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The Influence of Detraining After Duration of Aerobic Exercise on Blood Plasma Lipoproteins and Highest Oxygen Intake

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Abstract

The research found that aerobic exercise for eight weeks significantly increased High-Density Lipoprotein (HDL) and maximum oxygen absorption while significantly lowering LDL and VLDL levels. Female basketball players had negative effects on their blood lipoproteins' maximal oxygen consumption after 4 weeks of training. As a consequence, 24 female Premier Basketball League players between the ages of 18 as well as 28 consented to take part in the study. Before the training phase, Maximum Oxygen Consumption (MOC) tests were carried out and blood samples were obtained after 4 weeks of detraining and 8 weeks of aerobic activity. The 8 aerobic exercise plans included three 60-minute workout sessions each week at 70 to 75 percent of one's maximal oxygen intake. The data were analyzed using a dependent t-test as well asinvestigated to test the significance level of $P \leq 0.05$. According to the study's findings, aerobic exercise for 8 weeks significantly increased maximum oxygen uptake and High-Density Lipoprotein (HDL), while lowering levels of Low-Density Lipoprotein (LDL) and Very-Low-Density Lipoprotein (VLDL). Also, it was shown that after 4 weeks of practice, female basketball players' blood lipoproteins' maximal oxygen consumption had been adversely affected.

Keywords:(LDL), (HDL), (MOC), Aerobic exercise, (VLDL), Blood plasma lipoprotein

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1. Introduction

Maintaining an athlete's aerobic power across many training seasons is one of the most crucial jobs for coaches and players. Aerobic power is one of the crucial qualities for an athlete to perform well. Detraining, or the athlete's break such as consistency or regularity of applicable exercise, is the main barrier impeding the development and growth of biometry skills [1]. It is uncertain how regular exercise affects the hazard element for CoronaryHeart Disease (CHD) in a child with teenagers. This work presents an understanding of how exercise affects blood lipids including lipoproteins, namely Total Cholesterol (TC), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), Triglycerides (TG) in adolescents as well as children [2]. Numerous investigations have shown that aerobic exercise improves young, healthy women's body composition, cardiorespiratory fitness, and lipoprotein-lipid profiles. The majority of known and freshly discovered cardiovascular risk variables have been shown to stabilize in a very favorable manner when aerobic energy expenditure is enhanced [3]. Figure 1 illustrates the preventive benefits of exercise on the cardiovascular system.

Many studies have shown that exercise is a very effective non-pharmacological therapy for Metabolic Syndrome (MS), improving blood lipid levels including TC, TG, and LDL-C, body composition, and also sleep quality in patients [4]. Dyslipidemia, which includes hypercholesterolemia, hypertriglyceridemia, and high LDL-C levels, along with decreased HDL-C levels, is hypothesized to promote atherogenesis. It's a difficult condition that increases the risk of heart attacks and strokes. The high stage of LDL-C as well as the low stage of HDL-C have been linked to Myocardial Infarction (MI) and stroke 1, 2, 3, and 4. Data show that in 2017, the age-adjusted death rate regard to CardioVascular Disease (CVD) was 219.4 per 100,000 individuals [5].

After 20 weeks of aerobic exercise, 34 healthy, untrained middle-aged males ($x = 58.2\ years$) agreed to participate in a longitudinal study to examine changes in lipoprotein profiles [6]. The study [7] looked at how aerobic exercise training affected the stage of serum Fibroblast Growth Factor-21 (FGF21) in overweight with obese males. The article [8] investigated in adults Circulating Endothelial Cell (CEC), HDL-C, and a Cluster of Differentiation 146

(CD146) levels to determine the preventive CVD benefit of Aerobic Exercise Training (AET). The research [9] looked at how combined exercise would affect inflammatory markers, paraoxonase 1 (PON1) activity, and plasma lipid levels in obese people. The author [10] looked at how a 10-week aerobic exercise program affected women with type II diabetes' atherogenic indices, lipid profiles, and body composition. The sequence of acai pulp consumption combined with AET at a high-fat diet-influenced Non-Alcoholic Fatty Liver Disease (NAFLD) has been studied. The study [11] examined the effectiveness of AET in reducing Insulin Resistance (IR), atherogenesis, and dyslipidemia brought on by a severe low-sodium diet (LS). The author [12] investigated how eight weeks of moderate-intensity exercise, together with cardio and weight training, affects women's cardiovascular risk factors.The research [13] evaluated the impact of Ziziphusjujuba (ZJ) extract and high-intensity training (HIT) on rat tissue nesfatin-1. The study [14] proposed that aerobic exercise reduces obesity by influencing downstream target genes of the Long noncoding RNA steroid receptor peroxisome proliferator-activated receptor gamma (RNA activator SRA/p38/JNK/PPAR pathway). The study [15] examined the effects of pregnancy-related lipid concentrations of exercise, DocosAhexaenoic Acid (DHA), and EicosaPentaenoic Acid (EPA). The allocation of participants to aerobic exercise or control groups was random. The research [16] determined if study/intervention variables are related to changes in the Standard Lipid Profile (SLP) of persons with three or more metabolic syndrome (MetS) risk factors after AET.

2. Materials and methods

The research methodology combines practical and quasi-experimental elements. All female basketball players that participated in Basketball Premier League in 2009 made up this statistical population. 60 gamers voluntarily provided samples and participated in the research. Nonetheless, a total of 31 participants were picked, with 24 serving as the sample due to homogeneity.Participants' blood was collected using autoanalyzer equipment, and their mental and physical health was assessed by a questionnaire. They also underwent a 1.5-mile running test to see who could consume the most oxygen. The procedure for gathering data was as follows: After completing the questionnaire and confirming that there was no medical history, the individual's height and weight were assessed. The maximum quantity of oxygen consumed was determined by adding the subjects' weight (kg) and distance walked (meters) to equation (1) below. Participants were instructed to walk 1.5 miles.

$$[Maximum\ oxygen\ consumption\ (moc) = 88.02 - 0.16(weight\ (w)\ inkg) - 2.76(Time\ (t)\ inmins) + 3.716 + Gender]$$

Males obtain a score of 1 and women a score of 0.The next eight weeks of the program were performed in two cycles of four weeks each, with three sessions of 60 minutes each planned each week at a heart rate of 55 to 70% for 3 weeks of overburden with one week of no overburden. Warm-up exercises, this primary exercise, intermittent aerobic activity with a heart rate goal of 55% for the first week, and then 5% steadily increasing exercise intensity each week are all included in the training session schedule. After a five-minute warm-up, stretching, and two sluggish movements, the workouts comprised endurance running and circular motions. Also, at a significance threshold of $P \leq 0.05$, descriptive statistical techniques like standard deviation, and mean and it is based on inference t-tests with the retest were used to examine the variances throughout several stages.

3. Results and Discussions

The maximal oxygen intake is determined by how well the three respiratory, circulatory, and muscle systems work, the data show. Each of these three devices is impacted by aerobic activity. These 3 devices adjust as a consequence of endurance training, which raises people's maximal aerobic capacity and tolerance for more strenuous activity. Hence, as was amply shown in this research, an increase in aerobic power after eight weeks of aerobic training is acceptable. One of the most significant changes brought on by endurance training anytime the plasma volume rises is an increase in plasma volume. MOC rises as blood volume, stroke volume, and cardiac output all rise.

It demonstrates how well the pulmonary function metrics in trained individuals quickly decline following detraining.This decline manifests as an improvement in respiratory exchange, a reduction in the highest amounts of respiration, a fall in the MOC utilized, a decline in oxygen consumption each pulse, and just a decline in oxygen consumption overall. The greater oxygen consumption of sports-trained athletes during the detraining phase declines through 6 to 20% in around 8 weeks with eventually recovering to the pre-workout condition, still, it subsequently remains stable at a superior stage either on a level with the sedentary individuals in the control group.

Tables 1 and 2 correspond to earlier studies that examined many physiological markers, including MOC after glycogen depletion and the impact on 4 weeks with glycogen depletion followed by 8 weeks of aerobic activity on MOC. The findings of the previous study conflict with the four weeks of detraining, which reduced endurance capacity by around 21% and reduced performance from an average of 79 to 62 minutes.Reduced endurance capacity might be the result of changes to material consumption or electrolyte control. The maximal oxygen intake of athletes was maintained throughout the stoppage phase, according to several published research, for example. The quantity of physical activity the athletes engaged in during the earlier phases may be the cause of this variation. Detraining causes diverse responses in various indications. For instance, during the first weeks of glycogen depletion, the localized durability factors that were created during the training phase are lost far more quickly than the strength factor.

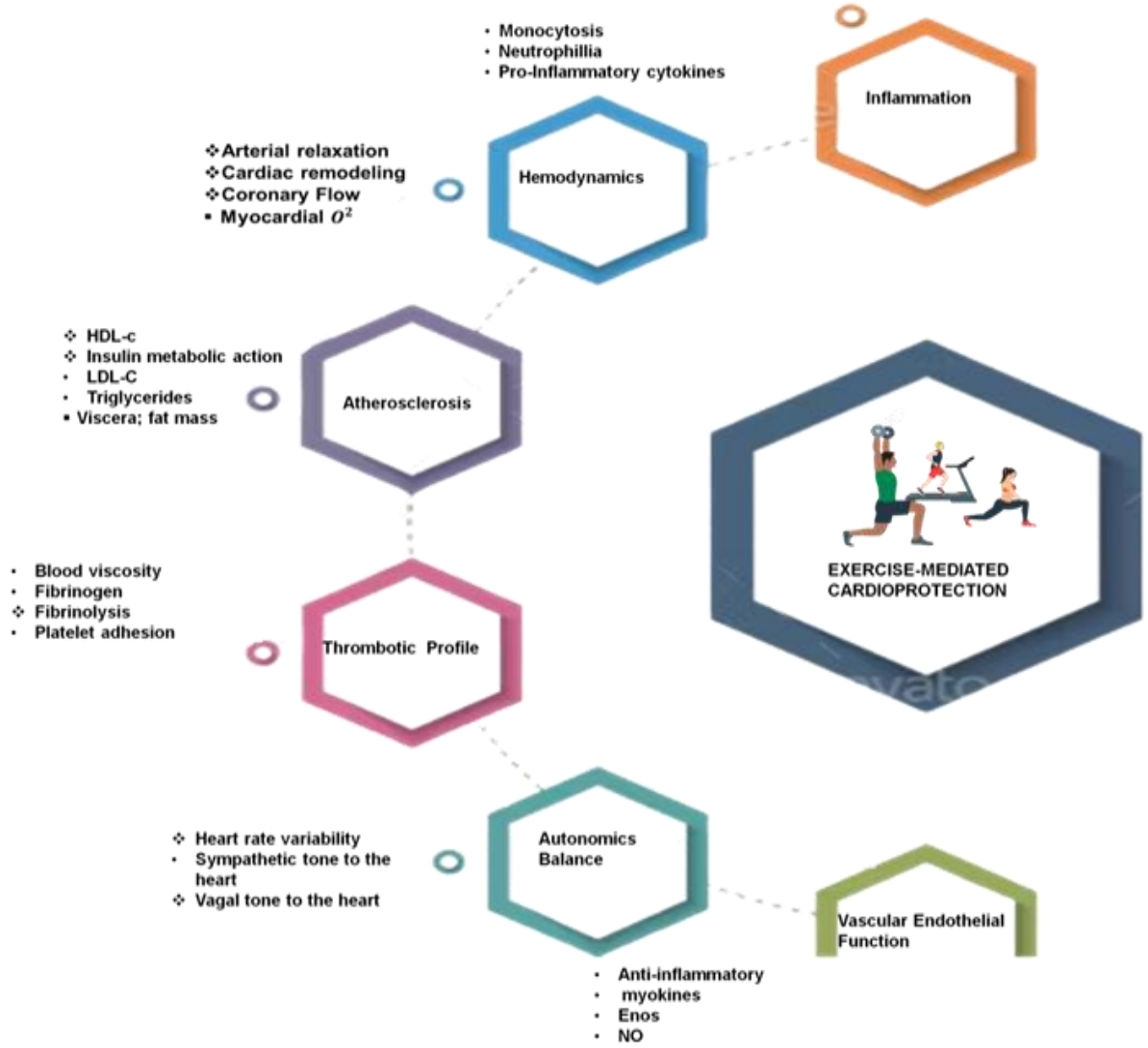

Figure 1: Cardiovascular system protection brought by exercise

Table 1: Provided pre- and post-test criteria for the research variables

Statistical indicators Variables		Number of samples	Avg changes	Min	Max	SD
(HDL) (mg/dl)	pre-test	24	41.91	29.97	24.38	740
	Post-test	24	44.03	33.89	56.43	6.98
	After 4 weeks ofdetraining	24	42.41	31.04	55.02	7.22
	After eight weeks of detraining	24	42.04	31	56	7.49
(VLDL) (mg/dl)	pre-test	24	24.69	14.62	50.42	9.17
	Post-test	24	21.26	13.22	43.02	7.66
	After 4 weeks of detraining	24	24.32	15.62	48.02	8.67
	After eight weeks of detraining	24	25.73	16.36	49.101	8.96
LDL) (mg/dl)	pre-test	24	80.95	42.02	128.02	1.79
	Post-test	24	75.02	34.02	110.02	1.71
	After 4 weeks of detraining	24	76.27	36.02	121.02	1.74

	After eight weeks of detraining	24	80.53	41.03	128.03	1.81
(vo_2max)	pre-test	24	45.27	38.23	50.26	3.42
	Post-test	24	52.29	42.12	58.93	4.34
	After 4 weeks of detraining	24	47.93	40.77	50.58	3.96
	After eight weeks of detraining	24	45.23	38.03	50.09	3.48

Table 2: An analysis of a female Premier League basketball player's avg MOC (vo_2max) before with after 8 weeks of aerobic exercise

(vo_2max) maximum	Mean (m)	Mini	Max	SD	T-test	p
Before exercise	46.24	39.22	51.28	4.50	-19.918	0.001
After exercise	53.28	43.10	59.92	5.42		

Table 3: Female basketball players' pre- and post-8 weeks of aerobic exercise avg LDL, HDL, and VLDL values were compared

Statistical Indicators / Variable		Mean(m)	Min	Max	SD	T-test	P
VLDL (mg/dl)	Before exercise	25.68	15.61	51.41	10.15	8.628	0.001
	After exercise	22.25	14.21	44.00	8.65		
LDL (mg/dl)	Before exercise	80.94	42.00	118.00	2.77	11.46	0.001
	After exercise	76.01	35.02	120.02	2.83		
HDL (mg/dl)	Before exercise	42.90	31.10	25.37	8.48	-5.88	0.001
	After exercise	45.01	34.97	57.51	7.97		

Table 4: Following 8 weeks of training and 4 weeks of detraining, the variation in group averages from the stage

Statistical indicators / Various tests	The variance between the maximal oxygen consumption implies (vo_2 max)	Significance level
After exercise – after 4 of detraining weeks	5.844	0.001

Table 5: The difference between the mean difference LDL, VLDL, HDL

Statistical indicators	Mean Difference (MD)			Significance Level
Difference tests	LDL (mg/dl)	VLDL (mg/dl)	HDL (mg/dl)	
After exercise and detraining weeks	0.26	3.06	2.728	0.001

Adenosine Triphosphate (ATP) levels were 37 to 70% higher than pre-workout levels, however, in inactive individuals who had undergone a six-week endurance training program, ATP production reduced through around 12 to 28% in the course of the 3 weeks of post-workout glycogen depletion. Consequently, it is acceptable to assume that the activity of a mitochondrial enzyme is the cause of this drop.

Only by continuing to exercise consistently is it able to maintain the consistency of endurance training. Exercise-induced changes during detraining periods vanish. A few variables have a rapid rate of change. For former professional athletes, the elements in a way period are particularly risky because, as the detraining period lengthens with the HDL, LDL ratio rises over a certain threshold, hypertri-glyceridemia, which up to six times increases the risk of coronary heart disease, develops. Notwithstanding what some studies indicate, exercise after a detraining period may be beneficial for both athletes and inactive people. The study discovered that the number of lipoproteins had altered along with the effects of aerobic exercise on lipoproteins had lessened after less than five weeks of

detraining. Table 3-5 is valid given how strongly lipoproteins are influenced by the length of glycogen depletion.

An increase in leptin synthesis, a hormone that activates lipase and insulin and aids in the acceleration of body fat levels, is one of the reasons underlying the fast rise in body fat during the detraining phase. The hormones lipase and Photo Luminescent Long Persistent Luminescence (PL-LPL), which both inhibit and stimulate fat production, are inhibited by an increase in leptin levels. In this research, there is a substantial difference in the means of LDL, VLDL, and HDL levels during exercise and four and eight weeks after detraining.

Higher HDL-C levels, lower LDL-C levels, with greater insulin sensitivity to glucose are the key advantages of exercise. Moreover, it makes postprandial blood lipoproteins like chylomicrons more transparent in plasma. This catalytic enzyme lipase greatly reduces the quantity of blood lipoprotein during chylomicron migration. The PL-LPL activity is influenced by aerobic exercise. The PL-LPL levels, which are lowered by detraining, were greater in regular aerobic exercises. The study's findings are both consistent and different from its findings. Longer courses, the kind of exercise, participants' ages, and genders may all contribute to this discrepancy.

So far, additional study is needed to determine if other variables may also be at play in the changes in lipoprotein levels that occur during aerobic exercise and detraining. So, athletes must continue to engage in regular exercise to retain adaptations from aerobic activity.

4. Conclusions

Maintaining aerobic power over the many training seasons is one of the most crucial responsibilities for coaches and players. Aerobic power is one of the crucial qualities for an athlete's effective performance that develops throughout the training stages.The primary issue endangering the growth and improvement of biomotor abilities is detraining, or the athlete's departure by the structure and coherence of regular as well as suitable activity. The study's findings demonstrated that aerobic exercise for 8 weeks significantly increased maximum oxygen uptake and HDL while significantly lowering LDL and VLDL levels. After 4 weeks of practice, female basketball players' MOC of blood lipoproteins also decreased.

References

[1] H. Shirvani and E. Arabzadeh. (2020). Metabolic cross-talk between skeletal muscle and adipose tissue in high-intensity interval training vs. moderate-intensity continuous training by regulation of PGC-1α. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity, 25, 17-24.

[2] Y. Escalante, J.M. Saavedra, A. García-Hermoso, and A.M Domínguez. (2012). Improvement of the lipid profile with exercise in obese children: a systematic review. *Preventive medicine*, *54*(5):pp.293-301.

[3] D. Kostrzewa-Nowak, R. Nowak, Z. Jastrzębski, A. Zarębska, M. Bichowska, I. Drobnik-Kozakiewicz, Ł. Radzimińsk, A. Leońska-Duniec, K. Ficek, and P. Cięszczyk. (2015). Effect of 12-week-long aerobic training programme on body composition, aerobic capacity, complete blood count and blood lipid profile among young women. *Biochemia medica*, *25*(1):pp.103-113.

[4] Y. Zhou, W. Wu, Y. Zou, W. Huang, S. Lin, J. Ye, and Y. Lan. (2022). Benefits of different combinations of aerobic and resistance exercise for improving plasma glucose and lipid metabolism and sleep quality among elderly patients with metabolic syndrome: a randomized controlled trial. *Endocrine Journal*, *69*(7):pp.819-830.

[5] R.I. Doewes, G. Gharibian, B.A. Zaman, and R., Akhavan-Sigari. (2022). An updated systematic review on the effects of aerobic exercise on human blood lipid profile. *Current problems in cardiology*:p.101108.

[6] F. Morales-Palomo, M. Ramirez-Jimenez, J. F. Ortega, A. Moreno-Cabañas and R. Mora-Rodriguez. (2020). Exercise training adaptations in metabolic syndrome individuals on chronic statin treatment. The Journal of Clinical Endocrinology & Metabolism, 105(4), e1695-e1704..

[7] M. Matsui, K. Kosaki, K. Myoenzono, T. Yoshikawa, J. Park, M. Kuro-o, and S. Maeda. (2022). Effect of aerobic exercise training on circulating fibroblast growth factor-21 response to glucose challenge in overweight and obese men: a pilot study. *Experimental and Clinical Endocrinology & Diabetes*, *130*(11):pp.723-729.

[8] K. Kumboyono, I.N. Chomsy, D.H. Firdaus, M. Setiawan, and T.A. Wihastuti. (2022). Protective cardiovascular benefits of exercise training as measured by circulating endothelial cells and high-density lipoprotein in adults. *Journal of Taibah University Medical Sciences*, *17*(4):pp.701-706.

[9] A.R. Streb, P.G.S. Braga, R.F. de Melo, L.J. Botelho, R.C. Maranhão, and G.F. Del Duca. (2022). Effects of combined physical exercise on plasma lipid variables, paraoxonase 1 activity, and inflammation parameters in adults with obesity: a randomized clinical trial. *Journal of Endocrinological Investigation*, *45*(10):pp.1991-1997.

[10] B. Ghorbanian, and Y. Saberi. (2022). The Effect of a Ten-Week Aerobic Training on Atherogenic Indices, Lipid Profile, and Body Composition in

Postmenopausal Women with Type II Diabetes. *Journal of Obstetrics, Gynecology and Cancer Research*, *2*(2).

[11] V.N. Lavorato, D.C. de Miranda, F.R. Drummond, L.M.T. Rezende, M. doCarmo Gouveia Pelúzio, M.E. Silva, and A.J. Natali. (2022). Combined action of açai and aerobic exercise training on the development of NAFLD induced by a high-fat diet: a preliminary exploration. *Sport Sciences for Health*:pp.1-7.

[12] A.P.G. Bochi, G.D.S. Ferreira, V. Del Bianco, P.R. Pinto, L.G. Rodrigues, M.D.S. Trevisani, L.N.S. Furukawa, K.C.S. Bispo, A.A. da Silva, A.P.P. Velosa, and E.R. Nakandakare. (2022). Aerobic Exercise Training Reduces Atherogenesis Induced by Low-Sodium Diet in LDL Receptor Knockout Mice. *Antioxidants*, *11*(10):p.2023.

[13] A. Ghanbari-Niaki, F. Hosseini, D.R. Broom, B. Tejenjari, and S. Rahmati-Ahmadabad. (2022). Combined Effects of High-Intensity Aerobic Exercise Training and Ziziphus jujuba Extract on Tissue Nesfatin-1 in Rats. *Frontiers in Endocrinology*, *13*.

[14] G. Wood, E. Taylor, V. Ng, A. Murrell, A. Patil, T. van der Touw and N. Smart. (2022). Determining the effect size of aerobic exercise training on the standard lipid profile in sedentary adults with three or more metabolic syndrome factors: a systematic review and meta-analysis of randomised controlled trials. British Journal of Sports Medicine, 56(18), 1032-1041.

[15] B. Wu, J. Ding, A. Chen, Y. Song, C. Xu, F. Tian, and J. Zhao. (2022). Aerobic exercise improves adipogenesis in diet-induced obese mice via the lncSRA/p38/JNK/PPARγ pathway. *Nutrition Research*, *105*:pp.20-32.

[16] C. Serviente, M. Chalvin, and S. Witkowski. (2022). The influence of menopause and cardiorespiratory fitness on lipoprotein particles in midlife women. *Applied Physiology, Nutrition, and Metabolism*, *47*(4):pp.447-457.