

Modulation Of Lactate Dehydrogenase Activity Induced By Fartlek Training Among Novice Student Athlete

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Abstract

Lactate dehydrogenase (LDH) is an enzyme found in nearly all body tissues particularly in muscles, liver, kidneys, and red blood cells that converts sugar into cellular energy. The purpose of this study was to examine the effects of Fartlek training on lactate dehydrogenase (LDH) levels in Novice Student Athletes. An experimental research study was conducted among twenty (20) novice student-athletes registered at Superior University, Lahore, Pakistan. Thus, they were randomly placed into two groups, i.e. Control Group (CG) and Experimental Group (EG). Each group comprised ten (10) subjects. A self-made eight (08) weeks Fartlek training protocol was applied to EG. LDH level was measured by taking the blood samples prior to and after the training period. Analysis of the data was performed using the statistical package for social sciences (SPSS, version-26), and thus suitable statistical tools were applied according to parametric data. Based on data analysis and findings, the researcher concluded that

eight (08) weeks of Fartlek training has a significant effect on the LDH level of EG. The study also concluded that there is better physiological adaptation to exercise and management of muscle stress in the EG than in the CG.

Author Details

Keywords: Fartlek Training, Lactate Dehydrogenase (LDH), Novice Athletes.

Received on 15 Dec 2025

Accepted on 20 Jan 2026

Published on 30 Jan 2026

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Introduction

Lactate dehydrogenase (LDH) is a pivotal enzyme in skeletal muscle metabolism, catalyzing the reversible conversion of pyruvate to lactate while regenerating nicotinamide adenine dinucleotide (NAD⁺), a process essential for sustaining glycolytic adenosine triphosphate (ATP) production during conditions of elevated metabolic demand. When oxygen availability is limited or exercise intensity exceeds oxidative capacity, LDH activity facilitates continued energy generation by maintaining glycolytic flux. As a result, LDH serves as a sensitive biochemical indicator of metabolic stress and anaerobic contribution during physical exercise (1,2). Traditionally, lactate accumulation and increased LDH activity were viewed as markers of muscular fatigue and metabolic inefficiency. However, contemporary research has fundamentally shifted this perspective, recognizing lactate as an important metabolic intermediate rather than a waste product. Lactate functions as a transportable energy substrate and a precursor for gluconeogenesis, supporting the redistribution of energy between tissues through the lactate shuttle mechanism. This paradigm has established LDH as a key regulator of metabolic flexibility, linking anaerobic glycolysis with aerobic energy production during both rest and exercise (3,4).

Exercise intensity and training modality are major determinants of LDH activity. Continuous moderate-intensity aerobic exercise predominantly relies on oxidative metabolism and generally induces minimal acute changes in LDH. In contrast, variable-intensity and high-intensity exercise impose greater glycolytic stress, leading to elevated lactate production and transient increases in LDH activity. Repeated exposure to such metabolic stressors promotes enzymatic and mitochondrial adaptations that enhance lactate clearance, oxidative capacity, and overall metabolic efficiency (5,6). Interval-based and mixed-intensity training modalities significantly influence lactate metabolism and LDH activity. Acute bouts of high-intensity or fluctuating-intensity exercise are associated with elevated circulating LDH levels, reflecting increased glycolytic flux and muscle membrane disruption. Over time, chronic training adaptations include increased expression of oxidative LDH isoforms, improved mitochondrial density, and enhanced lactate transport capacity, collectively contributing to improved metabolic control during exercise (7,8).

Untrained individuals typically show lower aerobic efficiency, reduced lactate clearance, and heightened reliance on anaerobic glycolysis, resulting in greater elevations in blood lactate and LDH following exercise (9,10). LDH plays a central role in exercise metabolism by linking glycolytic and oxidative pathways and facilitating efficient energy distribution during variable exercise intensities. (13,14,15,16). Fartlek training, characterized by continuous exercise interspersed with irregular changes in speed and intensity, provides a unique physiological stimulus that simultaneously challenges aerobic and anaerobic energy systems. For novice athletes, this training approach may accelerate improvements in lactate threshold, metabolic flexibility, and LDH regulation. Beyond performance enhancement, optimized lactate metabolism may support improved recovery, reduced exercise-induced fatigue, and greater training tolerance, making fartlek training a practical and adaptable method for beginner athletic populations (11,12).

MATERIALS AND METHODS

The study was conducted at the campus of Superior University, with novice student-athletes serving as the study population. An experimental research study was conducted among twenty (20) novice student-athletes registered at Superior University, Lahore, Pakistan. Thus, they were randomly placed into two groups, i.e. Control Group (CG) and EG(EG). Each group consisted of (ten) subjects aged 18–25 years who met the predefined inclusion and exclusion criteria. The EG underwent a progressive Fartlek training program consisting of dynamic warm-up exercises,

variable-intensity running sessions, and structured cool-down activities, while the control group maintained their routine physical activity. Lactate dehydrogenase (LDH) activity was measured through laboratory-based plasma analysis before the initiation of the training program and after the completion of the eight-week intervention. LDH levels served as the primary dependent variable to assess metabolic adaptations to training. Analysis of the data was performed using the statistical package for social sciences (SPSS, version-26), and thus suitable statistical tools were applied according to parametric data.

RESULTS:

Table 1: Descriptive Analysis of Age, BMI and LDH of EG (N = 10)

The EG consisted of 10 participants with a mean age of 21.40 ± 1.89 years (range 19-24), height of 173.00 ± 7.88 cm (range 160-185), pre-test weight of 76.30 ± 7.10 kg decreasing to post-test 74.70 ± 6.53 kg, and BMI from 25.47 ± 1.38 kg/m² to 24.95 ± 1.29 kg/m². LDH levels increased from pre-test 146.90 ± 17.08 U/L (range 131-190) to post-test 177.10 ± 17.66 U/L (range 151-205).

	N	Range	Minimum	Maximum	Mean	Std. Deviation	Variance
Age (yrs)	10	5	19	24	21.40	1.897	3.600
Height (cm)	10	25	160	185	173.00	7.888	62.222
Pre-Weight (kg)	10	21	67	88	76.30	7.103	50.456
Pre-BMI (kg/m ²)	10	4.39	23.37	27.76	25.4770	1.38492	1.918
Post-Weight (kg)	10	19	66	85	74.70	6.533	42.678
Post-BMI (kg/m ²)	10	4.3	22.8	27.1	24.950	1.2921	1.669
Pre-Test LDH (U/L)	10	59	131	190	146.90	17.084	291.878
Post-Test LDH (U/L)	10	54	151	205	177.10	17.666	312.100
study group	10	0	2	2	2.00	.000	.000
Valid N (listwise)	10						

a. study group = experimental group

Table 2 Descriptive Analysis of Age, BMI and LDH of CG (N = 10)

The CG (N=10) had a mean age of 21.70 ± 1.49 years (range 19-24), height of 173.30 ± 8.63 cm (range 163-185), pre-weight of 74.40 ± 7.55 kg (BMI 24.82 ± 2.35 kg/m²) decreasing slightly to post-weight 73.40 ± 7.59 kg (BMI 24.42 ± 1.80 kg/m²). LDH levels showed minimal change from pre-test 148.70 ± 5.91 U/L (range 139-158) to post-test 148.60 ± 8.45 U/L (range 136-159).

		N	Range	Minimum	Maximum	Mean	Std. Deviation	Variance
Age (yrs)		10	5	19	24	21.70	1.494	2.233
Height (cm)		10	22	163	185	173.30	8.629	74.456
Pre-Weight (kg)		10	25	63	88	74.40	7.545	56.933
Pre-BMI (kg/m ²)		10	7.40	20.45	27.85	24.819	2.34831	5.515
						0		
Post-Weight (kg)		10	23	63	86	73.40	7.589	57.600
Post-BMI (kg/m ²)		10	6.1	21.0	27.1	24.420	1.7999	3.240
Pre-Test LDH (U/L)		10	19	139	158	148.70	5.908	34.900
Post-Test LDH (U/L)		10	23	136	159	148.60	8.449	71.378
study group		10	0	1	1	1.00	.000	.000
Valid N (listwise)		10						

a. study group = Control group

Descriptive Statistics of the Experimental Group

Table 3: Table one normality test EG

	Kolmogorov-Smirnov ^b			Shapiro-Wilk		
	Statistics			Statistics		
	c	df	Sig.	c	df	Sig.
Height (cm)	.137	10	.200*	.973	10	.921
Pre-Weight (kg)	.179	10	.200*	.940	10	.550
Pre-BMI (kg/m ²)	.209	10	.200*	.930	10	.449
Post-Weight (kg)	.164	10	.200*	.937	10	.515
Post-BMI (kg/m ²)	.177	10	.200*	.943	10	.590
Pre-Test LDH (U/L)	.239	10	.111	.787	10	.670
Post-Test LDH (U/L)	.116	10	.200*	.967	10	.861

*. This is a lower bound of the true significance.

a. study group = experimental group

b. Lilliefors Significance Correction

Table 4: Table one normality test CG and EG

Normality of the data was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests (Table 4). The results indicated that all variables for both the CG and EG were normally distributed ($p > .05$). This confirmed the suitability of parametric statistical tests for subsequent analyses, including group comparisons and pre-post intervention assessments.

	Kolmogorov-Smirnov ^b			Shapiro-Wilk		
	Statisti			Statisti		
	c	df	Sig.	c	df	Sig.
Height (cm)	.232	10	.136	.875	10	.114
Pre-Weight (kg)	.121	10	.200*	.978	10	.950
Pre-BMI (kg/m ²)	.141	10	.200*	.945	10	.614
Post-Weight (kg)	.134	10	.200*	.958	10	.758
Post-BMI (kg/m ²)	.145	10	.200*	.973	10	.914
Pre-Test LDH (U/L)	.134	10	.200*	.981	10	.971
Post-Test LDH (U/L)	.199	10	.200*	.886	10	.152

*. This is a lower bound of the true significance.

a. study group = Control group

b. Lilliefors Significance Correction

Table 5: Paired Samples Statistics Body Mass Index (BMI) and Lactate Dehydrogenase (LDH) of CG and EG

The CG showed minimal changes in BMI (slight decrease to 24.42 kg/m²) and LDH levels (148.70 to 148.60 U/L), indicating no significant physiological impact. In contrast, the EG exhibited a notable BMI reduction (25.48 to 24.95 kg/m²) and LDH increase (146.90 to 177.10 U/L), reflecting greater metabolic and muscular activity due to the intervention.

		Mean	N	Std. Deviation	Std. Error Mean
Control group	Pre-BMI (kg/m ²)	24.8190	10	2.34831	.74260
	Post-BMI (kg/m ²)	24.420	10	1.7999	.5692
Experimental group	Pre-BMI (kg/m ²)	25.4770	10	1.38492	.43795
	Post-BMI (kg/m ²)	24.950	10	1.2921	.4086
Control group	Pre-Test LDH (U/L)	148.70	10	5.908	1.868
	Post-Test LDH (U/L)	148.60	10	8.449	2.672
Experimental group	Pre-Test LDH (U/L)	146.90	10	17.084	5.403
	Post-Test LDH (U/L)	177.10	10	17.666	5.587

The CG showed minimal changes in BMI (slight decrease to 24.42 kg/m²) and LDH levels (148.70 to 148.60 U/L), indicating no significant physiological impact. In

Table 8: Effect Sizes (Cohen d and Hedges g) of Pre -Post changes in BMI and LDH of Experimental and CGs

The EG showed very large effect sizes for BMI reduction (Cohen d=2.571, Hedges g=2.463) and LDH increase (Cohen d=2.216, Hedges g=2.122), indicating significant changes due to fartlek training. In contrast, the CG exhibited negligible, non-significant effects for both BMI (Cohen d=0.288, Hedges g=0.276) and LDH (Cohen d=-0.013, Hedges g=-0.012).

					Point	95% Confidence Interval		
					Standardizer ^a	Estimate	Lower	Upper
Experimental group & Control Group								
Experimental group	Pair 1	Pre-BMI (kg/m ²)	Cohen's d	.20494	2.571	1.236	3.879	
		& Post-BMI (kg/m ²)	Hedges' correction	.21401	2.463	1.184	3.715	
	Pair 2	Pre-BMI (kg/m ²)	Cohen's d	1.38462	.288	-.353	.914	
		& Post-BMI (kg/m ²)	Hedges' correction	1.44585	.276	-.338	.875	
Control Group								
Experimental group	Pair 1	Pre-Test LDH (U/L)	Cohen's d	13.628	2.216	-3.382	-1.019	
		& Post-Test LDH (U/L)	Hedges' correction	4.231	2.122	-3.239	-.976	
	Pair 2	Pre-Test LDH (U/L)	Cohen's d	7.752	-.013	-.607	.632	
		& Post-Test LDH (U/L)	Hedges' correction	8.095	-.012	-.582	-.606	
Control Group								

DISCUSSION:

The present study aimed to investigate the effects of an 8-week Fartlek training program on body composition, as measured by body mass index (BMI), and lactate dehydrogenase (LDH) activity among novice male undergraduate student athletes. The findings underscore the physiological and biochemical adaptations elicited by variable-intensity endurance training in untrained individuals, demonstrating significant improvements in the EG while the CG exhibited negligible changes. These results align with existing literature on high-intensity intermittent training (HIIT), including Fartlek, which has been shown to induce metabolic and anthropometric responses in beginner athletes' Similar findings were observed by Abustan et al (17,18).

A key outcome was the significant reduction in BMI observed in the EG($t = 8.132$, $p < 0.001$), contrasting with the non-significant change in the CG ($t = 0.911$, $p = 0.386$) (19). This suggests that structured variable-intensity aerobic training can enhance body composition in relatively untrained individuals over a short 8-week period. The intervention likely promotes energy expenditure, fat oxidation, and cardiovascular efficiency, as supported by prior research on Fartlek training. Similar findings were observed by DUSU, et al. (20,21). Furthermore, the strong positive correlation between pre- and post-test BMI in the EG($r = 0.991$, $p = 0.001$) indicates consistent responses to the training, reflecting its systematic impact on body composition. In comparison, the moderate correlation in the CG ($r = 0.809$, $p = 0.005$) may be attributed to natural variability in BMI without structured intervention (Similar

findings were observed by Bellicha et al., (22). These anthropometric changes highlight Fartlek's potential as an accessible modality for novices, facilitating macroscopic shifts in body composition through increased caloric expenditure and metabolic demands (23).

In addition to anthropometric alterations, LDH levels—a biomarker of anaerobic metabolism and cellular stress—demonstrated substantial changes. The EG exhibited a significant elevation in LDH activity post-intervention ($t = -7.007$, $p = 0.001$), whereas the CG showed no significant alteration ($t = 0.041$, $p = 0.968$) (24). This indicates that Fartlek training imposes considerable metabolic stress, stimulating anaerobic glycolysis and enzymatic adaptations to support ATP production during high-intensity bouts. The moderate positive correlation between pre- and post-LDH levels in the EG ($r = 0.693$, $p = 0.026$) further evidences the consistency of these biochemical responses, suggesting adaptive enzymatic shifts in response to repeated anaerobic challenges. Conversely, the weak and insignificant correlation in the CG ($r = 0.463$, $p = 0.178$) underscores the absence of such adaptations without targeted training. Similar findings were observed by Asgari & Kushkestani. LDH elevation reflects the body's compensatory mechanisms, such as enhanced pyruvate-to-lactate conversion under hypoxic conditions, to maintain energy homeostasis during strenuous activity. Similar findings were observed by Kane et al. Similar findings have been reported in HIIT and endurance studies, where LDH activity increases in untrained individuals exposed to novel stimuli. Similar findings were observed by Lundahl et al. (25,26,27).

Fartlek training's flexibility distinguishes it from traditional interval methods, allowing participants to modulate intensity through continuous running interspersed with speed variations. This approach fosters both aerobic and anaerobic adaptations while potentially reducing perceived exertion, thereby improving compliance among beginners. Physiologically, it enhances mitochondrial efficiency, activates glycolytic enzymes, and optimizes lactate clearance, corroborating the observed LDH changes. Similar findings were observed by Abustan et al. Integrating BMI and LDH data provides a comprehensive view of Fartlek-induced adaptations: BMI reflects macroscopic body composition improvements, while LDH signifies cellular-level metabolic shifts. Similar findings were observed by Szlachta et al. This dual impact positions Fartlek as an evidence-based training method for enhancing metabolic capacity and body composition in novice athletes. Similar findings were observed by Stuart (28,29,30).

CONCLUSION

In conclusion, the study demonstrates that 8-week Fartlek training positively influences BMI reduction and LDH elevation in novice student athletes. The study also concluded that there is better physiological adaptation to exercise and management of muscle stress in the EG than in the CG.

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