

## Effect of Training on the Lactate Dehydrogenase (LDH) levels of Athletes

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**Abstract:** The influence of training on serum lactate dehydrogenase (LDH) levels of athletes was investigated in this study. The study involved a 6 week-long high intensity exercise programme to establish the effect of training on athletes. These studies preceded the clinical trials of some nutritional supplements, on two (200) athletes and exercise enthusiasts randomly assigned to a test group and a control group. There were significant increase in LDH values after training for both groups:  $307.0 \pm 7.80$  IU/L (Pre-training) and  $493.40 \pm 12.60$  IU/L (Post-training), for the test group and  $353.70 \pm 13.10$  IU/L (Pre-training) and  $521.10 \pm 8.30$  (Post-training), for the control group. These findings suggest that training may cause moderate to extensive microscopic muscles tissue damage as a result of lipid peroxidation of muscle cell membranes due to increased production of free radicals. This causes the leakage of intracellular LDH into the capillaries of skeletal muscles and then to the general circulation.

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### 1. Introduction

Lactate dehydrogenase (LDH) is an oxidoreductase enzyme that catalyses the interconversion of pyruvate and lactate. Cells release LDH into the bloodstream after tissue damage or red blood cell hemolysis. At high concentrations of lactate, the enzyme exhibits feedback inhibition, and the rate of conversion of pyruvate to lactate is decreased. It also catalyzes the dehydrogenation of 2-hydroxybutyrate, but it is a much poorer substrate than lactate. There is little to no activity with beta-hydroxybutyrate (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011).

The lactate dehydrogenase (LDH) test measures the amount of LDH in the blood. A blood sample is needed. Subjects are instructed to stop all medications including vitamins. Drugs that can increase LDH measurements include anesthetics, aspirin, clofibrate, fluorides, mithramycin, narcotics, and procainamide (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011). LDH is most often measured to check for tissue damage. The protein LDH is in many body tissues, especially the heart, liver, kidney, muscles, brain, blood cells, and lungs (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011). Other conditions under which the test may be done include anemia of vitamin B-12 deficiency, leukemia or lymphoma, megaloblastic anaemia and pernicious anaemia (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011).

Higher-than-normal levels may indicate blood flow deficiency (ischemia), cerebrovascular accident (such as a stroke), heart attack, hemolytic anemia, infectious mononucleosis, liver disease (for example, hepatitis), low blood pressure, muscle injury, muscular dystrophy, new abnormal tissue formation (usually cancer), pancreatitis and tissue death (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011).

LDH assay is a screening test in sports medicine and serves as an indicator for acute or chronic muscle tissue damage. Tissue breakdown releases LDH, and therefore LDH can be measured as a surrogate for tissue breakdown, e.g. hemolysis. Other disorders indicated by elevated LDH include cancer, meningitis, encephalitis, acute pancreatitis, and HIV (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011).

A typical range is 105 - 333 IU/L (international units per liter). Normal value ranges may vary slightly among different laboratories. Some laboratories use different measurements or test different samples (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011). If the LDH level is raised, an LDH isoenzymes test may be required to establish the definitive diagnosis (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011).

However, in this study comprehensive physical examinations and tests were carried out on each participant to exclude the existence of heart disease, liver disease, autoimmune diseases and muscular

dystrophy. In any case, athletes are among the fittest persons in any community (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011). In this study, we report the effect of training on lactate dehydrogenase (LDH) of athletes.

## 2. Material And Methods

### 2.1. Population and sample

One hundred (100) student-athletes and 100 non-athletes of the University of Port Harcourt, Nigeria were randomly placed in each of two study groups. Samples of blood were collected from the antecubital veins of each participant. The training programmes of the participants during a 42-day period were similar and properly designed. All participants consumed the same meals as provided in the University of Port Harcourt athletes training camp. Food records were monitored during the study. The participants were all treated for malaria prior to the commencement of the study. The use of prescription drugs, vitamins, mineral supplements and other sports nutritional supplements was forbidden during the study. Written informed consent was obtained from participants after detailed explanations of the risks involved in the study. Detailed physical examination was carried out on all the participants to exclude any heart or musculoskeletal disease.

### 2.2. Endurance clinical trial

The endurance of each participant was measured by a treadmill run with increasing workload (stepwise every minute), until muscle fatigue (time-to-fatigue). The time-to-fatigue which was adopted as the fatigue threshold was measured pre-study. At day 42, each participant was made to stop the exercise activity at his fatigue threshold. LDH levels measurements were assayed the same interval immediately after exercise and also 50 minutes after exercise using lactate dehydrogenase test kit (manufactured by Randox Laboratories Ltd., 55 Diamond Road, Crumlin, Co. Antrim, United Kingdom).

### 2.3. Data Analysis

The data generated were analyzed by multivariate statistical methods. For statistical analysis SPSS software (version 20.0, Chicago, USA) was used, the paired *t* test and independent samples-test were used to compare values of the experimental treatment and control group. A comparison was considered statistically significant if the *P* value was < 0.05.

## 3. Results

The paired *t*-test results for enzyme assay showed that there were significant differences in pre-training and post-training lactate dehydrogenase

(IU/L) levels for the experimental group ( $p=0.001$ ) and for the control group ( $p=0.022$ ).

### 3.1. Pre-Training and Post-Training lactate dehydrogenase (IU/L) levels in athletes (Experimental Group)

Using qualitative correlation analysis, there was a rise in the LDH level after training among the female athletes (Figure 1a). However, in a sample size of 50, about three athletes show no significant rise in the LDH level after training. This could be attributed possibly to not being involved in strenuous training. There was also an increase in LDH levels among the male athletes after training although about 30% of the sample size does not show any significant rise (Figure 1b). The same reason could be attributed as in the female athletes after training.

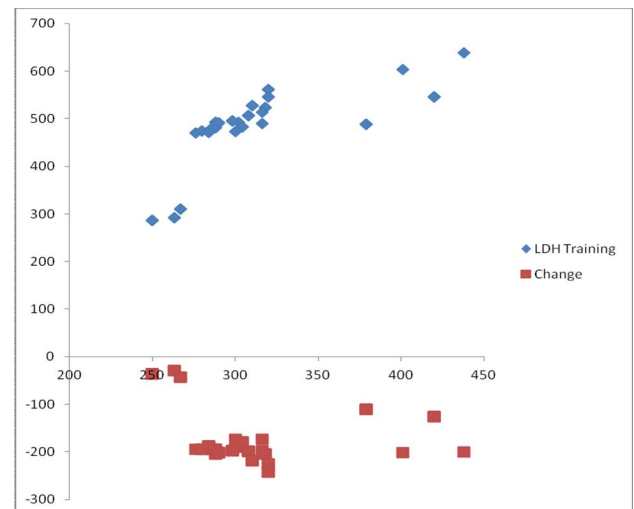


Figure 1a: Rise in LDH levels in female athletes after training

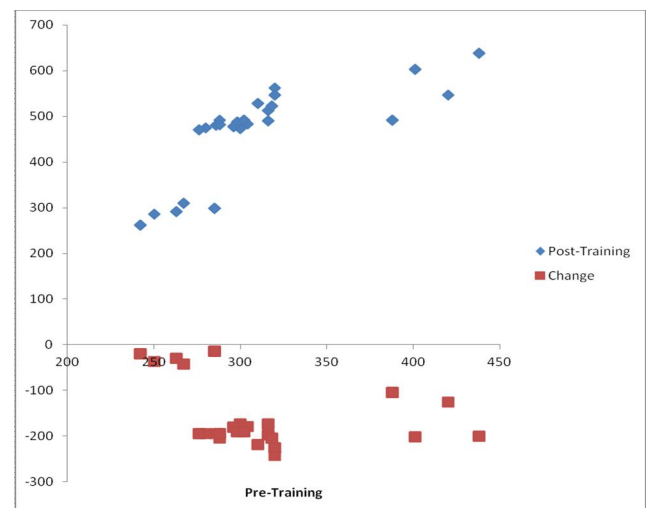
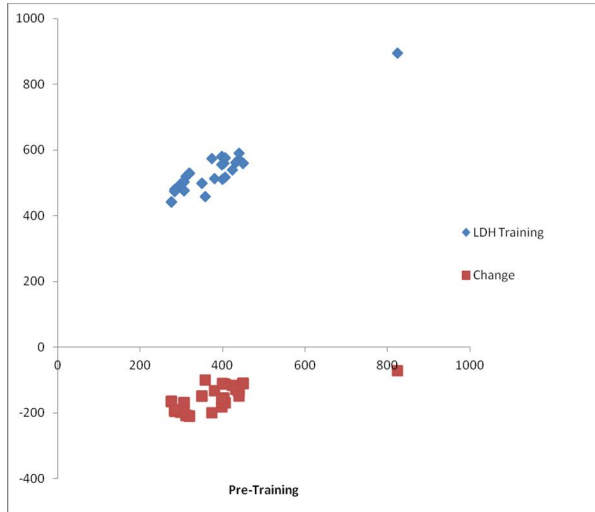


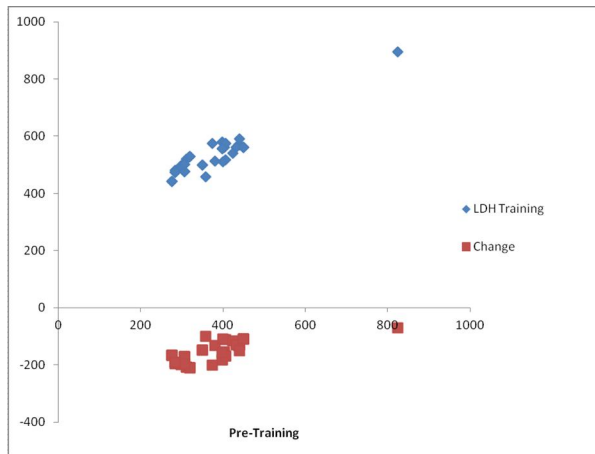
Figure 1b: Rise in LDH levels in male athletes after training

**3.2. Pre-Training and Post-Training lactate dehydrogenase (IU/L) levels in athletes (Control) using qualitative correlation analysis**

There was a rise in LDH level after training in female athletes (control) which indicates that the control group shows significant rise in the LDH levels after training (Figure 2a). The response of LDH to training among male athletes was similar to that of the female athletes; showing significant rise in LDH values after training (Figure 2b).



**Figure 2a: Rise in LDH levels after training in female athletes (control)**

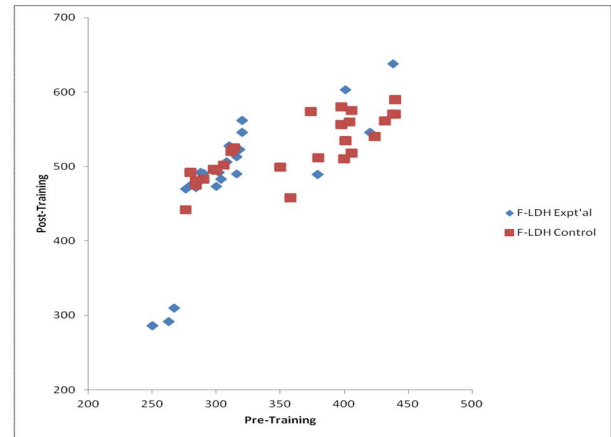


**Figure 2b: Rise in LDH levels in male athletes (control) after training.**

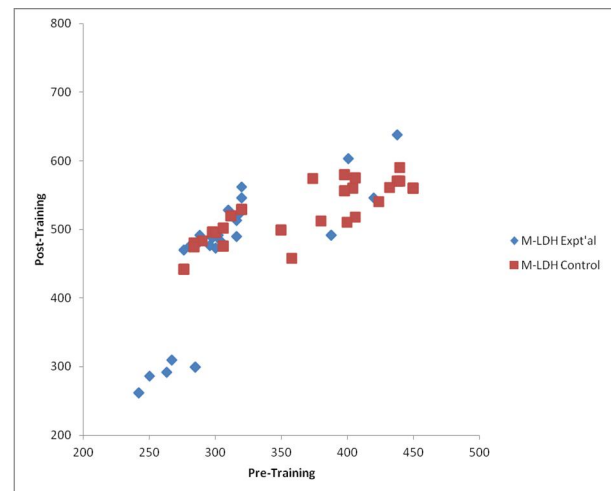
**3.3. Comparison of the rise in LDH levels after training between the experimental and control athletes using qualitative correlation analysis**

A comparison carried out to determine the rise in LDH levels after training between the

experimental and the control athletes showed that there was no significant difference in the rise in LDH levels between the females in both the experimental and control groups although, some athletes in the experimental group had higher LDH levels compared to their control counterparts (Figure 3a). However, the result was similar when the rise in LDH levels after training between the experimental and control groups male athletes were compared (Figure 3b).



**Figure 3a: Comparison of the rise in LDH levels after training between the experimental and control female athletes**

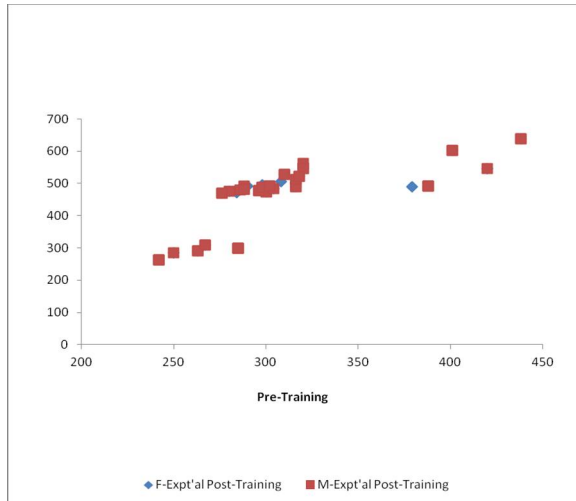


**Figure 3b: Comparison of the rise in LDH levels after training between the experimental and control male athletes**

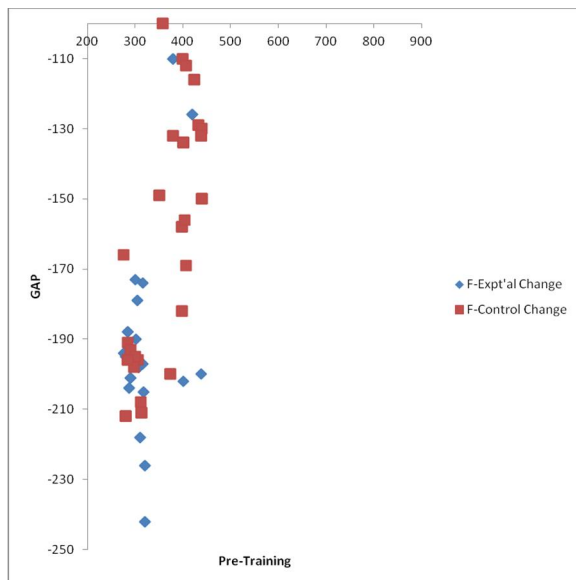
**3.4. Comparison of the rise in LDH levels after training male and female athletes of the experimental and control group.**

More so, a comparison of the rise in LDH values between the female and male athletes indicated no gender bias (Figure 4a). The control group showed a lower LDH levels after training than

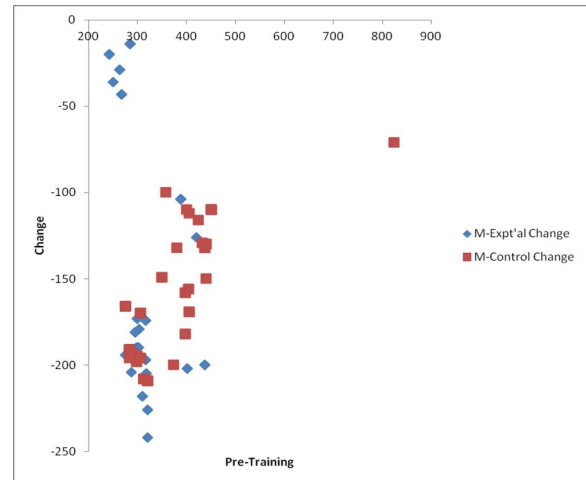
the experimental group among the female athletes (Figure 4b). The male athletes in the experimental group show higher LDH levels than their control counterparts (Figure 4c). However, a few subjects in the experimental group show very low increase in LDH level after training.



**Figure 4a: Comparison of the rise in LDH levels after training between the male and female athletes of the experimental group.**



**Figure 4b: Comparison of the change in LDH levels after training between females in the experimental and control groups.**



**Figure 4c: Comparison of the change in LDH levels after training between male experimental and control groups.**

**4. Discussion**

The study showed significant increase in serum lactate dehydrogenase levels of both subjects and controls after exhaustive exercise. Serum levels of skeletal muscle enzymes are markers of the functional status of muscle tissue and vary widely in both pathological and physiological conditions (Brancaccio *et al.*, 2007). An increase in these enzymes may represent an index of cellular necrosis and tissue damage following acute and chronic muscle injuries (Brancaccio *et al.*, 2007).

Changes in serum levels of muscle enzymes and isoenzymes are also found in normal subjects and athletes after strenuous exercise and the amount of enzyme from muscle tissue to blood can be influenced by physical exercise (Brancaccio *et al.*, 2007). LDH occurs in many tissues of the body, especially in the heart, muscle, liver, red blood cells, lungs, kidneys, stomach, pancreas and brain. LDH levels in blood can be elevated when cells in these tissues are damaged. This occurs in the following conditions: stroke, heart attacks, haemolytic anaemia, hepatitis, low blood pressure, pancreatitis and leukaemia. Furthermore, certain medications cause raised LDH levels in blood. These medications include aspirin, mithramycin, clofibrate, and fluorides (Brancaccio *et al.*, 2007).

As a result of the numerous possible sources of the elevation of LDH, it was important to narrow down the source(s). In this study, it was not feasible to carry out LDH isoenzyme tests to determine the primary source of the additional LDH. To obviate this limitation, an appropriate and detailed physical examination was carried out on each participant in order to rule out any health problems. From the results of the physical examination, participants with

the suggestion of heart disease, liver disease, autoimmune diseases and muscular dystrophy were excluded. Emphasis was placed on the cardiovascular system, the musculoskeletal system and vital intra-abdominal organs. This was important for the non-athletes, since the athletes were all in confirmed reasonably good health.

In addition, all the participants were forbidden from taking any medication or supplement throughout the duration of the study. These measures narrowed the option of possible sources of LDH to the skeletal muscles. This inference would be reinforced by the assay of another skeletal muscle enzyme, creatine kinase. In any case, athletes are among the physically fittest persons in any community (Gregg and Prchal, 2008; Gallagher, 2011; Selwood and Jaffe, 2011).

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