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Variations in haematological parameters in obese, sedentary, aerobic-training and resistance-training populations

Haematological differences among resistance-training populations

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Abstract
Aim: Although physical activity has a considerable impact on various laboratory markers, evidence on haematological alterations, cardiovascular risk factors and inflammatory mediators after resistance-training is limited. The aim of this study is to investigate alterations in hematological parameters among populations engaging in obesity, sedentary behaviour, aerobic exercise, and resistance training.
Material and Methods: A study with 122 male volunteers aged 18-45 divided into four groups based on exercise type and Body Mass Index. It compared blood parameters of resistance exercisers to normal-weight sedentary, overweight/obese individuals, and normal-weight aerobic exercisers. Blood count included RBC, HCT, leukocytes, reticulocyte, platelet counts, hemoglobin, MCV, MCH, RDW, and MPV. Neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII) were determined based on hemogram.
Results: Monocytes and HCT levels were significantly higher in resistance exercisers, whereas MPV and MCHC levels were significantly lower. It is also noteworthy that resistance training does not alter any of the inflammatory indices.
Discussion: Resistance training may have negative effects on individuals at cardiovascular risk, but it was found to be advantageous for athletes or sedentary adults with a good vascular endothelium to raise HCT and, possibly, enhance tissue blood supply by inducing vasodilation. Furthermore, extended resistance workouts did not enhance the inflammatory indices measured in the blood, and it was proven to be critical for the body's defence because they increase the number of monocytes, which subsequently spread to the tissues as macrophages.

Keywords
Platelet-to-LYmphocyte Ratio (PLR), Systemic Immune-Inflammation Index (SII), Neutrophil-to-LYmphocyte Ratio (NLR), Cardiovascular Risk, Exercises

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Introduction

Physical activity is essential for the promotion of health. However, incorrect exercise could be hazardous, and cardiovascular complications during exercise could result in sudden death [1]. Many epidemiologic studies have shown that exercise protects against coronary artery disease (CAD). A recent meta-analysis revealed that moderate to vigorous physical exercise is connected with a decreased risk of coronary artery disease. In particular, intense and moderate exercise reduced the incidence of coronary artery disease by 27% and 11%, respectively, as compared to persons with minimal or no physical activity [2]. Several variables, such as dehydration, an increase in hematocrit, plasma viscosity, red blood cell (RBC) aggregation, and a decrease in RBC deformability caused by intense exercise, may contribute to a decrease in blood fluidity [1]. Resistance exercise training (RET) combined with dietary protein supplementation is a frequent technique among athletes and leisure exercisers with the goal of improving RET-induced increases in muscle development and strength [3].

Muscle injury brought on by exercise triggers an augmented immune response, leading to the production of inflammatory signalling molecules from leukocytes and their various cellular subtypes. The final stage of this process is the entry of inflammatory cells into the injured region [4]. There is a correlation between high-intensity exercise and a biphasic shift in the number of circulating leukocytes. An increase in the overall number of leukocytes is seen in the immediate post-exercise phase. This rise in the total number of leukocytes happens mostly at the cost of lymphocytes, neutrophils, and, to a lesser degree, monocytes [4]. Atherosclerosis is linked to the presence of persistent inflammation at a low grade. Inflammatory cells, such as white blood cells (WBC), are critical to the progression of atherosclerosis in the arterial artery wall. This disease may be prevented by eliminating inflammatory cells. Because of this, having a high WBC count is linked to an increased risk of developing cardiovascular illnesses [5].

Obesity is a chronic metabolic disorder that is related to cardiovascular disease as well as increased morbidity and mortality rates . In recent years, it has come to be regarded as the nation's important public health problem and an epidemic [6]. Numerous studies have shown that obesity and being overweight are connected with an increased platelet reactivity [7, 8]. As a result, official guidelines propose that each person should undertake the cardiorespiratory exercise for 20–60 minutes three–five days each week. These guidelines are based on research that has shown that strenuous physical activity decreases the risk of cardiovascular disease [9].

We planned prospective research to examine the changes of some parameters of the complete blood cell count (CBC) in resistance exercisers, normal-weight sedentary, normal-weight regular exercisers, and overweight or obese in order to determine whether the increased cardiovascular problems and inflammation that are sometimes observed after certain kinds of exercise may be reflected by variation of red cell distribution width (RDW) , mean platelet volume (MPV) and Systemic immune-inflammation index (SII). There was no other research in the literature that compared the hematological effects of resistance exercise to those of obesity and regular exercise.

Material and Methods

This research was conducted at the xxxx University Faculty of Medicine, Department of Physiology laboratory, and 122 healthy adult males participated in this study. All individuals were informed of the objectives of the study and permission was acquired prior to their participation.

The research was carried out in a manner that was compliant with both the Declaration of Helsinki and the standards for Good Clinical Practice. After receiving ethical approval and having a signed permission form on file, we started recruiting participants. Authors had access to information that could identify individual participants during or after data collection.

Individuals were placed into one of four categories based on their preferred type of exercise and their body mass index (BMI);

(1) Resistance Exercise Group (REG): This group consisted of 31 healthy male volunteers who participate in bodybuilding on a regular basis and have a BMI that is greater than what is considered normal (28,11). The mean duration of exercise for participants was determined as 6.45 ± 5.45 (year ± standard deviation)

(2) Sedentary Group (SG): This group consisted of 29 healthy male volunteers who did not exercise regularly (Sedentary) and had a normal BMI (22.70).

(3) Obesity and Overweight Group (OG): This group consisted of 30 healthy male volunteers who did not participate in any regular activities and whose body mass index was much higher than the average (28.75).

(4) Regular Aerobic Exercise Group (AEG): This group consisted of 32 healthy male volunteers with a normal body mass index (22.56) who participated in regular aerobic exercise in the form of brisk walking and running on a regular basis. The mean duration of exercise for the participants was determined as 5.93 ± 3.10 (year ± standard deviation).

Collection of Blood and Hemogram Measurement

Following the collection of the essential demographic data, 2 ml of venous blood was drawn from each volunteer participant following an 8-hour overnight fasting into tubes containing ethylenediaminetetraacetic acid. Within 2 hours of blood withdrawal, the Abbott Cell-DYN Ruby Hematology Analyzer (USA) was used to analyse whole blood. Red blood cell (RBC), haematocrit (HCT), reticulocyte, and platelet counts, haemoglobin, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), RBC distribution width (RDW), and mean platelet volume (MPV) were included in the complete blood count. Also, systemic immune-inflammation index (SII), the neutrophil-to-lymphocyte ratio (NLR), the platelet-to-lymphocyte ratio (PLR), and the HGB-to-RDW ratio were calculated. Total neutrophil and platelet counts were divided by lymphocyte counts to get the NLR and PLR, respectively. The peripheral platelet (P), neutrophil (N), and lymphocyte (L) counts were used to arrive at the Systemic immune-inflammation index (SII) using the formula SII = (P N)/L [10].

Statistical Analysis

For statistical analysis, the Windows-compatible IBM SPSS 25.0 (IBM SPSS Inc, Chicago, IL, USA) package program was used. Using the Shapiro-Wilk test, skewness, kurtosis, histogram, and Q-Q plot, we assessed the conformity of the data to the normal distribution. Continuous variables following the normal distribution were represented by the Mean (M) ± Standard Deviation (SD), whereas continuous variables not following the normal distribution were expressed as the Median (interquartile range of values). Between groups, non-normally distributed data were analysed with the Kruskal Wallis H test (Bonferroni correction was used for within-group comparisons), and normally distributed data were analysed with one-way analysis of variance (Tukey's test for within-group comparisons). In the analysis, 95% was accepted as the confidence interval. $p<0.05$ was statistically significant.

Ethical Approval

This study was approved by the Ethics Committee of Harran University Clinical Research (Date: 2022-09-05, No: 2022/17/27).

Results

There was no statistically significant difference in the height and age values of the groups when their demographic information was evaluated (Table 1). As predicted, the BMI and weights of obese and muscle-obesity resistance exercisers were considerably greater than those of lean groups (CE and REG).

The results of the study's hemogram analysis are outlined in Table 2, which provides a summary of the findings. In the groups that were under investigation, there were no significant changes found in WBC, NEU, LYM, HGB, MCH, RDW and PLT. Similar outcomes were observed for inflammatory indexes such as SII, NLR, PLR, and the HGB-to-RDW ratio.

Discussion

There is compelling evidence that unhealthy lifestyle factors, such as smoking, alcohol consumption, physical inactivity, and being overweight or obese, contributed to approximately 60% of deaths and were linked to chronic inflammation status, highlighting the potential modifiability of the effects of chronic inflammation by these factors [11]. It has been shown in recent years that the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII) are all valid indicators of inflammatory responses at the cellular level.

Studies have shown a rise in NLR in response to acute or intense exercise [12]. However, prolonged (chronic) exercise regimens appear to have contradictory findings. In the study conducted by Makras et al. (2005) on healthy male adults, four weeks of military training consisting of intermittent moderate activity decreased neutrophils and the neutrophil-to-lymphocyte ratio [13]. In the study conducted by Wang et al. on obese male teenagers, a 4-week diet and activity intervention dramatically decreased the neutrophil to lymphocyte ratio. The intervention was linked with a considerable reduction in pro-

Table 1. Demographic data

	REG (n=31) Mean ± SD	SG (n=29) Mean ± SD	OG (n=30) Mean ± SD	AEG (n=32) Mean ± SD	p
Age (Year)	28,55 ± 4,83	27,10 ± 5,98	28,67 ± 5,49	26,63 ± 5,52	0.357
Height (cm)	176,45 ± 6,63	177,21 ± 5,28	178,00 ± 5,90	179,38 ± 5,68	0.244
Weights (kg)	87,62 ± 7,17[a,c]	71,34 ± 7,40[d]	91,22 ± 8,34[f]	72,62 ± 6,11	<0.001
BMI (kg/m²)	28,11 ± 0,99[a,c]	22,70 ± 1,97[d]	28,75 ± 1,64[f]	22,56 ± 1,43	<0.001

Variables were represented by the Mean (M) ± Standard Deviation (SD). RGE: Resistance Exercise Group, SG: Sedentary Group, OG: Obesity and Overweight Group, AEG: Regular Aerobic Exercise Group. a: Defined as a statistical difference between REG and SG. c: Defined as a statistical difference between REG and AEG. d: Defined as a statistical difference between SG and OG. f: Defined as a statistical difference between OG and AEG

Table 2. Hematological Parameters

	REG (n=31) Mean ± SD	SG (n=29) Mean ± SD	OG (n=30) Mean ± SD	AEG (n=32) Mean ± SD	p
WBC	8,55 (3,00)	7,67 (2,29)	7,65 (2,64)	6,86 (2,02)	0,134
NEU	4,58 (1,91)	4,08 (1,71)	3,94 (1,53)	3,92 (1,12)	0,771
LYM	2,54 (1,73)	2,39 (0,58)	2,77 (1,06)	2,31 (0,77)	0,073
MONO	0,67 (0,20)[c]	0,64 (0,22)	0,54 (0,18)	0,51 (0,20)	0,005
HGB	16,60 (1,50)	16,50 (1,60)	16,30 (1,27)	16,40 (4,03)	0,618
HCT	53,15 ± 4,03[a,b,c]	50,32 ± 3,12	50,74 ± 3,21	50,96 ± 2,50	0,005
MCV	87,25 (7,90)[b]	87,70 (3,79)	85,30 (3,80)	87,60 (5,28)	0,024
MCH	28,00 (1,70)	28,35 (1,80)	27,50 (2,17)	28,50 (2,10)	0,253
MCHC	31,80 (2,30)[b,c]	32,20 (0,90)	32,50 (1,45)	32,45 (1,83)	0,018
RDW	11,55 (1,93)	10,90 (1,30)	11,15 (1,03)	11,00 (0,78)	0,564
PLT	268,44 ± 50,06	283,40 ± 46,60	288,60 ± 50,25	277,53 ± 59,79	0,495
MPV	6,96 ± 1,38[a]	7,84 ±1,19	7,64 ±1,27	7,39±1,00	0,038
NLR	1,56 (0,90)	1,67 (0,63)	1,37 (0,56)	1,69 (0,58)	0,39
PLR	108,36 (61,82)	112,90 (36,30)	102,34 (49,02)	123,95 (29,04)	0,203
SII	426,51 (395,13)	501,19 (245,01)	418,57 (163,12)	482,71 (202,67)	0,602
HGB/RDWR	1,46 ± 0,17	1,46 ± 0,16	1,47 ± 0,13	1,48 ± 0,15	0,897

Variables were represented by the Mean (M) ± Standard Deviation (SD) or Median (interquartile range of values, IQR). RGE: Resistance Exercise Group, SG: Sedentary Group, OG: Obesity and Overweight Group, AEG: Regular Aerobic Exercise Group. a: Defined as a statistical difference between REG and SG. b: Defined as a statistical difference between REG and OG. c: Defined as a statistical difference between REG and AEG inflammatory cytokine concentrations[14]. In contrast, there was no significant difference between the obese, resistance exercise, sedentary, and regular exercise groups in this study. The absence of long-term change in blood neutrophil and lymphocyte levels may be related to the body's adaptability to this environment, despite the possibility of acute increases in blood neutrophil and lymphocyte levels in chronically applied regular exercise regimens.

PLR levels were significantly affected by high intensity exercise, with values almost twice as high as at rest. Possible explanations include exercise-dependent platelet mobilization in peripheral circulation [15]. Regarding the effect of persistent exercise on the PLR, after 3 weeks of endurance exercise in a group with multiple sclerosis, no changes were seen [16].Similarly, there was no significant difference in PLR values between the chronic exercise groups and the obese group in our investigation.

Recently, the systemic immune-inflammation index (SII), which is derived using neutrophil, lymphocyte, and platelet counts in peripheral blood, was proposed as a measure of the equilibrium between systemic inflammation and immunological state. Similar to NLR, SII readings are highest when neutrophil and platelet numbers are high and lymphocyte numbers are low. Prior research suggested that a high SII level was related to a worse outcome for individuals with certain cancers [11]. Also, It was hypothesized that chronic inflammation, which might cause reactive nitrogen species, reactive oxygen species, genomic instability, and cell senescence, increased the risk of cardiovascular disease and death in participants with higher SII levels [11]. Schlagheck et al., who aimed to evaluate the differences in cellular immunological alterations between acute endurance exercise and resistance exercise, determined that resistance exercises have no effect on SII [17]. Taranu et al. discovered no significant changes in the mean values of NLR, SII, and PLR between the overweight obese and severely obese weight groups [18]. In our study, resistance exercisers were compared to the obese, regular exercise, and sedentary groups. However, there was no significant difference between the groups.

During exercise recovery, under the effect of glucocorticoids, substantial numbers of lymphocytes and monocytes enter the circulation. Monocytes activated by exercise are likely to penetrate skeletal muscle and develop into tissue-resident macrophages that enable repair and regeneration, especially following strenuous exercise sessions that produce severe skeletal muscle damage. Additionally, monocytes with effector characteristics are redeployed preferentially after exercise [19]. Among the interesting findings of our study is that the monocyte count was highest in the resistance-exercising group. This increase suggested that resistance workouts, which place a greater demand on the skeleton's muscular tissue, can increase the number of monocytes.

MPV, an indication of platelet activation, plays a significant role in the pathogenesis of cardiovascular disorders [20]. MPV is a crucial biological characteristic, and bigger platelets possess a greater thrombogenic potential . Based on the findings of this study, resistance exercise has been found to lower MPV and may lessen the thrombogenic potential of blood.

Several studies have shown that exercise increases haematocrit and blood viscosity. It is hypothesized that many mechanisms, including fluid shift, water loss, release of sequestered RBCs from the spleen, and water retention in the muscle, are responsible for this increase [21]. Young erythrocytes, the number of which rises as a consequence of the hemolysis that occurs during exercise, have a lower MCHC as well as a higher MCV and deformability. Also, It is well established that a lower MCV and a higher MCHC both contribute to an increase in the internal viscosity of RBCs, which in turn reduces their deformability [22]. Kilic Toprak E. et al. reported an increase in haematocrit and blood viscosity following 12 weeks of progressive resistance training [23]. In contrast, aerobic exercise research demonstrates that extended outdoor walking does not affect blood viscosity or haematocrit [24]. High blood viscosity has generally been believed to have a detrimental effect on performance from an exercise physiology standpoint. An excessive rise in blood viscosity during exercise has been hypothesized to be potentially harmful to the cardiovascular system because it is believed to enhance vascular resistance and the post-load work of the heart [21]. But it is also known that the increased wall shear stress may induce nitric oxide (NO) generation by endothelial cells to provide a vasodilatory compensation in response to an increase in blood viscosity [25].

The study's limitations include the fact that it did not use a bigger sample size by including other cardiovascular imaging modalities, blood viscosity, and other inflammation indicators. Furthermore, the study is limited by the absence of longitudinal data analysis that tracks changes in hematological parameter responses to resistance training over time and by the lack of information regarding dietary factors that may have influenced hematological parameter levels. These restrictions may be considered in future research. Nevertheless, this compensating reaction is contingent on the health condition of the endothelium, and a rise in blood viscosity may be more harmful to the vascular system in the event of endothelial dysfunction.

Conclusion

According to the findings of our study, resistance training may have negative effects on individuals at cardiovascular risk, but it was found to be beneficial for athletes or sedentary individuals with a healthy vascular endothelium to increase HCT and thereby increase tissue blood supply by stimulating vasodilation. In addition, prolonged resistance workouts did not produce any rise in the inflammatory indices assessed in the blood in our study, and it was determined to be crucial for the body's defence since they increase the number of monocytes, which then spread to the tissues as macrophages.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or compareable ethical standards.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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