory rate to approximately 30 beats per minute and tidal volume to approximately 2000 millilitres^[37]. To supply oxygen to the tissues, the respiratory and cardiovascular systems collaborate. Exercise causes the respiratory system to instantly increase pulmonary ventilation by stimulating the brainstem's respiratory centers through the motor cortex and proprioceptors in the muscles and joints^[38].

Further increases in respiratory rate are triggered by the surge in CO₂ generation, hydrogen ions, and body warmth during exercise. In adults, pulmonary ventilation can increase from approximately 10 liters/minute at rest to more than 100 liters/minute at high-intensity efforts^[8]. The cardiac output to the pulmonary circuit is identical to that of the systemic circuit. The available surface area for gas exchange increases in response to the increased cardiac output, resulting in a decrease in the alveolar dead space. Blood gas and pH balance can be maintained with more alveolar surface area available for gas exchange and increased alveolar ventilation due to increased frequency and volume of respiration^[37].

One of the metabolic byproducts of muscle action is CO₂, which is usually transported as bicarbonate from peripheral active tissue. When attached to haemoglobin in red blood cells, a fraction moves as carbamino-hemoglobin and dissolved CO₂ in plasma. CO₂ is readily incorporated into the RBC cytosol, where it is metabolized into carbonic acid by the enzyme carbonic anhydrase^[39]. After then, carbonic acid

breaks down spontaneously into two ions: Hydrogen and bicarbonate. Carbonic anhydrase catalyses the opposite reaction to create CO_2 , which is exhaled and eliminated from the body once bicarbonate reaches the lungs. In higher-intensity activities, the volume of carbon dioxide expelled per unit of time is sustained by the decreased alveolar dead space and increased tidal volume [8].

Long-Term physiological adaptations to regular exercise

Humans' skeletal muscle undergoes phenotypic changes in response to exercise, including changes in the amount and type of metabolic enzymes, the amount of contractile protein, the stiffness of the connective tissue, and the store of nutrients. Exercise frequency, intensity, and duration, together with an individual's age, gender, genetics, fuelling, and training history, all contribute to a change in phenotype [40]. As a result, although while exercise is frequently referred to as a single stimulus and we have searched for generalised responses, each person's response to exercise training will differ depending on what we know and (probably) a lot more that we do not.

The idea that a small amount of high-intensity training or a

big amount of moderate-intensity training can produce comparable skeletal muscle adaptation is the basis for the role of exercise intensity. Certain metabolic enzyme mediators are significantly increased as training intensity increases when volume is matched, resulting in gene expression and mitochondrial biogenesis, as well as physiological adaptations including raising $\text{VO}_{2\text{p}}$. The volume increase also increases the mitochondrial content when training is matched at a high intensity. Furthermore, in both males and females, a single high-intensity sprint session raised the concentration of growth hormone (GH) and plasma catecholamines after exercise, which are hormones linked to muscle growth and fat metabolism.

Long-term endurance activity typically results in an initial constriction of plasma volume due to fluid loss, which is followed by an expansion of plasma volume. Regular training can sustain this growth, which can be reversed three to five days after stopping [41]. By increasing plasma volume and enhancing $\text{VO}_{2\text{peak}}$ and exercise performance, endurance training lowers blood viscosity [42]. Blood viscosity reduction enhances oxygen delivery to the tissues and microcirculatory blood flow.

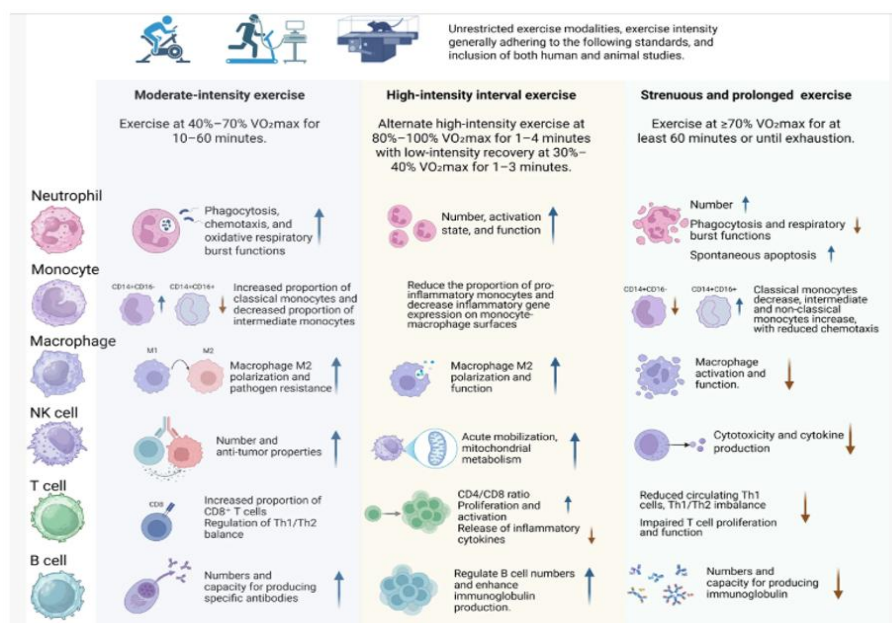


Fig 2: The impact of various exercise workloads on immune cells. Adopted from [43]

Full Blood Count (FBC) Test

The full blood count (also known as complete blood count CBC) and white blood cell (WBC) differential provide useful information on the peripheral blood's primary biological components: Red blood cells (RBCs), WBCs, and platelets (Table 3) [44]. The FBC's WBC differential section classifies WBCs into five distinct types: Neutrophils, lymphocytes, monocytes, eosinophils, and basophils. Determining the number of each type of WBC provides additional insight into the underlying issue. For instance, the majority of the rise in WBCs during the initial phases of an infection can be attributed to the rise in neutrophils [45]. Lymphocytes rise while the infection persists. While allergy diseases like hay fever cause an increase in basophils, worm infections can cause an increase in eosinophils. Impedance, conductivity tests, light scatter, and fluorescence are among the laboratory methods used. Leukopenia, or a drop in WBC count from baseline, might be a sign of either changed immunity or an

infection that depletes supply of specific WBCs [46]. The resolution of an infectious process may be indicated by a lower WBC count in comparison to a previously high count (i.e., becoming more within normal ranges). The WBC absolute value is more informative than the relative value (percentage) and has clinical significance, as it illustrates the medullary response to inflammatory stimuli [45]. An etiological diagnosis is made possible by the relative value, which is useful for determining which WBC population is most involved in the inflammatory process.

The erythrocyte count in circulating blood is roughly $4 \times 10^{12}/\text{L}$ under healthy conditions, making up almost half of the total blood volume [47]. The myeloid stem cell line's erythroblastic progenitors give rise to erythrocytes, which have high haemoglobin and oxygen content because their nuclei are extruded before they reach the reticulocyte stage. An estimated 10^{11} erythrocytes are produced and eliminated from the bloodstream every day, making erythropoiesis a

dynamic process that strictly controls the amount of erythrocytes in the blood^[47].

The average size of the erythrocytes as well as the average amount and concentration of haemoglobin in the erythrocytes can be estimated using different indices. With the use of contemporary haematology analysers, these indices can be assessed directly or computed electronically. They can be helpful in identifying the pathologic process producing the anaemia or in further defining anaemia based on the size or quantity of haemoglobin in the red blood cells. The Mean Cell Volume (MCV), Mean Corpuscular Hemoglobin Concentration (MCHC), and Mean Corpuscular Hemoglobin (MCH) are examples of erythrocyte indicators. The average size of circulating erythrocytes is measured by the MCV^[48]. Anaemia can be measured as either macrocytic (big cells) or microcytic (small cells). Hyperviscosity/polycythemia and anaemia brought on by a folate or vitamin B₁₂ deficiency are both associated with an increased MCV^[49]. The MCHC measures the hemoglobin concentration in a given volume of red blood cells. The amount of haemoglobin in the RBC determines its colour, which can be classified as normochromic, hypochromic, or hyperchromic^[50]. The MCH calculates the mean haemoglobin content per red blood cell in a blood sample.

Table 3 displays the reference ranges of each component of CBC. Previous epidemiological studies have examined the combination of CBC components, such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR), in addition to the WBC, RBC, and platelet parameters per se^[51].

Table 3: Complete Blood Count parameter with reference range. Adopted from^[51]

Test Acronym	Normal Range Values (Male)	Normal Range Values (Female)
RBC	4.0–10.8 × 10 ³ /μL	4.0–10.8 × 10 ³ /μL
WBC	4.5–6.1 × 10 ⁶ /μL	4.0–5.4 × 10 ⁶ /μL
Hb	13.0–17.0 g/dL	12.0–16.0 g/dL
HCT	40.0–52.0%	37.0–47.0%
MCV	80–98 fL	80–98 fL
MCH	27.0–33.0 pg	27.0–33.0 pg
MCHC	31.5–37.0 g/dL	31.5–37.0 g/dL
RDW	11.5–14.5%	11.5–14.5%
PLT	150–400 × 10 ³ /μL	150–400 × 10 ³ /μL
Neutrophils %	40–73%	
Lymphocytes %	19–48%	
Monocytes %	0.4–10.0%	
Eosinophils %	0–7.0%	
Basophils %	0–2.0%	

Effect on RBC Count and Hemoglobin

Haematocrit and plasma viscosity, two important factors that determine blood viscosity, are the main causes of the well-documented rise in blood viscosity that occurs after exercise. This increase in blood viscosity following exercise is caused by a number of mechanisms, including fluid shifts, an increase in circulating erythrocytes as a result of splenic contraction and their redistribution, elevated plasma protein concentrations, and water loss from thermoregulatory mechanisms and subsequent entrapment in muscle cells^[52]. Similarly, it was also observed that exercise leads to red blood cell (RBC) damage and hemolysis on account of osmotic and mechanical stress generated by exercise, foot stroke hemolysis and free-radical driven lipid peroxidation of RBC membrane^[53].

Changes in plasma viscosity may be more important for tissue oxygen supply because a rise in haematocrit can decrease blood flow while increasing oxygen-carrying capacity^[54].

Reticulocyte counts and reticulocyte index calculations are becoming increasingly important in sports medicine, particularly when it comes to anti-doping testing. However, the automated analysers employed determine the outcomes. It is possible to ascertain the distribution of maturity stages within the reticulocyte population. Because it rises before the reticulocyte count, the immature reticulocyte fraction is a trustworthy metric^[55].

Due to increased iron metabolism, athletes' bone marrow is constantly stimulated. Evaluation of the Hb content of reticulocytes (CHr, Ret-He) is essential because it enables prompt diagnosis and monitoring of iron deficiency and therapy outcome^[56]. Both hyperproliferative erythropoiesis and inefficient erythropoiesis are indicated by the quantity of RNA-rich immature reticulocytes. Athletes had higher levels of immature reticulocytes than non-athletes, with values over the reference interval^[55]. One powerful progenitor cell that is thought to be an indirect indicator of haematopoietic activity is the CD34+ cell, also known as a haematopoietic progenitor cell (HPC)^[22]. Athletes' HPC concentration is four times higher than that of inactive controls, but it drops again a few hours after the race^[55]. However, following endurance exercise, the reticulocyte system generally exhibits relatively little alteration.

Effect on WBC Count

It is generally recognised that depending on the intensity and length of the work, exercise can cause an acute leukocytosis to varied degrees. Acute high-intensity exercise elevated white blood cell (WBC) count due to enhanced trafficking of blood cells into circulation^[57]. The leukocyte count can be twice as high after high-intensity exercise (up to 31.8 × 10⁹/L in some situations, given a baseline of 6.4 × 10⁹/L)^[55]. The leukocyte count returns to normal in 30 minutes following 30 minutes of exercise. According to McCarthy DA's theory, exercise produces catecholamines that raise the ratio of circulating to non-circulating leucocytes, which causes the body to experience physiological stress and release adrenaline^[58]. Research indicates that the degree of variation in leukocyte count is contingent upon the intensity, duration, and individual fitness level. Neutrocytosis may be influenced by the length of exercise rather than its intensity, which is also influenced by the release of adrenocorticotrophic hormone^[58]. Short-term, intense exercise led to changes in plasma catecholamine levels and a rise in leukocytosis, which included lymphocytes, granulocytes, and monocytes^[59].

Consistent moderate exercise enhances immune function, however high-intensity exercise or overtraining can lead to detrimental alterations in the immune system^[60]. Other investigations indicated no substantial effect of exercise trials on leukocyte subgroup counts. High-intensity exercise resulted in notable leukocytosis primarily driven by transient lymphocytosis and monocytosis, subsequently followed by a delayed neutrophilia in young adults. Sustained neutrophilia may remain for 1 to 5 hours post-exercise^[53]. The elevated total leukocyte counts, particularly neutrophil levels, are commonly utilised as indicators of the inflammatory response linked to exercise-induced skeletal muscle injury and heart stress^[54]. Regular aerobic exercise is expected to raise blood serum levels of leukocytes, cytokines, interleukins, and tumour necrosis factor alpha. Athletic effort has no effect on

adaptive immunity, but the innate system reacts differently to long-term stress by lowering neutrophil activity and raising natural killer cell activity^[4].

Immediately following endurance activity, the leukocytes (neutrophils and lymphocytes) increase by 50–100%. The lymphocytes drop to 30–50% below pre-exercise levels after 30 minutes and continue to do so for three to six hours^[55]. At the same time, there is noticeable, protracted neutrophilia. This causes a bi-phase pattern in the leukocyte response to endurance exercise, with a second peak occurring two to four hours later. While cortisol is believed to initiate the latter phase by releasing leukocytes from the bone marrow, catecholamines and the subsequent rise in cardiac output are responsible for early leukocytosis, which depletes the marginal stores from leukocytes. These leukocytoses can be seen for at least eight hours, and in certain situations, they can continue more than twenty-four hours. Regarding the various leukocyte kinds, there are no conclusive conclusions. Eosinopenia, lymphocytosis, and neutrophilia are seen. There is a modest drop in basophilic granulocytes and a minor increase in monocytes.

The quantity of leukocytes and the function of lymphocyte subpopulations are significantly impacted by physical activity and exercise (Fig 2). NK cells have the strongest reaction out of the three main subpopulations of lymphocytes (T, B, and NK cells). NK cell numbers are temporarily elevated by acute moderate-intensity exercise and revert to baseline in a few of hours. NK cells have been shown to be mobilised by prolonged moderate-intensity exercise, increasing their activity and anti-tumor potential^[43]. According to Parent-Roberge *et al.*, (2024) moderate-intensity cycling dramatically raised NK cytotoxic activity (NKCA) and peripheral NK cell numbers^[61]. Additionally, the cytotoxic NK cell fraction in whole blood increased after six weeks of moderate-intensity exercise, suggesting improved antitumor potential^[62].

Effect on Platelet Count

An increasing amount of research suggests that platelet function is impacted by both regular physical activity and acute exercise. The platelet is essential for stopping bleeding. Temporary haemostasis by platelet plug formation is the most significant role of platelets. The variations in platelet count, mean platelet volume, platelet distribution width, and other platelet functions that are linked to exercise in both healthy individuals and patients have drawn more attention over the past few decades^[63]. In addition to playing a crucial part in thrombosis and homeostasis, platelets also play a key role in supporting and regulating immunological and inflammatory responses^[64]. Frequent, regular exercise improves the haemostatic profile at rest and during exertion, which lowers the incidence of CVD^[65]. Conversely, in individuals with or without underlying vascular illness, intense exercise without training may also result in venous thromboembolism and sudden cardiac mortality^[66]. Because of these impacts on the cardiovascular system, platelets' reactions to various forms of exercise vary in both healthy and patient populations based on a variety of factors, such as the subject's level of physical fitness and the intensity and length of the exercise. While intense activity promotes platelet activation and aggregation, moderate-intensity exercise inhibits platelets. A 12-week regular exercise program reduces platelet hyperactivity in response to acute exercise in healthy adults, whereas a 20-minute cycling session at 70–80% of maximum heart rate

significantly increases platelet aggregation to agonists^[64]. Increased adhesiveness, aggregation, and intraplatelet calcium were therefore seen after an incremental exercise test till exhaustion, but these effects were diminished or nonexistent after consistent training.

It appears that only intense activity increases the thrombotic tendency, even if acute exercise can activate platelets in sedentary healthy persons. Platelet aggregation has been demonstrated to result from increased shear stress and a notable release of platelet agonists in plasma after intense exercise^[64]. Additionally, the impacts of physical exercise on many cell/tissue types and pathways can have an impact on platelets. For example, intense exercise increases oxidative stress, shear, and catecholamine release, all of which cause platelet activation. Catecholamine effects in the modulation of platelet activation are a mechanism that results in a hypercoagulable response to acute exercise^[64]. Epinephrine stimulates platelet adhesion, aggregation, and fibrinogen binding through alpha2-adrenergic receptors^[67]. Although physiological levels of epinephrine might not be sufficient to initiate the pathways involved in these aspects of platelet response, epinephrine plus ADP increases following vigorous exercise but not during moderate-intensity exercise^[68] and activates platelets *in vivo*, which is consistent with research showing hyperactivity of platelets during high-intensity but not moderate-intensity exercise. Exercise-induced increases in arterial blood flow and shear rate should also be taken into account^[64].

Mechanisms linking exercise and Blood cell Counts Hormonal and metabolic responses

Hormone secretion and the neuroendocrine system are strongly impacted by physical activity. It is well known that conditioning the adaptive reaction to training to become physically fit causes elite athletes to have alterations in their hormonal makeup^[69]. Athletes' hormone systems are complicatedly affected by physical training. The hypothalamus and pituitary gland's reactions to physical strain are influenced by a number of variables, such as training duration and intensity, nutrition and energy levels, gender, sex, age, and stage of sexual development. Specifically, the hypothalamic-pituitary-adrenal (HPA) axis is stimulated by exercise intensity and duration, resulting in elevated cortisol levels, whereas reproductive hormones are suppressed by an inadequate diet and limited energy availability^[70]. The hormonal response is modulated by gender and sex differences, with variations associated with levels of oestrogen and testosterone. The hypothalamic-pituitary axis's responsiveness is influenced by age, with older adults showing a weaker reaction than younger people^[69]. Furthermore, the endocrine response is greatly altered by sexual developmental stages including puberty and menopause, underscoring the significance of a customised training regimen^[71].

Basal hormone levels usually exhibit particular variations with extended exercise training, either slightly increasing or slightly decreasing. Table 4 summarizes gender-specific hormonal differences are summarised. The baseline levels of different hormones are shown in the first column along with the gender in which they are more prevalent. The final column indicates whether these gender disparities have widened or remained unchanged. The hormonal differences between acute physical activity and regular training are reported in the intermediate columns. This phenomena is

significantly influenced by the "basement effect," which limits noticeable declines in hormone levels, particularly when values approach zero ^[69]. As a result, significant additional reductions become difficult to identify.

Table 4: General changes in hormone levels induced by physical activity. Adopted from ^[69]

	Basal F/M	Training	Acute Physical Exercise	Acute Physical Exercise F/M
GH	↑ F	↑/=/↓	↑	↑ F
IGF-1	↑ M	↑/=/↓	↑/=	↑ M
CATECHOLAMINES	F = M	↑/=/↓	↑	↑ M
ACTH	F = M	↑/=/↓	↑	↑ F/= M
CORTISOL	↑ M	↑/=/↓	↑	↑ M-↑ F
TSH	↑ F/= M	↑/=/↓	↑/=/↓	↑ F/= M
T3-T4	F = M	↑/=/↓	↑/=/↓	F = M
LH-FSH	F = M	=/↓	↑/=/↓	F = M
TESTOSTERONE	↑ M	=/↓	↑/=/↓	↑ M-↑ F
ESTRADIOL	↑ F	=/↓	↑/=/↓	↑ F
INSULIN	F = M	↓	↓	F = M

Key Legend: F, female; M, male; ↑, increase; ↑, substantial increase; =, no variations; ↓, decrease. Growth hormone (GH); insulin-like growth factor-1 (IGF-1); thyroid-stimulating hormone (TSH); triiodothyronine (T3); thyroxine (T4); luteinizing hormone (LH); follicle-stimulating hormone (FSH)

Many of the changes that take place in muscles during exercise are regulated by cortisol, a vital chemical in our bodies. The amount of it in our blood is influenced by our nutrition, circadian rhythm, degree of fitness, and the length and intensity of our exercise. Although training may involve a greater physical burden, Nikolovski *et al.*, (2023) found a rise in cortisol levels during contests due to higher physical and psychological stress ^[72]. During games, there is more physical and psychological strain due to intense physical contact and explosive behaviours, which affect cortisol levels. Consequently, a number of factors, such as the competition atmosphere, playing time, number of interactions, and psychological pressure, influence cortisol levels ^[73]. In sports, cortisol is frequently utilised as a sign of extreme overtraining and stress, which can lower performance ^[69]. An imbalance between training and recuperation, exercise and capacity, and stress and stress tolerance is known as overtraining syndrome (OTS) ^[74]. As a result, cortisol can decrease the synthesis of anabolic hormones, which encourage muscle growth, and has a catabolic effect on muscular tissues, encouraging the breakdown of muscle fibres.

Young Adult Population

WHO defines 'Adolescents' as those in the 10-19 years age group and 'Youth' as the 15-24 year age group ^[75]. While 'Young People' includes the age span 10-24 years. There are over 360 million teenagers comprising about 20% of the population in the countries of the South-East Asia Region (SEAR). The journey from infancy to adulthood involves substantial physical, sexual, psychological and social developmental changes, all taking place at the same time. In addition to chances for development this change poses threats to their health and welfare. Contrary the widespread assumption that this is a health age group, the teenagers actually have various public health challenges ^[75]. From a developmental perspective, young adulthood is marked by typical biological and psychological maturation; however, the

specific social roles and responsibilities assigned to each cohort of young adults are influenced by the attributes of the society at a given historical moment. In modern American society, young adulthood is characterised by significant diversity in transitional experiences, with notable variation in the timing, order, and nature of social duties and responsibilities ^[76].

Youth are characterised not alone by bodily transformations but also by cultural identity and social norms inside their contextual framework as they navigate adulthood. The transition from infancy to adulthood is contingent upon various factors within the socio-cultural setting. Personal transformation include addressing issues associated with social identity, education, training, recreation, employment, and familial development ^[77]. The self-image of young individuals is significantly influenced by their self-perception and the nature of their social identity, which encompasses how they are perceived by others. Certain scholars have highlighted the distinctive characteristics of young individuals, perceiving them as 'creative agents' who actively construct their cultural identities through the exploration of youth culture. The definition of young people has evolved in a context where perceptions of aspirations and expectations for this demographic are shifting. Historically, as noted by Hochberg & Konner, (2020) ^[78], the shift to adulthood has been marked by the assumption of adult roles, including marriage, parenthood, and stable job. Adults typically assume these tasks prior to or subsequent to the age of 20. Hochberg & Konner, (2020) also stated that while the idea of adult development is evident in industrialised nations ^[78], research is scarce in poor countries. Although there has been a trend to individuality as a result of globalization, there is still a profound community of individuals. Their responsibilities are notably limited to parents, but personal exploration and self-development during adult life are limited. For young individuals in North America and Europe, financial independence is an important indicator of maturity ^[77]. Conversely, Asian colleagues consider the ability to financially support parents as one of the most essential characteristics. These attitudes towards the family can limit the quest for personality and education and professional choices.

Physiological Characteristics of Young Adults

Puberty involves an endocrine transition with noticeable physical and behavioral changes, especially in body image, sex identity, aggression, and impulsivity. To identify a maturational stage between adolescence and adulthood, we need first to define the conclusion of youth. During this phase, growth velocity decelerates, blood and tissue hormone levels increase, aggression becomes less overt, and learning and maturity lessen hormonal impact ^[78].

Using maturational measurements avoids the difficulties of defining emerging maturity according to chronological age. For example, the Tanner scale of teenage development is based on exterior primary and secondary sex traits. Tanner stage V acknowledges the finish of puberty in males when the testicular capacity is >20 ml and the length of the penis is >14 cm and in girls when the breast reaches final adult size and the areola returns to the contour of the surrounding breast with a protruding central papilla ^[79].

According to Hochberg and Konner (2020) ^[78], emerging adulthood is a phase of life between adolescence and full-fledged adulthood, with particular demographic, social, and

subjective psychological traits. This life- historical stage pertains to individuals aged between 18 and 25 years, the period during which they become more economically independent via training and/or education. Previously, the psychodynamic theorist Erik Erikson described a stage that he called a protracted adolescence or psychosocial hiatus in young people in developed countries^[80]. Much more recently, Hopwood and colleagues explored genetic and environmental influences on personality development during the transition to adulthood in same-sex male and female monozygotic and dizygotic twins assessed in late adolescence (approximately age 17 years), emerging adulthood (~24 years), and young adulthood (~29 years)^[78]. Their genetically-informed results support a life-course approach on personality development during the transition to maturity.

Lifestyle Factors Affecting Health in Young Adults

Young adulthood is associated with growing responsibilities, including financial independence, job development, and establishing a family. Young Americans between the ages of 25-34 spend more time at work and caring for others, and have less leisure time than younger age groups^[81]. With greater time allocated to these duties, young individuals may not have as much time to maintain a healthy lifestyle. Over time, less healthy lifestyle practices define health trajectories into adulthood, raising the chances of morbidities and chronic disease in the future. Lifestyle and health related behavioral decisions are essential variables for understanding health. Physical inactivity and poor diet are key risk factors for obesity and other chronic diseases such as hypertension, cardiovascular disease, diabetes, and several malignancies. Inadequate sleep is also connected to poor health status, including obesity, hypertension, and Type 2 diabetes.

Why exercise is recommended in Young Adults

According to the American Heart Association, a sedentary lifestyle is one of the five main risk factors for cardiovascular disease, along with high blood pressure, abnormal blood lipid readings, smoking, and obesity^[82]. Numerous scientific studies have demonstrated that lowering these risk factors lowers the likelihood of having a heart attack or other cardiac event, like a stroke, as well as the likelihood of requiring a coronary revascularisation procedure (coronary angioplasty or bypass surgery)^[84, 83]. Many of the known risk factors for cardiovascular disease are positively impacted by regular exercise. Exercise, for instance, can lower blood pressure and encourage weight loss. Exercise can increase "good" cholesterol (high-density lipoprotein level [HDL]) and decrease "bad" cholesterol (low density lipoprotein [LDL] level and total cholesterol). Regular exercise improves the body's capacity to use insulin to regulate blood glucose levels in diabetic people^[65]. When paired with other lifestyle changes (including healthy eating, quitting smoking, and taking medication), the impact of consistent, moderate exercise on overall cardiovascular risk can be significant, even if the effect of an exercise program on any one risk factor may typically be minimal.

Exercise has many physiological advantages, two of which are increases in muscle strength and function and the body's capacity to absorb and utilise oxygen (maximal oxygen consumption or aerobic capacity)^[85]. Regular everyday tasks can be carried out with less weariness as one's capacity to carry and use oxygen improves. Patients with cardiovascular disease, whose exercise ability is usually lower than that of

healthy individuals, should pay special attention to this^[65]. Additionally, there is evidence that exercise training enhances the blood vessels' ability to dilate in response to hormones or exertion, which is associated with increased vascular wall function and an enhanced capacity to supply oxygen to the muscles during exercise. Research comparing muscle strength and flexibility before and after exercise regimens indicates benefits in bone health and everyday functioning, along with a decreased risk of back discomfort and disability, especially in older age groups^[82].

Additional advantages of physical activity include:

- Increase in exercise tolerance
- Reduction in body weight
- Reduction in blood pressure
- Reduction in bad (LDL and total) cholesterol
- Increase in good (HDL) cholesterol
- Increase in insulin sensitivity.

Empirical review

Review of Previous Studies on Exercise Frequency and Hematological Parameters

A study done by Musa *et al.*, (2024) show that in sedentary young men, a 90-minute football game causes an increase in plasma volume within the physiological range and a drop in red blood cell indices^[52]. Even if these alterations are substantial, it is doubtful that they will affect cardiovascular health in healthy people. Leukocyte and erythrocyte parameters are not significantly altered by short-term aerobic exercise, whereas platelet parameters are in University students involved in short term aerobic exercise. Additionally, there was a gender-based rise in granulocytes and lymphocytes after exercise that persisted for 24 hours^[86]. RBC, Hb, and Hct increase significantly during incremental physical exercise of highly trained athletes of different sport specialization, and then return to resting values within 30 min upon test termination. Regardless of the physiological profile of the athletes, Ciekot-Sołtysiak *et al.*, (2024) noticed a uniform pattern of exercise-induced changes in Hct and RBC characteristics. However, throughout the progressive test, the changes in Hct and RBC parameters are proportionate to the level of exercise^[48]. The higher haemoglobin concentration linked to exercise intensity is probably an adaptation to the increased oxygen demand of tissues, primarily skeletal muscles.

Anwer & Babiker (2021) found that after an hour of football activity, Sudanese football players had considerably higher levels of total white blood cells (TWBCs)^[58], absolute neutrophils, absolute lymphocytes, and platelet counts. Physical exercise whether leisure or strenuous affects leukocytosis and hemostasis in male and female genders^[59]. Johannsen *et al.*, (2012) gave a contrasting view on the effects of aerobic exercise on TWBC in post-menopausal women^[87]. Their findings show that aerobic exercise training reduces total WBC and neutrophil counts, in a dose-dependent manner, in overweight/obese postmenopausal women and is especially beneficial for those with systemic low grade inflammation.

For a year, a set of participants has been exercising for at least an hour every day, five days a week. Hb, total WBC count, and platelet count were found to increase significantly following exercise. Following exercise, the RBC count was also markedly elevated. A comparison of the differential leukocyte count before and after an hour of exercise revealed a highly significant rise in neutrophil and monocyte counts as

well as a considerable increase in lymphocytes^[88].

Fifty girls and fifty guys are chosen at random by Kilim *et al.*, the platelet count was assessed both prior to and following five minutes of moderate cycling ergometer activity^[89]. Following a thorough analysis of all the data, they came to the conclusion that moderate exercise increases platelet count without causing platelet activation^[89]. Twelve handball players who took part in a five-day competition had their platelets measured. Prior to and following the competition, every haematological parameter was measured. The author ends by stating that, in comparison to baseline data, the platelet count increased significantly following the handball tournament^[90]. Lippi *et al.*, evaluated the platelet counts of thirty-one athletes following a half-marathon run of 21.1 kilometres^[91]. Blood samples were taken prior to, during, and three and twenty hours after the run. According to their findings, the platelet count increased following the competition compared to the pre-marathon run and returned to baseline after 20 hours^[91].

Conclusion

Physical exercise influences hematological parameters among young adults, with effects varying according to exercise frequency, intensity, duration, individual characteristics, nutritional status, and environmental conditions. Moderate, consistent exercise may support healthy immune and blood cell function, whereas excessive training may produce adverse changes. Promoting regular moderate physical activity among young adults is therefore essential for maintaining hematological health, preventing disease, and establishing sustainable healthy lifestyle practices that reduce future risks of chronic illnesses.

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