An-Najah National University
Faculty of Graduate Studies

The Effect of Altitude on Blood Biochemistry of Soccer Players in West Bank

By

Faiha Othman Albahsh

Supervision of

Dr. Suleiman Al-Khalil **Dr.Abdel Naser Qadumi**

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This thesis was defended successfully on 24-Jun-2001 and Approved by

Committee members		Signature
Dr. Suleiman Al-Khalil	(Supervisor)	
Dr. Abedl Naser Qadumi	(Co-Supervisor)	
Dr. Reiad Amain	(External Examiner)	
Dr. Jumana Saleh	(Internal Examiner)	

Dedication

To My Beloved Parents

And Husband

Acknowledgments

It was by the support guidance of many individuals specially my great father and mother, who provided me with great opportunities, I am so grateful to them, and what ever I do will not ever thank them. Another thanks will be to my supervisors Dr. Suleiman Al-Khalil and Dr.Abdel Naser Al qadomi for their guidance, continuous support, encouragement and help through out my research work.

Last but not least,I would express all my deep thanks to my husband Tareq, family, Mr. Zaher,soccer players in each team in Nablus,Qalqelia, and Jerico for there cooperation and for the facilities which enable me to continue my research.

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ABSTRACT

The current study aimed at evaluating the effect of altitude variation on blood biochemistry of soccer players. To achieve this aim, the study was conducted on a sample of 40 players belonging to three different teams in different geographical areas. These teams were; AL-AHLI (Qalqelia) at sea level , AL-ITHAD (Nablus) above sea level, and AL-HILAL (Jerico) below sea level .

Measurements of Heamoglobin, Glucose, Total Iron Binding capacity, Cholesterol, Lactate dehydrogenase , Triglyceride , Hematocrete and Insulin prior and post exercise were conducted.

Result for the diffirences on the pre exercise blood biochemistry measures according to area variable show that computed (F) values on the pre exercise of (TIBC,and Cholesterol) are respectively (0.47 and 1.54),such two values are lower than critical (F) value (3.26) this mean that there are no significant differences at(α =0.05)on these two variables due to the area variable.While computed (F) values for (Hb,Glu. LDH, Trig. Insulin and Hematocrate)are respectively (6.66, 9.11,7.52, 3.68, 14.17, and 4.15)all of these values are greater than critical (F) value (3.20),this means that there are significant differences at (α =0.05) on these variables due to the area variable.

While the result for post exercise showed that computed (F) value for (Glu, TIBC, LDH, and Hematocrate)variables are respectively (1.22, 0.83, 0.97,and 2.19) all of these values are lower than critical (F) value (3.26),this means that there are no significant differences at (α =0.05) on these variables due to the area variable.While computed (F) values for (Hb,Cholesterol, Trig. and Insulin)are respectively (4.12, 6.004, 3.81, and 3.98) all of these values are greater than critical (F) value.This means that there are significant differences at (α =0.005) on these variables due to area variable.

Chapter one

Introduction and Theoretical Background

Chapter one

Introduction:

In the last 20 years there has been worldwide acceleration especially in the United States, in exercise and sports participation by people of all ages and both sexes.

However, there are some problems accompanying this generally positive development. Some physicians saw that, regular exercise has many health benefits. But what wasn't widely appreciated is that exercise under certain conditions can be harmful and even deadly, especially playing at altitude without adaptation or adjust.

1-1 Physical Fitness:

When one tries to explain how some change in body function comes, as a result of exercise, it can be said that one wants to know as mechanism and the response, that is one wants to know as much as possible about the physical and chemical responses, responsible for the change in function.

Several changes occur to the physiological system of the body which help it adapt to the decreased or increased atmospheric pressure at altitude. Some of these changes take place immediately, other requires several weeks.

1.2 Variables that may change:

1.2.1 Hemoglobin

When a percentage of person travel from see level to high a latitude, the percentage of the hemoglobin changes.

Also exercises have physiological response even during or after exercise. During exercise the working muscles may use oxygen at a rate ten or twenty times greater than at rest in order to supply the extra oxygen required, not only must cardiac out put increase, but the circulation of blood through the working muscles must be increased.

As I mentioned before, hemoglobin concentration changes even in response to altitude and to exercise. Many researches talk about the changes occurring to hemoglobin.

Studies have shown that there was a significant increase in hemoglobin at moderate altitude. There was no doubt that training at moderate altitude, improves exercise performance at moderate altitude, but results concerning sea level performance after altitude training are controversial (Friedmann, Wasseran, 1998).

Whereas some studies report no effects especially in well-trained athletes training in hypoxia condition. (Klausen, Telford, 1996).

However hypoxia is an important stimulus for erythropoietin, within (15) minutes to (2) hours after onset of hypoxia erythropoietin levels in peripheral blood begin to rise and remain significantly elevated for about one week during exposure to moderate altitude (Berglund, Klausen, 1996). Some authors report that these reactions are more pronounced in athletes exercising in hypoxia compared to normal oxygen level and conclude that the erythropoietin stimulus of hypoxia might be enhanced by training (Mairbaur, 1986).

1.2.2 Glucose:

Glucose is an important fuel for exercising skeletal muscle. Physical endurance is significantly improved by carbohydrate feeding in the days before exercise and by glucose infusion during exercise (Vissing, Henrik, 1989).

It is generally assumed that mobilization of glucose during exercise is accurately coupled by feedback mechanisms to the metabolic needs of working muscles. Maintenance of resting glucose levels in plasma during exercise, in spite of the great increase in glucose from rest to exercise, has been argued as being precise feedback control of hepatic glucose production during exercise (Jenking, 1987; Finegood, 1989). Many studies were done on fed rats and humans and the results show that, the increase in hepatic glucose production can exceed the increase in hepatic glucose utilization, resulting in an increased plasma glucose concentration (Sonne, 1985; Vissing, 1998).

It thus seems as if hepatic glycogenolysis is directly related to hepatic glycogen content during exercise. A similar correlation between the size of glycogen stores and glycogenolysis during exercise was performed by Colling in 1999. Other studies showed that during prolonged exercise, the glycogen concentration in the working muscles decreases and the concentration in plasma of glucagon and insulin increase and decreases, respectively (Richter, 1981).

These findings indicated that during exercise, adrenomedullary hormones enhance muscular glycogenolysis and glucagon secretion but inhibit insulin secretion (Finegood, 1989; Sonne, 1985). on the other hand other studies show the effect of altitude on glucose concentration, during exercise after acclimatization to high altitude, the net degradation rate of muscle glycogen decreases compared with that determined for the same exercise power output before acclimatization has been interpreted to reflect an enhanced ability to utilize lipid energy sources, (Brooks, 1999).

In human, Ahlbor et al, found that blood glucose could account for as much as (40%) of total oxidative substrate by the legs during prolonged exercise of low intensity.

During rest and exercise the liver releases glucose that is produced by either glycogenolysis or gluconeogenesis. The intensity and duration of exercise are the major factors that determine both the magnitude of the rise in hepatic glucose production and the relative contributions of hepatic glucose production and the relative contributions of hepatic glycogenolysis and gluconeogenesis to the increased glucose production (Brooks, 1999

1.2.3 Triglyceride and Cholesterol

Triglyceride, and cholesterol interest in total plasma cholesterol and Triglyceride concentration has long been recognized as the strongest and most consistent risk factors for atherosclerotic disease, thus a reduction in plasma cholesterol and Triglyceride has received much attention as a means of preventing disease. From this point many researches try to find the effect of altitude and exercises, on Triglyceride and cholesterol.

Several recent investigations have demonstrated the importance of dietary fatty acids in the regulation of blood cholesterol level (Robert, Rogers, 1997).

During exercise after acclimatization to altitude, the net degradation rate of muscle glycogen decreases compared with that determined for the same exercise power out put before acclimatization (Young, 1992; Green, 1989).

This glycogen effect of acclimatization is associated with increased circulating free fatty acid levels during exercise (Yong, 1992).

The lesser rate of change in glycogen utilization after acclimatization has been interpreted to mean that acclimatization causes an enhanced ability to utilize lipid energy source (Young, 1992).

However, no direct evidence has been observed on utilization of intramuscular Triglyceride dependence or cholesterol to support these interpretations of increased dependence on lipid energy sources at high altitude, nor was it considered that elevated concentration of free fatty acids after acclimatization may be indicative of decrease utilization (Roberts, 1995).

Thus it is possible that elevated levels of free fatty acids seen with acclimatization are a function of epinephrine induced lipolysis and decreased fatty acid uptake and are not indicative of an increased dependence on fat (Catherine, 1994).

While during exercise carbohydrate and lipids represent the major part of the substrates used for the production of energy. The contribution of one fuel or the other to muscle energy production depends on the level on mobilization and the level of oxidation of the substrate in the muscle (Vissing, 1998).

It has also been suggested that availability of extracellular glucose acts on the level of mobilization of fatty acids during exercise indirectly indicated by lower plasma fatty acid and glycerol levels than in controls (Ahlborg, 1982).

So this fatty acid and triglycerides are oxidized and stored intracellurarly in skeletal muscle (Show, 1976).

Several conditions associated with insulin resistance such, as non-insulin- dependent diabetes mellitus is associated with increased intramuscular triglyceride content. (Coggan, 1997).

In light of the strong association between intramuscular triglyceride and reduced insulin-mediated glucose disposal (Abonetti, 1988). It is likely that these lipid stores are of considerable importance. Unfortunately little is known about the metabolism of these lipid pools.

For example the immediate precursor pool (s) for intramuscular triglyceride synthesis and the mechanism for the increased triglyceride stores are unknown (Chapman, 1998).

It is known that the mobilization of fatty acids takes place in the adipose tissue by hydrolysis of triglycerides under the effect of lipase sensitive to several hormones (Fain, 1980) and in particular, epinephrine whose level in the plasma is known to increase significantly during exercise (Galbo, 1983).

Many studies show that a close correlation has been shown to exist between muscle glycogen decrease and duration of exercise (E.vara, 1986).

This result suggests that depletion of muscle glycogen stores is one major limiting factor in the ability to perform long-term exercise (Hickson, 1977; Hermansen, 1986).

1.2.4 Insulin

In muscle cells, glucose transport is stimulated by insulin and by agents that increase the demand for glucose (Brooks, 1999).

Recent studies of insulin- stimulated skeletal muscle suggest that insulin cause a translocation and redistribution of glucose transporters from an intracellular pool to the plasma membrane (Hirshman, 1990).

Additional studies in muscle have shown that exercise stimulate glucose transporter several folds (Hirshman, 1990). Of the many known stimulators of glucose transport, in skeletal muscle, two major mediators are insulin and fiber contraction. Douen et al have measured intracellurar glucose transporters and conclude that, although both exercise and insulin cause an increase in plasma membrane glucose transporters (Brooks., 1991). Many studies show that during prolonged exercise the secretion of

glucagon and catecholamines increases while the secretion of insulin decreases (Galbo, 1977).

The ability of physiological concentration of insulin to stimulate glucose utilization and glycogen synthesis in rat skeletal muscle is enhanced after both high and moderate-intensity exercise (Brooks, 1999). In addition after high –intensity exercise, glucose utilization is enhanced in the absence of insulin as long as the muscle still deplete glycogen (Garetto, 1984).

As many studies show that after acclimatization to high altitude the netdegradation rate of muscle glycogen decreases compared with that determined for the same exercise power output before acclimatization (Brooks, 1993). However, no direct evidence has been obtained that nor was it considered the decreased muscle glycogen degradation observed during exercise after acclimatization could be due to increased muscle glucose uptake (Brooks, 2000). Moreover, results of one report indicated that lower blood glucose in men after exposure to (4,300m) was due to increased glucose disappearance and oxidation (Brooks, 2000).

In neither rats (Brooks, 2000) nor dogs (Green, 1989) was the increased dependence on glucose in exercising anemic animals associated with secretion of insulin. While acute altitude exposure and acclimatization result in enhanced dependence on blood glucose.

1.2.5 Lactate dehydrogenates (LDH)

A conventional view indicates that blood lactate increases during exercise, because as the availability of oxygen to the exercising muscle is not adequate to meet the metabolic demands of the muscle and this limitation increases with increasing altitudes (Cathrine, 1994).

Lactate is formed as a waste product in the muscle and spills over into the blood. The development of muscle hypoxia is often postulated to account for accumulation of blood and muscle lactate during exercise at sea level (Aviat,Green,1989) but that concept has been challenged (Lary, 1994). O2 limited metabolism in the working muscle cannot explain the increased net lactate release (Brooks. 1990).

At high attitude, the arterial lactate response to a given exercise power out put is blunted (Sutton, 1983). This observation has been interpreted as reduction in muscle lactate production (Guo. 1998) although neither blood lactate flux nor the influences of factors that might affect the balance on blood lactate entry and removal have been ascertained at altitude. The role of hypoxia and correlated factors on blood lactate concentration are uncertain (Cathrine, 1994).

It has been concluded that much of the blood glucose pool as well as muscle and whole body glycogen reserves are disposed of after conversion to lactate (Brooks, 1991).

Brooks's show that blood lactate concentration at rest and exercise are higher on arrival at high altitude has not been known whether the higher values reflected increased production or decreased disposed. Other research of Brooks et al (1991) suggest that changes in lactate concentration at high altitude reflect increased production, i.e. rate of appearance, or arrival and the subsequent decrease with acclimatization.

Because these changes in lactate appearance were associated with epinephrine, these authors suggest that epinephrine stimulates muscle glycogenolysis, glycolysis and muscle lactate production.

Further, Brooks et al. infer with acclimatization and improved arterial oxygenation epinephrine level decrease and muscle glycogenolysis and lactate production fall.

1-3 Review of literature:

The next section of this study provides an introductory detailed review of glucose metabolism and the other varieties.

1.3.1 Glucose supply during rest and exercise:

During exercise the liver plays the essential role of increasing the glucose production to meet the increasing demands of the working muscle. The increased rate of glucose production can be accomplished by increasing the rates of hepatic glycogenolysis and gluconeogenesis early in exercise. Later during the course of prolonged exercise, liver glycogen is depleted and all glucose must be produced by gluconeogenesis from lactate, pyruvate, alanine glycerol and other precursors (Catherine, 1994).

Hepatic glycogen is the major source of hepatic glucose production at the onset of exercise. However, glycogen stored in the liver in times of glucose abundance may be depleted by an overnight fast and by (90-120) minutes of moderately intense running or swimming (Galbo, 1977). The liver glycongenolytic rate increases as a function of workout (Winder,1979). The relative contribution of glycogenolysis to total glucose production varies with intensity and duration of exercise and previous food intake (Antezana, 1992).

Hepatic glucose supply during exercise is modulated by hormones, neural control mechanisms, substrate availability and substrate competition (Butler, 1989).

1.3.2 Role of hepatic glycogenolysis in supplying glucose during exercise:

Hepatic glycogen is the major source of hepatic glucose production at the onset of exercise. At the onset of exercise central drive (feed forward control) increases the secretion of epinephrine, with an ensuing increase in glucagon and decrease in insulin (Wasserman, 1990).

Nutritional state must be taken into account when considering the role that liver glycogenolysis has on glucose production during exercise. Sonne and Galbo (1996) studies rats with high and low liver glycogen levels. High liver glycogen levels were brought about by fructose tube feeding in the morning on the experimental day, and low liver glycogen levels were brought about by giving the rates only half the amount of food consumed. At a given work load the increase in glucose production and plasma glucose concentration and the decrease in liver glycogen concentration were directly related to proexercise liver glycogen level in the two groups. In the food restricted rats plasma insulin level was lower and hepatic cAMP and total glycogen phosphorylase concentration higher during exercise that in fructose fed rats indicating higher neuroendocrine stimulation of the liver in the former compared to latter group of rats.

The correlation between the glucose production and liver glycogen content during exercise therefore suggests that hepatic glycogen contents is an important determinant of hepatic glycogenolysis contribution to total hepatic glucose production.

While for glucose and altitude some data shows the increased rates of appearance of glucose in the transition from rest to exercise after altitude acclimatization in the presence of elevated norepinephrine levels suggesting a hepatic postganglionic effect of norepinephrine or glycogenolysis as well as on gluconeogenesis (Brooks, 1999).

As well, Sonne and Galbo (1985) reported that distruction of the liver sympathetic in rats had no effect on the rate of liver glycogenolysis during exercise. Thus it seems it may be combination of stimuli are necessary for the increased glucose production.

1.3.3 The role of insulin:

The actions of catecholamines and of glucagon on liver glycogenolysis are antagonized by insulin (Winder , 1978). Specifically, insulin inhibits the glucagon-induced increases in cAMP and phosphorylase in isolated rat hepatocytes. Insulin also inhipits the alpha-adrenergic responses to catecholamines. Thus it has been shown by Issekutz (1990).

That a decrease insulin resulted in a 2- fold increase in glucose production. Furthermore, when insulin was infused this response was reversed. The authors show that the decline in glucose production as a result of insulin infusion was due to a (38%) decrease in glucose production and a (37%) increase in glucose clearance. Thus it was determined that the exercise induced (epinephrine mediated) decline in plasma insulin is essential for allowing adequate hepatic glucose production.

1.3.4 Role of hepatic gluconeogenesis in supplying glucose during exercise:

Gluconeogenesis is the process whereby lactate, pyruvate, glycerol and alanine are converted to glucose and glycogen. The liver is the major site of gluconegenesis, although the Kidney becomes important during prolonged starvation. The most important function of the gluconeogenesis is the maintenance of blood glucose levels when food intake is restricted and / or glycogen stores are depleted. Thus during prolonged exercise

gluconeogenesis is primarily responsible for the increase in glucose production (Wasserman, 1984, Wasserman, , 1987, Turcotte, 2000).

Gluconeogenesis is controlled by the balance between insulin and counter regulatory hormones, glucagon, epinephrine and cortisol (Winder. 1985; Wasserman, 1999).

Gluconeogenesis is increased during prolonged exercise by:

1. An increase in the uptake of substrates (Aloborg, 1982, Wasserman , 1999).
2. Inhibition of glycolytic enzymes by hormone mediated mechanisms.
3. Changes in intracellular regulatory factors (Winder, 1998).
4. And increases in gluconeogenic enzyme activities (Dohm, 1983).

All gluconeogenic precursors as well as glucose reach the liver in substrating concentration. Thus regulation of substrate release into the blood from extrahepatic tissue provision of substrate from the diet will directly affect hepatic glucose formation and utilization. The primary gluconeogenic precursors are lactate and alanine (pilikis, 1988). Stanley et al (1998) report that (10-25%) of the glucose during prolonged submaximal exercise in man is from lactate. During prolonged exercise the extraction of lactate from the liver. Wasserman (1990) also reports that the hepatic uptake of lactate and alanine is increased during prolonged submaximal exercise in dogs.

In summary, the sum of changes in hepatic glucogenolysis and gluconeogenesis are closely coupled to the increase in glucose uptake by the working muscle. The increase in the rate of glucose production from these pathways are essential for glucose homeostasis. The exercise induced rise in glucagon and full in insulin interact to stimulate hepatic glycogenolysis, where as the increase in gluconeogensis is due to an

increase in the supply of substrate for this pathway and glucagon action. Glucagon exerts stimulatory effects on gluconeogenesis by increasing the precursor extraction by the liver and the channeling into glucose within the liver. It may be that a combination necessary for the increase hepatic glucose supply which may be that a combination of stimuli are involve both direct sympathetic nervous system stimulation of the liver and increased circulation of catecholamines to attenuate the effects of insulin there for potentiating the effects of glucagon.

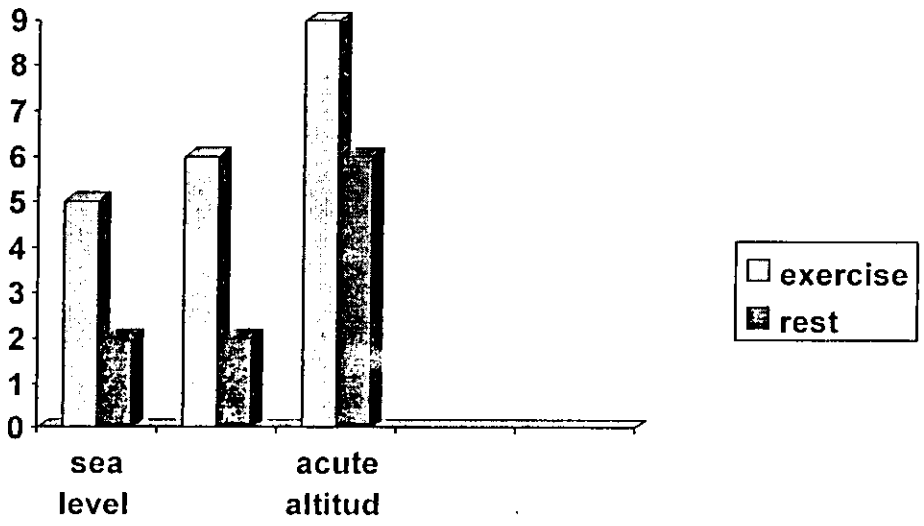
1.3.5 Altitude exposure:

Exposure to hypobaric hypoxia of high altitude results in a complete set of circulatory, endocrine and tissue responses which operate to maintain O2 delivary and utilization (Wolfel, 1999) as well as cellular energy state (Green, 1989).

At high altitude the partial pressure of O2 in the air is reduced leading to the condition of hypobaric hypoxia. The decrease in the partial pressure of oxygen greatly reduces the pulmonary O2 pressure which decreases the amount of O2 diffused across the lungs consequently, circulatory transport of O2 is reduced because arterial O2 partial pressure saturation are reduced. This condition is termed (hypoxemia). However, it is unlikely that a slight reduction in the availability of oxygen in the mitochondria would be enough to interfere with oxidative phosphorylation and ATP formation during rest or submaximal exercise (Connett,1990). Especially since circulatory adjustments allow maintenance of whole body as well as tissue O2 compensation and ensure significant reserves to increase systemic O2 transport tissue extraction and utilization during exercise as well as rest.

Several studies have demonstrated that both acute and chronic exposure to high altitude or hypoxic conditions increase dependency on

blood glucose (Brooks, 1991, 1992; Cartee, 1996). A shift toward an increased dependency on glucose and other carbohydrate energy sources optimizes the energy yield per unit O2 consumed (see the figure (1)).



Figure(1.1) Effect of altitude, acclimatization and exercise on arterial blood glucose metabolic clearance rate

This increased dependency is characterized by increased hepatic glucose production and increased muscle glucose uptake. Because on compensated hypoxia oxygenation is adquate to maintain wholebody and tissue VO2, and because ATP and creatine phosphate levels are maintained thus the classic explanation of response to O2 limitation is of little help in explaining the observed increased utilization of glucose.

Brooks et at (1991) demonstrated that when calories were sufficient, acclimatization to high altitude (4,300m) resulted in a greatly increased dependence on blood glucose as a fuel. Another reports show that blood glucose utilization is increased in exercising, but not resting.

Taken together, reports in the literature can be interpreted to suggest that hypoxia in its various form result in a shift in substrate utilization to a more O2 efficient carbohydrate fuels. The perspective which has emerged is that glucose, which provides a greater energy yield per volume of O2 consumed than fats or amino acids (Brooks, 1997) becomes a preferred substrate.

Moreover, because hepatic function is apparently protected in iron deficiency (1962) and at high altitude (Brooks, 1992) lactate the consequences of rapid glycolysis, can be recycled to glucose in the liver and the kidneys or directly oxidized in heart or highly oxidative skeletal muscle fibers (Brooks, 1997). From the perspective of comparative physiology discovery of enhanced glucose dependency in hypoxia comes as no surprise because a shift to carbohydrate- based metabolism in muscles of high altitude- adapted species which would allow them to take advantage on increased energy yield per unit of O2 consumed has been suspected for some time (Circulation, Respiration and Metabolism).

1.3.6 lactate and altitude exposure:

A conventional view indicates that blood lactate increases during exercise because the availability of oxygen to the exercising muscle is not adequate to meet the metabolic demands of the muscle and this limitation increases with increasing altitude. The controlling role of oxygen fits the well-established Pasteur effect where by lack of oxygen inhibits substrate oxidation to CO2 and water. Lactate is formed as a waste product in the muscle and spills over into the blood. The development of muscle hypoxia is often postulated to account for accumulation of blood and muscle lactate during exercise at sea level (Katz, 1989) but that concept has been challenged (Stainsby, 1984).

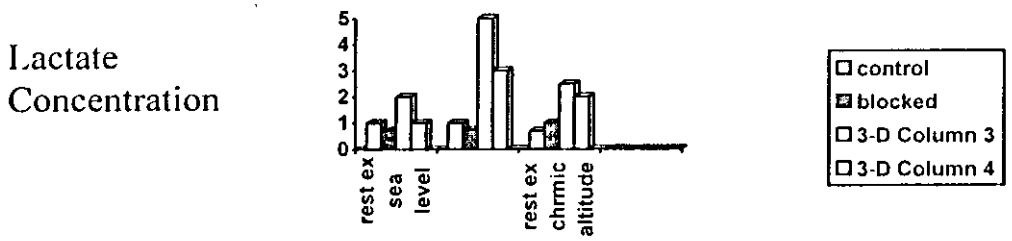
Muscle blood flow increases under acute hypoxia, and arterial O2 delivery is sufficient to maintain muscle O2 demand during submaximal exercise (Bender, 1998). The efficiency of muscular contraction as assessed by the relationship between power output and O2 consumption is undiminished (Bender, 1988). Under such conditions O2 limited metabolism in the working muscle cannot explain the increased net lactate release (Connett, 1990).

Neither blood lactate flux nor the influences of factors that might affect the balance of blood lactate entry and removal have been ascertained at altitude. The roles of hypoxia and correlated factors on blood lactate concentration are uncertain.

From initial studies on rats using isotopic tracers (Donovan, 1983) which were subsequently confirmed on humans (Mazzeo, 1986, Stanley, 1985, 1988), it appears that during exercise the rates of lactate appearance and oxidation can equal or exceed the glucose disappearance and oxidation.

Therefore, it has been concluded that much of the blood glucose pool as well as muscle and whole body glycogen reserves are disposed of after conversion to lactate (Brooks, 1996). Moreover, it has become apparent that net muscle lactate release is associated with epinephrine stimulation (Mazzeo , 1989, Brooks, 1991), and that blood lactate appearance is highly correlated with circulating epinephrine (Green, 1989, Stainsby, 1990). Although results of isotope-tracer studies are controversial (Mazzeo, 1986). It is appropriate to conclude that changes in blood metabolite concentrations in response to any stress need to be evaluated in terms of the balance of appearance and disappearance and within the context of an integrated model of blood metabolite flux (Lehman, 1990, Lehman, 1998).

In summary, since the initial observations of Edwards (1936) it has been repeatedly shown (Brooks, 1991) that blood lactate concentration at rest and exercise are higher on arrival at high altitude than subsequently during acclimatization, but it has not been known whether the higher values reflected increased production or decreased disposal. Recent research of Brooks et at (1991) suggest that changes in lactate concentration at high altitude reflect increased production, i,e, rate of appearance on arrival and the subsequent decrease with acclimatization. Because these changes in lactate appearance were associated with epinephrine, these authors suggest that epinephrine stimulate muscle glycogenolysis glycolysis and muscle lactate production. Further Brooks et al inter with acclimatization and improved arterial oxygenation epinephrine levels decrease muscle glycogenolysis and lactate production fall.



Figure(1.2)Mean arterial lactate concentration during rest and exercise at sea level upon acute and chronic exposure to (4300m) altitude

1.3.7 Lipoprotein composition and metabolic pathways:

Plasma lipoproteins are the prominent constituent of a complex transport system that provides for the movement of exogenous and endogenous lipid between the liver, the intestine and peripheral tissue.

Since lipid are not water soluble, they must combine with a polipoprotein to form micelle lipid-protein complexes or lipoprotein (young, 1992). These water-soluble macromolecules are spherical, have finite dimensions, and contain cholesterol (both free and esterified) triglyceride, phospholipid, and various apolipoproteins.

Four basic classes of lipoproteins have been categorized according to their gravitation density, chylomicron, derived from intestinal absorption of triglyceride (or trigcylglycerol) very low-density lipoprotein (VLDL or pre-13- lipoprotein)derived from the liver for the export of triglyceride, lowdensity lipoprotein (LDL, or B-lipoprotein) representing a final stage in the catabolism of VLDL and high-density lipoprotein (HDL, or α-lipoprotein) involved in the reverse transport of cholesterol. However other lipoprotein subtractions exist and include intermediate-density lipoprotein subtractions exist and include intermediate-density lipoprotein(IDL), an intermediate step in VLDL, catabolism, found in a density range of (1.006 to 1.019 g/ml) lipoprotein (a) (LP(a)) with adinsity range of (1.055 to 1.120 g/ml) and HDL that is typically studied as two separate subfraction HDL, and the more dense HDL (Tsopanakin, 1986).

The tables show more details describe the lipoprotein classes

Table(1.1)Composition of human plasma lipoproteins

Fraction	Source	Protein (%)	Total lipid	TG	Chol free
Chylomicron	Intestine	1-2	98-99	88	1
VLDL	Liver and intestine	1-10	90-93	56	8
IDL	VLDL and chylmiric	11	89	29	9
LDL	VLDL and chylomiric	21	79	13	10
Lpca	Liver	29	69	19	8
HDL	Intestine and liver	33	67	16	10

VLDL= very low-density lipoprotein.

LDL= low-density lipoprotein.

IDL= intermediate-density lipoprotein.

HDL= high-density lipoprotein.

TG= triglyceride.

Chol= cholesterol.

Lipids have important beneficial biologic functions that include the use of triglyceride for energy production or as stored fat in adipose tissue and the use of cholesterol as a component in conjunction with phospholipids, of cellular membranes or in the synthesis of steroid hormones. As many as (17) or more a polipoproteins have been identified, with one or more a polipoproteins associated with each lipoprotein complex (Brooks, 1993, Brooks, 1996). Though most apolipoproteins are synthesized by the liver or intestine, and some are in both.

Several enzymes have important roles in the metabolism of plasma lipoproteins. Lipoprotein lipase is bound to capillary walls and has been

isolated in extracts of many tissues (e.g. heart, adipose tissue, skeletal muscle). Lipoprotein lipase is involved in the hydrolysis of the triglyceride core of chylmicrons and VLDL and enhances the uptake of the released fatty acid into extrahepatic tissue.

A sequence of metabolic steps involved in the delivery of cholesterol to peripheral tissues (referred to as the LDL receptor pathway) has facilitated the overall understanding of cholesterol (Brown, 1986). Though we have increased our knowledge about the transport of cholesterol and triglyceride to the peripheral tissues less is known, about the return of cholesterol from peripheral tissues to the liver a process referred to as reverse cholesterol transport (Glomset, 1968).

Triglyceceride:

Generally, regular participation in physical activity is associated with lower plasma triglyceride concentrations. Results from cross-sectional studies using endurance athletes (Marti, 1991, Martin, 1990, Thompson, 1984), cross-country skiers (Lehtonen, 1978) and tennis players (Vodak, 1980) indicate lower triglyceride concentration in active persons than in active controls.

In some cases where endurance athletes don't have a significant low triglyceride concentrations than controls (especially in the study of women), the lack of difference was likely due to lower than average values for the inactive controls (Durstine, 1997). Endurance exercise training usually reduces plasma triglyceride concentrations (Kluttenun, 1979, Thompson, 1998).

When baseline concentrations are elevated, but not always (Leon, 1979). The amount of this reduction is related to the pertaining concentration and the volume of exercise completed during the training program (Zavaroni, 1981).

This exercise training reduction in triglyceride is believed to result from both a single exercise session and habitual exercise.

Some attention has been given to blood lipids lipoproteins, and physical activity levels of children as they advance through developmental stages. Since children generally have low triglyceride concentrations that differ during development periods, and definitions of activity levels differ among studies, one must be cautious when comparing the results from various studies (Lobo, 1992).

Cholesterol:

Although cholesterol is an important component of cell membranes and is needed for the synthesis of steroid hormones, elevated plasma cholesterol concentrations have been implicated in the development of cell (Goodman, 1988).

Some observational studies have reported lower plasma cholesterol concentrations for endurance-trained male (Williams, 1986) and female runners (Wood, 1977), but most studies observe no difference in plasma cholesterol concentrations for male runners (Thompsone, 1991) female runners (Durstine, 1987), and other endurance –trained athletes (Tsopanakis, 1986) compared with inactive counterparts. Similar (Berg, 1980) or higher plasma cholesterol concentrations (Farrell, 1982) have been observed for speed-trained and power –trained athletes, compared with inactive controls.

Result from endurance-training studies have not been any more encouraging. Most well-designed and executed studies accounting for gender, dietary intake modifications, changes in plasma volume body weight change, and altered body composition have not produced significant plasma cholesterol reductions (Thompson, 1998) some studies have reported reductions in plasma cholesterol after exercise training, but they did not concurrently maintain an inactive control group. Thus in these investigations seasonal variations and/ or interference from other variables may confound interpretation of the results for both men (Tsopanakis, 1985) and women (Lobo, 1992).

Plasma cholesterol differences for age and gender have been reported. Men have higher total cholesterol concentrations than women between the third and sixth decades of life, after this women have higher concentrations (Roberts, 1998).

Reaven et al (Reaven, 1993) completed across-sectional study comparing several levels of physical activity in older women and men. Women had higher total cholesterol concentrations than men at all activity levels, but regular exercise had no effect on total cholesterol of women or men. In addition, no exercise training-induction have been reported for older adults (Seals, 1984), furthermore in children cross-sectional (Roberts, 1998) and differences in plasma cholesterol between physically active and inactive children do not exist.

1-4 Statement of the problem:

Many researches were done to consider the effect of high altitude from sea level on blood biochemistry. But there is no studies made on low altitude, and considering its effect on blood biochemistry. From here the problem come in order to know and measure the effect of high and low altitude, and the effect of exercise on blood biochemistry for soccer players.

1-5 Purpose of the study:

The study aims at achieving the following purposes:

1. The difference between rest and exercise on blood biochemistry in different areas.
2. Making a comparison between the three areas on blood biochemistry for soccer players in order to determine the effect of altitude exposure or adaptation on blood.

1-6 Hypotheses of the Study:

The study aims at testing the following null hypotheses:

1- There are no significant differences at ($\alpha = 0.05$) between pre and post exercise blood biochemisty in different areas (Nablus, Jerico, Qalqilia).

2- There are no significant differences at ($\alpha = 0.05$) on the pre exercise blood biochemistry due to the area variable.

3- There are no significant differences at ($\alpha = 0.05$) on the post- exercise blood biochemistry due to the area variable.

1-7 Significance of the study:

1. It is the first study in Palestine that aim in studding the changes will be on blood biochemistry in soccer's at different altitude (high, low, and at sea level).
2. The study also will supply us a clear idea about the effect of exercise and rest on blood biochemistry.
3. The availability of three different areas (high, low, and sea level) makes this study so special, because our country has the lowest area in the word which is Jerico, this specialty makes our countries so important and suitable for such a study.

1-8 Limitation of the study:

The study were made on three teams of soccer players in different areas, they were from Nablus as a high altitude, Qalqelya as sea level altitude, and Jericho as a low altitude. Each team contains (12) subjects from the ages (18-25) most are nonsmokers.

A blood test taken twice, one time before training while they are relaxed, and the second one were taken after running (5km) and then rested for about (10) minutes.

The idea of making these soccer's to run (5km) is that many examine made to measure the distance the soccer run while he is playing a match and found it (5km).

Chapter Two

Methodology

Chapter Two

Methodology

2-1 Study Sample:

Twelve subjects were selected ,as each team from one of the three different areas of different altitude above sea level , below sea level , at sea level(Nablus, Jerico, Qalqelia , respectively). These subjects are in the ages (18-25) years. Some of those subjects are smoking, others are not. Some data taken from the subjects before starting the test as:

- The place of living.
- Smoking.
- Kind of work.
- Age.
- Weight.
- Kind of food he prefere.

The research sheet is provided.

Table (2.2) The effect of high and low attitude on blood biochemistry in soccer players

Test	Hb	Hct	Glu	Ins.	LDH	Trig	Chol
Pre exercise							
Post exercise							

*research sheet given to the subjects before starting the test.

2-2 Study procedure:

Measurements were made at rest and during steady state exercise, after running about (5km). They were relaxed for about (10) minutes and then blood samples were collected from the subjects.

The first team which we examine was from Nablus, the samples were collected first before running at about (6)PM then the subject who examined go to run, then after running he relaxed and another blood sample was collected.

The same procedure were performed on Qalqelia and Jerico teams.

Venous and arterial arm blood samples were taken. The blood samples were immediately placed on ice and some are analyzed with in (2) hours.

Blood samples obtained were put in (2) kinds of sterile vacationers tubes, some tubes contain EDTA in order to measure hemoglobin and hematocrite, while the other vacationers tubes contain no EDTA, these tubes were used to measure the other variables. The amount of blood taken was (6) ml in each tube.

All samples were immediately stored on ice until being centrifuged at the end of the collection period, the plasma (supernatant) was then removed with transfer pipetts and stored in tubes at 20-C until transported for future analysis.

Plasma was later analyzed for the following metabolites. Glucose, lactate, Triglyceride, insulin, cholesterol. All metabolite concentrations were determined spectrophotometrically.

Hemoglobin concentration were measured independently hematocrit (Hct) was determined by the microhematorcrit method.

For each assay to prepare the subjects' froze plasma samples for analysis, they were removed from the freezer an removed before ten minutes.

2-3 Variables:

There were two kinds of variables:

1- Independent variables which were the different altitude we measured (Nablus, Qalqilia, and Jerico).

2- The dependant variables, these variable were the analytical measurement we made like Glucose, Triglyceride, cholesterol, hemoglobin hematocrate, insulin, and LDH.

2-4 Chemical analysis

2.4.1 Glucose analysis

Serum glucose concentration was determined by a kit from SPINREACT (Spain). The content of one bottle of enzyme (Glucose oxidase peroxides, 4-Aminophenazone) were dissolved with one bottle of buffer solution.

Three kinds of tubes were used, tube as a blank, tube as standard, and tubes of the samples.

- 20µl of standard solution were put into the standard tube.
- 20µl of the sample were added to each tube of sample.
- 2ml of reagent solution were added to each tube of blank tube, standard tube, and each sample tube.
- Mixing and incubated in a water bath at (37°C) for (10) minutes.
- Each sample was put into a (1ml) cuvette and the extinction was measured at (505nm) with the spectrophotometer.

- Glucose concentration was calculated as

$$\text{Glucose (mg/dl)} = \frac{\text{Ext. Sample}}{\text{Ext. standard}} \times \text{standard conc.}$$

2.4.2 Cholesterol analysis.

Serum samples were used:

- 20µl of standard solution were added to the standard tube.
- 20µl of the sample were added to each tube of the samples.
- 2ml of the working reagent were added to blank tube standard tube and to samples tubes.
- After mixing the tubes, incubate for (5) minute at (37°C) in water bath.
- Then the extinction were measured of standard and sample against Blank reagent at (505m).
- To calculate cholesterol conc. $= \frac{\text{Ext. sample}}{\text{Ext. Standard}} \times$ Standard conc.

Standard conc. = 200mg/dl

2.4.3 Insulin analysis:

Insulin analysis

Add to wells with anti-insulin antibodies	standards	Unknown
1. Standards.	25µl	-
2. Samples.	-	25µl
3. Conjugate solution.	100µl	100µl
4. Incubated on a shaker for I hour at room temperature.		
5. Washing (4) times with automatic washer.		
6. 200µl-peroxidase substrate were added.		
7. Incubate for (15) minutes.		
8. 50µl stop solution were added. The plate were put on the shaker for approx (5) seconds to ensure mixing of substrate and stop solution.		
9. The absorbance were measured at (450) nm and evaluated.		

2.4.4 LDH analysis.

Serum samples were used as in the kit of SPINREACT(Spain):

- The test were made in temperature of (37°C).
- 3ml- of working reagent were put in each test tube.
- 50µl- of sample were added to each test tube too.
- The tubes were mixed and then wait for (1) minute.
- The extinction were measured at (340)nm.

2.4.5 TIBC (Total iron binding capacity).

Iron in serum is bound to the protein. Normally this protein is about one third saturated with iron.

Serum transferrin is assayed by saturating with iron by absorption in magnesium carbonate powder. The iron is then assayed on the supernatent.

Two kinds of reagent used from the kit used from SPINREACT company(Spain).

Serum sample were used as the following:

1- Sample (serum) 0.50ml

Saturating solution 1.50ml

2- mixed and allowed to stand at room temperature for 10min.

3- A precipitation agent was used (1) drop.

4- Mix and incubated for (10)min, at room temperature. Then centrifuged (10)min.

5- The supernatant was processed according to instructions of iron determination.

The calculation is indicated as:

$$\text{Mg/dl TIBC} = \frac{\text{Ext. sample} \times \text{standard conc. (ml/dl)} \times 4 \times 0.179}{\text{Ext. standard}} = \text{mol/L}$$

- The multiplication's factor (4) in the calculation is the factor of initial dilution of sample.

2.4.6 Hemoglobin and hematocrite analysis:

determined by the microhematocrit method.

2-5 Statistical analysis.

For analyzing data (SPSS) program was used using the following statistics:

1- Paired –t-test.

2- One- Way ANOVA and Scheffes' Post –hoc Test.

Chapter Three

Results

Chapter Three

Results

This chapter contains a detailed analysis of the data which are presented reports the characteristics of the sully sample and section two presents the results of the study hypotheses.

Section one: characteristics of subjects

A total of (40) soccer players between the ages of (18 to 25) completed all blood biochemistry measures at rest and after exercise. The means and standard deviations in table (3).

Table (3.3) Means and standard deviations for subject characteristics

Statistics	Age (yrs)	HT (M)	WT (kg)	BMI(kg/m²)
Means	22.70	1.74	68.77	22.69
Standard deviations	4.35	0.09	6.99	2.46

Results of hypotheses:

Hypothesis no 1:

There are no significant differences at ($\alpha = 0.05$)between pre and post exercise blood biochemistry of soccer players due to the altitude variable (sea level, above sea level and under sea level).

For testing this hypothesis paired t-test was used for each area and all areas as in tables (4, 5, 6, and 7).

A- Sea level (Qalqelya):

Table (3.4) Results of paired t-test for the differences between pre and post exercise blood biochemistry measures in Qalqelya (sea level)

Measures	Unite of measure	Pre-test N=13		Post-test N= 13		t.test	Sig*	% of change
		Mean	St. deviation	Mean	St. deviation			
Hb	mg/dl	15.40	1.67	15.74	2.19	1.19	0.25	2.20
Glu.	mg/dl	107.42	19.17	121.92	48.37	1.43	0.17	13.49
TIBC (Fe)	mg/dl	309.70	50.19	312.60	48.89	2.35	0.03*	-2.59
Cholesterol	mg/dl	146.77	40.33	120.99	26.48	3.35	0.006*	-17.56
LDH	U/l	279.53	34.19	289.87	34.76	7.91	0.000*	3.69
Trig	mg/dl	154.58	85.68	138.58	70.69	1.50	0.15	-10.35
Insulin	mg/dl	14.31	4.79	9.98	3.70	5.84	0.000*	-30.25
Hematocrate	%	46.65	5.22	47.56	6.76	1.05	0.31	1.95

*significant at (0.05) critical t value (1.78)

The results of table (4) show that computed t-test values on (Hb, Glu, Trig, and Hematocrate) are respectively (1.14, 1.43, 1.50 and 1.05) all of these values are lower than critical t-value (1.78).

This means that there are no significant differences at ($\alpha = 0.05$) between pre and post exercise on these variables for the soccer players in Qalqelya. While computed t-test values for (Fe, Cholesterol, LDH, and

insulin) are respectively (2.35, 3.35, 7.91, and 5.84) all of these values are greater than critical t-value (1.78).

This means that there are significant differences at ($\alpha = 0.05$) on these variables between pre and post exercise in favor of post exercise. Such results are clear in figures (3),(4), (5), (6).

figure (3) means of pre and post exercise Fe

of soccer players in Qalqelya (Sea level)

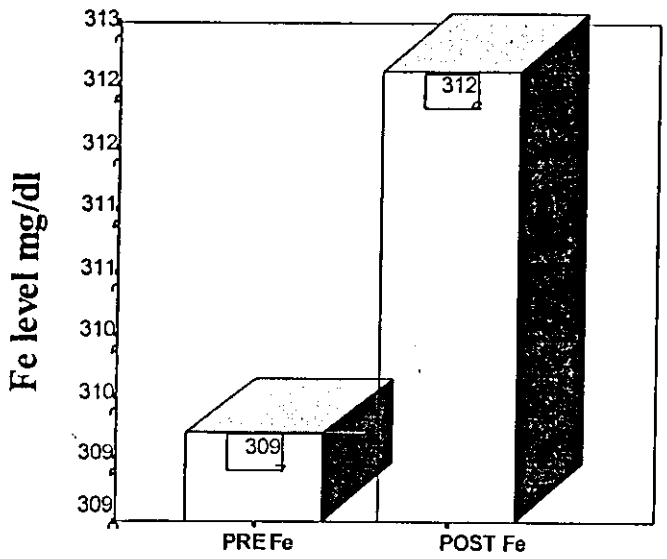


figure (4) means of pre and post exercise cholesterol

of soccer players in Qalqelya (sea level)

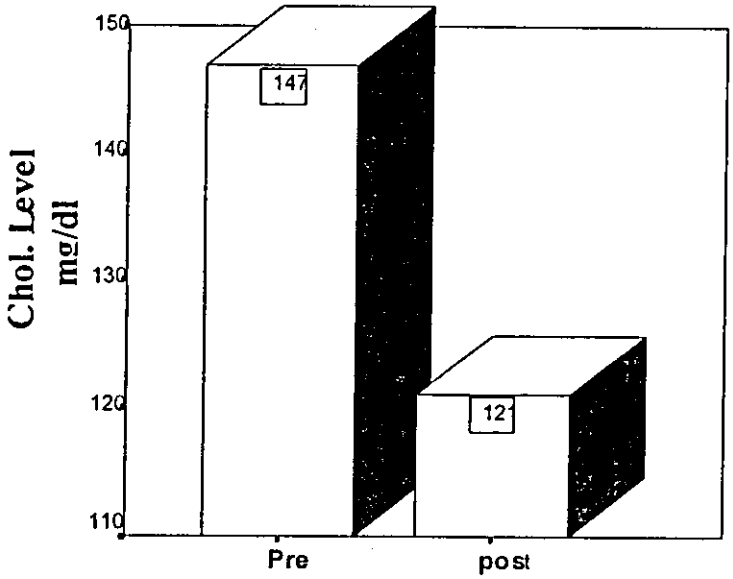


figure (5) means of pre and post exercise LDH

of soccer players in Qalqelya (sea level)

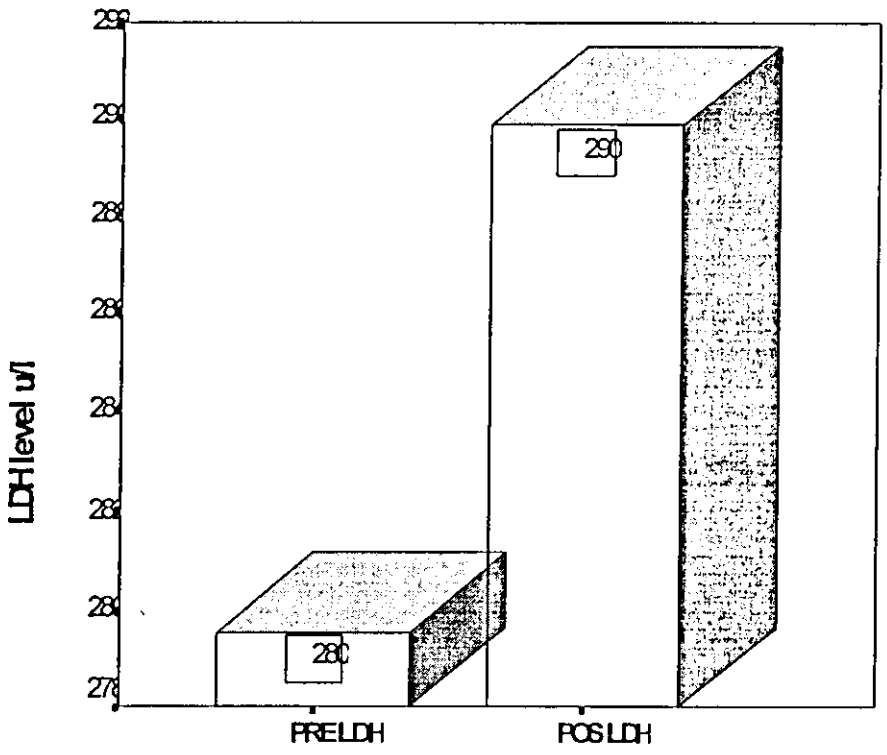
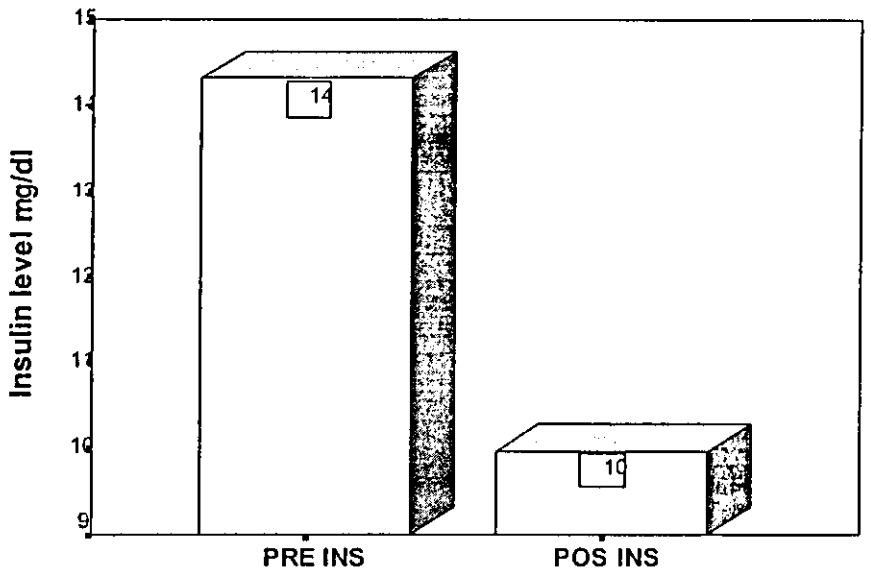


figure (6) means of pre and post exercise Insulin

of soccer players in Qalqelya (sea level)



B-Above sea level (Nablus):

Table(3.5) Results of paired t-test for the differences between pre and post exercise blood biochemistry measures in Nablus (above sea level)

Measures	Unite of measure	Pre-test N=13		Post-test N=13		t.test	Sig*	% of change
		Mean	St. deviation	Mean	St. deviation			
Hb	mg/dl	15.55	1.36	15.95	1.70	2.16	0.04*	2.57
Glu	mg/dl	110.18	23.63	121.32	27.55	1.48	0.16	10.11
TIBC (Fe)	mg/dl	315.95	59.72	319.07	55.17	1.03	0.32	-0.09
Cholesterol	mg/dl	172.98	39.89	171.77	46.61	0.12	0.90	-0.06
LDH	U/l	239.93	58.23	274.37	72.52	1.81	0.09	14.35
Trig	mg/dl	118.78	53.25	94.95	42.43	3.17	0.007*	-20.06
Insulin	mg/dl	6.37	4.26	5.40	2.40	1.60	0.13	-15.27
Hematocrate	%	45.00	3.16	45.64	3.15	2.69	0.01*	1.42

*Significant at (0.05) critical t value (1.78)

The results of table (5) show that computed t-test Nablus on (Glu, TIBC, Cholesterol, LDH, and insulin) are respectively (1.48, 1.03, 0.12, 1.81, and 1.60), all of these values are lower than critical t-test value (1.78) this means that there are no significant differences at ($\alpha = 0.05$) between pre and post exercise on these variables for the soccer players in Nablus. While computed t-test values on (Hb, Trig and Hematocrate) are respectively (2.16, 3.17 and 2.69) all of these values are greater than critical t-test value (1.78) this means that there are significant differences at ($\alpha = 0.05$) on these variables between pre and post exercise in favor of post exercise such results are clear in figures (7) , (8), and (9).

figure (7) means of pre and post exercise Hb of soccer players in Nablus (high altititude)

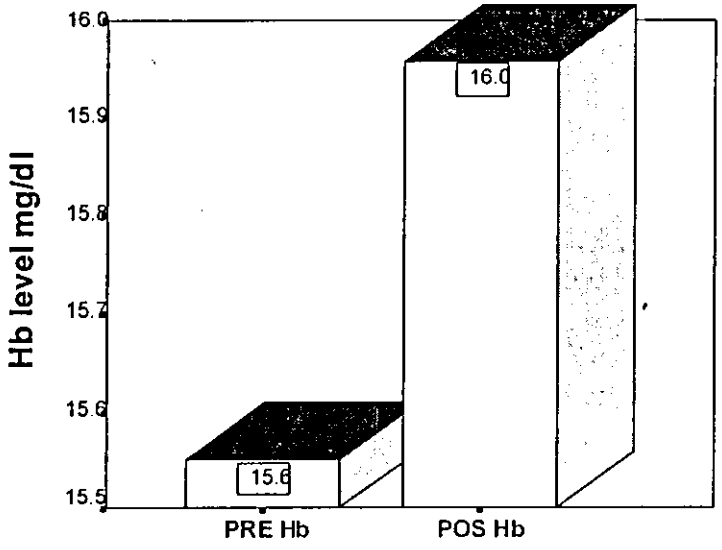


figure (8) means of pre and post exercise Trig of soccer players in Nablus (high altitude)

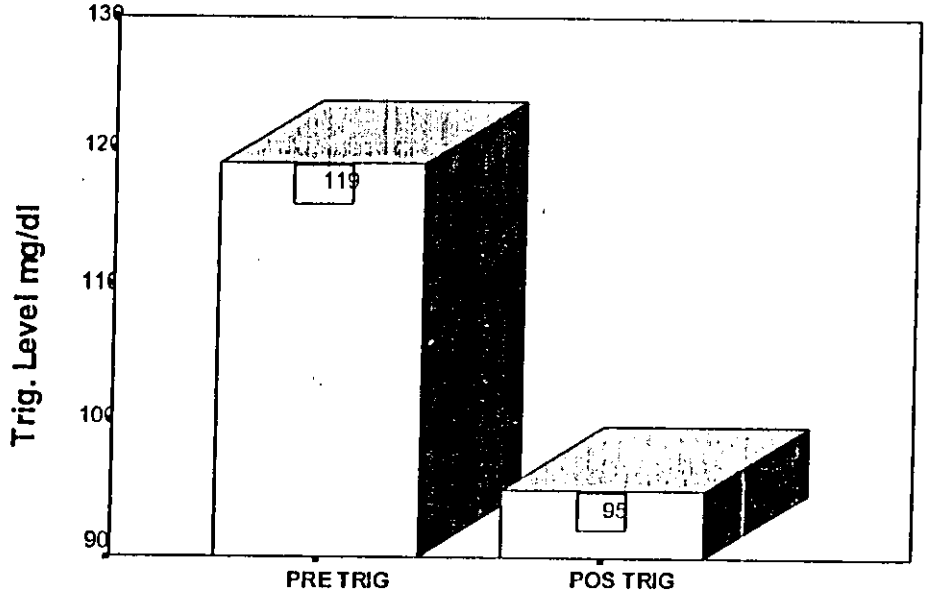
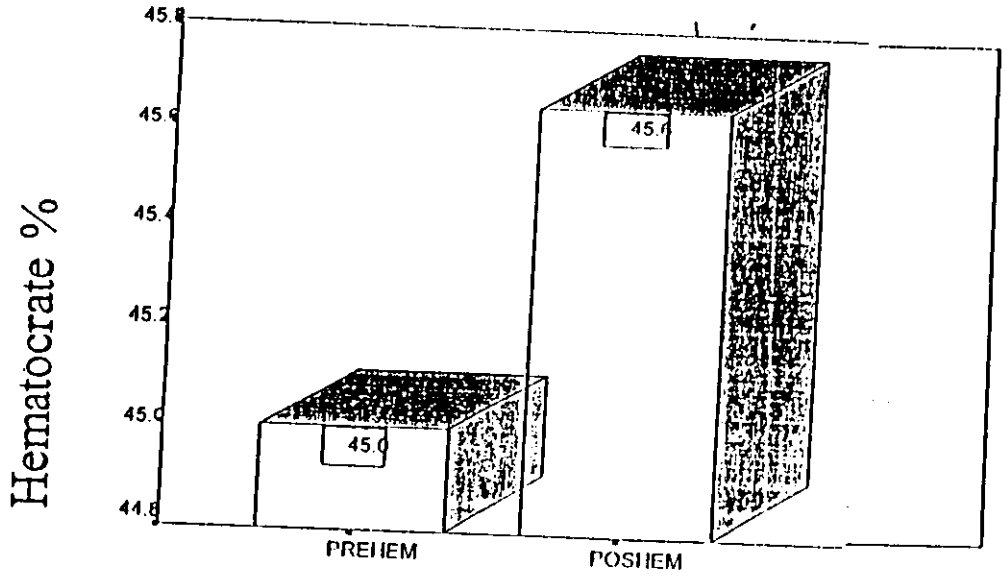


figure (9) means of pre exercise Hct



C- Under sea level (Jerico):

Table(3.6) Results of paired t-test for the differences between pre and post exercise blood biochemistry measures in Jerico (under sea level)

Measures	Unite of measure	Pre-test N=14		Post-test N=14		t.test	Sig*	% of change
		Mean	St. deviation	Mean	St. deviation			
Hb	mg/dl	13.96	1.13	14.44	1.21	5.57	0.000*	3.43
Glu	mg/dl	79.45	17.33	101.20	32.60	2.58	0.02*	27.37
TIBC (Fe)	mg/dl	300.71	84.51	297.97	77.07	0.36	0.72	-0.009
Cholesterol	mg/dl	156.86	87.33	169.86	57.08	0.32	0.75	2.11
LDH	U/l	333.47	42.90	317.85	116.96	0.58	0.78	-4.68
Trig	mg/dl	92.24	25.12	90.50	34.84	0.27	0.79	-1.88
Insulin	mg/dl	7.75	5.68	8.10	6.64	0.18	0.85	4.51
Hematocrate	%	42.46	3.09	43.97	3.34	4.70	0.000*	3.55

*Significant at (0.05) critical t value (1.77)

The results of table (6) show that computed t-test values on (TIBC, Cholesterol, LDH, Trig, and Insulin) are respectively (0.36, 0.32, 0.58, 0.27, and 0.18) all of these values are lower than critical t-test value

(1.77) this means that there are no significant differences at ($\alpha = 0.05$) on these variables between pre and post exercise under sea level.

While computed t-test values for (Hb, Glu, and Hematocrate) and respectively (5.57, 2.58, and 4.70) all of these values are greater than critical t-test value (1.77), this means that there are significant differences at ($\alpha = 0.05$) on these variables between pre and post exercise in favor of post exercise such aresults are clear in figures (10),(11),and (12).

figure (10) means of pre and post exercise Hb of soccer players in jerico (lower sea level)

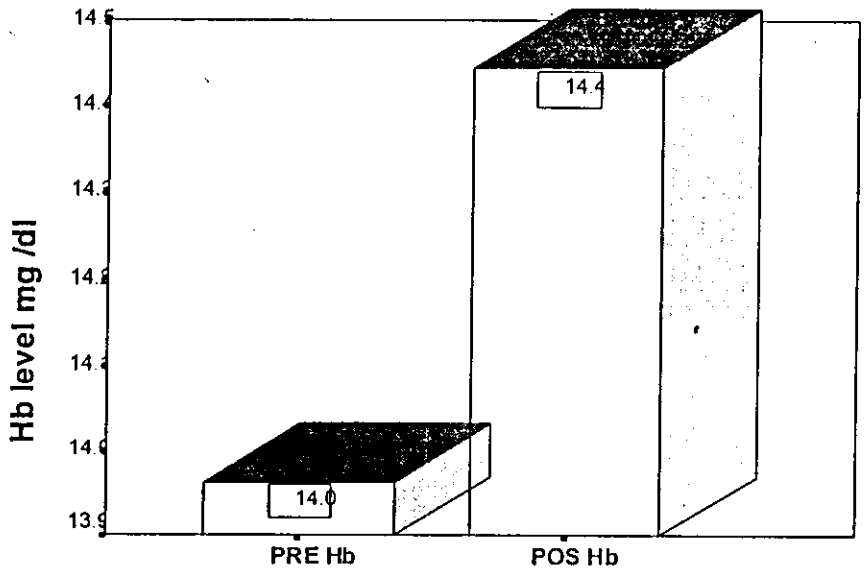


figure (11) means of pre and post exercise glu of soccer players in jerico (lower sea level)

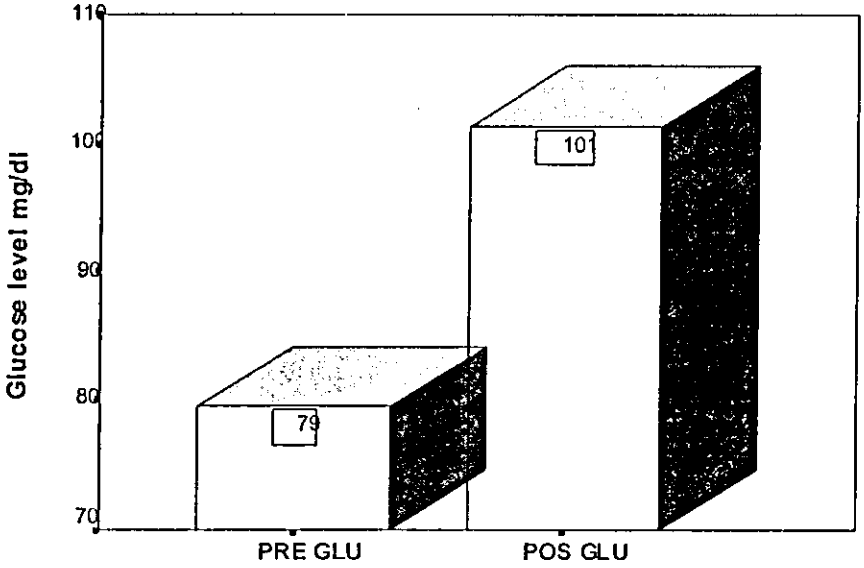
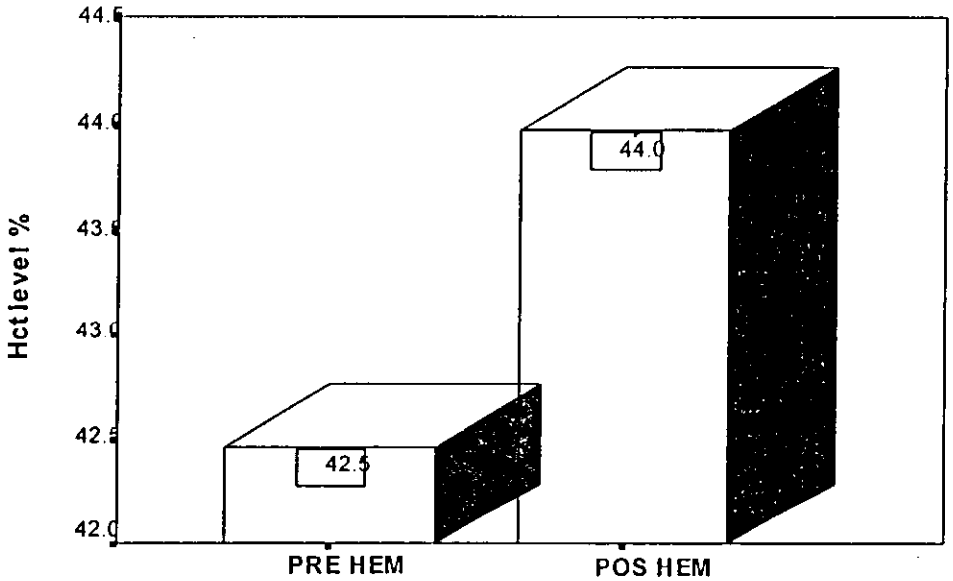


figure (12) means of pre and post exercise hematoc. of soccer players in jerico (lower sea level)



D- Total (all areas):

Table(3.7) Results of paired t-test for the differences between pre and post exercise blood biochemistry measures in total (all areas)

Measures	Unite of measure	Pre-test N=40		Post-test N=40		t.test	Sig*	% of change
		Mean	St. deviation	Mean	St. deviation			
Hb	mg/dl	15.01	1.53	15.44	1.79	3.79	0.001*	2.86
Glu	mg/dl	98.15	24.33	113.22	36.62	3.01	0.005*	15.35
TIBC (Fe)	mg/dl	311.49	63.84	312.36	59.50	0.31	0.75	-0.002
Cholesterol	mg/dl	162.53	42.03	156.20	50.32	1.02	0.31	-3.89
LDH	U/l	284.57	74.92	294.31	83.85	0.83	0.41	3.42
Trig	mg/dl	120.64	63.89	106.39	54.70	3.01	0.005	-11.81
Insulin	mg/dl	9.10	4.92	7.69	4.92	1.87	0.06	-15.49
Hematocrate	%	44.77	4.18	45.86	4.73	3.31	0.002*	2.43

*Significant at (0.05) critical t value (1.68) with Df (39)

The results of table (7) show that computed paired t-test values on variables (TIBc, Cholesterol, LDH, and Insulin) are respectively (0.31, 1.02, 0.83, and 1.77) all of these values are lower than critical t-test value (1.78). this means that there are no significant differences at ($\alpha = 0.05$) on these variables between pre and post exercise, while computed t-test

values on variables (Hb, Glu, Trig, and hematorcte) are respectively (3.79, 3.01, 3.01, and 3.31) all of these values are greater than critical t-test value (1.68) this means that there are significant differences at (α = 0.05) on these variable between pre and post exercise such results are clear in figures (10), (11), and (12).

Hypothesis no 2:

There are no significant differences at (α = 0.05)on the pre exercise blood biochemistry of soccer players due to the altitude variable (sea level, above sea level and under sea level).

For testing this hypothesis One Way ANOVA test was used ,where table (8) shows the means and table (9) shows the results of ANOVA.) .

Table (3.8) Means of blood biochemistry measures of pre exercise according to area variable

Measures	Qalqelya (N = 13)	Nablus (N = 13)	Jerico (N = 14)
Hb	15.40	15.74	13.96
Glu	107.42	109.04	79.45
TIBC (Fe)	309.70	324.87	300.71
Cholesterol	146.77	174.69	165.86
LDH	279.53	236.94	333.47
Trig	154.58	117.28	92.24
Insulin	14.31	5.33	7.75
Hematocrate	46.65	45.36	42.46

Table(3.9) Results of ANOVA for the differences on the pre exercise blood biochemistry measures according to area variable

Measures	Source of variance	Sum of squares	DF	Mean squares	F	Sig*
Hb	Between. G	24.33	2	12.16	6.66	0.003*
	Within .g	67.56	37	1.82		
	Total	91.89	39			
Glu	Between. G	7556.84	2	3778.42	9.11	0.001*
	Within .g	15342.35	37	414.65		
	Total	22899.19	39			
TIBC (Fe)	Between. G	3996.64	2	1998.3	0.47	0.62
	Within .g	154978.40	37	4188.60		
	Total	1658975.05	39			
Cholesterol	Between. G	5305.81	2	2652.90	1.54	0.22
	Within .g	63605.81	37	1719.08		
	Total	68911.79	39			
LDH	Between. G	63302.18	2	31651.09	7.52	0.002*
	Within .g	155658.06	37	4206.97		
	Total	218960.25	39			
Trig	Between. G	26419.15	2	13209.57	3.68	0.03*
	Within .g	132778.92	37	3588.62		
	Total	159198.07	39			
Insulin	Between. G	5363.54	2	281.77	14.17	0.000*
	Within .g	735.7	37	14.88		
	Total	1299.26	39			
Hematocrate	Between. G	125.23	2	62.61	4.15	0.02*
	Within .g	557.37	37	15.06		
	Total	682.60	39			

Critical (F) value (3.20) with DF (2.37).

The result of table (9) show that computed (F) values on the pre-exercise of (TIBC, and Cholesterol) are respectively (0.47 and 1.54), such two values are lower than critical (F) value (3.26) this means that there are no significant differences at ($\alpha = 0.05$) on these two variables due to the area variable.

While computted (F) values for (Hb, Glu, LDH, Trig, Insulin, and Hematocrate) are respectively (6.66, 9.11, 7.52, 3.68, 14.17, and 4.15) all of thes values are greater than critical (F) value (3.20). this means that there are significant differences at ($\alpha = 0.05$) on these variables due to the area variable. To determine these differences Scheffes' post- hoc test conducted as in tables (10), (11), (12), (13), and (14).

A. Hb Variable:

Table(3.10) Scheffes' post-hoc test for the differences of pre exercise Hb according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		-0.34	1.43*
Nablus			1.78*
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (10) show that there is a significant difference at ($\alpha = 0.05$) in (Hb) between Qalqelya and Jerico in favor of Jerico. While there are no significant difference between Qalqelya and Nablus. Such results are clear in figure (13).

figure (13) means of pre exercise according to area variable

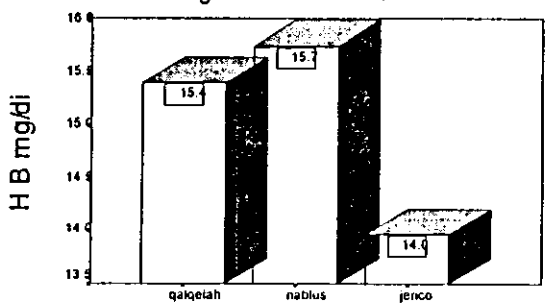


Figure (13)

Means of pre exercise (Hb) according to the area variable

B. Glu variable:

Table(3.11) Scheffes' post-hoc test for the differences of pre exercise (GLU) according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		-1.61	27.97*
Nablus			29.59*
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (11) show that there is a significant difference at ($\alpha = 0.05$) in (GLU) between Qalqelya and Jerico in favor of Qalqelya, Nablus and Jerico in favor of Nablus. While there is no significant difference between Qalqelya and Nablus. Such results are clear in figure (14).

figure (14) means of pre exercise according to area variable

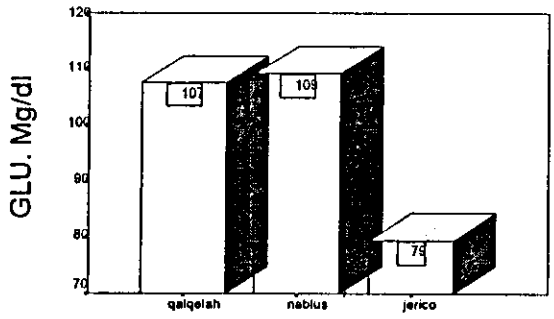


Figure (14)

Means of pre exercise (GLU) according to the area variable

C. LDH variable:

Table(3.12) Scheffes' post-hoc test for the differences of pre exercise (LDH) according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		42.59	-53.93
Nablus			-96.53*
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (12) show that there is a significant difference at ($\alpha = 0.05$) in (LDH) between Nablus and Jerico in favor of Nablus. While there are no significant difference between (Qalqelya and Nablus), (Qalqelya and Jerico). Such results are clear in figure (15).

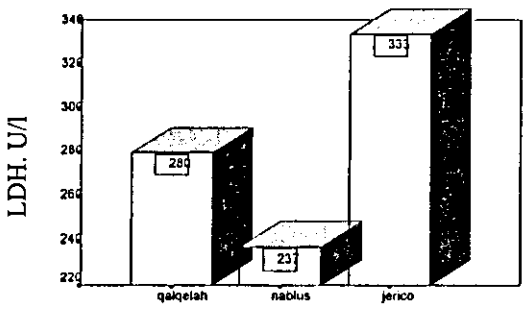


Figure (15)

Means of pre exercise (LDH) according to the area variable

D.Trig variable:

Table (3.13) Scheffes' post-hoc test for the differences of pre exercise (Trig) according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		37.30	62.34*
Nablus			25.04
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (13) show that there is a significant difference at ($\alpha = 0.05$) in (Trig) between Qalqelya and Jerico in favor of Qalqelya. While there are no significant difference between (Qalqelya and Nablus), (Nablus and Jerico). Such results are clear in figure (16).

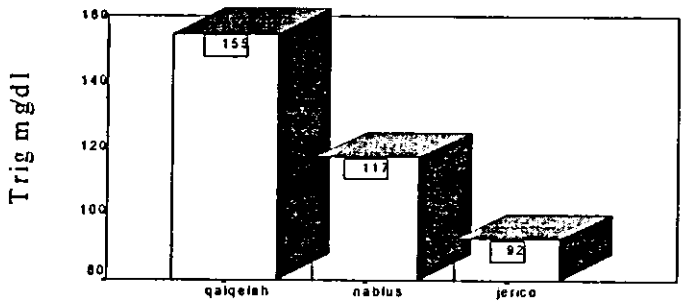


Figure (16)

Means of pre exercise (Trig) according to the area variable

E. Insulin Variable:

Table (3. 14) Scheffes' post-hoc test for the differences of pre exercise Insulin according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		8.98*	6.55*
Nablus			-2.42
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (14) show that there is a significant difference at ($\alpha = 0.05$) in insulin between Qalqelya and Nablus in favor of Qalqelya, Qalqelya and Jerico in favor of Qalqelya. While there are no significant difference between Nablus and Jerico. Such results are clear in figure (17).

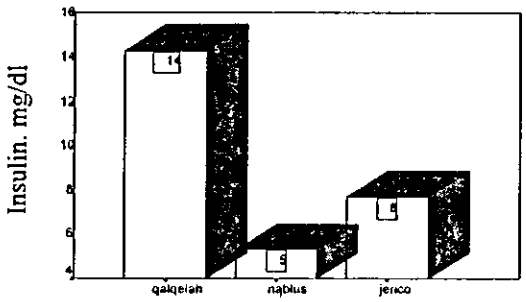


Figure (17)

Means of pre exercise insulin according to the area variable

F. Hematocrate variable:

Table (3. 15) Scheffes' post-hoc test for the differences of pre exercise Hematocrate according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		1.28	4.18*
Nablus			2.90
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (15) show that there is a significant difference at ($\alpha = 0.05$) in Hematocrate between Qalqelya and Jerico in favor of Qalqelya. While there are no significant difference between (Qalqelya and Nablus), (Nablus and Jerico). Such results are clear in figure (18).

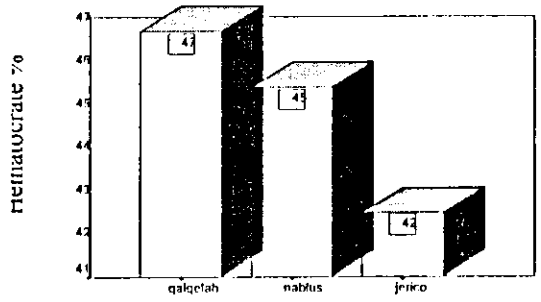


Figure (18)

Means of pre exercise Hematocrate according to the area variable

Hypothesis no 3:

There are no significant differences at (α = 0.05)on the post exercise blood biochemistry of soccer players due to the altitude variable (sea level, above sea level and under sea level).

For testing this hypothesis ANOVA test was used ,where table (16) shows the means and table (17) shows the results of ANOVA.) .

Table (3. 16) Means of blood biochemistry measures of post exercise according to area variable

Measures	Qalqelya (N = 13)	Nablus (N = 13)	Jerico (N = 14)
Hb	15.74	16.21	14.44
Glu	121.92	117.46	101.20
TIBC (Fe)	312.60	327.63	297.97
Cholesterol (Fa)	120.99	176.72	169.85
LDH	289.87	273.39	317.85
Trig	138.58	91.32	90.50
Insulin	9.98	4.96	8.10
Hematocrate	47.65	46.12	43.97

Table (3.17) Results of ANOVA for the differences on the post exercise blood biochemistry measures according to area variable

Measures	Source of variance	Sum of squares	Df	Mean squares	F	Sig*
Hb	Between. G	22.95	2	11.47	4.12	0.02*
	Within .g	102.98	37	2.78		
	Total	125.93	39			
Glu	Between. G	3241.86	2	1620.93	1.22	0.30
	Within .g	490.62.11	37	1326.003		
	Total	52303.97	39			
TIBC (Fe)	Between. G	3930.78	2	2965.39	0.83	0.44
	Within .g	132184.92	37	3572.56		
	Total	138115.70	39			
Cholesterol (Fa)	Between. G	24199.15	2	12099.57	6.004	0.006*
	Within .g	4756.47	37	2015.28		
	Total	98764.63	39			
LDH	Between. G	13071.46	2	6850.73	0.97	0.38
	Within .g	260535.40	37	7041.49		
	Total	274236.87	39			
Trig	Between. G	19963.37	2	9981.49	3.81	0.03*
	Within .g	96761.56	37	2615.17		
	Total	116724.94	39			
Insulin	Between. G	167.44	2	83.72	3.98	0.02*
	Within .g	778.07	37	21.02		
	Total	945.51	39			
Hematocrate	Between. G	92.66	2	46.33	2.19	0.12
	Within .g	781.50	37	21.12		
	Total	874.16	39			

Critical (F) value (3.26) with DF (2.37).

The results of table (17) show that computed (F) value for (Glu, TIBC, LDH, and Hematocrate) variables are respectively (1.22, 0.83, 0.97, and 2.19) all of these values are lower than critical (F) value (3.26). this means that there are no significant differences at (α = 0.05) on these variables due to the area variable. While computed (F) values for (Hb, Cholesterol, Trig and Insulin) are respectively (4.12, 6.004, 3.81, and 3.98) all of these values are greater than critical (F) value. This means that there are significant differences at (α = 0.05) on these variables due to the area variable. To determine these differences Sheffes' post-hoc test was conducted as in tables (18), (19), (20), and (21).

A. Hb variable:

Table (3.18) Scheffes' post-hoc test for the differences of post exercise Hb according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		-0.46	1.30
Nablus			1.77*
Jerico			

*Significant at (α = 0.05).

The result of table (18) show that there is a significant difference at (α = 0.05) in (Hb) between Nablus and Jerico in favor of Nablus. While there are no significant difference between (Qalqelya and Nablus), (Qalqelya and Jerico). Such results are clear in figure (19).

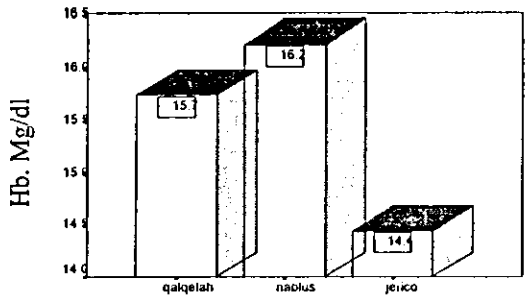


Figure (19)

Means of post exercise Hb according to the area variable

B. Cholesterol variable:

Table (3.19) Scheffes' post-hoc test for the differences of post exercise cholesterol according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		-55.73*	-48.85*
Nablus			6.87
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (19) show that there is a significant difference at ($\alpha = 0.05$) in cholesterol between Qalqelya and Nablus in favor of Qalqelya, between Qalqelya and Jerico in favor of Qalqelya. While the difference is no significant between Nablus and Jerico.. Such results are clear in figure (20).

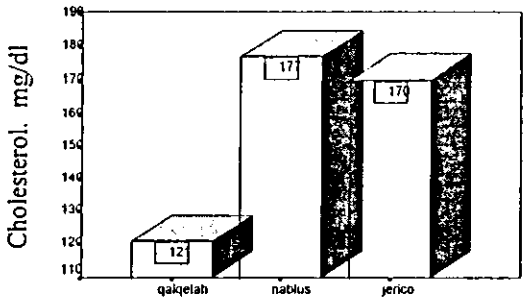


Figure (20)

Means of post exercise cholesterol according to the area variable

C. Trig variable:

Table (3.20) Scheffes' post-hoc test for the differences of post exercise Trig according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		47.26*	48.05*
Nablus			0.82
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (20) show that there is a signifioant difference at ($\alpha = 0.05$) in post exercise Trig between Qalqelya and (Nablus, Jerico) in favor of Qalqelya. While there are no significant difference between Nablus and Jerico. Such results are clear in figure (21).

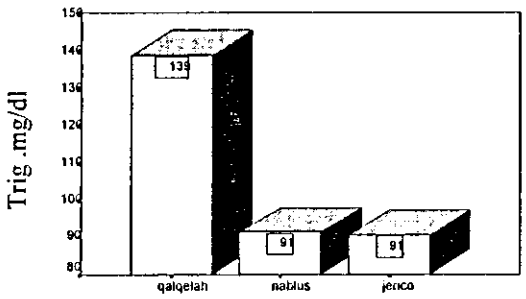


Figure (21)

Means of post exercise Trig according to the area variable

D. Insulin variable:

Table (3.21) Scheffes' post-hoc test for the differences of post exercise Insulin according to area variable

Area variable	Qalqelya	Nablus	Jerico
Qalqelya		5.02*	1.88
Nablus			-3.13
Jerico			

*Significant at ($\alpha = 0.05$).

The result of table (21) show that there is a significant difference at ($\alpha = 0.05$) in (Insulin) between Qalqelya and Nablus in favor of Qalqelya. While there are no significant difference between Nablus and Jerico, between Qalqelya and Jerico. Such results are clear in figure (22).

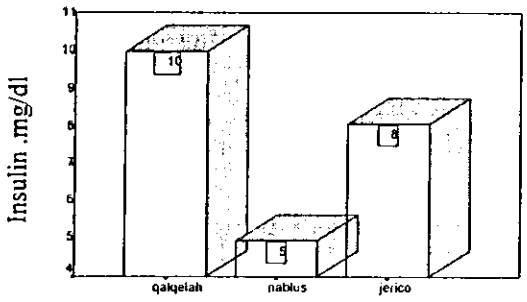


Figure (22)

Means of post exercise Insulin according to the area variable

Chapter Four

Discussion and Recommendation

Chapter Four

Discussion and Recommendation

The primary purpose of this study was to investigate the effect of Altitude on blood biochemistry of soccer players at rest and after exercise. To achieve this purpose the study was conducted on (40) first grade soccer players, and they were distributed according to altitude to three areas Qalqelya (Sea level) Nablus (Above sea level) and Jerico (under sea level). The subject ranged in age from (18 to 25) years.
The blood biochemistry measure were conducted two times pre and post exercise, and the discussion of the results are as follow:

Findings on Glucose :

Data presented in table (4, 5, 6) showed that there was no significant change at sea level and above sea level, while there is a significant change under sea level (27.37%) such finding could be attributed to exposure to high attitude or hypoxic conditions increased dependency on blood glucose, our findings in this respect are in agreement with the findings of previous studies (Brooks, 2001, Brooks, 1992, Cartee, 1991, Cooper, 1986).

During exercise muscle glucose uptake is influenced by changing in glucose tarnsporter proteins (Good year, 1991, Goodyear,1990, Goodyear, 1991, Rodrick, 1991).
Such change was attributed to that exercise can induce a change in the intrinsic activity of the transporters (Goodyear et al , 1991).

After exercise the diminution of glucose uptake correlated with a reduction in the availability of glucose transporters as well as with a decline in their intrinsic activity which decreased at a greater rate than the actual change in transporter number (Goodyear, 1990).

Brooks et al (1991) demonstrated that when calories were sufficient, acclimatization to high attitude (4.300m) resulted in a greatly increased dependence on blood glucose as a fuel.

Some researchers found that acclimatization to high attitude increases glucagon and norepinephrine concentration and that arterial glucagon concentration correlates with norepinephrine, but not insulin during rest and exercise are interpreted to suggest that gluconeogenesis is essential for maintenance of euglycemia during rest and exercise after high attitude acclimatization (Cathrine, 1994).

As well, these researchers demonstrated increased dependance on blood glucose as a fuel during exercise after acclimatization, cumulative effects of altitude exposure and acclimatization resulted in very high rates of glucose appearance, disappearance, and clearance.

Cholesterol and Triglycerides

There were decreased (17.56%) and (10.35%) respectively at sea level. While above sea level (Nablus) there was a decrease in cholesterol (0.06%) and for trig, a decrease for (20.06%), while under sea level there is no significant change on cholesterol while there is a decrease of about (1.88%) on trig.

The total of the three altitude show a decrease in cholesterol (3.89%) and a decrease in Trig (11.81%). The researcher attributed such

change that during exercise after acclimatization to high attitude the net degradation rate of muscle glycogen decreases compared with that determined for the same exercise power out put before acclimatization (Yong,1982, Green,1989, Young,1992). Furthermore, this glycogen sparing effect of acclimatization is associated with increase circulation of free fatty acid levels during exercise (Young,1982, Young, 1992). The lesser rate of change in glycogen utilization after acclimatization has been interpreted to mean that acclimatization causes an enhanced ability to utilize lipid energy sources (Nicolosi, 1997, Young, 1982, Suton, 2001).

The latter possibility was suggested by research which demonstrated that hypoxia potentiates exercise-induced sympathetic neural activation at sea level inhabitants (Seals, 1991). and in high altitude residents (Wanger, 2001).

Thus it is possible that elevated level of free fatty acids seen with acclimatization are a function of epinephrine induced lipolysis and decreased free fatty acid uptake and are not indicative of an increased dependence on fat.

Finally, recent evidence by (Butterfield et al ,1992) and (Brooks, et al, 1991) suggest that precious interpretation of substrate utilization after altitude exposure have been influenced by energy imbalance, a circumstance where fat utilization is known to be involved, as well as hypoxia.

Brooks et al (1991) demonstrated that when energy balance was maintained i.e. energy intake was increased to meet increased rates of energy expenditure on dialy basis, altitude exposure increased glucose dependency at rest and during exercise.

Specifically, the decreased rate of muscle glycogenolysis seen after acclimatization to high attitude was attributed to increased rates of glucose uptake by muscle from blood.

However, in that investigation free fatty acid consumption was not measured. Thus, the cause or purpose of elevated circulating free fatty acids in hypoxia is not clear.

For Hemoglobin, Heamatocriate, TIBC.

At sea there were no significant change for hemoglobin and heamatocrate while there is a decrease (2.59%) in TIBC while the result at above sea level show that there is an increase (2.57%) for hemoglobin and increase of heamatocrate (1.42%) and a decrease for (TIBC) for (0.09%).

Under sea level Hb and Hct show a increase of about (3.43%) and (3.55%) respectively, while TIBC show a decrease (0.009%), the researchers attributed such a change to that during sub-maximal exercise, it is generally thought that VO2 depends on energy requirements of the effort undertaken and not on O2 availability to mitochondria.

However, at maximal exercise the majority of evidence points to a maximal VO2 (VO2max) that is reached and limited by O2 a valiantly (Wager, 1996). This appears to be most evident in highly trained athletes (Powers et al. 1989).

At extreme altitude VO2 max may by only 25-30% of sea level values (West et al. 1983a, Cymerman et al. 1989).

While studies at extreme altitude necessarily require substantial periods of acclimatization, and raise questions of factors other than hypoxia.

On the argument that maximal VO2 is reduced due to reduced O2 availability as altitude is gained, several authers have analyzed theoretical execration of decrements in O2 uptake due to altitude (West, 1983, Diprampero and Ferretti, 1990).

None of these analyses have employed a moder permitting quantitative feedback between the lung and the muscles in terms of O2 transport. For example, if muscle diffusions conductance for O2 were reduced for some reasons this would not only impair O2 unloading from the muscle microcirculation directly but it would also effect pulmonary gas exchange in a secondary manner. In these example, reduced O2 extraction in muscle would raise venous (O2) which would increase the rate of diffusion equilibration of O2 in the lunges, due to a reduction in mean slope of the O2-Hb dissociation curve (Wagner, 1982). Thus it is not just the ability of the lungs to exchange O2, that determines this pressure drop but also circulatory properties (blood flow, hemoglobin concentration). The same reservation applies to exchange with in the muscles.

So many researchers agree that acute exposure of humans or animals to environmental hypoxia results in a decrease in the maximal rate of O2 uptake (VO2 max), which is proportional to the decrees in O2 delivery that follows the reduction in blood O2 content (Grover, 1986). As acclimatization proceeds, blood O2 content increase because of the stimulation of red blood cell production, however this is not accompanied

by a proportionate increase in VO2 max which show no change (Gonzales, 1998) or a modest increase (Gonzalez, 1991, 1993) during the course of acclimatization.

On one hand, it is possible that an increase in cardiac out put, by shortening capillary transit time, may result in incomplete O2 diffusion equilibration at the pulmonary or the tissue level.

The former could limit arterial blood oxygenation and offset the effect of the increased blood flow on the rate of connective O2 delivery to the tissues. The latter would limit the rate of O2 transfer from capillary to mitochondrion. Second the increase in myocardial O2 consumption (mVO2 secondary to an increase in heart rate could exceed the rate of O2 supply to the myocardium thereby compromising cardiac function (Gonzales, 1998).

Other researchers show that the compromised oxygen uptake and higher blood lactate values are thought to be a consequence of reduced blood flow to the exercising muscle in the skating position.

The Effect of Altitude and Exercise on LDH:

At sea level LDH show an increase (3.69%), above sea level, LDH has no significant change, and under sea level the result show no significant change too.

Some researcher attributed such a change to that blood lactate increases during exercise because the availability of oxygen to the exercising muscle is not adequate to meet the metabolic demands of the muscle and this limitation increases with in creasing attitude. The controlling role of oxygen fits the well-established "Pasteur-effect" (Cathrine, 1994) where by lack of oxygen inhibits substrate oxidation to CO2 and water. Lactate is formed as awaits product in the muscle and spills over into the blood. The development of muscle hypoxia is often postulated to account for accumulation of blood and muscle lactate during exercise at sea level (Katz, 1999) but that concept has been challenged (Stainsby, 2000).

Arterial lactate concentration at a given subamximal exercise power out put is greater under acute hypoxia than in normoxia (Bender, 1988, Sutton, 1983,1998) however, muscle blood flow increases under acute hypoxia, and arterial O2 delivery is sufficient to maintain muscle O2 demand during sub maximal exercise (Bender, 1998).

The efficiency of muscular contraction as assessed by the undiminished (Bender,1998). Under such conditions, O2 limited metabolism in the working muscle cannot explain the increased net lactate release (Connet, 1999).

At high altitude the arterial lactate response to a given exercise power out put is blunted (Sutton,1983). This observation has been interpreted as a reduction in muscle lactate production (Cathrine, 1994), although neither blood lactate flux nor the influences of factors that might effect the balance of blood lactate entry and removal have been ascertained at altitude. The roles of hypxia and correlated factors on blood lactate concentration are uncertain.

Many studies on rats using istopic tracers (Cain, 1983) which were subsequently confirmed on humans (Stanley, 1988, 2001) it appears that during exercise the rates of lactate appearance and oxidation can equal or exceed the glucose disappearance. Therefore, it has been concluded that much of the blood glucose pool as well as muscle and whole body glycogen reserves are disposed of after conversion to lactate (Brooks, 1999), moreover it has become apparent that net muscle lactate release is associated is highly correlated with circulating epinephrine (Stainsby, 1990).

It is appropriate to conclude that changes in blood metabolite concentrations in response to any stress (e.g. acute hypobaric hypoxia).
In summary, since the initial observation of many researchers has been repeatedly shown (Bender, Sutton, 1983, Brooks, 1999) that blood lactate concentration at rest and exercise are higher on arrival at high altitude than subsequently during acclimatization, but it has not been known whether the higher values reflected increased production or decreased disposal. Recent research of Brooks et al. (1999) suggest that changes in lactate concentration at high altitude reflect increased production i.e rate of appearance, on a rival and the subsequent decrease with acclimatization. Because these changes in lactate appearance were

associated with epinephrine, these authors suggest that epinephrine stimulates muscle glycogenolysis, glycolysis and muscle lactate production.

Present data can not be interpreted as previous data about the regulatory role of epinephrine on lactate metabolism. Previously, it was suggested that hypoxia stimulated epinephrine release which in turn stimulates muscle glycogenolysis, glycolysis and muscle lactate production (Brooks, 1991). However other researchers measured both arterial and venous concentrations of lactate across the legs. (Cathrine, 1994).

The effect of altitude and exercise on Insulin

The result show at sea level that there is a decrease (30.25%) in insulin, and at above sea level there is also a decrease (15.17%), while at under sea level there is no significant change on insulin, the researcher attributed such change to that enhances sympathodrenal activity and depressed circulation insulin are the main stimuli of lipolysis in man during exercise.

Our findings of reduced cholesterol and Triglyceride concentration are in agreement with reports in the literature some researches show that effects of epinephrine and insulin on leg (FFA) uptake and consumption are not documented others show that there is no difference in insulin values after, acute or chronic altitude exposure during rest or exercise, and insulin had minimal effect on the increase in (FFA) concentrations in control subjects after altitude exposure.

Such a result is in agreement with the result of studies of (Brooks, 2001, Cathrene, 1994, Wagner, 1996). On the other hand researchers show that at sea level, resting arterial insulin was the same on both control and blocked subjects and did not change with altitude exposure. During exercise at each phase arterial insulin concentration decreased but again was not different between control and blocked subjects not was insulin concentration different after altitude exposure despite increased rates of glucose uptake across the legs.

Recommendations

Based on the study finding, the researcher recommended the following:

1- Further research study should be conducted to determine the effect of high and low altitudes on selected physiological parameters such as aerobic and anaerobic powers.

2- Further research study should be conducted on ordinary persons, and making comparisons in physiological functions between high and low altitude residence.

3- Further research study should be conducted to determine aerobic and anaerobic co-enzymes response to high and low altitude.

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الملخص

أثر الانخفاض والارتفاع عن سطح البحر على بيوكيميائية الدم لدى لاعبي كرة القدم

إعداد

فيحاء عثمان البحش

إشراف

الدكتور سليمان خليل الدكتور عبد الناصر القدومي

هدفت الدراسة التعرف إلى أثر الارتفاع عن سطح البحر على بيوكيميائية الدم لدى لاعبي كرة القدم، إضافة إلى إجراء مقارنات في بيوكيميائية الدم أثناء الراحة وبعد المجهود تبعاً لمتغير الارتفاع والانخفاض عن سطح البحر.

لتحقيق ذلك أجريت الدراسة على عينة قوامها (40) لاعباً لكرة القدم من أندية الدرجة الممتازة، تم توزيعهم تبعاً للارتفاع عن سطح البحر إلى ثلاث مجموعات (مستوى سطح البحر) ويمثله النادي الأهلي في قلقيلية، (فوق مستوى سطح البحر) ويمثله نادي الاتحاد في نابلس، (تحت مستوى سطح البحر) ويمثله نادي هلال أريحا.

وقد تم قياس الهموجلوبين، والجلكوز، TIBC، والكوليستيرول، وأنزيم نازعات الهيدروجين، وثلاثي الجليسرايد، والانسولين، والهموتوكريت، قبل وبعد التمرين في كل منطقة.

وبشكل عام أظهرت النتـــائج وجـــود تـــأثير للتمريـــن علـــى متغـــيرات (الهموجلوبين، والجلكوز، والهيموتكريت) كذلك أظهرت النتائج وجود فـــروق ذات دلالة إحصائية عند مستوى الدلالة (α = 0.05)في الهموجلوبين والجلكوز، وانزيـــم نازعات الهيدروجين وثلاث الجليسرايد، والانسولين،و الهيموتكريت بيـــن أفـــراد المجموعات الثلاث أثناء الراحة.

أما فيما يتعلق بأثر التمرين والمقارنات على القياس البعدي تبيـــن وجـــود فروق على متغيرات الـــهيموجلوبين، والكلوليســـتيرول، وثلاثـــي الجليســـيرايد، والانسولين تبعاً للمناطق المختلفة.