40218

National Library of Canada | Bibliothèque nationale du Canada | CANADIAN THESES ON MICROFICHE | THÈSES CANADIENNES SUR MICROFICHE

NAME OF AUTHOR/NOM DE L'AUTEUR LUC ALBERT LEGER

TITLE OF THESIS/TITRE DE LA THÈSE THE EFFECTS OF CONTINUOUS AND INTERMITTENT TRAINING UPON ATP, PC, CPK, AK AND "M" AND "H" LDH IN SKELETAL MUSCLE, HEART AND LIVER OF THE RAT

UNIVERSITY/UNIVERSITÉ UNIVERSITY OF ALBERTA

DEGREE FOR WHICH THESIS WAS PRESENTED/GRADE POUR LEQUEL CETTE THÈSE FUT PRÉSENTÉE DOCTOR OF PHILOSOPHY

YEAR THIS DEGREE CONFERRED/ANNÉE D'OBTENTION DE CE GRADE 1978

NAME OF SUPERVISOR/NOM DU DIRECTEUR DE THÈSE Dr. HOWARD WENGER

Permission is hereby granted to the NATIONAL LIBRARY OF CANADA to microfilm this thesis and to lend or sell copies of the film.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

L'autorisation est, par la présente, accordée à la BIBLIOTHÈQUE NATIONALE DU CANADA de microfilmer cette thèse et de prêter ou de vendre des exemplaires du film.

L'auteur se réserve les autres droits de publication; ni la thèse ni de longs extraits de celle-ci ne doivent être imprimés ou autrement reproduits sans l'autorisation écrite de l'auteur.

DATED/DATÉ 27/6/78 SIGNED/SIGNÉ [signature]

PERMANENT ADDRESS/RÉSIDENCE FIXE 6400 DES JALESNES

ANJOU, QUÉ.

H1M 1Y2

NL-91 (3-74)

NOTICE

The quality of this microfiche is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us a poor photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this film is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30. Please read the authorization forms which accompany this thesis.

THIS DISSERTATION
HAS BEEN MICROFILMED
EXACTLY AS RECEIVED

AVIS

La qualité de cette microfiche dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de mauvaise qualité.

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, examens publiés, etc.) ne sont pas microfilmés.

La reproduction, même partielle, de ce microfilm est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30. Veuillez prendre connaissance des formules d'autorisation qui accompagnent cette thèse.

LA THÈSE A ÉTÉ
MICROFILMÉE TELLE QUE
NOUS L'AVONS REÇUE

THE UNIVERSITY OF ALBERTA

THE EFFECTS OF CONTINUOUS AND INTERMITTENT TRAINING UPON ATP, PC, CPK, AK AND "M" AND "H" LDH IN SKELETAL MUSCLE, HEART AND LIVER OF THE RAT

by

© LUC ALBERT LEGER

A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE
OF DOCTOR OF PHILOSOPHY

DEPARTMENT OF PHYSICAL EDUCATION

EDMONTON, ALBERTA

FALL 1978

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled THE EFFECTS OF CONTINUOUS AND INTERMITTENT TRAINING UPON ATP, PC, CPK, AK AND "M" AND "H" LDH IN SKELETAL MUSCLE, HEART AND LIVER OF THE RAT

submitted by LUC ALBERT LEGER

in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

.....
Supervisor

.....

.....

.....

.....

.....
External Examiner

Date ..May 14 1975.....

ABSTRACT

The purpose of the present study was to investigate the effects of sprint and endurance training upon enzymes associated with anaerobic glycolysis and high energy compound metabolism in rat tissue. Thirty rats were randomly assigned to either sedentary, sprint training or endurance training group. Sprint training consisted of 10 x 1 min run/4 min relief at 70m/min, 8% slope, 5 days/week for 6 months and endurance training, 45 min at 31 m/min, 8% slope, 5 days/week for 6 months. Both training regimens resulted in a decreased activity of LDH, M-LDH and CPK in the fast twitch gastrocnemius, plantaris and tibialis anterior muscles without altering AK activity and PC stores. In the heart and slow twitch soleus, LDH, M-LDH, CPK and AK activities as well as PC stores were unchanged as a result of chronic exercise. No LDH, CPK and AK changes were present in the liver. Both training programs reduced the body weight gain and increased the relative weight of the heart and skeletal muscles. All enzymes and metabolites of heart, liver and slow and fast twitch muscles are affected in a similar manner by sprint and endurance training in the laboratory rat. Sprint training in these animals is presently very empirical and requires further investigation.

ACKNOWLEDGEMENTS

I wish to sincerely thank my advisors Dr. R.C. Glassford, Dr. A.W. Taylor and Dr. M. Wenger, and the members of my committee, Dr. N. Kravchuk, Dr. S. Nendryk and Dr. J. Russel for their guidance and assistance in the completion of this thesis.

I would also like to thank Dr. R.J. Barnard (University of California at Los Angeles) who served as external examiner.

I would like to thank Prof. A. Sheedy and Dr. G. Larivière (University of Montreal), both directors of the Physical Education Department at one time or another, who facilitated my teaching load so I could complete this work.

Appreciation is extended to Dr. A. Imbach and Dr. J. Leduc (University of Montreal, Medical School) for the use of their laboratory facilities. I would also like to thank Dr. R. Gianetto and R. Chassé (University of Montreal, Biochemistry Department) who helped me to set a gel electrophoretical method for LDH and Axelle Charlot who patiently did the enzyme analyses. I would further like to thank Dr. R. Cléroux (University of Montreal, Mathematics and Statistics Department) for counseling and help in running the computerized statistical analyses.

Finally, I would like to express my love to my wife Carole who patiently waited for the end of this adventure.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF PLATES

TABLE 1 Abbreviations, Symbols and Definitions

ADP	adenosine-5-diphosphate
Aerobic energy	energy metabolized via the citric acid cycle, O_2 being the final electron carrier
AK	adenylate kinase or ATP:AMP phosphotransferase (EC 2.7.4.3) or myokinase
AMP	adenosine-5-monophosphate
Anaerobic energy	energy metabolized without O_2 via the adenylate kinase, the creatine phosphokinase and the lactate dehydrogenase reactions as well as the use of the ATP stores
ATP	adenosine-5-triphosphate
ATPase	ATP phosphohydrolase (EC 3.6.1.3)
Cgr	continuous training group
CPK	creatine phosphokinase or creatine kinase or ATP: creatine phosphotransferase (EC 2.7.3.2)
Fiber types	The muscle fiber type classification used in this study is that of Edington and Edgerton (1976). There are three types of muscle fibers in the rat: fast-twitch glycolytic (FG), fast-twitch high-oxidative glycolytic (FOG) and slow-twitch oxidative (SO). Fast and slow-twitch (FT and ST) are also used to designate muscle fibers without making any inference to their metabolic state. FT and ST are also used to identify muscles predominantly composed of FT and ST fibers.
FG	fast twitch glycolytic or fast twitch white (see also fiber types)
FOG	fast twitch high oxidative glycolytic or fast twitch red (see also fiber types)
FT	fast twitch or type II (see also fiber types)
LDH	lactate dehydrogenase or total LDH activity or L-lactate: NAD oxidoreductase (EC 1.1.1.27)

TABLE 1 (Continued)

LDH_{21}	LDH activity at PA = 21×10^{-4} M
LDH_3	LDH activity at PA = 3×10^{-4} M
M-LDH	muscle type monomer of LDH or LDH activity due solely to muscle type monomers
NAD	nicotinamide-adenine dinucleotide
NADH	nicotinamide-adenine dinucleotide, reduced
NADP	nicotinamide-adenine dinucleotide phosphate
NADPH	nicotinamide-adenine dinucleotide phosphate, reduced
GM	gastrocnemius medialis
G6P	D-glucose-6-phosphate
G6P-DH	glucose-6-phosphate dehydrogenase or D-glucose-6-phosphate: NADP oxidoreductose (EC 1.1.1.49)
High energy compounds	ATP + PC
H-LDH	heart type monomer of LDH or LDH activity due solely to heart type monomers
HK	hexokinase or ATP: D-hexose-6-phosphotransferase (EC 2.7.1.1)
Igr	intermittent training group
IU	international unit for enzyme activity (= amount of enzyme which convert 1 micromole of substrate per minute under specific conditions, optimal ionic strength of the buffer, optimal pH, wave length)
n	number of rats per group
P	plantaris or probability level
PA	pyruvate (pyruvic acid)

TABLE 1 (Continued)

PC	N-phosphorylcreatine (creatine phosphate or phosphocreatine)
PH	phosphorylase or α-1,4-glucan: orthophosphate glucosyltransferase (EC 2.4.1.1)
PK	pyruvate kinase or ATP: phosphorylase phosphotransferase (EC 2.7.1.40)
PFK	phosphofructokinase or ATP: D-fructose-6-phosphate 1-phosphotransferase (EC 2.7.1.11)
S	soleus
Sgr	sedentary group
SO	slow-twitch oxidative muscle fiber or slow twitch intermediate (see also fiber types)
ST	slow twitch or type I (see also fiber types)
TA	tibialis anterior
$\dot{V}O_2max$	maximal oxygen uptake
W_{abs}	absolute weight
W_{reg}	regressed weight
W_{rel}	relative weight (i.e. organ weight to body weight ratio)

CHAPTER I

STATEMENT OF THE PROBLEM

Introduction

Muscular contraction is a major element of sport performance. Physical training is carried out in order to improve the various mechanisms that control muscular contraction and hence, sport performance. The present study deals with one aspect of muscular performance, namely, anaerobic metabolism and some of its related components: ATP*, phosphorylcreatine (PC), adenylate kinase (AK, E.C. 2.7.4.3), creatinephosphokinase (CPK, E.C. 2.7.3.2) and lactate dehydrogenase (LDH, E.C. 1.1.1.27). (Figure 1)

Muscular contraction depends on the interaction of the myofibrillar proteins: actin, myosin, troponin and tropomyosin. Contraction is initiated by nervous depolarization via the T tubule'system which results in the liberation of the Ca^{++} from the sacroplasmic reticulum, which in turn makes it possible for the myofibrillar proteins to react with ATP (Ashley, 1971; Fabiato and Fabiato, 1977; Margreth et al., 1973; Porter and Franzini-Armstrong, 1965).

---

* All abbreviations, symbols and definitions used in this study are explicated in Table 1 (p. xiii).

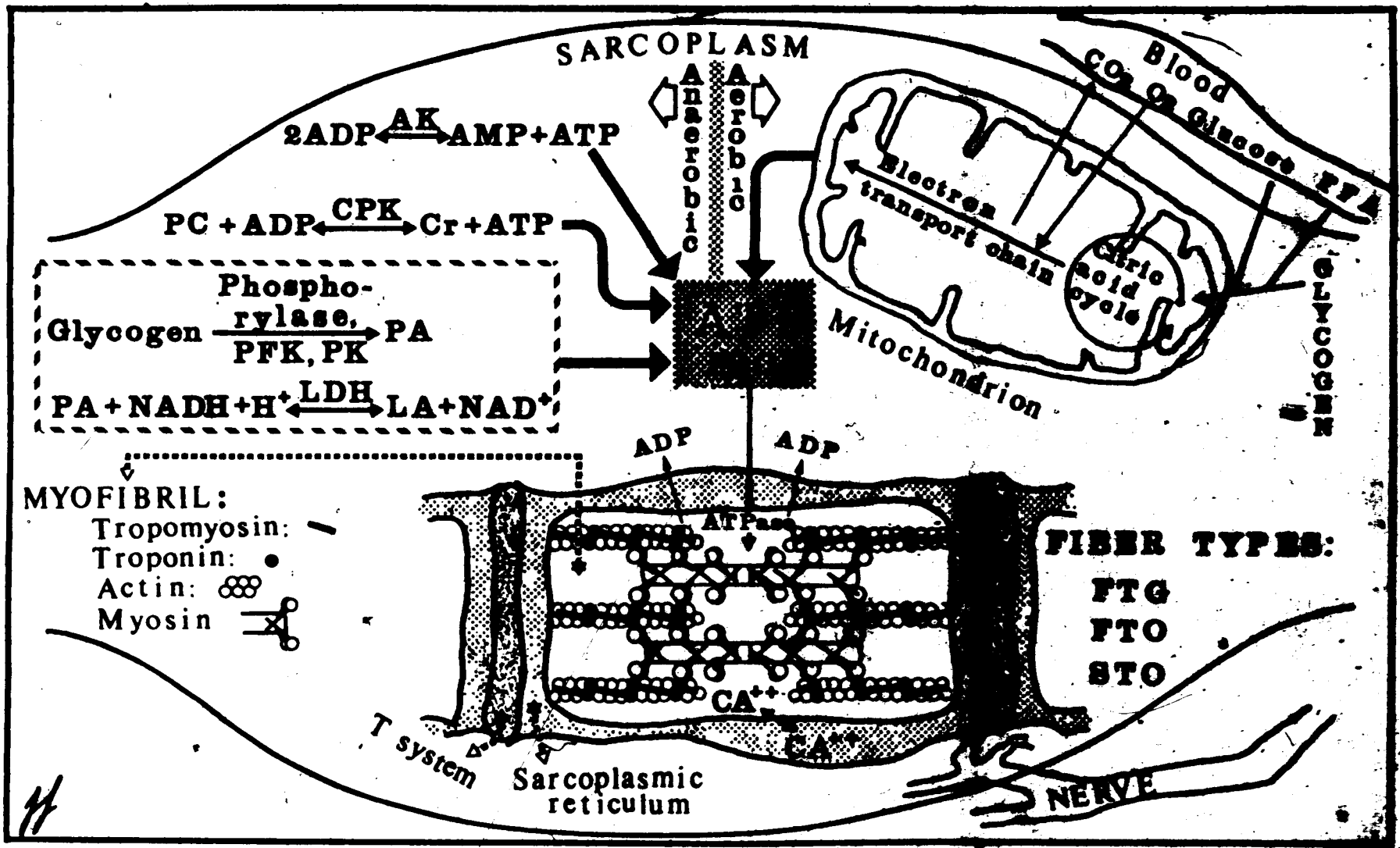

FIGURE 1. CONTROL MECHANISMS OF MUSCULAR CONTRACTION

There are two distinct forms of myosin with different myofibrillar ATPase activities (Samaha et al., 1970; Streter et al., 1966) and contractile properties (Barany, 1967; Barnard et al., 1970 b and 1971; Close, 1972). Some authors (Barnard et al., 1970 a and 1971; Close, 1972; Edstrom and Nystrom, 1969; Engel, 1974; Muller, 1974; Peter, 1970) have used these properties to classify skeletal muscle fibers into two main groups: slow twitch (ST) or type I fibers with low ATPase activity and fast twitch (FT) or type II fibers with high myofibrillar ATPase activity. The recruitment of these fibers might be specific to the nature of the exercise training (Edington and Edgerton, 1976).

Muscular contraction is finally dependent on the availability of ATP. As ATP stores in the muscle are very limited, the resynthesis of ATP is obviously another important factor related to muscle performance. A small amount of ATP can be regenerated immediately from either phosphorylcreatine and ADP in the presence of creatine-phosphokinase (CPK, E.C. 2.7.3.2) or from ADP in the presence of myokinase or adenylate kinase (AK, E.C. 2.7.4.3). As for ATP, the stores of PC and ADP are limited and, for more prolonged work, ATP resynthesis must occur via other metabolic pathways: anaerobic glycolysis and oxidative metabolism. In the final step, anaerobic glycolysis is characterised by the reduction of pyruvate into lactate

with NADH being oxidized to NAD^+. Although ATP resynthesis is very fast via this pathway, lactate accumulates and this has been associated with early local muscular fatigue (Ferris, 1969; Hermansen and Osnes, 1972; Keul, 1973; Margaria, 1972; Osnes and Hermansen, 1972; Wenger and Reed, 1976). Nevertheless, in sports such as wrestling, hockey and long sprint-races, anaerobic glycolysis is the main energy production pathway (Astrand and Rodahl, 1970; Keul et al., 1972).

The pyruvate to lactate reaction is catalysed and may be regulated by lactate dehydrogenase (LDH, E.C. 1.1.1.27). LDH is composed of two subunits: a muscle specific (M) and a heart specific (H) type, which are combined in a tetramer molecule giving five isozymes (H_4, H_3M, H_2M_2, HM_3, M_4) with different properties (Cahn et al., 1962; Dawson et al., 1964; Dietz and Lubrano, 1967; Everse and Kaplan, 1970 and 1973; Kaplan, 1960-1962; Plageman et al., 1960a and b; Stambaught and Post, 1966a and b). M - LDH favours the reduction of pyruvate whereas H - LDH favours the oxidation of lactate.

Finally, the ATP resynthesis can occur via the oxidation of fat or carbohydrate as acetyl CoA via the citrate cycle and the electron transport chain. The aerobic energy production is the common energy source for most cellular activities in mammalian tissue, including those affected by physical exercises of low to moderate intensities.

Justification for the Study

Most physical activities can be grossly classified as one of the two following types:

1. High intensity, short duration or anaerobic*
2. Low to moderate intensity, long duration or aerobic

Sport performance is usually based on either one or both of these types of exercise. Therefore the importance of understanding the possible mechanisms of adaptations in either type of performance is obvious.

Adaptations to aerobic exercise are well documented. Maximal oxygen uptake (Astrand and Rodahl, 1970; Karlsson et al., 1967; Saltin et al., 1968), maximal cardiac output (Ekblom et al., 1968; Saltin et al., 1968), and the activities of certain oxidative enzymes (Baldwin et al., 1972; Barnard and Peter, 1971; Benzi et al., 1975; Fitts et al., 1975; Gollnick et al., 1969, 1970; Holloszy, 1967 and 1971) are all increased with prolonged exercise training executed at an intensity of greater than 50% of the maximal oxygen uptake. Cardiovascular adjustments (Bevegard and Shepherd, 1967; Dempsey and Rankin, 1967; Rowell, 1969, 1974) and

---

* Anaerobic exercises are those based on ATP synthesis that do not use O_2 as the final electron carrier. In other words, the anaerobic metabolism includes the adenylate kinase, the creatine phosphokinase and the lactate dehydrogenase reactions as well as the ATP already stored and ready to be used by the muscle.

substrate mobilisation and/or interaction (Ahlborg, 1967; Felig and Wahren, 1975; Fredholm, 1970; Horstman et al., 1971; Issekutz et al., 1966; Jorfeldt, 1971; Jorfeldt and Wahren, 1970; Pruett, 1970; Wahren, 1970; Weil et al., 1965) in acute exercise are also well described in the literature.

On the other hand, adaptation to anaerobic exercise and/or adaptation of the anaerobic parameters to exercise is less well documented. With the exception of blood lactate response (Issekutz et al., 1965, 1966; Jorfeldt, 1971; Margaria et al., 1933, 1968, 1972), and plasma enzyme response to exercise (Bloor, 1969; Doty et al., 1971; Fowler et al., 1962, 1968; Garbus et al., 1964; Haralambie, 1972; Hunter et al., 1971; Papadopoulos et al., 1968; Raven et al., 1970; Rose et al., 1970 a and b), few studies on the adaptation of anaerobic parameters to exercise, particularly those using anaerobic exercise, exist.

Glycogen stores have been shown to restrict long lasting muscular effort (Hultman, 1967; Piehl, 1974; Saltin et al., 1973; Taylor et al., 1971). A specific depletion pattern in ST and FT fibers with aerobic and anaerobic exercise does occur (Gollnick et al., 1973 a ; Piehl, 1974; Saltin et al., 1973). Phosphorylase (Baldwin et al., 1973; Bylund et al., 1977; Edgerton et al., 1970; Gould and Rawlinson, 1959; Holloszy et al., 1971; Kowalski et al., 1969; Morgan et al., 1971; Saubert et al., 1973; Taylor et al., 1972; Zika et al., 1971), hexokinase (Baldwin et al., 1973,

1977; Barnard and Peter, 1969; Bylund et al., 1977; Holloszy et al., 1971; Suominen and Kaikkinen, 1975), phosphosfructokinase (Baldwin et al., 1973, 1977; Gollnick et al., 1973; Henricksson and Reitman, 1976; Holloszy et al., 1971; Morgan et al., 1971; Saubert et al., 1973; Thorstensson et al., 1976), and pyruvate kinase (Baldwin et al., 1973; Bostrom et al., 1974; Holloszy et al., 1971; Morgan et al., 1971; Saubert et al., 1973) activities in muscles have been shown to increase or remain stable with different types of exercise training. Many of the previously cited authors have used these parameters as indicators of anaerobic adaptation. The above mentioned enzymes (e.g. PH, PFK, HK and PK) are solicited whether pyruvate is converted to lactate or is oxidized through the citric acid cycle and the electron transport chain. On the other hand, LDH, CPK and AK activities as well as ATP and PC stores appear to contribute to non-oxidative energy production without being involved in aerobic breakdown of fuel. LDH is often seen as a regulatory enzymes of anaerobic metabolism (Everse and Kaplan, 1973; Fritz, 1965; Karlsson et al., 1974; Sjodin, 1976).

Training may induce an increase in the high energy compounds of the heart (Gangloff et al., 1961) and skeletal muscles (Ericksson et al., 1973; Gale and Nagle, 1971; Haralambie, 1972; Rogozkin, 1976), but the literature is not conclusive (Gale and Nagle, 1971; Haralambie, 1972; Karlsson

et al., 1972; Saltin and Karlsson, 1971; Thorstensson et al., 1975). There may be an optimal form of exercise that leads to increased stores of ATP and PC, but this optimal form of exercise has not yet been determined. Age might also be at the origin of some reported increases (Ericksson et al., 1973).

Very few studies have dealt with the response of CPK activity to training. Some have reported increases in tissue CPK (Kendrick-Jones and Perry, 1965; Wagner and Critz, 1970) and others have noted no changes with training (Bohmer, 1969, Gangloff et al., 1961; Oscai and Holloszy, 1971; Rawlinson and Gould, 1959; Thorstensson and Karlsson, 1974). Newsholme and Start (1973, p. 113) believe that CPK may amplify the control of glycolysis when coupled with the ATPase reaction resulting in an increase in inorganic phosphate which stimulates PFK activity.

Studies showing an increase in AK activity with chronic exercise (Collowick as quoted by Kendrick-Jones and Perry, 1965; Thorstensson and Karlsson, 1974) have been reported. On the other hand, others have observed no change with training (Oscai and Holloszy, 1971). The AK reaction appears to be involved in the control of glycolysis via PFK: the small and transient decrease in ATP must be accompanied by a relatively greater increase in AMP to stimulate PFK (Newsholme and Start, 1973, p. 113).

Skeletal muscle LDH activities have been shown to remain constant (Bohmer, 1969; Gollnick et al., 1967; Hickson et al., 1976; Holloszy, 1971; Molé et al., 1973; Peter, 1970) or to decrease with training in animals (Baldwin et al., 1972, 1973; Hickson et al., 1976; York et al., 1974). In the heart, LDH activity is usually increased with training (Gollnick et al., 1961 and 1967; Walpurger and Anger, 1970; York et al., 1975 and 1976). The literature is equivocal with regard to the response to training of the LDH subunits or isoenzymes. Some authors have reported no change (Molé et al., 1973) while others have indicated increases in the H or the M type of LDH depending upon whether the training was primarily aerobic or anaerobic in nature (Karlsson et al., 1975; Peter et al., 1970 and 1971; Sjodin, 1976).

The conflicting literature may be a reflection of the fact that most studies have been looking at the effects of endurance training (aerobic type usually) on aerobic and anaerobic parameters and that few studies have looked at the specific effects of both aerobic and anaerobic training on the anaerobic parameters. The physiological age of the subjects during the training regimens and the choice of sampled tissues may have added to the confusion. Finally, the assay techniques used in many studies, particularly those investigating the LDH and high energy compound response to training, might be at the origin of some of the discrepancies reported in the literature. In some cases,

the discrepancies are unexplained, a fact which further justifies the reinvestigation of these parameters.

The Problem

The purpose of this study was to determine the comparative effects of chronic, moderately intense, continuous running and high speed interval running on the following: ATP and PC stores, AK, CPK and LDH activities and LDH subunit distribution in heart (H), liver (L) and resting skeletal muscles, namely, soleus (S), medial and lateral gastrocnemius (GM , GL), plantaris (P) and tibialis anterior (TA).

Limitations and Delimitations of the Study

1. The study was confined to Sprague Dawley rats,
2. The training regimens started at 6 weeks of age and lasted 6 months, covering both the growth and adult periods,
3. The two long-term training regimens were intended to primarily overload the aerobic and anaerobic systems separately,
4. The physical activity used in the training regimen was limited to running on a motor-driven treadmill,
5. Sampled tissues were limited to the heart as an index of cardiovascular involvement, the liver as the locus of many biochemical substrate transformations and several skeletal muscles of the lower limbs to provide a picture of the adaptations of

different muscles involved in the work (agonist vs antagonist, slow twitch vs fast twitch),

6. Biochemical assays were conducted in vitro on whole muscle homogenates rather than using muscle fiber type as an index. However, the fiber composition of the chosen muscles was identified (Table 20 in Discussion),
7. Although the training programs used in this study have been shown to induce in situ improvement of the fatigue curve (Barnard and Peter, 1971) and of the oxidative capacity of the muscles (Barnard and Peter, 1971; Fitts et al., 1975; Gollnick et al., 1970), this was not verified in the present study. The only common measures used to check the effectiveness of the training regimens were body and organ weights,
8. Except for LDH, no glycolytic, glycogenolytic, or oxidative substrate or enzyme activities were measured.

CHAPTER II

REVIEW OF LITERATURE

Lactate Dehydrogenase

LDH (E.C. 1.1.1.27) catalyses the following reaction:

$$\text{pyruvate} + \text{NADH} + \text{H}^+ \xrightleftharpoons{\text{LDH}} \text{lactate} + \text{NAD}^+$$

This reaction is the last and only specific step of anaerobic glycolysis. LDH is composed of two subunits: a muscle specific (M) and a heart specific (H) type. M and H subunits are combined in a tetramer molecule giving five isoenzymes:* H_4, H_3M, H_2M_2, HM_3 and M_4 which are also respectively identified as LDH-1 to LDH-5. When isolated with electrophoresis, LDH-1 is the fastest moving band toward the anode (Dietz and Lubrano, 1967; Everse and Kaplan, 1973; Plageman et al., 1960a and b). Figure 2 from Rosalki (1969) illustrates the classical separation of LDH isoenzymes in serum and tissues. Isoenzyme H_4, found in highly aerobic tissue, has a relatively low turnover number with pyruvate and is maximally active only at low concentrations of this substrate, which strongly inhibits the enzyme at higher concentrations. On the other hand, in relatively more anaerobic tissues, isoenzyme M_4 favors the pyruvate to lactate reduction for

---

* "Isozyme" is also used in the literature, but the term "isoenzyme" is recommended by the Standing Committee on Enzyme of the International Union of Biochemistry (Wilkinson, 1970).

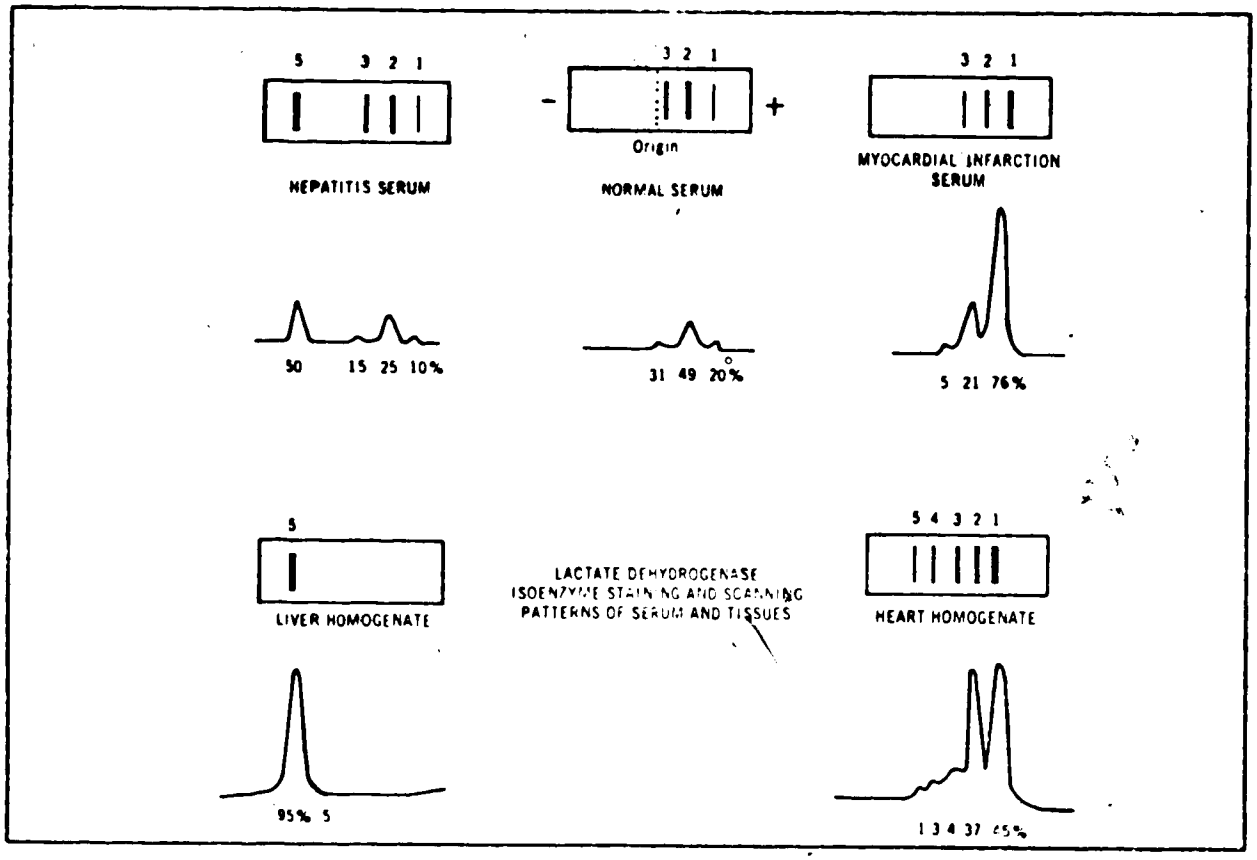

FIGURE 2. GENERAL PATTERN OF LDH ISOENZYMES (FROM ROSALKI, 1969)

immediate release of energy. Between H_4 and M_4, hybrid isoenzymes are found with proportional intermediate characteristics (Everse and Kaplan, 1973; Kaplan et al., 1961; Karlsson et al., 1974; Rosalki, 1969; Sjodin, 1976).

The M and H composition of LDH appears to be controlled by two separate and independent genes. The synthesis of M subunits is stimulated during hypoxic conditions and suppressed with high O_2 tension, while the reverse is true for the H subunits (Cahn et al., 1962; Dawson et al., 1964; Everse and Kaplan, 1973; Karsten et al., 1973; Latner and Skillen, 1968, p. 85; Thorling and Jensen, 1966). Embryologic development of M and H LDH differentiation does exist toward their future functional role. Foetal rabbit gastrocnemius appears to develop the heart muscle enzyme first, whereas the skeletal type develops postnatally (Dawson et al., 1964). Brain LDH isoenzyme patterns show more M-LDH for humans living at high altitude (Hellung et al., 1973). Muscles which contract tonically* or rythmically have relatively more H-LDH, while muscles which contract phasically have more M-LDH (Dawson et al., 1964). Function seems to be more important than tissue type, e.g. migrating birds have more H-LDH in their breast muscle than domestic birds (Wilson et al., 1963).

---

* Posture and anti-gravimetric muscles (e.g. soleus, flying muscles) and the heart contract tonically and rythmically, whereas other muscles that are used only occasionally are said to be phasic muscles. This functional classification corresponds to the fast twitch (phasic) and slow twitch (tonic) muscles.

Other kinds of adaptations have been reported. For instance, hormonal or vitamin treatment (Acebal et al., 1974; Beitner et al., 1973; Dawson et al., 1964; Garbus et al., 1964; Hirota et al., 1976), sideropenic anemia (Penney et al., 1974) and denervation (Dawson et al., 1964) have been shown to induce LDH isoenzyme changes. It is interesting to note that 10 days after sciatic section, only a slight decrease in enzyme activity had occured and it is only after 31 days that M-LDH decreased in rabbit leg muscles. Such adaptations may be tied to the disappearance of the anaerobic stimuli. Duration is obviously an important factor to control when studying isoenzyme adaptation. Intermittent long-term stimulation (8 hr/day) of fast rabbit tibialis anterior up to 28 days with a frequency pattern resembling that of a slow muscle (10 impulses/sec) resulted in a decrease of total LDH activity with concomitant decrease in M-LDH % (Pette et al., 1973).

The Aerobic-Anaerobic Theory. According to Kaplan's group (Cahn et al., 1962; Dawson et al., 1964; Everse and Kaplan, 1973; Everse et al., 1970) and to Pfleiderer's group (Pfleiderer and Wachsmuch, 1961, as quoted by Latner and Spillen, 1968, p. 80 and Thorling and Jensen, 1966), LDH of the H type (heart) has evolved to operate as a regulator of pyruvate metabolism in highly aerobic cells and functions as a lactate dehydrogenase because it is inhibited by high pyruvate concentration; LDH of the M type (skeletal muscle

and liver) has evolved to prevent this inhibition in anaerobic tissue and operates as a pyruvate reductase. Wuntch, Vesell and Chen (1970a), Vesell and Pool (1966) and Amarasingham and Uong (1968), have contested this theory and suggested other functions than metabolic ones for LDH isoenzymes. First, they pointed out that liver with its high M-LDH content is a highly aerobic tissue which is, however, in opposition to Pfleiderer's findings (Latner and Spillen, 1968, p. 80). Second, they were unable, at high LDH concentrations similar to cellular concentrations ($7x10^{-6}M$) to show the classical inhibition of H-LDH to high pyruvate concentration obtained with the usual hundred fold dilution of homogenate procedure. They suggested other metabolic roles for LDH isoenzymes: 1) a regulatory function for LDH-5 M_4 considered as an allosteric protein, 2) an association of isoenzymes in different proportions with various subcellular particles, in particular LDH-5 within the nucleus, 3) predominance of LDH-5 in rapidly dividing cell or in tissue capable of rapid cell proliferation, and 4) a conservative metabolic role in which one isoenzyme would be required to maintain critical enzymatic function in a tissue where another isoenzyme was rapidly degraded.

Latner et al., (1966) were unable to reproduce the above experiment. Similarly, Everse, Berger and Kaplan (1970, 1973) and Stambaugh and Post (1966) obtained LDH-1 usual inhibition at high enzyme concentrations with high

pyruvate concentrations. According to them, inhibition is due to the formation of an abortive ternary complex between pyruvate, nicotinamide-adenine dinucleotide (NAD^+) and the LDH. They obtained different results because the pyruvate substrate was added to the LDH-NADH solution in the presence of NAD^+ whereas the other groups (Amarasingham and Uong, 1968; Vesell and Pool, 1966; Wuntch et al., 1970a) excluded the NAD^+ resulting in the LDH reaction without inhibition because the fast reaction occured before any ternary complex was formed.

Wuntch, Chen and Vesell (1970b) further stated that the inhibition in vivo is also diminished with competition by other enzymes (glyceraldehyde -3- phosphate dehydrogenase, malate dehydrogenase) or albumin with NAD^+. These authors (Vesell and Pool, 1966 and Wuntch et al., 1970b) noted that pyruvate in vivo never reaches levels high enough to inhibit LDH and that NAD^+ is probably more determinant of the LDH reaction.

However, Kaplan and collaborators (Everse and Kaplan, 1973) still believed that the binding of NAD^+ to other proteins in vivo, resulting in a net decrease in the concentration of the free NAD^+, does not affect the concentration of free NAD^+ in the cell to such an extent that formation of the abortive LDH complex is no longer feasible. They also added that the results obtained by Vesell and his co-workers could be due to the limiting amount of NAD^+ that

was used in their experiments (14:0 μM). Another possible explanation for these discrepancies may be that a significant part of H-LDH is present in an inactive form in the fresh tissue extracts (Everse and Kaplan, 1973).

Stambaugh and Post (1966a) reported that H-LDH inhibition in the forward reaction is more a matter of product (i.e. lactate) inhibition than substrate (i.e. pyruvate) inhibition with in vivo concentrations. Karlsson (1971a) has shown that muscle pyruvate concentration increased from 0.06 at rest to 0.13 mmoles/kg wet weight at maximal work load; even maximal pyruvate concentration found in vivo is about 10 times lower than that required to produce the substrate inhibition reported by Kaplan's group (Everse and Kaplan, 1973; Kaplan et al., 1960, 1962, 1968). On the other hand, Everse et al., (1970) did show substrate inhibition at physiological enzyme concentrations. The possibility that the intracellular concentration of the pyruvate at the actual locations of the isoenzymes may reach inhibitory levels could not be excluded but was not considered likely (Vesell and Pool, 1966; Wuntch et al., 1970b). Although it seems that there exists no study on LDH inhibition with in vivo concentrations for all the reagents, most authors believe in some kind of inhibition in vivo (Everse et al., 1970; Everse and Kaplan, 1973; Karlsson et al., 1974b; Latner and Skillen, 1968; Sjodin, 1976; Stambaugh and Post, 1966).

To understand the functional role of LDH isoenzymes, one must realize that pyruvate, lactate, NADH and NAD^+ compete together to bind with LDH and either form the ternary abortive complex or the ternary complex that catalyses the reaction. It seems that pyruvate/lactate and NAD^+/NADH ratios are more important than the absolute concentrations of these substrates and products.

Table 2, reproduced from Everse and Kaplan (1973), illustrates the probable role of H-LDH with different NAD^+/NADH and PA/LA ratios. H-LDH appears to be under metabolic control and is regulated by its own oxidized products as well as the oxidized coenzymes. Therefore, in normal concentration, H-LDH is prevented from reducing pyruvate to lactate. Lactate can be produced by the heart during ischemia and myocardial infarction due to the lack of oxygen supply and the NADH increase which displaces the abortive complex with H-LDH and favors the reduction of pyruvate. Reactions 2 and 3 (Table 1) appear to be geared to assure an optimal concentration of reduced coenzyme under various physiological conditions.

The aerobic-anaerobic theory is in accordance with isoenzyme distribution in muscle fibers and cellul[redacted] localizations. M-LDH is more predominant in fast twitch fibers and H-LDH, in slow twitch fibers (Blanchaer an[redacted] 1962; Brody and Engel, 1964; Fine et al., 1963a[redacted]on et al., 1974b; McMillan, 1967; Peter et al., 1971; Sjodin,

TABLE 2 Effects of Substrate and Coenzyme Concentrations on H-LDH in Aerobic Tissues.*

Reaction No.	Concentration Ratios		Action of H-LDH
	NAD^+/NADH	Pyruvate/Lactate	
1	high	high	Formation of E-NAD^+-pyruvate abortive complex**
2	high	low	Oxidation of lactate (E-NAD^+-lactate)
3	low	high	Reduction of pyruvate (dissociation of E-NAD^+-pyruvate complex as in myocardial infarction (E-NADH-pyruvate)
4	low	low	Formation of E-NADH-lactate complex

* From Everse and Kaplan, 1973.
** E = Enzyme (H-LDH)

1976a; Van Wijhe et al., 1964). The higher LDH activity in fast twitch fibers, as demonstrated biochemically or histochemically (Karlsson et al., 1974b; McMillan, 1967; Meijer, 1973; Peter et al., 1971; Sjodin, 1976a), seems to be related to higher M-LDH content, although this was not the case for endurance trained athletes (Karlsson et al., 1975) due to the absence of a linear relation between total LDH and ST fibers (Karlsson et al., 1975). It is interesting to note that most LDH is located in the sarcoplasm (Brody and Engel, 1964; Sjodin, 1976a), but LDH isoenzymes with predominant H subunits are also located in mitochondria, and LDH isoenzymes mainly composed of M subunits have a strong binding affinity for membranes, e.g. sarcoplasmic reticulum or external mitochondrial membrane (Brody and Engel, 1964; Sjodin, 1976) or other subcellular particulate fractions (Ratner et al., 1974). Specific LDH isoenzyme compartmentalization within subcellular units may increase the difficulty with which NADH reaches LDH and may be related to or explain some physiological function attributed to LDH isoenzymes (Ratner et al., 1974; Sjodin, 1976a).

LDH as a Regulatory Enzyme of Glycolysis. As shown previously, LDH may play an important role in controlling the amount of lactate produced and in oxidizing NADH accumulated in the cell. But it seems that this is not under the exclusive control of LDH. According to Boxer and Devlin (1961) and Keul et al. (1972, pp. 14, 87, 131), two other shuttle reactions can assure the oxidation of NADH. First,

dihydroxyacetone-P is reduced to alpha-glycerophosphate in the sarcoplasm with the glycerophosphate dehydrogenase; the cycle is closed in the mitochondria by the reverse reaction. Second, acetoacetate is reduced to beta-hydroxybutyrate in a similar way. Like lactate, alpha-glycerophosphate increases in anaerobic states, but it is not a "dead end" product like lactate and its significance in terms of fatigue is unknown.

Actual evidence does not suggest, however, that endurance training preferentially accentuates the glycerophosphate dehydrogenase compared to the LDH since changes were of the same order of magnitude and in the same direction for both enzymes (Baldwin et al., 1973; Morgan et al., 1971). Baldwin et al. (1973) reported a decrease from 4 (n.s.) to 27% ($P < 0.02$) and 15 ($P < 0.05$) to 23.3% ($P < 0.001$) for the alpha-glycerophosphate dehydrogenase and the LDH respectively in the red and white quadriceps of the rat; in the soleus alpha-glycerophosphate dehydrogenase and LDH increased respectively by 54.7% ($P < 0.001$) and 12% (n.s.). Morgan et al. (1971) reported non significant decreases of 26.9% and 22% for alpha-glycerophosphate dehydrogenase and LDH of human quadriceps after endurance training. Holloszy and Oscai (1969) had previously found similar results. In rat heart, Kraus (1971) reported a 85% increase either with a strenuous swimming program and a voluntary running program. Staudte et al. (1973) reported no change in either LDH or

alpha-glycerophosphate dehydrogenase after sprint training in the rat. According to Boxer and Devlin (1961), the NADH shuttles can work only in aerobiosis or partial anaerobiosis since they are based on citric acid cycle intermediates. As the direct oxidation of cytoplasm formed NADH in the mitochondria is not possible, only pyruvate can oxidize NADH in anaerobiosis.

Concerning the regulation of LDH itself, it seems that the mass action law is not sufficient to explain muscle lactate formation since pyruvate and lactate do not increase at the same rate in exercise (Karlsson, 1971a; Keul et al., 1967 and 1972). According to Fritz (1965), M_4-LDH but not H_4-LDH behaves like an allosteric enzyme and is also activated by the seven citric acid cycle intermediates as well as by aspartic and glutamic acids which are directly converted to citric acid cycle intermediates. Although there was a significant increase (50%) in malic enzyme (L-malate: NADP oxidoreductase, E.C. 1.1.1.40) after endurance training in the rat (Molé et al., 1973), the very low absolute levels of this enzyme do not seem to play an important role in the pyruvate metabolism (e.g. lipogenesis) in skeletal muscle (Molé et al., 1973).

According to Felig and Wahren (1971, 1973 and 1975), another pathway, pyruvate conversion to alanine, may interact with the LDH reaction, decreasing the pyruvate*

* There are other alternative fates for pyruvate (Malher and Cordes, 1966, p. 435), but their importance and functional role in exercise is presently unknown.

available for oxidation of NADH via anaerobic glycolysis. According to these authors, the alanine pathway is a non-toxic alternative to ammonia in the transport of amino groups from the periphery to the liver, where alanine is converted back to glucose. The lower muscle or blood lactate levels observed in submaximal exercise in trained individuals (Astrand and Rodahl, 1970, p. 379; Karlsson, 1968, 1971a; Molé et al., 1973; Robinson et al., 1941) may be explained by an increase in glutamate pyruvate transaminase (GPT, E.C. 2.6.1.2.) and more pyruvate being converted to alanine (Felig and Wahren, 1971, 1975; Molé et al., 1973).

However, at maximal and supra-maximal work loads (e.g. $\dot{V}O_2$max or maximal voluntary contraction), trained subjects have higher lactate accumulation (Astrand and Rodahl, 1970, p. 379; Ericksson et al., 1973; Molé et al., 1973; Robinson et al., 1941), suggesting a greater contribution of LDH and probably of M-LDH. Reciprocal behaviour in lactate formation at low and medium work load compared to high work load appears to be ruled by the inhibition of lactate and free fatty acids on each other at these work loads (Issekutz et al., 1965, 1966; Felig and Wahren, 1975; Fredholm, 1970; Horstman et al., 1971; Molé et al., 1973; Weil et al., 1965).

Lactate can be used as substrate by the heart (Everse et al., 1973; Keul, 1971, 1973), the liver (Keul, 1973; Rowell, 1966, 1971) and more or less by the skeletal muscles (Issekutz et al., 1965; Jorfeldt, 1971; Keul, 1971; Felig and Wahren, 1975). Lactate diffuses from the organs where it is produced (mainly the skeletal muscle) to the blood (Jorfeldt, 1971; Keul, 1967, 1971, 1973a and b; Margaria, 1968, 1972). When there is a sudden rise in anaerobic metabolism, a delay is observed before equilibrium is reached between the blood and the muscle. The "peak" blood lactate is usually reached between 3 to 10 minutes after exercise (Karlsson, 1971a; Margaria, 1968, 1972). Blood and muscle lactate concentrations are also dependant on the equilibrium between uptake and production. The peak lactate in the blood, although smaller than the muscle concentration, is always representative of the muscle concentration (Karlsson, 1971a). It is worthwhile to note that blood lactate reflects an average situation for all the muscles of the body taken together, even though lactate may be found in different amounts in different muscles or even in different muscle fibers (Essen and Haggmark, 1975) where possible product inhibition can occur (Karlsson et al., 1971, 1975; Sjodin, 1976a and b).

High lactate concentrations have been associated with exhaustion in exercise of high intensity (Ahlborg, 1972; Karlsson, 1971a; Keul, 1973, Margaria, 1972, 1968). Lactate may be either a cause or consequence of fatigue, but it has

been suggested (Ferris, 1969) that the lactate anion itself is related to anxiety and other similar symptoms. Others, (Hermansen and Osnes, 1972; Keul, 1973; Osnes and Hermansen, 1972) suggested that the increase in acidity linked with lactate production may be the cause of fatigue. There seems to be a good correlation between lactate concentration and exhaustion feelings, at least in short lasting-high intensity work. According to Margaria (1968, 1972) and Keul (1973), anaerobic glycolysis has the second fastest energy production rate after the immediate utilization of high energy phosphate stores. Therefore, when less intense work is done, energy can be produced via oxidative pathways and exercise can be performed longer before exhaustion is reached. With prolonged exercise, however, lactate concentration and production is low and cannot be related to exhaustion (Karlsson, 1971a; Keul, 1973).

Lactate is produced and accumulates when the energy demand is greater than the energy that can be produced with aerobic metabolism. In supra-maximal exercise, lactate is produced to a greater extent than it is taken up and this is reflected by the greater accumulation of lactate in the muscle and the blood. At low work loads (30% $\dot{V}O_2max$), lactate is probably produced but does not accumulate (Di Prampero et al., 1976; Margaria, 1968, 1972). At medium intensity (30-60% $\dot{V}O_2max$), which can be sustained for a long time, lactate increases at the beginning but returns

to resting levels with time (Jorfeldt, 1971; Felig and Wahren, 1973; Karlsson et al., 1968; Keul et al., 1972). Even maximal work after prolonged exercise does not produce the usual lactate rise (Astrand, 1963).

To explain the sudden rise of lactate at the beginning of medium work load, Jorfeldt (1971) and Felig and Wahren (1975) suggested a net release of lactate from white fibers which are mainly composed of M-LDH. When the delay due to circulatory adjustments is finished, the red fibers are able to meet the energy demand aerobically and assume the responsibility of muscle contraction, perhaps using lactate as a substrate. It is known that during prolonged exercise, glycogen depletion as measured at 20, 60, 120 and 180 mm of exercise, first occurs in ST fibers but as the exercise progresses, the FT fibers are also depleted (Gollnick et al., 1973d). But this fiber specific depletion pattern could not be observed before 60 min of exercise and is not necessarily in contrast with the early transient lactate rise that was observed at the 20 min mark (Gollnick et al., 1973d). During repeated 1 min sprints of high intensity interspaced with 10 min of rest, glycogen is first depleted in FT fibers as opposed to the glycogen depletion pattern of prolonged exercise (Gollnick et al., 1973a). Others (Baldwin et al., 1973; Piehl, 1974) have reported the possibility of a selective recruitment pattern of different fiber types at the onset of work and with various types of

exercise. Essen and Haggmark (1975) recently measured lactate in single muscle fibers* and in pooled muscle fibers of the same type in exercises known to result in lactate formation. With single fiber measurements, they found great variations in post exercise lactate, both for type I and type II fibers reflecting a selective fiber pattern. Lactate from pooled or single fibers was higher in some cases for type II fibers.** The exercise stimuli used in this study were bicycle work at 100% VO_2max and static contraction at 50% of MVC which, if the selective recruitment theory is good, should preferentially involve fast twitch or type II fibers. It would be interesting to have a similar study with exercise of medium intensity where lactate is known to increase at first and to decrease thereafter.

Isoenzyme Pattern Modifications. The crucial and central point of glycolytic regulation by LDH is the LDH subunit or isoenzyme pattern and its possible modification with physiological demand (e.g. training). According to Millar (1974), lactate dehydrogenase must be in an "activated state" to hybridize in vivo. Simple dissociation

---

* Single muscle fibers were dissected from freeze-dried samples and the ends cut off for identification with myosin ATPase reaction. Single fibers were placed directly in a fluorometric tube for analysis. Pooled fibers (25-50 fibers of the same type) were first digested in HCL and the supernatant analysed conventionally.

** No statistical analyses were made in this preliminary study.

to dimers is not enough for hybridization. In vivo, therefore, an environment must be created in which the enzyme is not membrane bound, in which the anti-hybridizing effects of substrate, coenzyme and protective ions are prevented from occurring and in which "activation" can take place. These are highly restrictive conditions. The possibility exists that, in vivo, activating agents are present which negate the influence of the inhibitors and accelerate hybridization.

Newly and preferentially synthesized LDH subunits appear to be the route by which isoenzyme patterns are modified. The half-life of LDH may be as long as 31 days in rat skeletal muscle and somewhat shorter in the liver and the heart (Fritz et al., 1969). Opposite trends have been shown for the time of occurrence of peak specific radioactivities (Don and Master, 1975; Fritz et al., 1969). Heart M_4 has been shown to possess a much shorter half-life than any other isoenzyme in the heart, the liver or the skeletal muscle of the rat (Fritz et al., 1973).

Acute Exercise and LDH

In serum, LDH and more specifically M-LDH may temporarily increase by as much as 400% in trained and untrained humans and animals if the relative intensity and duration of the work load are sufficient (Block et al., 1969; Bloor and Papadopoulos, 1969; Doty et al., 1971; Fowler et al., 1962; Garbus et al., 1964; Hallonen and Konttinen, 1962;

Haralambie, 1972; Hunter and Critz, 1971; Novosadova, 1969; Papadopoulos et al., 1968; Raven et al., 1970; Rose et al., 1970a and b; Schmidt and Schmidt, 1969; Siest and Galteau, 1974; Wolfson et al., 1972). Many mechanisms, including cellular necrosis, membrane disruption, increased permeability due to hypoxia or to increased circulating catecholamines, increased blood flow, carriers, etc... have been proposed to explain the increased release of LDH and M-LDH from tissue to serum (Atland and Highman, 1961; Doty et al., 1971; Garbus et al., 1964; Highman and Altland, 1963; Karlsson et al., 1968; Raven et al., 1970; Sanders and Bloor, 1975; Schmidt and Schmidt, 1969; Siest and Galteau, 1975; York et al., 1976). Such a release from tissue must rely on some assumptions. For instance, tissue LDH either maintains the same activity but its concentration is decreasing as a result of cellular leakage to serum or increases its activity or its concentration with a concomitant increase in serum. Cellular leakage is not directly related to LDH activity itself and thus does not seem to be related to a possible training effect in tissue LDH. Such a decrease in rat tissue LDH after acute exercise has been reported by Doty et al. (1971). Novosadova (1969) reported that heart, liver and skeletal muscle LDH decrease in trained and untrained rats with acute exercise but serum LDH, paradoxically, increased in untrained rats and decreased in trained rats. Other trends have however been reported for tissue LDH after acute exercise. Garbus et al. (1964) observed

no consistent changes. Gollnick et al. (1967) reported no significant change in LDH activity of rat heart or skeletal muscle after acute exercise. Finally, Karlsson et al. (1968) reported a significant increase in human muscle LDH after prolonged exercise. The reasons for these different findings are unclear at the present time. Karlsson et al. (1968) believed that the increase in muscle LDH was due to an increased enzyme concentration and possibly to a change in the Michaelis constant. Due to the long half-lives of LDH (Fritz et al., 1969, 1973), it seems that such changes must result from a transient change in activity (inhibition or facilitation). This reported increase in human muscle LDH after strenuous prolonged exercise appears to be paradoxical and is unique in the literature. It seems paradoxical because 1) such prolonged exercise is known to yield low lactate levels (Astrand et al., 1963; Jorfeldt, 1971; Felig and Wahren, 1973; Karlsson et al., 1968; Keul et al., 1972), 2) LDH activity is well above the maximal rate of lactate formation in vivo (Karlsson et al., 1968), 3) training with prolonged exercise does not increase human skeletal muscle LDH (Bylund et al., 1977; Morgan et al., 1971; Sjodin et al., 1976a and b), 4) and all other human and animal studies showed similar increases in serum LDH after acute exercise and all other animal studies* either showed a decrease or no change in tissue LDH after acute exercise. Species

---

* A recent preliminary study (Sjodin, 1976a) indicated that LDH and M-LDH did not change in human muscle after a 100 km race.

differences are not excluded. In this regard, it is interesting to note that in rat serum about 80% of the LDH is present as M-LDH (Bloor and Papadopoulos, 1969; Garbus et al, 1964; Raven et al., 1970) as compared to 20-30% for human serum (Barengo and Itoiz, 1972; Block et al., 1969; Dietz and Lubrano, 1967; Rose et al., 1970). Acute effects of exercise on LDH appear to be transitory and quite independant of chronic effects of exercise. Thus the acute effects of exercise on LDH are not a very useful aid to understanding the chronic effects of exercise.

Chronic exercise and LDH

Although the relationship between serum and tissue LDH is puzzling, training does effectively reduce the LDH rise in serum at a particular submaximal work load (Bloor and Papadopoulos, 1969; Garbus et al., 1964; Hunter et al., 1971; Novosadova, 1969; Papadopoulos et al., 1968; Rose et al., 1970a; Wolfson et al., 1972) and at rest (Hallonen and Konttinen, 1962). Another study has reported, however, a mild but significant increase in resting serum after training (Hunter and Critz, 1971).

In tissues, LDH changes appear to be related to the type of training as well as to the type of tissue. Endurance training, either running or swimming, appears to increase the LDH activity of the heart (per mg of N_2 or mg of fresh tissue) by 10 to 30% (Gollnick et al., 1961, 1967; Walpurger and Anger, 1970; York et al.,

1975 and 1976) and more specifically, the M-LDH* activity by 22 to 30% as well as the M-LDH% by 3 to 5% (Peter, 1970; York et al., 1975 and 1976). The LDH increase in the heart may be a function of the intensity and duration of training as well as of the age at which the training regimen was started (York et al., 1975 and 1976). Two other studies (Peter, 1970; Ruhling et al., 1973) reported nonsignificant changes in total LDH activity of the heart after endurance training.

In skeletal muscles, LDH adaptation to endurance exercise appears more complex and more confusing. Generally, fast twitch skeletal muscle of endurance trained athletes have 30 to 68% less LDH activity than sedentary subjects (Costill et al., 1976; Karlsson et al., 1975; Suominen and Heikkinen, 1975). Genetic endowment may explain these differences since none of the endurance training studies in humans showed significant changes in mixed skeletal muscle (Bylund et al., 1977; Morgan et al., 1971; Sjodin et al., 1976a and b). In each case studied, however, there was a decrease of 6 to 22%. Most training studies in animals also showed decreases of 7 to 58% in fast twitch skeletal muscles. These trends were often non significant in the mixed FG and FOG gastrocnemius and plantaris (Böhmer, 1969;

---

* In many cases, M-LDH activity and M-LDH% have been estimated from isoenzyme activities or % using the following formula:

$$\text{M-LDH} = 0.25\ H_3M + 0.5\ H_2M_2 + 0.75\ HM_3 + M_4$$

Gollnick et al., 1967; Hickson et al., 1976; Holloszy, 1971; Molé et al., 1973) and sometimes significant in FG muscles (Baldwin et al., 1972, 1973; Hickson et al., 1976; York et al., 1974). Two studies reported no change (Gollnick and Hearn, 1961; Gould and Rawlinson, 1959) and another reported a 39% nonsignificant increase in rat biceps brachii with tonic training (Zika et al., 1973). In slow twitch muscle, like the soleus, there may be an opposite trend with an increase of about 12% in LDH activity after endurance training on a treadmill (Baldwin et al., 1972b and 1973). Hickson et al. (1976) have, however, reported a small but significant decrease in soleus LDH using a running wheel device. It was also found that FOG muscles had a greater decrease in LDH activity than FG muscles (Baldwin et al., 1973; York et al., 1974).

The effects of sprint training on tissue LDH are less well documented and appear even more confusing than the effects of endurance training. Fast twitch skeletal muscles of sprint and strength trained athletes have significantly higher LDH and M-LDH activity than endurance trained athletes and sedentary persons (Costill et al., 1976; Karlsson et al., 1975). Although present, none of the increasing trends were significant in sprint training studies dealing with humans (Sjodin et al., 1976b; Thorstenson et al., 1975). Hickson et al. (1976) did report a significant 15-20% decrease with sprint training in fast twitch skeletal muscles of rats whereas Staudte et al. (1973) reported no change

after a 21 day sprint training study. In the heart, Ruhling et al. (1973) found no myocardial LDH change with sprint training. Isometric training that brought exhaustion in 4 to 5 min, 3 times twice a day for 25 days in a row with at least 30 min recovery between each exercise bout, had no effect on LDH activity of fast twitch rectus femoris of female rats but decreased the LDH activity in the soleus (Exner et al., 1973a). In male rats, there were no change in LDH activity of either rectus femoris or soleus after a similar isometric training (Exner et al., 1973b).

To summarize, endurance training appears to decrease LDH and M-LDH activity in fast twitch skeletal muscle, and to increase these activities in the heart and soleus. The changes in LDH are a function of the fiber type composition of the muscles. On the other hand, the few sprint training studies reported either indicate a similar or an opposite LDH behaviour in mixed skeletal muscle of humans and other mammals.

The decrease in LDH of fast twitch skeletal muscle with endurance training could be explained by the increased oxidative capacity of these muscles (Baldwin et al., 1972) and by the greater contribution of aerobic metabolism to energy demand. It has been shown that during endurance training more pyruvate is converted to alanine or is directed toward the citric acid cycle and more fat is oxidized (Felig and Wahren, 1975; Gollnick et al., 1969, 1970, 1972;

Holloszy, 1971; Molé et al., 1973). Therefore, less energy is coming from the LDH reaction, particularly from the M-LDH reaction, and the observed LDH decrease might be a secondary side effect of the increased oxidative capacity. On the other hand, the increase of LDH in the heart, which has been attributed to the most anaerobic subunit (i.e. M-LDH) is surprising, particularly in view of the fact that endurance training increases the utilisation of lactate as a substrate (Keul, 1973a). This heart specific adaptation is not exclusive to LDH, and is consistent with other findings. In this regard, it is interesting to note that Hearn and Gollnick (1961) reported increased ATPase activity in the heart but not in the gastrocnemius of endurance swim-trained rats. Heart muscle also usually shows less oxidative adaptation to endurance training than skeletal muscle (Baldwin et al., 1977a; Holloszy, 1975; Oscai et al., 1971b).

It is known that LDH half-lives were estimated to 1.6, 16 and 31 days in the heart, liver and skeletal muscle respectively (Fritz et al., 1969). In addition, M_4 isoenzyme of the heart has a much shorter half-life than any other isoenzyme of the heart, liver or skeletal muscle (Fritz et al., 1973). These facts are consistent with the heart LDH and M-LDH activity changes, but could not explain the long term adaptation that resulted from a 6 month training regimen.

York et al. (1975 and 1976) have shown that experimen-

tal hypoxia resulted in M-LDH concentrations twice as large as those found after running or swimming, in both ventricles and atria, even though hypertrophy was present only in the right ventricle and atria. Hypoxia appears more important for M-LDH synthesis than hypertrophy and its accompanied cellular and chemical changes. If hypertrophy per se would increase the activities of LDH, as has been hypothesized (York et al., 1975), one would expect relative increases in other glycolytic enzyme activities. To date, this phenomenon has not been demonstrated in the literature (Baldwin et al., 1973; Gollnick and Hermansen, 1973; Saubert et al., 1973; York et al., 1975). Hypertrophy might still explain partially, at least, some of the LDH increases in the heart. In this regard, Walpurger and Anger (1970) reported significant heart LDH increases and hypertrophy only in the running training group and not in the swimming group.*

Therefore, hypoxia appears more important than hypertrophy to explain LDH and M-LDH increases in the heart. Such hypoxic changes would be consistent with the aerobic-anaerobic theory previously described and other reports on the effects of hypoxia on LDH (Dawson et al., 1964; Fox and Reed, 1969; Hellung et al., 1973; Thorling and Jensen, 1966). Since oxygen delivery to the heart might not be limited during moderate sustained exercise (Holloszy, 1973; Rowell, 1974; York et al., 1975 and 1976), York et al. (1975 and

* Baldwin et al. (1972a, 1977a and b) reported that swimming usually resulted in greater heart hypertrophy than running in rats.

1976) believed that M-LDH could increase in the absence of hypoxia but gave no alternative explanation for this behavior. The situation is somewhat similar to the oxidative enzyme rise after aerobic training since, O_2 tension in muscle appears more than adequate (Rowell, 1974; Stainsby, 1973) and where the oxygen uptake, even at maximal rate, is well below the oxidative enzyme activities (Holloszy, 1967 and 1971).* Thus, the oxidative enzymes as well as LDH appear to adapt to a situation where the energy demand is increased or the oxygen availability is decreased even though it does not reach a critical level. In skeletal muscle, the oxidative enzymes appear to adapt first to the demand resulting in a decrease in LDH. In the heart, the oxidative enzymes adapt to a lesser extent (Baldwin et al., 1977a; Holloszy, 1975; Oscai et al., 1971b) maybe because oxidative enzyme levels in the heart are already in a trained state and closer to a possible physiological limit so that the extra energy demand of training has to be met by increased LDH activity. The increase in the soleus LDH activity is harder to explain on the hypoxia basis. It is possible that this slow twitch muscle might have been under a greater stess than fast twitch muscles if equally solicitated by the running action. It is known that glycogen is depleted faster in ST fibers as compared to FT fibers in prolonged exercise (Gollnick et al., 1973a , d).

---

* The oxygen supply as a limiting factor to muscular contraction is still an open question however (Bostrom et al., 1974; Kaijser, 1970 and 1973).

Those facts might indicate a possible rôle of anaerobic glycolysis in this muscle.

Hypoxia might be a stimulus for LDH adaptation but it is hard to reconcile the facts that on one side trained heart oxidizes more lactate in moderate exercise and on the other side, has higher LDH and M-LDH activity. The fact that other glycolytic enzymes of the heart do not increase and that more fat is being oxidized might be at the origin of the LDH changes. Trained skeletal muscles also use more lactate as substrate and training might reduce the circulating lactate.

With sprint training which does not increase the VO_2max (Sjodin et al., 1976b and Thorstenson et al., 1975), an increase in LDH activity seems to be mandatory to meet the energy demand. The fact that animal studies did not support such an adaptative pattern may be due to the insufficiency of the training programs. Hickson et al. (1976) used only 10 sec work intervals and 40 sec rest intervals. Staudte et al. (1973) used longer work intervals (45 sec) but their rats did only 4 repetitions a day and trained only for 21 days.

Other reasons seem to warrant further investigation on the effect of chronic exercise on LDH activity and LDH subunits. As endurance and sprint training might have opposite effects on LDH, one form of these exercises has to be used exclusively. The use of mixed regimens (e.g. sprint

intervals superimposed to endurance running) might explain some of the nonsignificant results reported earlier (Holloszy, 1971; Molé et al., 1973) and should be avoided. Since many of these studies were significant only when studying the quadriceps muscles (Baldwin et al., 1972a and 1973; Hickson et al., 1976; York et al., 1974) and not significant when dealing with the gastrocnemius or the plantaris (Böhmer, 1969; Gollnick et al., 1961, 1967; Hickson et al., 1976; Holloszy, 1971 ; Molé et al., 1973), there may be specific muscle recruitement that should be further investigated. Gould and Rawlinson (1959) have reported no LDH change in the rat biceps brachii after swimming training, but it is not clear that this muscle is a prime mover for this motion. A biochemical comparison of "agonist" and "antagonist" muscles appears necessary. Finally, in many of the reviewed studies, total LDH activity was often measured at only one pyruvate concentration and M-LDH% was estimated without the previous determination of the species-specific optimal pyruvate concentrations for M-LDH and H-LDH. This may explain some of the many nonsignificant but large percentage changes reported in the literature.

Metabolism of High Energy Compounds*

ATP is the immediate source of energy for muscular contraction (Cain et al., 1962; Davies, 1971; Maréchal, 1972; Mommaerts, 1969). ATPase catalyses the conversion of ATP

---

* See footnote on next page.

into ADP. Because ATP stores are very limited (Davies, 1971; Karlsson, 1971a), ATP has to be resythesized continually to pursue muscular contraction. Most newly resynthesized ATP comes from oxidative metabolism and anaerobic glycolysis. In addition, minute amounts come from the conversion of PC and ADP into ATP through the two following reactions:

$$PC + ADP \xleftrightarrow{CPK} Creatine + ATP$$

and

$$2\ ADP \xleftrightarrow{AK} AMP + ATP$$

Adenosine Triphosphate and Phosphorylcreatine

Energy liberated from the reverse of PC is the most rapidly available (Cain et al., 1962; Di Prampero et al., 1970; Hohorst et al., 1962; Hultman et al., 1967; Karlsson, 1971a; Keul et al., 1972; Margaria, 1972; Piiper and Spiller, 1970). PC breakdown seems to provide most of the energy necessary for a 6 to 20 seconds work bout of high intensity like a 100 meter run (Keul, 1973; Margaria, 1968, 1972).

---

* Ennor and Morrisson (1958) have reviewed the origin of the term "Phosphagen" and suggested that "Phosphagens" should be regarded as a generic name embracing all (and restricted to) those naturally occurring phosphorylated guanidine compounds which function as stores of phosphate-bond energy from which phosphoryl groups may be transferred to ADP to form ATP as a result of enzymatic catalysis. Thus ATP itself is not a phosphagen as it is often implied (Cerretelli and Di Prampero, 1969; Gollnick and Hermansen, 1973; Karlsson, 1971a; Pernow and Saltin, 1971). In addition, phosphorylcreatine is preferred to phosphocreatine or to creatine phosphate since it does contain a phosphoryl group (creatine-PO_3H_2) and not a phosphate group ($-PO_4H_2$). Thus, "High Energy Compounds" or "Energy Rich Compounds" expressions appear more adequate to describe the combination of ATP and PC compounds than the term "Phosphagens".

Even with limited stores, ATP and PC initial levels have a definite effect on this kind of sport performance. PC stores are depleted very soon at work (Cain et al., 1962; Davies, 1971; Di Prampero et al., 1970; Ericksson et al., 1973; Hohorst et al., 1962; Hultman et al., 1967; Karlsson et al., 1971a; Keul et al., 1972; chapt. III; Knutgen and Saltin, 1973; McGilvery and Murray, 1974) whatever the working intensity (Di Prampero et al., 1970; Karlsson, 1971a; Keul et al., 1972, chapt. III). ATP levels are usually maintained at equilibrium or more or less depleted in very severe work of if CPK is inhibited (Cain et al., 1962; Davies, 1971; Hohorst et al., 1962; Hultman et al., 1967). Most PC stores are replenished very rapidly, within 2 minutes in man or animals (Fox et al., 1969; Hultman et al., 1967; Margaria 1968, 1972; Piiper and Spiller, 1970).

Other facts support the importance of high energy compounds on contractile performance. Borredon (1967) showed a negative correlation between PC levels of the heart and electrocardiographic ST depression. Feinstein (1962), Fox and Reed (1966) and Rabinowitz and Zak (1975) reported up to 24 and 54% decrease in ATP and PC with experimental congestive heart failure. Total tension developed in the anaerobic state seems to be related to ATP and PC levels (Cerretelli and Di Prampero, 1969; Harris et al., 1975). Running performance in rats was reduced when the creatine reserve was itself decreased with a synthetic creatine analog diet (Shields et al., 1975). Cairella and Vecchi (1966) and

Cier (1965) reported an increased swimming endurance time in rats after ingestion of PC supplements.

Many experimental conditions - PC, K^+ and Mg^{++} Aspartate, amino-Acids, Monosodium Phosphate, vitamin C administration - have been shown to increase ATP and PC levels (Pourel, 1968). Rabinowitz and Zak (1975) recently reviewed several forms of cardiac hypertrophy (e.g. acute cardiac overload, developing cardiac hypertrophy, compensated cardiac hypertrophy) and found a decrease or no significant changes in ATP and PC. Degenri[illegible] (1975) and Scheuer et al. (1970) reported similar results for endurance swimming training in rat heart. In 1961, Gangloff et al. reported a paradoxical 32% increase in rat heart after endurance treadmill training, but the values (e.g. 1.5 mmole/kg wet weight) were well under the usual reported range (5-11 mmole/kg wet weight). [illegible]ny of the results reported before 1960 are low due to poor sampling and extraction techniques (Wollenberger et al., 1960) and thus, are questionable.

In the skeletal muscles, the effect of physical training on ATP and PC stores is somewhat confusing. According to Yakolev (1965) and Yampolskaya (1952) (as quoted by Haralambie, 1972), PC levels increased up to 75% after training with short exercises of high intensity as compared to lower intensity training. Russian workers are currently assessing the efficiency of their training programs with the specific PC response to training (Rogozkin, 1976). There

may be an opposite trend in PC adaptation in heart and skeletal muscle (Harren, 1938, quoted by Haralambie, 1972). Other studies, however, are no more conclusive. Compared to sedentary rats, Gale and Nagle (1971) found that neither sprint nor endurance running-trained rats had significantly different ATP and PC levels of the soleus and gastrocnemius plantaris muscle group. Absolute values reported by these authors were only about 50% of the usual values. Saltin and Karlsson (1971) studied the effect of physical conditioning in man on ATP and PC but reported no significant change on these parameters. Karlsson et al. (1972) showed that endurance training increases ATP but not PC levels of the quadriceps. Thorstensson et al. (1975) did not show any significant change in ATP and PC after short sprint training in man. Swimming also failed to alter significantly the ATP levels of rat gastrocnemius (Böhmer, 1969).

Ericksson et al. (1973) reported an 11 and 39% increase of ATP and PC respectively in vastus lateralis of 11-13 years old boys after 6 months of general conditioning. They explained the ATP increase by the concomitant mitochondrial increase but could not explain the unusual increase in PC. It is interesting to note that the pre-training values were 14.5 as compared to 20.2 mmole x kg^{-1} wet weight after training. Scandinavian groups (Bergstrom et al., 1971; Harris et al., 1974; Hultman et al., 1967; Karlsson et al., 1971a) usually have reported values of 17 to 21 mmole x kg^{-1} wet weight in human adult quadriceps. Since there was no

control group for age effect, it may be that the increase reported by Ericksson et al. (1973) reflected an aging effect rather than a training effect. Casten (1950) and Quarto di Palo (1960) previously reported an increase in PC but not in ATP in maturing rats.

To summarize, it seems that training induces a decrease in ATP and PC levels of the heart. In the skeletal muscle, the situation is not conclusive and requires further investigation. Some authors (Ericksson et al., 1973; Jacobs and Klingenberg, 1964; Haralambie, 1972; Keul et al., 1972, chapt. III; McGilvery and Murray, 1974; Rabinowitz and Zak, 1975; Saks et al., 1974; Seraydarian et al., 1974) questioned the physiological importance of possible ATP and PC changes. They believe that enzyme activities (CPK and AK) are more efficient ways to adapt to the energy demand and that PC may be more important as a regulator of other energy synthetic reactions or as a form of energy that can be transported from the mitochondria to the myofibrils rather than being a storage form of energy.

Creatine phosphokinase

Creatine phosphokinase (CPK) is a dimer composed of a brain type (B) and a muscle type subunit (M), but the nature and the function of the three isoenzymes (BB, BM, MM) is very unclear (Traugott et al., 1973). As the amount of brain type CPK is usually very low in muscle (Saks et al., 1974), more concern is given to total CPK.

The effects of acute exercise on serum CPK are well documented (Block et al., 1969; Fowler et al., 1962 and 1968; Haralambie, 1972, 1973; Hunter and Critz, 1970; Kendrick-Jones and Perry, 1965; Nuttall and Jones, 1968; Rose et al., 1970; Sanders and Bloor, 1975; Schmidt and Schmidt, 1969; Siest and Galteau, 1971; Wagner and Critz, 1970). It seems that serum CPK may increase up to 400% (Wagner and Critz, 1970) and is a better index of the work load intensity than other enzymes (Sanders and Bloor, 1975). Kendrick-Jones and Perry (1965) and Wagner and Critz (1970) reported a muscle CPK rise after acute exercise or in vitro contraction (isometric or isotonic) but this could not be confirmed by Oscai and Holloszy (1971) and Dieter (1970) unless there were Vit C deficiencies. On the other hand, Bostrom et al. (1974a and b) reported a decrease in tissue CPK after swimming or in vitro stimulation (isometric or isotonic). Serum and tissue changes after acute exercise are generally transient and have probably little significance for the understanding of the chronic effects of exercise on tissue CPK.

The effects of chronic exercise on tissue CPK appear inconsistent from one report to another. Endurance training has been shown to increase the animal heart, soleus and gastrocnemius CPK by more than 10% (Wagner and Critz, 1970). Other studies (Böhmer, 1969; Dart and Holloszy, 1969; Dieter, 1970; Oscai and Holloszy, 1971; Rawlinson and Gould, 1959; Walpurger and Anger, 1970) reported no CPK change in either mitochondrial or cytoplasmic extracts of heart or skeletal

muscles. Bohmer (1969) and Kendrick-Jones and Perry (1965) showed a CPK decrease in human and animal muscles after immobilisation, but this does not imply a tissue CPK increase after training. As a matter of fact, Suominen and Heikkinen (1975) and Thorstensson et al. (1974, 1976a and b) failed to show a CPK increase in human muscle after endurance and strength training respectively. On the other hand, Thorstensson et al. (1975) found a 35% increase in human muscle after sprint training. In animals, sprint training resulted in a 12% CPK increase in the soleus but did not change the CPK activity of the rectus femoris (Staudte et al., 1973), whereas isometric training had a reciprocal effect in these two muscles, both in male and female rats (Exner et al., 1973).

To summarize, tissue CPK does not seem to change with training except perhaps with sprint and isometric training. Further research is required to substantiate CPK adaptation to chronic exercise.

Adenylate Kinase

ATP can be resynthesized through the adenylate kinase (AK) reaction. According to Newsholme and Start (1973), the energy provided by this reaction can suffice for 3 seconds at most. From the athlete's point of view, this could be very important. For biochemists, however, this is a very small amount of energy as compared to other energy sources. Newsholme and Start (1973) considered this reaction as an amplification mechanism for the regulation of glycolysis

since the AK reaction is alway close to equilibrium and the ATP concentration is 50 times larger than the AMP concentration. Relatively small changes in ATP are amplified by the relatively large change in the AMP effector. Other functions have been attributed to AK, such as a rephosphorylation of the adenylic acid accumulating with the oxidation of the fatty acid (Pette, 1971) or an extra intramitochondrial adenosine nucleotide exchange (Klingenberger, 1965). Thus, the biochemical function of AK is not clearly determined. Adenylate kinase adaptations to chronic exercise are not more conclusive. Oscai and Holloszy (1971) indicated no change in mitochondrial and cytoplasmic adenylate kinase after endurance running in rat gastrocnemius. In heart muscle, however, Walpurger and Anger (1970) found a 50% and a 30% rises in cytoplasmic, but not in mitochondrial, AK after endurance swimming and running in the rat respectively. Dart and Holloszy (1969) failed however to demonstrate any AK adaptation after experimental heart hypertrophy in the rat using arteriovenous fistula. In human skeletal muscles, Thorstensson et al. (1975 and 1976b) failed to show any AK change after sprint and strength training although they observed a 7.8% significant increase with strength training in a previous study (Thorstensson et al., 1976a).

In conclusion, it seems that skeletal muscle AK changes rarely with training, except maybe with strength training. There is also a possible increase in the heart AK activity.

CHAPTER III

METHODS AND PROCEDURES

Animals

Forty male Sprague Dawley rats (Canadian Breeding Farm and Laboratories, Ltd., St. Constant, Quebec) were used in the study. The rats were approximately six weeks of age and weighed 160 ± 17 grams (X ± SD) at the time of arrival. The animals were placed in 60 X 60 X 30 cm self-cleaning cages at 25°C, ≈50% relative humidity and 755 mm Hg in groups of ten for the first 8 weeks and in groups of five thereafter. Cage locations in the rack were rotated in a random fashion once a week. Rats were exposed to the usual 12hr daylight and 12hr darkness. Rats were identified with a color code on the proximal end of the tail with the use of a marking pen. Rats were fed ad libitum with tap water and standard rat chow (Charles River Rat and Mouse Formula) containing 22% protein, 5% fat, 5% fiber and 11% moisture.

Experimental Groups

After an initial exercise program, the thirty best runners were numbered in order by weight and randomly assigned to one of three experimental groups*:

---

* At that time, the rats were relocated according to their new group, always 10 per cage. This resulted in aggressive behaviour which was somewhat reduced by putting 5 rats per cage. Individual cages were unavailable due to restricted facilities.

Sgr: Sedentary group

Cgr: Continuous training group

Igr: Intermittent training group

Mean group weights of the rats ($\bar{X} \pm SD$) were 305.0 ± 26.5, 317 ± 18.0, 311.7 ± 20.4 grams for Sgr, Cgr and Igr respectively at time of group assignment. Due to the limited availability of the "trainer", running time had to be changed a few times during this 6 month experimental period. On a few occasions, training was conducted in the evening with the lights on. At all other times, the rats were trained in day light either in the morning at 8:00 or at the end of the afternoon at 17:00. The attrition rate for the experimental animals was 2 of 10 per group.

Initial Exercise Program. After one week of only cage activity for adaptation to the new environment, an initial exercise program, consisting of running five days per week for four weeks, was provided. Running took place on a motor-driven treadmill accomodating 10 animals at a time and incorporating a shock grid at the rear of the compartments to motivate the animals to keep pace with the belt movement (Quinton Instruments, Seattle, Wash. U.S.A., Small Animal Treadmill, Model 42-15). The rats learned to run continuously for 6 minutes on an 8% grade with the speed progressively increased from 10 to 31 m/min. This progression is low enough to avoid significant training effects on the activities of the oxydative enzymes (Benzi et al., 1975; Fitts et al., 1975; Holloszy, 1967; Molé et al., 1973).

In addition, following the continuous work, the initial exercise program included low intensity intermittent running, starting with 10 X 15 sec work at 15m/min with 15 sec rest intervals and finishing with 10 X 15 sec work at 35-40 m/min with 10 sec rest intervals. The initial training program was deleted thereafter for the entire 6 month experimental period. Knowing the reversible effect of training, it was assumed that the initial training program would have no training effect at the time of sacrifice.

The sedentary group (Sgr) was restricted to normal cage activity: eating, drinking, sleeping, walking, fighting and weekly weighing.

The continuous training group (Cgr) ran for 6 months, five days per week. The treadmill was set at 31 m/min and 8% grade. The duration of the training session was gradually increased from 10 to 50 minutes over 3 months, maintained at this level for 1.5 month and reduced to 40 minutes for the last 1.5 month.* This training stimulus is believed to be the maximal that can be handled by these rats. It was therefore impossible to reproduce Holloszy's training

---

* The training load had to be reduced because the rats failed to run after 40 minutes even with increased electrical stimulation. This behavior may be linked to the cage re-assignment that followed the initial exercise program since the rats were harder to train at that time. Reduced training had also been reported by others (Barnard and Peter, 1971; Peter, 1970).

regimen (1967) as originally intended. Holloszy was able to have young Wistar rats running for 2 hours after 3 months and demonstrated significant changes in the oxidative capacity of the rats (Baldwin et al., 1972; Holloszy, 1967; Molé et al., 1973; Pattengale and Holloszy, 1967). Nevertheless, Fitts et al. (1975) and Gollnick et al. (1970) have reported intensive glycogenolytic and lipolytic responses in rats with a training regimen similar to the one used in this study.

The intermittent training program (Igr) was designed to cause a greater effect on the anaerobic metabolism. Consequently, the intensity was higher than for the continuous training. Each training session started with a 5 minute warm-up period at 31 m/min and 8% grade. The rats ran 10 X 1 minute at an intensity that was gradually increased from 40 to 75 m/min over the first 5 months and maintained at 70 m/min (8% slope) during the 6th month. Each work interval was interspaced by a 4 minute rest interval.

Dependent Variables

The dependent variables mostly represent key metabolites or enzymes of the anaerobic metabolism in different tissues. These include:

1. Absolute weight of the body or the organs, W_{abs}. These measures provide a simple check of the effectiveness of the training programs;

2. Relative weight of the organ, W_{rel}. The ratio of organs W_{abs} over body W_{abs} is used as a parameter per se as well as a relative index of organ weight since the usual decrease of body weight with training may compensate for the expected organ hypertrophy. For the second purpose, regressed weights (Gollnick et al., 1967; Héroux and Gridgeman, 1958; Muller, 1975; Tanner, 1949) are usually better than W_{rel}, but will not be reported here since no significant correlations between organ weights and body weights were observed in this study. In addition, it has been reported that W_{rel} is similar to the regressed weight of muscles (Héroux and Gridgeman, 1958; Muller, 1975);
3. Adenosine triphosphate, ATP;
4. Phosphorylcreatine, PC;
5. ATP + PC;
6. Creatine phosphokinase, CPK (E.C. No. 2.7.3.2, ATP: creatine phosphoryltransferase);
7. Adenylate kinase, AK (E.C. No. 2.7.4.3, ATP: AMP phosphotransferase);
8-13. Lactate dehydrogenase, LDH (E.C. No. 1.1.1.27, L-Lactate: NAD oxidoreductase);
8. LDH activity at 21×10^{-4} M PA, LDH_{21}; at high pyruvic acid concentration, there is inhibition of the heart type of LDH subunit;

9. LDH activity at $3x10^{-4}$M PA, LDH_3; at low pyruvic acid concentration, there is inhibition of the muscle type of LDH subunit;
10. The ratio of LDH_{21} over LDH_3, LDH_{21}/LDH_3, is a good indicator of the percentage of heart and muscle types of LDH subunits, an important aspect of anaerobic metabolism since "H" type favors the lactate to pyruvate reaction whereas the "M" type favors the pyruvate to lactate reaction;
11. LDH activity related to muscle type of LDH subunits, M-LDH;
12. LDH activity related to heart type of LDH subunits, H-LDH;
13. Total LDH activity = M-LDH + H-LDH, LDH.

The dependent variables as well as their sampling sites are listed in Table 3.

Sampling Procedures

At the end of the training period, rats were sacrificed at rest, two days after the last work bout to avoid acute exercise effects on dependent variables.

The measurement of ATP and PC is critical for the sampling procedures due to the rapid hydrolysis in anaerobic conditions (Karlsson, 1971; Lamprecht, 1963; Lowry et al., 1964a and b). Rats were anesthetized with an intraperitoneal injection of 50 mg of Nembutal (sodium pentobarbital Abbott, 50 mg/ml) per kg of body weight. This anesthetic

TABLE 3 Listing of Dependent Variables

MEASURES*	1 BODY (BW)	2 TIBIALIS ANTERIOR (TA)	3 PLANTARIS (P)	4 GASTROCNEMIUS MEDIALIS (GM)	5 GASTROCNEMIUS LATERALIS (GL)	6 SOLEUS (S)	7 HEART (H)	8 LIVER (L)
1. W_{abs}	x	x	x	x	x	x	x	x
2. W_{rel}		x	x	x	x	x	x	x
3. ATP		x	x	x	x	x	x	
4. PC		x	x	x	x	x	x	
5. ATP + PC		x	x	x	x		x	
6. CPK		x	x	x	x		x	x
7. AK		x	x	x	x	x	x	x
8. LDH_{21}**		x	x	x	x	x	x	x
9. LDH_3**		x	x	x	x	x	x	x
10. LDH_{21}/LDH_3		x	x	x	x	x	x	x
11. M - LDH		x	x	x	x	x	x	x
12. H - LDH		x	x	x	x	x	x	x
13. TOTAL LDH		x	x	x	x	x	x	x

* See Table 1 for abbreviations.

** LDH at 21×10^{-4}M and 3×10^{-4}M PA respectively.

was preferred to ether in order to minimize excitation and also possible changes in enzyme activities that occur with ether (Ben et al., 1969; Katona, 1973). However, Nembutal has a depressive effect on the cardiovascular system (Sawyer et al., 1971) and definite effects on substrate concentrations of rat liver (Faupel et al., 1972). ATP levels are not affected by Nembutal (Faupel et al., 1972). The effects of Nembutal on heart and skeletal muscle substrates and on enzymes are not known but the number and the sites of sampled tissues required anaesthesia of the rats. A systematic error, if any, should not disturb comparisons of the three training groups. After discarding the skin and the superficial muscle layers, the muscles of the left leg were isolated in the following order GM, TA, S, P, GL, with minimum trauma, leaving intact the circulation, innervation and insertions.

Each muscle was isolated with a small plastic plate approximately 15 cm long, 1 mm thick, and 1.5 cm wide at one end and 3 cm at the other. The plastic plate had protuberant rounded edges increasing the thickness of the plate to 4 mm. This plate assured better thermal insulation and made it easier to grasp the suspended muscle with copper tongs precooled in liquid nitrogen (-190°C). The muscle was then cut immediately along the edge of the copper tongs and put into the liquid nitrogen. Protruding tissue was broken off the copper tongs to avoid contamination with

slowly frozen tissue. The samples were immediately wrapped in aluminum foil and temporarily stored in liquid nitrogen and then, in a deep freeze at -60°C (Revco, Ultralow, Model ULT-075-0-2) until chemical analysis was carried out. At these temperatures samples are stable almost indefinitely. (Lowry and Passonneau, 1972, pp. 120-122).

The tongs used were a pair of Vise-Grip pliers (Plate # 1) modified by soldering a pair of copper blocks to the original lips of the pliers in such a way that block surfaces always compressed the muscle into a parallel sheet of 1 mm thickness to ensure even, rapid and constant freezing within and between samples. Copper was preferred to the often used aluminum because it offers a slightly higher thermal conductivity and freezes 1.4 X more tissue for the same block size due to a higher density that largely compensates for its lower specific heat*. Each copper block was

---

* Physical characteristics (Handbook of Chemistry and Physics, 1973).

1. Thermal conductivity (Watt/cm)

	0°C	25°C	-173°C
Aluminum	2.36	2.37	3.0
Copper	4.01	3.98	4.83.

2. Specific heat (cal/g.C°)
   Al: 0.215; Cu: 0.093
3. Density (g/cm^3)
   Al: 2.7; Cu: 8.9
4. $\Delta Q = mc\ \Delta t$ or $m_1\ c_1\ \Delta t_1 = m_2\ c_2\ \Delta t_2$
   where ΔQ = heat transfer (calories)
   m = mass (grams)
   c = specific heat (cal/g.C°)
   Δt = change in temperature (C°)

PLATE 1. MODIFIED VISE-GRIP PLIERS WITH COPPER BLOCKS USED TO FREEZE THE TISSUES IN SITU

1 cm thick; other dimensions can be deduced from its cross section (Figure 3). Such a design allowed samples of various sizes and shapes to be taken.

It is believed that such a freezing technique is the simplest and the best available (Adam, 1963a; Cartier, 1967a; Eranko, 1954, Faupel et al., 1972; Hess, 1963; Lampretch et al., 1963a and b; Leunissen and Piatnek-Leunissen, 1968; Lowry and Passonneau, 1972; Pourel, 1968; Swynghedauw et al., 1967). Compared to immersion techniques using various refrigerants, "quick-freeze" tongs yielded better results. Even for isopentane, the best refrigerant, freezing (36° to 0°C) required over 7 sec with tissue samples as small as 200 mg due to the heat isolation of immersed tissues. This is caused by the development of a gaseous wrap of refrigerant (phenomenon of Leidenfrost) and is also due to the low thermal conductivity of the tissue itself. This delay is sufficient to cause hydrolysis of ATP and metabolic changes in many other substrates (Faupel et al., 1972).

The heart was extirpated next. It has been shown that the anoxia delay due to the opening of the thorax is enough to cause hydrolysis of ATP and PC (Adam, 1963a; Lampretch et al., 1963a and b). Therefore, a tracheotomy was performed for assisted ventilation using an artificial respiratory pump, (Rodent pump, Model 680-1, Harvard Apparatus Co., Dover, Mass. U.S.A.). The thorax was then opened and the heart was lifted by its apex and flattened

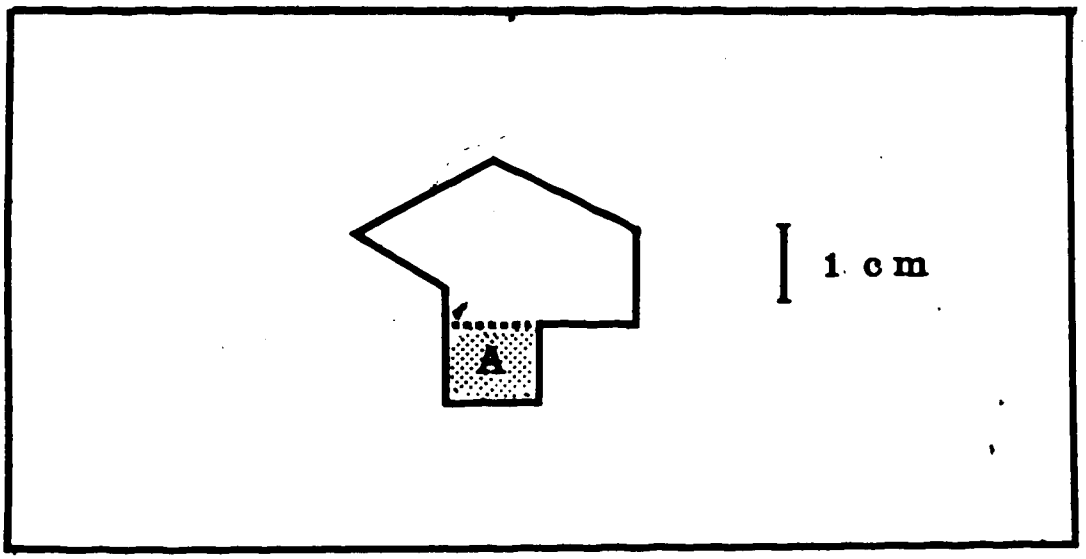

FIGURE 3. OVERHEAD DIMENSIONS OF COPPER BLOCKS USED IN FREEZING TISSUES. BLOCKS WERE SOLDERED TO THE ORIGINAL JAWS OF THE VISE-GRIP PLIERS WITH PART "A". BLOCKS WERE 1 CM THICK.

between the precooled copper tongs. The whole heart was maintained for weighing and enzyme analyses. Non-beating hearts due to poor ventilation were eliminated from ATP and PC analyses. Heart tissue was analysed similarly to the analyses mentioned previously for the left leg samples.

The liver was removed, frozen by immersion in liquid nitrogen and stored in a freezer at -60°C for further weighing and analyses. Finally, the muscles of the right leg were isolated, dissected, frozen by immersion, and stored at -60°C for weighing and enzyme analyses.

Analytical Methods

All chemicals used in this study and their source of supply are listed in Appendix G.

Tissue Preparation

Enzyme analyses. Fifty to 100 mg of wet tissue were blotted for blood, freed of connective tissue and weighed to one tenth of a milligram. Tissues were homogenized using 10 ml of 0.25 M sucrose per g of tissue with a Kontes glass pestle and tubes of size AA or A. (Canadian Scientific Company, Montreal, Canada, Cat. No. 885451 and 885452 for the pestles and the tubes respectively). The pestle was rotated by a motor (Fisher Scientific Co., Montreal, Canada, Dyna-Mix Model 143) at about 1000 rpm using rubber tubing as an universal joint. The tube was moved up and down manually (about 30 times to complete muscle disintegration) and frequently put in ice to prevent over-heating, (Hess,

1963). Crude homogenates were centrifuged at 4000 g and 4°C for 10-15 min (International Equipment Co., Centrifuge, No. PR-6000). Then, to bring the absorbance changes into the region of 0.02 to 0.05 absorbance units per minute with the assay mixtures, further dilutions were made as follows:

ENZYMES	SKELETAL MUSCLE	HEART	LIVER
AK	1/100	1/100	1/100
CPK	1/10000	1/1000	1/100
LDH	1/1000	1/1000	1/1000

and 0.05 ml of these dilutions were used to start the reaction in the assay mixture. The same sample was used for AK, CPK and LDH assays.

ATP and PC analysis. Tissue preparation (weighing, deproteinization, homogenization, centrifugation and neutralization) was conducted in a 4°C refrigerated room. Tissue samples of one rat were transferred from the freezer to a Dewar flask containing liquid nitrogen and maintained in this flask between manipulations. Until deproteinization, care was taken to limit exposure to 4°C air to less than 10 consecutive seconds. Tissue samples (50-400 mg) were rapidly weighed on a Roller Smith balance (Biolar Corporation, Model LG, North Grafton, Mass.) to one tenth of a mg and transferred back to the Dewar flask. The deproteinization was carried out according to Lampretch et al. (1963a

and b), using 6.5 ml of $HClO_4$ 6% W/V for 2 g of muscle tissue. Perchloric acid was preferred to trichloracacetic acid because the latter is reported to inhibit the G6P-DH used in the assay (Cartier et al., 1967a; Lowry and Passonneau, 1972, p. 123).

The exact amount of $HClO_4$ required was calculated and drawn into a pipet (Pipetman, Model P by Gibson, Analytical Instruments, Mississauga) for later use. Next the tissue was transferred to a porcelain mortar (30 ml capacity) containing liquid nitrogen and ground with a precooled porcelain pestle to a fine powder. As needed, 10 ml portions of liquid nitrogen were added to avoid complete evaporation. Then, the $HClO_4$ was slowly added and ground with the powdered tissue in liquid nitrogen. After grinding and evaporation of remaining nitrogen, the powdered mixture of muscle and $HClO_4$ were transferred to a Kontes glass homogenizing tube with a small plastic spatula and homogenized when the mixture started to melt, using the procedure described previously. Samples were left standing for 5-10 minutes to ensure complete deproteinization (Cartier et al., 1967a; Bucher et al., 1963), then centrifuged at 4°C for 10 minutes at 4000 g. Care was taken to minimize the time of deproteinization. The tolerance time for 0.6 M $HClO_4$ at 0°C is 1 hour for acid labile ATP and PC (Hess, 1963; Lowry and Passonneau, 1972, p. 124).

Supernatants of centrifuged homogenates were trans-

ferred to small test tubes, and neutralized to pH 7.4 according to Lamprecht (1963a and b) using Methyl Orange as the indicator and K_2CO_3 (5M) as titratant. Samples stood for 10 min. to permit $KClO_4$ sedimentation. The supernatant was transferred to another tube to avoid contact with any acid stable enzymes such as MK and ATPase in the sediment that might have affected PC or ATP assays. From this final solution, 0.05 or 0.1 ml was added quickly for the assay, because there is a slight hydrolysis of ATP and CP on standing (Lamprecht, 1963a).

Assays

All enzyme assays were conducted at 30° C, using 1 cm square cuvets containing 3 ml of reagent solution and recording the % transmittance change for 2 minutes at a wave length of 340 nm on a Beckman spectrophotometer (Model DB-6) coupled to a Fisher recorder (Recordall, Model 5223-51). Transmittance readings were transformed to absorbance and to enzyme activities using a small desk computer (Hewlett Packard, No. 9801 A).

Adenylate kinase was measured according to Oliver's method (1955). The reactions are basically:

$$ADP \underset{}{\overset{AK}{\rightleftharpoons}} ATP + AMP \qquad (1)$$

$$ATP + glucose \overset{HK}{\rightleftharpoons} ADP + G6P \qquad (2)$$

$$G6P + NADP \overset{G6P\text{-}DH}{\rightleftharpoons} 6\text{-phosphogluconate} + NADPH + H^+ \qquad (3)$$

According to Newsholme and Start (1973), the forward reaction is the fastest and is preferred to the reverse one (Collowick, 1955; Kleine and Chlond, 1967).

Creatine phosphokinase was measured according to Oliver's method (1955) as modified by Nielsen and Ludvigsen (1963) and Rosalki (1967), using commercial kits (Dade, CPK-UV-1 or CPK-UV-10) which have proven to be reliable and valid (Crowley and Alton, 1970; Rosalki, 1967). The reactions of this method are:

$$PC + ADP \xrightleftharpoons{CPK} \text{creatine} + ATP \qquad (1)$$

$$ATP + \text{glucose} \xrightleftharpoons{HK} ADP + G6P \qquad (2)$$

$$G6P + NADP \xrightleftharpoons{G6P\text{-}DH} \text{6-phosphogluconate} + NADPH + H^+ \qquad (3)$$

According to Rosalki (1967), the forward reaction is faster and is preferred to the reverse reaction.

Lactate dehydrogenase assay was more complicated. By measuring activity at two predetermined pyruvate concentrations, it is possible to obtain the percentage of "H" and "M" monomers of the LDH, plus activity due solely to "M" and "H" monomers or to total LDH. The method was essentially that used by Dawson and Kaplan (1964), Fox and Reed (1969), Hirota et al., (1976), Kaplan and Cahn (1962), Latner and Skillen (1968, p. 80), Plagemann et al. (1960a and b) and Stambaugh and Post (1966a). Thus, with two simple spectrophotometric assays, it is possible to obtain not only enzyme activity but also relative distribution of

"H" and "M" monomers. This method yields essentially the same information as the more complicated electrophoretic separation of the LDH isoenzymes since an equal mixture of LDH_5 (i.e. M_4) and LDH_1 (i.e. H_4), results in the same total activity as LDH_3 (i.e. M_2H_2) (Everse, 1973, p. 66; Kaplan and Cahn, 1962; Latner and Skillen, 1968, p. 30; Plagemann et al., 1960b).

LDH catalyses the following reaction:

$$PA + NADH + H^+ \rightleftharpoons^{LDH} LA + NAD^+ \qquad (1)$$

This LDH method is based on the specific calalytic properties of the relative composition in "H" and in "M" monomers. Each monomer has a different optimal pyruvate concentration for maximal velocity of the reaction. Therefore, at the two optimal pyruvate concentrations, the total velocities of the reactions are the following:

at optimal PA concentration for M-LDH,

$$V_1 = \text{M-LDH} + x\ \text{H-LDH} \qquad (1)$$

and at PA concentration for H-LDH,

$$V_2 = y\ \text{M-LDH} + \text{H-LDH} \qquad (2)$$

The optimal pyruvate concentrations as well as the values of x and y must be determined for each species (Cahn et al., 1962; Fine et al., 1963a; Latner and Skillen, 1968, p. 4, 20, 35). In this study, rat M and H were isolated with polyacrylamide gel electrophoresis using the method of Dietz and Lubrano (1967). Plate 2 (Appendix A) is an example of the isoenzyme separation. Additional heart and skeletal

muscle samples were treated in a similar fashion except for the staining procedure. The fastest and the slowest moving bands toward the anode, H_4 and M_4 respectively, were cut according to two stained samples run at the same time.* Then these unstained discs of M_4 and H_4 LDH were spectrophotometrically analysed to determine the optimal pyruvate concentrations (Figure 6 and Table 21 in Appendix A). Hence, it was found that the optimal pyruvate concentrations were the following:

PA = 21×10^{-4}M for M_4 or M-LDH

PA = 3×10^{-4}M for H_4 or H-LDH

The percentage of enzyme activity at these two concentrations were as follows:

PA	M_4	H_4
21×10^{-4}M	100%	80%
3×10^{-4}M	77%	100%

Therefore, equation (1) and (2) were re-written as:

$$LDH_{21} = 1.00\ M + 0.80\ H \qquad (3)$$

$$LDH_3 = 0.77\ M + 1.00\ H \qquad (4)$$

From (3) and (4), the ratio LDH_{21}/LDH_3 (V_1/V_2) which also indicated the percentage of H and M subunit (Figure 4), the activity solely due to H and M subunits (H-LDH and M-LDH) and the total LDH (i.e. M-LDH + H-LDH) can be computed.

---

* The ratio of the distance covered by the marker dye to that covered by the isoenzyme band was used. This ratio was constant for the same batch of analyses.

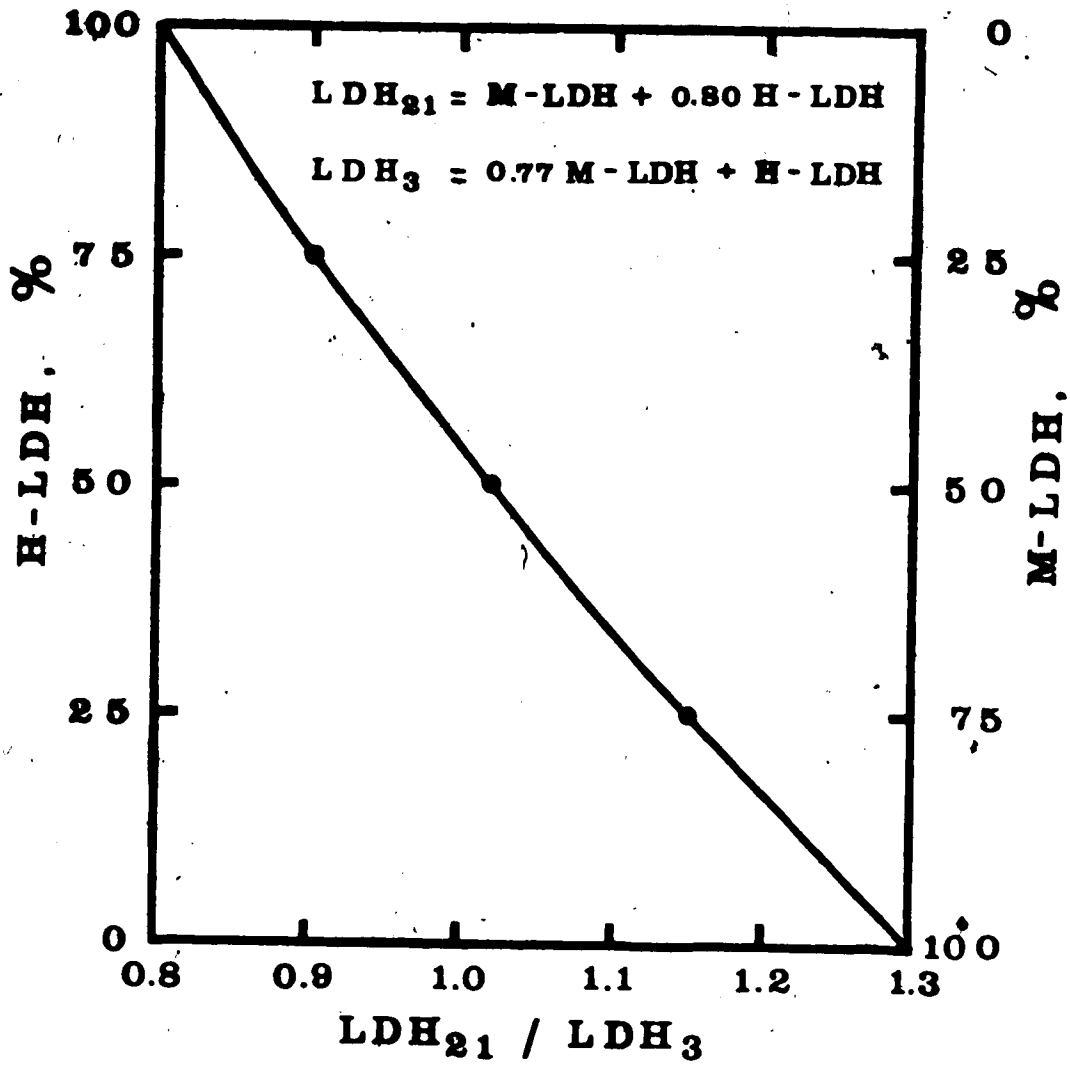

FIGURE 4. PERCENTAGE OF H-LDH AND M-LDH FROM THE LDH_{21}/LDH_3 RATIO. THE LINE HAS BEEN DRAWN BY SUBSTITUTING THE % OF M-LDH AND H-LDH IN THE INSERTED EQUATIONS WHICH HAVE BEEN OBTAINED FROM TABLE 21 AND FIGURE 6 OF APPENDIX A

Final spectrophotometric conditions of the reagents in the cuvets were pH 7.4, 0.067 M Phosphate buffer (Sorensen), 1.5×10^{-4} M NADH and 21×10^{-4} or 3×10^{-4} M pyruvate.

Adenosine triphosphate and phosphorylcreatine were measured in the same assay according to Lamprecht and Stein (1963b) and Lowry and Passonneau (1972, p. 151). The reactions, as for CPK, are as follows:

$$PC + ADP \xrightleftharpoons{CPK} \text{creatine} + ATP \qquad (1)$$

$$\text{glucose} + ATP \xrightleftharpoons{HK} G6P + ADP \qquad (2)$$

$$G6P + NADP \xrightleftharpoons{G6P\text{-}DH} \text{6-phosphogluconate} + NADPH + H^+ \qquad (3)$$

ATP was measured using Calbiochem Kits (ATP Stat-Pack No. 869206) which were modified for PC by adding other reagents to reach the final concentrations suggested by Lamprecht and Stein (1963b). This modification yielded identical results to the conventional approach but saved considerable time.

Statistical Methods

Assays were all done in duplicate and computation carried out on the means. Suspect values were rejected on a common sense basis. Calculated t for the rejected values were higher than the 1.9 and even the 2.44 rejection criteria ($\alpha = 0.05$ and 0.025 respectively for $n = 7$). Percentages of missing data appear in Appendix F. Data were analysed from two points of view: differences between training groups and differences between organs (or tissues). Group and organ means and standard deviations were calculated for all the dependant variables.

Two way analyses of variance (Winer, 1971, pp. 245-248) were made between group differences and between organ differences. Since the variance differed very much between some variables, and since there were no significant interactions between training groups and organs, and since the differences between organs were much larger than the ones between training groups (Appendix F), the two way analyses of variance were merely used to assess the general pattern between training groups. To assure more specific analyses, one way layouts (Winer, 1971, pp. 210-219) were done on each variable and each organ to assess training group differences and on each variable with combined data from all groups to assess organ differences.

For comparison between pairs of means, Scheffé's contrasts (Scheffé, 1959, pp. 66-67; Winer, 1971, pp. 198-201) were computed for α equal to 0.05 when the F ratios of the analysis of variance were significant ($p \leq 0.05$). Although less powerful than other a posteriori tests, Scheffé's method was used because it is clearly the most conservative with respect to type I error (Scheffé, 1959; Winer, 1971). Scheffé's procedure does not require equal n and is less sensitive to violations of normality and homogeneity of variance assumptions than Tukey's procedure, the only other comparable test with respect to Type I error (Myers, 1966, pp. 333-336). All statistics were computed with SCIRU (Service de consultation informatique pour la recherche universitaire), one of the services at the Université de Montréal.

CHAPTER IV

RESULTS

The raw data for the training groups are listed in Appendix C. The original statistics on dependent variables which showed significant differences ($P < 0.05$) between training groups appear in Appendices D, E and F. Training group comparisons for each dependent variable: means, standard deviations, ANOVA F ratios and Scheffé's contrasts, are found in Tables 4 to 17. The group body weight growth curves are plotted in Figure 5 from data found in Table 22 (see Appendix B).

Significant ANOVA F ratios ($P < 0.05$) were obtained between training groups for absolute body weight, relative organ weight, ATP and CPK. All other variables (PC, ATP + PC, AK, LDH_{21}, LDH_3, LDH_{21}/LDH_3, M-LDH, H-LDH and Total LDH) showed no significant trend with one way ANOVA. However, two way ANOVA revealed significant differences between training groups for these parameters (Appendix F).

The details of the aforementioned results are presented in the two following sections: "The Effect of Chronic Exercise" and "Organ Comparison".

The Effects of Chronic Exercise

Body Weight

The body weights obtained for the rats at each week during the study were averaged by group (Table 22, Appendix B). Weight progress is depicted graphically in Figure 5. During the initial exercise program, body weights were equal and progressed at the same rate for all groups. During the formal training period however, the increases in weight of the training groups progressed at a much slower rate than Sgr ($P < 0.005$, Table 4). Scheffé's contrasts were significant between S[illegible] (625 g) and the training groups (Cgr: 534 and Igr: 534 g) but not between the training groups themselves. Growth rate slowly declined with age and started to plateau by 25 weeks of age.

Organ Weights

Both absolute and relative weights of the organs were recorded at the time of sacrifice. There were no significant differences in absolute organ weights between the groups (Table 5). Relative organ weights (except liver) of the trained animals were heavier than those of Sgr (Table [illegible]). However, F ratios were significant only for the tibialis anterior, the plantaris, the gastrocnemius medialis and the heart. Furthermore, Scheffé's contrasts indicated that only the plantaris muscles of Cgr were heavier than those of Sgr.

TABLE 4 Final Body Weight for the Different Training Groups: Means, Standard Deviations, ANOVA F ratio and Scheffé's Contrasts.

Sgr[ab] g	Cgr[ab] g	Igr[ab] g	F	P<	Contrasts (P < .05)
625 70	534 42	534 44	7.698	0.005	Cgr < Sgr Igr < Sgr

a. abbreviations: see Table
b. n = 8

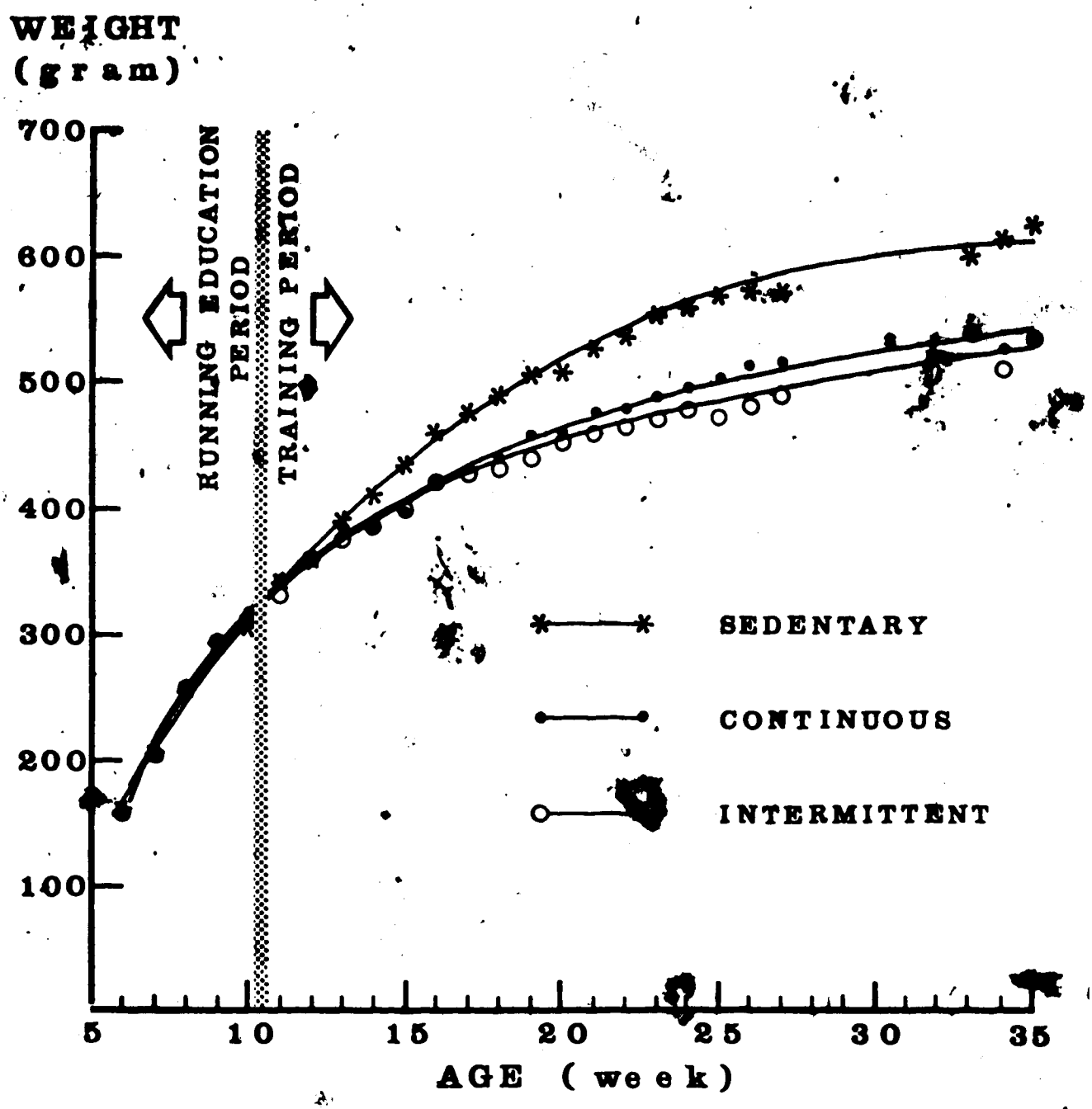

FIGURE 5. RAT BODY WEIGHT GAIN WITH AGE AND TRAINING

(DRAWN FROM TABLE 22, APPENDIX B)

TABLE 5 Absolute Weight of the Organs in the Different Training Groups: Means, Standard Deviations, and ANOVA F Ratios.

Organs[a]	Sgr[ab] mg	Cgr[ab] mg	Igr[ab] mg	F	P<
TA	998 124	1003 47	968 87	0.345	ns
P	562 58	582 53	551 58	0.591	ns
GM	1194 109	1244 100	1222 97	0.491	ns
GL	1440 173	1405 94	1450 189	0.176	ns
S	250 48	258 27	251[c] 31	0.116	ns
H	1496[d] 153	1471[d] 179	1499[c] 77	0.074	ns
L	15997 1364	13957 1544	12389 4852	2.827	ns

a. abbreviations: see Table 1.
b. n = 8
c. n = 7
d. n = 6

TABLE 6 Relative Weight of Organs in the Different Training Groups: Means, Standard Deviations, ANOVA F Ratios and Scheffé's Contrasts.

Organs[a]	Sgr[ab] mg/g	Cgr[ab] mg/g	Igr[ab] mg/g	F	P <	Contrasts (P < .05)
TA	1.62 0.07	1.88 0.10	1.82 0.22	3.635	0.05	Cgr & Igr > Sgr
P	0.91 0.17	1.09 0.05	1.04 0.14	3.783	0.05	Cgr > Sgr
GM	1.94 0.29	2.33 0.09	2.29 0.18	9.296	0.01	Cgr > Sgr Igr > Sgr
GL	2.34 0.47	2.64 0.14	2.72 0.35	2.584	ns	
S	0.41 0.10	0.48 0.03	0.47[c] 0.06	2.508	ns	
H	2.32[d] 0.20	2.73[d] 0.33	2.81[c] 0.27	5.814	0.05	Igr > Sgr
L	25.94 4.30	26.13 2.04	25.93 3.45	0.008	ns	

a. abbreviations: See Table 1.
b. n = 8
c. n = 7
d. n = 6

Adenosine Triphosphate and Phosphorylcreatine

Resting concentrations of ATP and PC as well as ATP + PC are reported for each group and each organ in Tables 7 to 9. Only ATP concentrations showed significant group differences. Scheffé's contrasts further revealed that for the tibialis anterior, ATP was lower in Igr compared to either Cgr or Sgr. For the gastrocnemius medialis and lateralis, ATP was lower in Igr compared to Cgr. Finally, the soleus of Cgr had higher ATP values than the two other groups. Therefore, the general tendency was to have a higher ATP concentration in Cgr, followed by Sgr and then Igr. This was confirmed by analysis of variance and contrasts run on the combined data from all organs (Appendix F).

Creatinephosphokinase and Adenylate Kinase

CPK and AK activities and training group comparisons are reported in Tables 10 and 11. None of the organs showed significant difference between training groups for AK. On the other hand, combined data from all organs indicated that CPK was higher in Sgr compared to the trained groups (Appendix F).

Lactate Dehydrogenase

LDH related variables for group comparisons are reported in Tables 12 to 17. Two way analysis of variance (Appendix F) revealed that LDH_{21}, LDH_3, M-LDH and total LDH

were higher in all organs of Sgr compared to the trained groups. One way analysis of variance showed less significant differences between training groups. LDH_{21} and LDH_3 activities were higher in the gastrocnemius lateralis of Sgr compared to Cgr whereas M-LDH was higher in the heart of Cgr than in Igr. No significant difference between groups characterized the LDH_{21}/LDH_3 ratio (or the percentage of M-LDH and H-LDH) and the H-LDH activity.

Organ Comparison

Organ means, standard deviations and ANOVA F ratios from combined group data* for each dependant variable appear in Table 18. Significant Scheffé contrasts are identified in Table 19. In general, the organs fell into four distinct categories: liver, heart, soleus and the other skeletal muscles.

Adenosine Triphosphate and Phosphorylcreatine (Tables 18 and 19)

PC was the lowest in the heart (5.5 mmoles/kg) slightly higher in the soleus (9.0 mmoles/kg) and much higher in other skeletal muscles (14.2 - 18.3 mmoles/kg). With a few exceptions, ATP and ATP + PC were characterized by a similar pattern. For instance, ATP was higher in the heart (4.2 mmoles/kg) than in the soleus (3.4 mmoles/kg).

---

* Since the differences were much larger between organs than between groups, group data were pooled.

TABLE 7 ATP for Each Organ in the Different Training Groups: Means, Standard Deviations, ANOVA F Ratios and Scheffé's Contrasts.

Organs[a]	Sgr[ab] mmoles/kg wet weight	Cgr[ab] mmoles/kg wet weight	Igr[ab] mmoles/kg wet weight	F	P <	Contrasts (P < .05)
TA	5.8 0.5	5.8 0.9	4.9 0.4	5.875	0.01	Igr < Sgr Igr < Cgr
P	5.1 0.7	5.3[c] 0.8	4.6 0.5	2.324	ns	-
GM	5.2 1.1	5.8 0.9	4.5 0.8	3.775	0.05	Igr < Cgr
GL	4.8 0.5	5.3 1.0	4.3 0.3	4.312	0.05	Igr < Cgr
S	3.3 0.2	3.9 0.5	3.2 0.5	6.924	0.01	Sgr < Cgr Igr < Cgr
H	4.0 0.4	4.5 0.5	4.3 0.4	2.402	ns	

a. abbreviations: see Table 1
b. n = 8
c. n = 7

TABLE 8 PC for Each Organ in the Different Training Groups: Means, Standard Deviations and ANOVA F Ratios.

Organs [a]	Sgr[ab] mmoles/kg wet weight	Cgr[ab] mmoles/kg wet weight	Igr[ab] mmoles/kg wet weight		P
TA	18.9 4.2	18.9 2.0	16.9[c] 2.4	0.979	ns
P	15.9 6.8	13.2 2.6	15.1[c] 3.3	0.617	ns
GM	15.5 2.5	16.8 3.6	16.7 3.5	0.365	ns
GL	14.7[c] 2.6	14.4 2.7	13.4[c] 3.0	0,385	ns
S	8.9 1.8	9.7 3.2	8.0[d] 1.0	0.[illegible]30	ns
H	5.3[c] 1.2	5.9[c] 0.7	5.2[c] 1.8	0.533	ns

a. abbreviations: see Table 1
b. n = 7
c. n = 6
d. n = 5

TABLE 9 ATP + PC for Each Organ in the Different Training Groups: Means, Standard Deviations and ANOVA F Ratios.

Organ[a]	Sgr[ab] mmoles/kg. wet weight	Cgr[ab] mmoles/kg wet weight	Igr[ab] mmoles/kg wet weight	F	P
TA	24.7 4.0	24.7 1.5	21.4[c] 2.4	2.825	ns
P	21.1 6.6	18.2 3.2	19.8[c] 3.6	0.629	ns
GM	20.6 2.9	22.5 4.2	21.3[c] 4.3	0.440	ns
GL	19.7[c] 2.4	19.7 3.3	17.6[c] 3.3	0.949	ns
S	12.2 1.9	13.7 3.4	11.0[d] 1.5	1.766	ns
H	9.3[c] 1.5	10.0[c] 1.5	9.6[c] 2.0	0.318	ns

a. abbreviations: see Table 1
b. n = 7
c. n = 6
d. n = 5

TABLE 10 CPK for Each Organ in the Different Training Groups: Means, Standard Deviations, ANOVA F Ratios and Scheffé's Contrasts.

Organ[a]	Sgr[ab] IU/g wet weight	Cgr[ab] IU/g wet weight	Igr[ab] IU/g wet weight	F	P<	Contrasts (P < .05)
TA	2870[d] 400	2540[c] 410	2420 330	2.463	ns	
P	2780 340	2440[c] 430	2230 440	3.757	0.05	Igr < Sgr
GM	2960 550	2340[c] 440	2350 410	4.492	0.05	Igr & Cgr < Sgr
GL	2770 460	1960 350	1900 390	11.723		Cgr < Sgr Igr < Sgr
S	1040 220	1060 170	1140[c] 90	0.737	ns	
H	810 160	910 180	890 120	0.907	ns	
L	10.2[c] 2.5	17.4 9.4	14.7 10.7	1.358	ns	

a. Abbreviations: see Table 1
b. n = 8
c. n = 7
d. n = 6

TABLE 11. AK for Each Organ in the Different Training Groups: Means, Standard Deviations and ANOVA F Ratios.

Organ[a]	Sgr[ab] IU/g wet weight	Cgr[ab] IU/g wet weight	Igr[ab] IU/g wet weight	F	P <
TA	133[d] 61	122 71	132[c] 36	0.082	ns
P	129[d] 62	124 72	125[c] 39	0.012	ns
GM	121[d] 53	122 74	126[c] 44	0.013	ns
GL	118[d] 52	96[c] 62	113[c] 38	0.389	ns
S	62[d] 25	52[c] 27	65[d] 28	0.439	ns
H	61[d] 18	65 32	71[c] 24	0.281	ns
L	20[d] 3	19[c] 4	20[c] 6	0.066	ns

a. abbreviations: see Table 1.
b. n = 8
c. n = 7
d. n = 6

TABLE 12 LDH_{21} for Each Organ in the Different Training Groups: Means, Standard Deviations, ANOVA F Ratios and Scheffé's Contrasts.

Organ[a]	Sgr[ab] IU/g wet weight	Cgr[ab] IU/g wet weight	Igr[ab] IU/g wet weight	F	P<	Contrasts (P < .05)
TA	587 164	452 123	469 136	2.161	ns	
P	599[c] 111	500 152	503 91	1.567	ns	
GM	482[d] 105	440 110	442[c] 109	0.306	ns	
GL	505 104	362 94	408[c] 121	3.752	0.05	Cgr < Sgr
S	131 40	138[e] 57	117[c] 25	0.459	ns	
H	280[d] 73	289[c], 129	270 66	0.074	ns	
L	325[c] 79	246[d] 90	300[d] 58	1.727	ns	

a. abbreviations: see Table 1.
b. n = 8
c. n = 7
d. n = 6
e. n = 5

TABLE 13 LDH_3 for Each Organ in the Different Training Groups: Means, Standard Deviations, ANOVA F Ratios and Scheffé's Contrasts.

Organ[a]	Sgr[ab] IU/g wet wright	Cgr[ab] IU/g wet weight	Igr[ab] IU/g wet weight	F	P	Contrasts (P < .05)
TA	486 142	364 108	387 120	2.207	ns	
P	510[c] 91	420 138	425 91	1.630	ns	
GM	423[d] 83	354 95	387[c] 93	0.976	ns	
GL	431 91	315 93	351[c] 91	3.667	0.05	Cgr < Sgr
S	143 38	151[e] 59	121[c] 33	0.879	ns	
H	327[d] 94	322[c] 149	334 91	0.019	ns	
L	279[c] 85	207[d] 82	258[d] 68	1.384	ns	

a. abbreviations: see Table 1.
b. n = 8
c. n = 7
d. n = 6
e. n = 5

TABLE 14 LDH_{21}/LDH_3 for Each Organ in the Different Training Groups: Means, Standard Deviations, M-LDH (%) and ANOVA F Ratios.

Organ[a]	Sgr[ab]	Cgr[ab]	Igr[ab]	F	P.
TA	1.21	1.26	1.22	0.445	ns
	0.09	0.11	0.11		
	85.3	91.1	85.6		
P	1.17[c]	1.21	1.20	0.259	ns
	0.12	0.10	0.09		
	79.6	82.7	81.4		
GM	1.14[d]	1.25	1.15[c]	2.182	ns
	0.10	0.11	0.12		
	73.5	90.9	74.2		
GL	1.17	1.17	1.16[c]	0.045	ns
	0.08	0.11	0.12		
	79.2	75.2	77.8		
S	0.92	0.93[e]	0.98[c]	0.538	ns
	0.13	0.13	0.09		
	28.7	28.3	39.6		
H	0.87[d]	0.90[c]	0.82	3.391	ns
	0.08	0.04	0.06		
	14.3	23.6	2.5		
L	1.18[c]	1.20[d]	1.18[d]	0.095	ns
	0.10	0.06	0.10		
	77.8	82.3	78.0		

a. abbreviations: see Table 1.
b. n = 8
c. n = 7
d. n = 6
e. n = 5

TABLE 15 M-LDH for Each Organ in the Different Training Groups: Means, Standard Deviations, ANOVA F Ratios and Scheffé's Contrasts.

Organ[a]	Sgr[ab] IU/g wet weight	Cgr[ab] IU/g wet weight	Igr[ab] IU/g wet weight	F	P	Contrasts (P < .05)
TA	516 164	419 126	415 136	1.284	ns	
P	497[c] 171	428 123	426 80	0.731	ns	
GM	374[d] 143	407 119	346[c] 140	0.398	ns	
GL	417 135	287 83	332[c] 149	2.258	ns	
S	44 54	41[e] 70	52[c] 20	0.092	ns	
H	48[d] 53	80[c] 35	8 58	3.884	0.05	Igr < Cgr
L	264[c] 47	210[d] 68	245[d] 39	1.769	ns	

a. abbreviations: see Table 1
b. n = 8
c. n = 7
d. n = 6
e. n = 5

TABLE 16 H-LDH for Each Organ in the Different Training Groups: Means, Standard Deviations and ANOVA F Ratios.

Organ[a]	Sgr[ab] IU/g wet weight	C[ab] IU/g wet weight	Tgr[ab] IU/g wet weight	F	P
TA	89 102	41 89	68 97	0.505	ns
P	127[c] 120	90 78	97 86	0.323	ns
GM	135[d] 99	40 81	120[c] 106	2.082	ns
GL	110 82	94 81	88[c] 80	0.157	ns
S	109 50	113[e] 65	80[c] 40	0.828	ns
H	290[d] 113	253[c] 116	327 118	0.760	ns
L	68[c] 74	45[d] 40	69[d] 72	0.271	ns

a. abbreviations: see Table 1.
b. n = 8
c. n = 7
d. n = 6
e. n = 5

TABLE 17 Total LDH for Each Organ in the Different Training Groups: Means, Standard Deviations and ANOVA F Ratios.

Organ[a]	Sgr[ab] IU/g wet weight	Cgr[ab] IU/g wet weight	Igr[ab] IU/g wet weight	F	P
TA	605 170	460 128	477 145		ns
P	624[c] 105	512 165	498 65	2.462	ns
GM	509[d] 103	448 114	466[c] 111	0.536	ns
GL	516 124	381 104	420[c] 116	2.916	ns
S	147 40	154[e] 59	133[c] 31	0.392	ns
H	338[d] 92	355[c] 154	335 86	0.060	ns
L	340[c] 91	255[d] 97	314[d] 70	1.578	ns

a. abbreviations: see Table 1.
b. n = 8
c. n = 7
d. n = 6
e. n = 5

TABLE 18 Weights, Metabolite Concentrations and Enzyme Activities in the Different Organs: Means and Standard Deviations from Combined Data for All Training Groups.

Variables[ab]	TA	P	GM	GL	S	H	L
Wabs (mg)	990 89	565 57	1220 100	1432 152	253 35	1489 132	14114 3276
Wrel (mg/g)	1.77 0.23	1.01 0.15	2.19 0.27	2.57 0.37	0.45 0.08	2.63 0.34	26.00 3.24
ATP mmole/kg	5.5 0.8	5.0 0.7	5.2 1.0	4.8 0.8	3.5 0.5	4.2 0.4	
PC mmole/kg	18.3 3.0	14.7 4.6	16.3 3.1	14.2 2.7	9.0 2.3	5.5 1.4	
ATP + PC mmole/kg	23.7 3.0	19.7 4.7	21.5 3.7	19.0 3.0	12.4 2.6	9.7 1.6	
CPK (IU/g)	2590 41	2490 45	2560 54	2210 56	1080 17	870 16	14.2 8.7
AK (IU/g)	128 56	126 57	123 56	108 50	59 26	66 25	20 4
LDH_{21} (IU/g)	503 149	531 124	452 105	426 119	128 39	279 89	292 80
LDH_3 (IU/g)	412 130	449 110	385 91	366 97	137 42	328 109	250 81
$\frac{LDH_{21}}{LDH_3}$	1.23 0.10	1.23 0.10	1.18 0.12	1.16 0.10	0.94 0.11	0.86 0.07	1.19 0.08
M-LDH (IU/g)	450 144	448 126	377 129	350 131	46 48	44 57	241 55
%[c]	87.2	81.2	80.0	77.6	32.0	12.9	79.0
H-LDH (IU/g)	66 94	104 92	94 100	99 78	100 50	292 115	61 62
%[c]	12.8	18.8	20.0	20.4	68.0	87.1	21.0
TOTAL LDH (IU/g)	514 157	541 127	471 108	440 124	144 41	343 109	305 90

a. All ANOVA F ratios for organ comparison were significant ($P < 0.001$).
b. The number of samples per cell was between 19 and 24.
c. Calculated from LDH_{21}/LDH_3 and Figure 4 (Methods and Procedures).

TABLE 19 Weights, Metabolite Concentrations and Enzyme Activities in the Different Organs: Scheffé's Contrasts from Combined Data for All Training Groups.

Contrasts*	W_{abs}	W_{rel}	ATP	PC	ATP + PC	CPK	AK	LDH_{21}	LDH_3	$\frac{LDH_{21}}{LDH_3}$	M-LDH	H-LDH	TOTAL LDH
P-TA	ns	ns	ns	-	-	ns	ns	ns	ns	ns	ns	ns	ns
GM-TA	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns
GM-P	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns
GL-TA	ns	ns	ns	-	-	ns	ns	ns	ns	ns	ns	ns	ns
GL-P	ns	+	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns
GL-GM	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns
S-TA	ns	ns	-	-	-	-	-	-	-	-	-	ns	-
S-P	ns	ns	-	-	-	-	-	-	-	-	-	ns	-
S-GM	ns	-	-	-	-	-	-	-	-	-	-	ns	-
S-GL	ns	-	-	-	-	-	ns	-	-	-	-	ns	-
H-TA	ns	ns	-	-	-	-	-	-	ns	-	-	+	-
H-P	ns	+	ns	-	-	-	-	-	-	-	-	+	-
H-GM	ns	ns	-	-	-	-	-	-	ns	-	-	+	-
H-GL	ns	ns	ns	-	-	-	ns	-	ns	-	-	+	ns
H-S	ns	+	+	-	ns	ns	ns	ns	+	ns	ns	+	+
L-TA	+	+				-	-	-	-	ns	-	ns	-
L-P	+	+				-	-	-	-	ns	-	ns	-
L-GM	+	+				-	-	-	-	ns	-	ns	-
L-GL	+	+				-	-	-	-	ns	ns	ns	-
L-S	+	+				-	ns	+	ns	+	+	ns	+
L-H	+	+				-	ns	ns	ns	+	+	-	ns

* ns: not significant ($P < 0.05$); "+" is a positive difference; "-" is a negative difference.

Creatine Phosphokinase and Adenylate Kinase (Tables 18 and 19)

CPK was much lower in the liver than in any other organ. Heart and soleus CPK were not different but had only half the values found in the other muscles. AK was twice as active in the tibialis anterior, the plantaris and the gastrocnemius as in the soleus, the heart and the liver.

Lactate Dehydrogenase (Tables 18 and 19)

LDH_{21} was found to be lower in the soleus than in the heart and the liver and highest in the other muscles. For LDH_3, the pattern was low activity in the soleus, slightly higher activity in the liver and highest activity in the other muscles including the heart.

The percentage of M-LDH and of H-LDH from Total LDH as indicated by LDH_{21}/LDH_3 ratios was lower in the heart and the soleus than in the liver and other skeletal muscles. M-LDH activity was found to be lowest in the heart and the soleus, much higher in the liver and highest in the other muscles. H-LDH was three times more active in the heart than in any other organ. Total LDH was lowest in the soleus, two times higher in the heart and liver and approximately three times higher in the other skeletal muscles.

CHAPTER V

DISCUSSION

The discussion will be divided into the following two sections: differences between selected muscles and liver and the chronic effects of exercise. For each of the selected parameters, differences between sampled muscles will first be analysed in order to compare the data in the present study with literature values. This comparison between sampled tissues will result in a classification that should make easier the following discussion of the chronic effects of exercise on the selected parameters.

Differences Between Selected Muscles and Liver (Tables 18 and 19)

Since the enzyme and metabolite concentrations usually vary from one fiber type to another, it is appropriate to identify the fiber composition of the muscles selected for this study (Table 20). According to the classifications used by Ariano et al. (1973), Barnard et al. (1970a), Edgerton et al. (1969 and 1975) and Peter (1970), it can be seen that except for the soleus which is predominantly composed of SO fibers, other skeletal muscles are mostly composed of FT fibers with equal proportions of FG and FOG fibers. The soleus and the other skeletal muscles will be identified as the ST soleus and the FT muscles in the following discussion.

TABLE 20 Literature Values of Fiber Type Composition of Selected Rat Skeletal Muscles.

Muscle	Reference	Fiber Types* (%) SO	FOG	FG
TIBIALIS ANTERIOR	Ariano et al. (1973)	2	66	32
	Close (1972)	15-20	40	40-45
PLANTARIS				
Total	Ariano et al. (1973)	6	53	41
Superficial	Edgerton et al. (1969)	15	10	75
Deep	Edgerton et al. (1969)	20	25	55
GASTROCNEMIUS				
Total	Schmalbruch et al. (1975)	15	50	30
Medialis	Ariano et al. (1973)	4	38	58
Lateralis	Ariano et al. (1973)	5	37	58
	Muller (1974)** Untrained	9	19-53	38-71
	Trained	12	34-50	38-55
SOLEUS	Ariano et al. (1973)	84	14	0
	Baldwin et al. (1972)	96	4	0
	Close (1972)	85-90	10-15	0
	Edgerton et al. (1969)	80	20	0
	Schmalbruch et al.(1975)	94	6	0
	Muller (1974)** Untrained	86	13	0
	Trained	96.3	2.7	0

* Slow twitch oxidative, fast twitch high oxidative glycolytic and fast twitch glycolytic respectively.

** Estimation from another classification.

Before considering the tissue or muscle differences for each of the studied parameters, it is worthwhile to note that the enzyme ratios (i.e. LDH/CPK, AK/CPK and LDH/AK) found in this study agree with those reported by Pette (1975).

Lactate Dehydrogenase in Various Tissues

As the assay techniques as well as the enzyme activity units vary from one study to another, activity ratios are more useful than absolute activity in comparing the validity of the present results with previously published findings. As a whole, this study (Tables 18 and 19) revealed that Total LDH activity was the lowest in the soleus (144 IU/g), 2X higher in the heart (343 IU/g) and the liver (305 IU/G), and approximately 3X higher in the other skeletal muscles (440 to 541 IU/g). Karlsson et al. (1975) and Sjodin et al. (1976b) reported that total LDH is proportional to the % of FT fibers which are mostly composed of M-LDH, as was also found in the present study. However, heart was not studied by these investigators and this muscle does not seem to follow a similar trend. The method for calculating total LDH in the present study may partially explain this difference. Total activity is the summation of M-LDH and H-LDH activity calculated from LDH activity at low and high pyruvate concentrations which were found to be optimal for H-LDH and M-LDH respectively (see Chapter III on Methods and Procedures). In other studies (Karlsson et al., 1974b; Peter et al., 1971; Sjodin, 1976a), total LDH was measured at one pyruvate concentration. It is possible that M-LDH

has been favored when compared to H-LDH. To illustrate this rationale, a comparison of the LDH values for the heart and the gastrocnemius lateralis (Tables 18 and 19) can be made. For LDH at high pyruvate concentration, the gastrocnemius (426 IU/g) is much more active than the heart (279 IU/g), which is consistent with the proposed lower LDH activity in ST muscles. However, for LDH at low pyruvate concentrations, the heart (328 IU/g) is similar to the gastrocnemius (366 IU/g); also, Total LDH for the heart 343 IU/g) and the gastrocnemius (440 IU/g) was not significantly different. Thus the ST % alone appears insufficient to explain the level of Total LDH activity. It is speculated that the LDH distribution pattern might be, partially at least, explained by the actual state and potential of oxidative activity of the muscles as well by their ST %. For example, FOG portions of muscles usually have a lower LDH activity than FG portions, although they have the same percentage of FT (or ST) fibers (Baldwin et al., 1973; Peter et al., 1971; York et al., 1974). The LDH activity of FOG and FG muscle portions behaves reciprocally with the muscle's oxidative capacity, as measured by different markers such as citrate synthase, carnitine palmityltransferase, cytochrome a and cytochrome c activities as well as the pyruvate -2-^{14}C and palmitate -U-^{14}C oxidations (Baldwin et al., 1972a, 1977a and b; Holloszy et al., 1975; Peter et al., 1971). Such a relationship is also supported by the two following facts: firstly, muscle samples from

endurance-trained athletes have been shown to possess higher oxidative capacity and lower LDH activity compared to muscle with the same FT/ST fiber ratio found in sedentary subjects (Karlsson et al., 1975; Sjodin et al., 1976b) and secondly, the heart, with the highest oxidative capacity (Baldwin et al., 1977a; Holloszy et al., 1975; Peter et al., 1971) also has lower LDH activity than FT muscles (Tables 18 and 19), and even FOG muscle portions (Peter et al., 1971). Since the oxidative capacity of the soleus is intermediary between the FG and FOG muscle portions, and much lower than the heart (Baldwin et al., 1972a, 1977a and b; Holloszy et al., 1975; Peter et al., 1971), the oxidative capacity of the muscle cannot explain the low LDH activity of the ST soleus found in this study and confirmed by others (Baldwin et al., 1973; Peter et al., 1971). Thus, both the oxidative capacity and the percentage of ST fibers appears to regulate the LDH activity levels. It is possible that at high intensity, work is carried out more by the FT muscles or fibers and less by the ST muscles or fibers, which would in turn, explain the lowest LDH activity of the soleus. In the other cases, and as long as the oxidative potential is not reached, the energy demand must be met either by the aerobic or the anaerobic metabolism and the higher the former, the lower the latter.

Besides the small differences that have been described above between Total LDH and LDH measured at one pyruvate concentration, the two values generally agree. From this

point of view, organ to organ ratios are similar to those reported in the literature for the rat and guinea pig (Baldwin et al., 1973; Doty et al., 1971; Gollnick et al., 1961, 1967; Hickson et al., 1976; Peter et al., 1971; Staudte et al., 1973; York et al., 1974, 1975, 1976), and human skeletal muscles (Gollnick et al., 1974; Karlsson et al., 1975; Sjodin et al., 1976a and b). There does not seem to be much difference between man and rodents although rabbit livers contain 40% M-LDH compared to 95% and more for man and rat (Fine et al., 1963).

M-LDH% values were similar for the soleus and the heart ($\bar{X}$ = 23%) and much higher in the liver and skeletal muscles ($\bar{X}$ = 84%). This finding agrees with other reported values for the rat, guinea pig and human (Fine et al., 1963; Hirota et al., 1976; Peter et al., 1971; Plageman et al., 1960a; Sjodin et al., 1976a and b; Thorling and Jenson, 1966; York et al., 1974, 1975, 1976). Thus these results support the effectiveness of the assay technique used in this study.

Creatine Phosphokinase and Adenylate Kinase in Various Tissues

As shown in Tables 18 and 19, CPK and AK were generally much higher in FT skeletal muscles (2500 and 120 IU/g respectively) than in the ST soleus (1080 and 59 IU/g), the heart (870 and 66 IU/g) and the liver (14 and 19 IU/g. These findings are in accord with the muscle to muscle activity ratios for rat and human tissue reported by others (Bernstein et al., 1973; Dieter, 1970; Jacob et al., 1964;

Haralambie, 1972 and 1973; Kleine and Chlond, 1967; Newsholme and Start, 1973; Oliver, 1955; Pette, 1971; Staudte et al., 1973). In general, CPK and AK are higher in FT muscle as compared to ST muscle. Such a specific pattern might have some implications for the effects of chronic exercise on these enzymes.

Adenosine Triphosphate and Phosphorylcreatine in Various Tissues

ATP and PC values in the present study showed similar patterns of difference from one tissue to the other and are as high as the ones reported in the literature for rat and human (Degenring et al., 1975; Fawaz et al., 1962; Harris et al., 1974; Hultman et al., 1967; Karlsson, 1971a; Leunissen and Piatnek-Leunissen, 1968; Keul et al., 1972, p. 32; Pool et al., 1976; Pourel, 1968; Rabinowitz and Zak, 1975; Swynghedauw et al., 1960). Thus, the difficult problem of rapid tissue fixation (particularly in the heart) seems to have been avoided. As shown in Tables 18 and 19, PC is twice as high in FT skeletal muscles (14 to 18 mmoles/kg) as in the ST soleus (9 mmoles/kg) and the heart (5.5 mmoles/kg).

In general, it can be stated that the glycolytic (LDH) and high-energy phosphate transferase enzymes (CPK and AK) as well as the ATP and PC stores are higher in the most anaerobic tissues.

The Chronic Effects of Exercise

Before discussing the effects of chronic exercise on the different parameters, some general aspects will be analysed. Firstly, the training effects reported in this study cannot be qualitatively characterized since there were no growth control groups in the study. In other words, although the trained groups might have a higher enzyme activity than the sedentary groups, which is a real difference, it cannot be stated that training has increased the enzyme activity because it may have prevented a possible decrease in activity with aging. Nevertheless, it seems appropriate to discuss the differences between the experimental groups.

Secondly, the fact that training was slightly reduced at the end of the training period might have a reversing effect on the parameters studied. However, as judged by the exhausted state of the rats after each training session, it was felt that the rats were still overloaded. Barnard and Peter (1971), who also had to reduce the training load of their animals, reported that cytochrome a and c continued to increase after this reduced work load. Thus, reduced work load might only have slowed down the improvement rate or maintained the activity level. The reasons for such a decrease in training load are not understood at the present time.

Body and Organ Weights (Tables 4 and 6)

As found in other studies (Baldwin et al., 1972; Crews et al., 1969; Dowell et al., 1976; Edington and Cosmos, 1972; Gollnick et al., 1961 and 1967; Holloszy, 1967; Hubbard et al., 1974; Oscai et al., 1971; Pattengale and Holloszy, 1967; Rogozskin, 1976; Ruhling et al., 1973), endurance training reduced by 15% the usual weight gain found in sedentary male rats (Table 4 and Figure 5). This adaptation supports the effectiveness of the endurance training regimen used in the present study. Sprint training has been shown to inhibit rat body weight gain to a similar extent. Staudte et al. (1973) have reported a 10% decrease in rat body weight after 21 days of sprint training and Rhuling et al. (1973) observed a 6-8% decrease in rat body weight after sprint training as compared to 14% and 4% after endurance running and swimming respectively.

The week by week rate of weight gain for trained and sedentary rats is depicted in Figure 5. Some points fell off the curve at one time or another. These variations appear to be normal as judged by other published curves (Booth, 1972; Freminet et al., 1975; Mayer et al., 1954; Muller, 1975) and surely reflect the precision and sensitivity of the mechanism of correction by successive compensation (or the long term regulation of the energy balance) discovered by André Mayer (Mayer, 1968).

Other possible explanations for the week by week abrupt

changes in body weight are 1) the irregularity in the weighing period time, 2) reduced population of the experimental group due to occasional death of a few animals (Table 22, Appendix B), 3) uncontrolled variations in diet or other living conditions of the animals under the care of the animal service of the University and 4) variations in the training load that were continuously adjusted to the estimated exhaustion levels of the rats. For these reasons and because the objective was to study the main effect of the exercise regimen on body weight, a curve of best fit was utilized. The exact causes of the abrupt changes remain to be however elucidated.

Organ weights were also measured to see if training induced hypertrophy. The absolute weights of the organs were similar in the three experimental groups (Table 5). However, the reduced body weight of both training groups may have masked organ hypertrophy since the relative weights of the organs (e.g. organ weight in mg/body weight in g) were higher in the heart and skeletal muscles but not in the liver of the trained groups (Table 6). Heart hypertrophy after endurance running and more particularly after endurance swimming training in rats is well documented (Baldwin et al., 1977a; Codini et al., 1977; Dowell et al., 1976; Gollnick et al., 1967; Hepp et al., 1974; Oscai et al., 1971; Penpargkul and Scheuer, 1970; Walpurger and Anger, 1970). Heart hypertrophy has also been reported after sprint training of the rat (Baldwin et al., 1977a). In skeletal

muscle, endurance training does not seem to induce hypertrophy (Baldwin et al., 1972 and 1977b; Holloszy, 1967; Oscai et al., 1971; Pattengale and Holloszy, 1967). Muller (1974), however, has found hypertrophy of the rat soleus, gastrocnemius and rectus femoris after 12 weeks using these same training regimens. Muller's findings do not differ very much from those studies previously reported. For example, as for most of the previously quoted studies, Muller used female rats and found no statistically significant difference for the body weights of endurance trained and control rats. Nevertheless, Muller believed he should use relative weights to evaluate hypertrophy. No significant hypertrophy, as judged by the absolute weight of the muscles, was reported. With trained male rats, which definitively differ in body weight when compared to control rats, absolute weights of the muscles cannot be used to assess hypertrophy unless the trained muscles were heavier than the non-trained muscles, which is usually not the case. Thus, relative weights were used in the present study, and significant hypertrophy of the muscles was observed. The only known study (Staudte et al., 1973) reporting the effect of sprint training on rat heart and muscles weights might have been too short (21 training days) to be conclusive. Nevertheless, no hypertrophy was found.

The use of organ to body weight ratios, as in the present study, has been questioned (Dowell et al., 1976;

Gollnick et al., 1967; Héroux and Gridgeman , 1958; Muller, 1974 and 1975a; Tanner, 1949). The heart weight, for example, is not linearly proportional to the body weight, and the use of heart to body weight ratios might reveal a false hypertrophy if, as is the case for male rats, there is a concomitant decrease in body weight. The use of female rats or food restriction techniques are often used to avoid this problem (Baldwin et al., 1977; Holloszy, 1967; Oscai et al., 1971). Otherwise, linear or better, logarithm regressed weights must be used (Dowell et al., 1976; Gollnick et al., 1967; Héroux and Gridgeman, 1958). However, the absence of significant correlation between the organ weights and the body weights made uncertain choices of a regressed weight equation. The lack of significant correlation was probably due to the small cell size. Thus, it was decided to use relative weight. As far as skeletal muscle is concerned, the use of muscle weight to body weight ratios is justified since muscle constitutes the major portion of the total body mass (Héroux and Gridgeman, 1958; Muller, 1974 and 1975).

There is one more assumption involved when relative weight or regressed weights are used to assess hypertrophy. It is assumed that the rest of the body, or more accurately, its comportments (fat and lean tissue), as compared to the studied organ, are changing in equal proportion. For example, if the fat is decreased as a results of training,

which is probably the case in the present study,* the organ to body weight ratio will no longer indicate hypertrophy.

True absolute hypertrophy is not completely excluded and may be necessary to meet the overload since muscle cell proliferation is absent in adult muscle tissues. This seems to be the case in pathological myocardial hypertrophy where the contractile properties of the heart are depressed, as opposed to physical training which improves the contractile properties of the heart (Dowell et al., 1976; Hepp et al., 1974; Penpargkul and Scheuer, 1970) and often leads to myocardial hypertrophy as reported earlier. Skeletal muscle might incur some degree of hypertrophy with physical training as indicated by increased fiber area (Gollnick et al., 1973b; Gordon et al., 1967; Muller, 1974). This increase in cell size with chronic but intermittent exercise is probably within the optimal and critical cell size (Goss, 1966; Hubbard et al., 1974 and 1975) and differs from permanent compensatory overload hypertrophy (Baldwin, 1977b; Dowell et al., 1976). Of course, the relative proportion of sarcoplasmic and myofibrillar proteins may change without external hypertrophy with training (Gordon et al., 1967). Edington and Edgerton (1976, p. 230), Goldberg et al. (1975) and Muller (1974) have reported some occasional hyperplasia (fiber splitting or development of satelite

---

* Although fat % was not assessed in this study, the decrease in body weight after training is usually the result of a decrease in fat % (Booth, 1972; Crews et al., 1969; Mayer, 1968).

cells) concomitant with skeletal muscle hypertrophy. In conclusion, both training programs used in this study appear to have increased the relative weight of the heart and skeletal muscle, but not of the liver.

Lactate Dehydrogenase Adaptation to Chronic Exercise (Tables 12 to 17, Appendix F)

From the two way analysis of variance (Appendix F), all the organs of both training groups have lower LDH and M-LDH activities as compared to the sedentary group. As training might have an opposite effect on LDH in different organs (Baldwin et al., 1972 and 1973; Gollnick et al., 1961 and 1967; York et al., 1974, 1975 and 1976) and as the analysis pools together all organs, a more stringent look at the results indicated that the main effect was due to changes in FT skeletal muscles and liver since either no change or a reversed trend was observed in the soleus and in the heart. One way analysis of variance conducted on each muscle revealed, however, only a few significant training effects (Tables 13 to 18). The fact that pooled data from the various muscles and liver resulted in significant effects in a greater number of cases, is acceptable from a statistical and mathematical point of view since pooling increases the degree of freedom and decreases the variance of the sample. Such a difference between one way and two way analyses of variance illustrates the necessity of larger sample groups, particularly when the coefficient of variation (CV) is large, which is the case with LDH activity ($CV = SD \times 100\bar{X}^{-1} = 30\%$).

Lactate Dehydrogenase Adaptation to Endurance Training.

The present results are consistent with the literature which either reports similar effects or no change with the utilization of small sample groups ($n \leq 10$). For instance, Baldwin et al. (1972 and 1973), Costill et al. (1976), Hickson et al. (1976), Karlsson et al. (1975), Suominen and Heikkinen (1975) and York et al. (1975) reported smaller LDH and M-LDH activity in FT muscles of endurance trained animals and man. In the heart, on the contrary, Gollnick et al. (1961 and 1967), Walpurger and Anger (1970), and York et al. (1975 and 1976) reported increased LDH and M-LDH activity. Other studies reported no significant effects of endurance training on the FT muscles (Bohmer, 1969; Bylund et al., 1977; Gollnick et al., 1967; Holloszy, 1971; Molé et al., 1973; Morgan et al., 1971) as well as the heart (Walpurger and Anger, 1970) and the ST soleus (Baldwin et al., 1973). It is interesting to note however that in all these cases, there was a tendency to follow the specific adaptation pattern reported previously. It seems that the small sample size used in these studies might have lead to a type II error. The lack of significant training effects might also be the result of different exercise conditions. For example, Walpurger and Anger (1970) reported a 15% and a 10% increase in myocardial LDH activity after endurance running and swimming respectively, but only the 15% increase was significant. Although the duration of the training regimen might affect the amplitude of the LDH

changes as shown by York et al. (1975 and 1976), this does not seem to be the case for the present study where the training lasted two months longer than others reported in the literature even though the intensity (31m/min, 8% slope) was similar to other studies (Baldwin et al., 1973; Gollnick et al., 1970; Holloszy, 1979; York et al., 1974, 1975, 1976). However, the duration of the training session was at the lower range of the reviewed studies which might partly explain the few significant training effects observed (1X ANOVA). On the other hand, Gollnick et al. (1970) and Fitts et al. (1975) have reported improved oxidative capacity of rat skeletal muscles with running training sessions of similar intensity and duration. In any case, the general tendency with endurance training appears to be a LDH decrease in FT muscle, an increase in the heart, and an increase or no change in the ST soleus.

There might be some exceptions to this pattern of adaptation. Firstly, the fact that training did not change significantly the LDH activity in human muscles (Bylund et al., 1977; Morgan et al., 1971; Sjodin et al., 1976a and b) and the fact that endurance trained athletes have 30 to 68% less LDH activity than sedentary subjects, would suggest some kind of genetic selection. However, training effects are not excluded since athletes are usually in a trained state for many years whereas training studies usually last only a few weeks. Thus, duration of training regimen appears to be an important factor that has not been fully

investigated. Again in each case reported above, there was a 6 to 22% decrease in human skeletal muscle LDH suggesting that the training regimen might have not been long enough to affect significant changes. This might also explain the apparent discrepancies between human and rat studies, where (in the latter case) endurance training significantly reduced the LDH activity of FT muscles as reported earlier. Indeed, the life span of a rat is much shorter than that of a human (1/15). A second fact that might also explain this human-rat controversy is that laboratory rats are relatively sedentary animals when restricted to normal cage activity whereas human control subjects are not. Thus the differences between "sedentary" controls and trained subjects might be greater and more easily significant in rats than in humans. In any case, all reviewed studies, whether dealing with human or other mammals, never show apposite trends. Only one study (Zika et al., 1973) reported a significant increase in the biceps brachii of rats trained "tonically." The undescribed nature of the training regimen as well as the sampled muscle make further discussion uncertain.

At the beginning of the present study, it was believed that the use of a more specific LDH assay technique (see Methodology chapter and this chapter section entitled "Lactate Dehydrogenase in Various Tissues) would yield more conclusive results. However, as shown by the two way and one way analyses of variance, this study simply confirmed what has been previously reported for endurance

training in FT muscles and the heart. It appears that the large coefficient of variation of the LDH values (30%) whether methodological or biological, as compared to other parameters (15% for PC and CPK), is a major problem in this study, and a larger "n" might improve the experimental design.

Heart muscle does not usually increase its oxidative capacity with endurance training (Baldwin et al., 1977a; Holloszy, 1975; Oscai et al., 1977b). The heart is continuously active and is probably closer to the genetic limits of its oxidative potential. It is therefore possible that any substantial increase in the work load and energy demand is met by an increased glycolytic capacity. In the skeletal muscles, the specific LDH adaptation may be explained by the specific recruitment of fiber types. With endurance running, glycogen is depleted faster in ST fibers as compared to FT fibers (Gollnick et al., 1973a and d). Also ST fibers are innervated by the smaller and more excitable motor neurons and are therefore the first to become active (Edington and Edgerton, 1976). Finally, the tension level might have been relatively higher in the ST fibers as compared to FT fibers or muscles. Therefore, the ST soleus has to increase or at least maintain both its oxidative and glycolytic capacities to meet the extra energy demand of endurance running. On the contrary, FT muscles working at a relatively lower intensity increase their oxidative capacity which results in a reduced LDH activity.

To summarize, it seems that endurance training increases Total LDH activity, and more specifically M-LDH of the heart, decreases activity in FT muscles and retains or possibly increases activity in the ST soleus.

Lactate Dehydrogenase Adaptation to Sprint Training. As stated earlier, both intermittent and continuous training groups showed similar LDH adaptations. High intensity, short duration (i.e. sprint) training is less well documented in the literature. Mixed skeletal muscles of sprint and strength trained athletes have been shown to have higher LDH and M-LDH (Costill et al., 1976; Karlsson et al., 1975). None of the sprint training studies on humans were able to demonstrate any significant LDH increases (Sjodin et al., 1976a and b; Thorstensson et al., 1975). On the other hand, sprint training in animals either kept LDH in mixed skeletal muscles at the same level (Staudte et al., 1973) or decreased it (Hickson et al., 1976a; York et al., 1974). These trends are similar to those reported in the present study. Sprint training has also been shown to induce opposite trends between humans (Costill et al., 1976; Thorstensson et al., 1975) and other mammals (Bagby et al., 1972; Saubert et al., 1973; Staudte et al., 1973) for other anaerobic enzymes, such as myosin ATPase, phosphorylase, triosephosphate dehydrogenase, PK, PFK, AK and CPK in mixed skeletal muscle. It must be said however that the equivalence of the sprint training regimens between animals and humans is not clear.

In humans, the energy sources of running at different speeds are well documented (Astrand and Rodahl, 1970, p. 314; Gollnick and Hermansen, 1973; Margaria, 1972). However, it is not certain that sprint training programs as used in animal studies (Bagby et al., 1972; Fitts et al., 1974; Hickson et al., 1976a and b; Ruhling et al., 1973; Saubert et al., 1973; Staudte et al., 1973) are mainly stressing the anaerobic metabolism as originally intended. The duration and the ratio of the work/rest intervals of these programs are quite different and the aerobic metabolism might have been the principal target in some of these training programs when the total duration of the work intervals were long enough. In the present study both forms of training, continuous at 31m/min and intermittent at 70-75m/min with a work/rest interval ratio of 1 min to 4 min, resulted in similar changes in body and organ weight. Also, it has been shown (Léger, 1975): 1) that such intermittent training could be more easily done than the continuous form; 2) that peak blood lactate was similar in both forms of training in the rat whereas; 3) humans can hardly double the speed at which they can run continuously for 1 hour, when training intermittently with the same work/rest intervals; 4) that humans had also higher blood lactate with this intermittent work as compared to the continuous form of running; and 5) that the blood lactate concentrations after such form of running (9 umole/ml) are somewhat lower than the ones reported by Baldwin et al. (1977c) after a 5 min

run at 48 m/min on a 17% slope (14.4 u mole/ml) and by Saubert et al. (1973) after 20 x 30 sec run at 67 m/min interspaced with 30 sec rest intervals (19.4 u mole/ml). This demonstrates that the intermittent training used in this study did not fully stress the anaerobic metabolism of these rats. More than the speed, the duration of the work/rest intervals appears to explain the lower blood lactate levels of the present study as the longer rest intervals (4 min) might have permitted the complete resynthesis of the PC stores before the start of the next work bout (Fox et al., 1969; Hultman et al., 1967a; Margaria, 1972; Piiper and Spiller, 1970). Fedak et al. (1974) have shown that the energy cost of running is double for bipeds as compared to quadripeds who are more efficient at higher speeds because they possess more gait options, passing from trot to gallop. The energy sources of running rats are unknown at the present time and may not follow the same pattern as humans. It has been reported that rats can run at 160 m/min (Guiness book of animal records), well above the speed used in this study. On the other hand, Ruhling et al. (1973) and Hickson et al. (1967a and b) have estimated the physiological limit at 100 m/min for rats in a running wheel. Saubert et al. (1973) have estimated 50 m/min to be the speed that corresponds to the $\dot{V}O_2$max of the rats. Nevertheless, Hickson et al. (1973) found similar decreases in LDH activity of FT and ST muscles with both sprint and endurance training with a concomitant increase in fumarase activity.

These authors used a work/rest interval ratio of 1/4 as in the present study with work intervals of 10 sec however, and the speed of the treadmill was set at 99 m/min. The intermittent training used by Staudte et al. (1973) was even closer to the present study: the speed was set at 80 m/min, slope, at 30°, work intervals, at 45 sec with at least 1 hour rest between the 4 repetitions. After 21 days of training, Staudte et al. (1973) observed no change in LDH of ST and FT muscles. In view of the half-life times for LDH (Fritz et al., 1969 and 1973), 21 days may have been too short to induce any LDH changes. Baldwin et al. (1977a) found greater oxidative capacity improvement when using continuous running with interspersed sprints as compared to steady state running training. Other studies disclosed no difference between sprint and endurance training in animals (Bagby et al., 1972; Fitts et al., 1974; Ruhling et al., 1973). This information suggests that some form of sprint training may stress the aerobic metabolism more than the anaerobic metabolism in rat muscles.

Saubert et al. (1973) did not study LDH, but reported an increase in other glycolytic enzyme activities (e.g. PFK, PH, PK) in the soleus but not in the red and white portions of the gastrocnemius. Staudte et al. (1973) also found increased glycolytic activity in the soleus but not in the fast rectus femoris. Even with endurance training, Baldwin et al. (1973) and Holloszy et al. (1975) found an increased glycolytic activity of the soleus instead of the

usual decrease found in the mixed skeletal muscle. It seems that, with previously reported types of sprint training, the ST soleus behaves reciprocally to FT muscles for the same reasons discussed for endurance training. It is not excluded however that, with other forms (i.e. more strenuous) of sprint training, FT muscles also increase their glycolytic activity as suggested by the higher LDH activity of highly trained human sprinters (Costill et al., 1976; Karlsson et al., 1975; Sjodin et al., 1976a; Thorstenson et al., 1974).

Another point in relation to sprint training is the smaller total training time as opposed to continuous endurance training. This alone may explain the fewer significant LDH adaptations with sprint studies. LDH might be less rate limiting than other glycolytic enzymes and take more time to adapt. It has also been shown that LDH has a longer half-life than other soluble proteins (Don and Master, 1975; Fritz et al., 1969; Schimke, 1973).

As far as liver is concerned, two way analyses of variance indicated that chronic exercise decreased its LDH activity. The decrease in total LDH was more closely related to M-LDH than the H-LDH. This is unexpected since liver is usually seen more as a site of lactate oxidation rather than a site of pyruvate reduction (Keul, 1973; Rowell, 1966 and 1971). However, 1 X ANOVA revealed no significant changes; therefore, one must be cautious in explaining any changes observed in liver LDH with training.

Metabolism of High Energy Compounds and Chronic Exercise.

Endurance and sprint training regimens used in this study failed to increase the high energy compound stores (ATP + PC) in any of the studied tissues (Tables 7 and 9), except for a slight ATP increase for the endurance group as seen from the pooled data from organs (Appendix F). On the other hand, intermittent training did reduce the ATP stores by 10 to 15% as compared to the other groups in all tissues except the heart (Table 7 and Appendix F). The training regimens were not more sufficient in increasing the CPK and AK activities (Table 10 and 11). On the contrary, CPK activities of the fast twitch muscles were generally depressed in both the continuous and intermittent training groups (Table 10 and Appendix F).

The concentration levels of high energy compounds and enzymes might not be very important limiting factors in the kind of work loads used in this study, assuming the overload principle of adaptation to training. The absence of changes in PC levels of the skeletal muscles after continuous and intermittent training is consistent with the findings of Karlsson *et al.* (1972) and Thorstensson *et al.* (1975) on endurance and sprint trained humans respectively. On the other hand, this lack of increase in PC stores as well as the slight decrease in ATP levels of the skeletal muscle of the sprint trained rats as opposed to the endurance train rats are at variance with the findings of Russian workers (Yakolev, 1965; Yampolskaya, 1952 as quoted by Haralambie,

1972; and Rogozskin, 1976). Since the details of the experimental design of these authors are not known, it is hard to make any comment at the present time. FT and ST muscles appear to behave similarly in this study although reciprocal trends have previously been reported by Gale and Nagle (1971). The PC levels reported by these authors as well as their sampling technique and statistical design are however questionable. As far as ATP is concerned, Karlsson et al. (1972) indicated an increase after endurance training in human skeletal muscle, whereas Bohmer (1969) reported no changes in rat gastrocnemius after swimming training which is consistent with the present study. It is possible that these discrepancies simply reflect a statistical artifact (small "n" and large variance) since there was an increasing tendency in each case. The nature of the training regimen as well as the subjects (humans or animals) might also be involved. Nevertheless, it seems that endurance training either increases the ATP level of skeletal muscle or has no effect on it. With sprint training the present study revealed no training effect on muscle ATP.

In the heart, endurance exercise (Degenring et al., 1975 and Scheuer et al., 1970) and other forms of experimentally-induced hypertrophy (Rabinowitz and Zak, 1975) usually result in a decrease or no change in the ATP and PC levels. This is in accord with the present study which demonstrates no training effect on myocardial ATP and PC with either endurance or sprint running. Gangloff et al.

(1961) reported a PC increase in the heart with training but their sampling technique as well as the very low published values are questionable.

As far as CPK is concerned, the present study supports the absence of change found by Walpurger and Anger (1970) but is in opposition to the increase reported by Wagner and Critz (1970) in the heart of endurance trained animals. Heart CPK after sprint training does not seem to have been investigated elsewhere. In the ST soleus, the present findings support the absence of change reported by Dieter (1970) but again are opposed to the increase reported by Wagner and Critz (1970) after endurance training. Since details of Wagner and Critz's study (1970) were not explicited (i.e. abstract), it is worthless to speculate on their results. In sprint-trained rats, Staudte et al. (1973) found an increase in soleus CPK but not in the rectus femoris, whereas in the present study CPK was found to stay at the same level in the soleus and to decrease in FT muscles. This decrease of CPK in fast twitch muscles in sprint trained rats is also in opposition with the increase found in sprint-trained humans (Thorstensson et al., 1975). The 5 second duration of the sprint intervals used by Thorstensson et al. (1975) as opposed to 1 minute intervals in the present study might have imposed greater and more exclusive stress on the CPK reaction. This is supported by the concomitant absence of change in LDH and $\dot{V}O_2max$ reported

by the same authors. Also, as discussed earlier, sprint training regimens might not be equivalent in man and animals. The decrease found in FT skeletal muscles of endurance trained rats is also in opposition with the increase (Wagner and Critz, 1970) or the absence of change reported for endurance trained rats (Bohmer, 1969; Dieter, 1970; Oscai and Holloszy, 1971) and endurance trained humans (Suominen and Heikkinen, 1975).

To summarize, it seems that ST and FT muscles behave reciprocally with either an increase or no change in ST muscles and either a decrease or no change in FT muscles with training depending on the nature of the working loads.

The fact that AK did not show any change with continuous and intermittent training is consistent with findings of Oscai and Holloszy (1971) in the gastrocnemius of endurance trained rats and the findings of Dart and Holloszy (1969) in the heart of rats after experimental hypertrophy (arteriovenous fistula). On the other hand, Walpurger and Anger (1970) reported a 50% and 30% rise in cytoplasmic myocardial AK after endurance swimming and running training respectively. The present data indicate a nonsignificant 20% rise in heart AK after intermittent training. As was the case for LDH, the large coefficient of variation (30-50%) might have hidden a possible rise in heart AK. In this regard, it is interesting to note that Thorstensson et al. (1976a and b) reported either an increase or no

change in strength trained human skeletal muscles on two different occasions using the same training regimen but different subjects. The only known sprint study carried out with humans (Thorstensson et al., 1975) is consistent with the absence of change in AK of FT skeletal muscles after intermittent training.

The absence of change in liver CPK and AK does not appear to be documented in the literature. However, this pattern seems consistent with the apparent lack of functional significance of liver CPK and AK in exercise.

The Effect of Chronic Exercise on Anaerobic Variables in Rat Tissues

Although the previous discussion revealed many unexplained discrepancies and many concurrences with literature data, there appears to be much more consistency when looking at the total metabolism. It seems that the usual increase in oxidative capacity after endurance training (Baldwin et al., 1972; Benzi et al., 1975; Fitts et al., 1975; Gollnick et al., 1970; Holloszy, 1967; Molé et al., 1973; Pattengale and Holloszy, 1967) is more important for fast twitch muscles and can explain the decrease in LDH, M-LDH% and CPK and the absence of change in AK and high energy compound stores. Such a reciprocal behavior between oxidative and non-oxidative enzymes has been previously reported by Pette et al. (1973). These authors found a decrease in LDH, M-LDH, CPK and AK activities concomitant to an increased oxidative activity in rabbit fast twitch

muscles stimulated intermittently for 8 hours a day. On the other hand, the increase in the oxidative capacity might be less important in the heart (Baldwin et al., 1977a; Holloszy, 1975a; Oscai et al., 1971b) and in the soleus (Holloszy et al., 1975) which would explain the retention or the increase of LDH, M-LDH and CPK activities of these muscles. As Baldwin et al. (1972 and 1973) and Holloszy et al. (1975) found similar increases in the oxidative capacity of the soleus as compared to fast twitch muscles, a higher intensity of work for the soleus might also explain the LDH and CPK activity retention in this muscle.

That intermittent and continuous training have similar effects on LDH, CPK and AK activities and on PC stores suggests that many of the selected forms of "sprint training" in animal studies, might be closer to endurance or continuous training. It is not excluded however that more strenuous forms of sprint training would bring more specific effects.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Summary

In view of the importance of anaerobic metabolism in some forms of physical activity, and in view of the few and conflicting related studies, it was decided to investigate the activities of the M and H forms of LDH, and the high energy compound stores (ATP + PC) and their regulatory enzymes (CPK and AK). These parameters were studied in the following tissues: liver, heart, slow twitch soleus, fast twitch gastrocnemius, plantaris and tibialis anterior of rats. Three experimental conditions were established: 1) a continuous endurance training program known to increase the oxidative capacity of the tissues; 2) a high speed intermittent training regimen using a 1 min work and a 4 min rest interval intended to stimulate both anaerobic glycolysis and high energy compound metabolism; and 3) a control or sedentary regimen restricting the rats to normal cage mobility.

The results indicated that similar adaptative changes occured for both training regimens. The forms of "sprint" training used with animals are still empirical and very unclear. Both training regimens resulted in a decreased activity of LDH, M-LDH, and CPK in the fast twitch muscles (tibialis anterior, plantaris, gastrocnemius), without

altering their AK activity and PC stores. In the heart and slow twitch soleus, LDH, M-LDH, CPK and AK activities as well ad PC stores were retained after chronic exercise. No LDH, CPK and AK changes were present in the liver. Both training programs reduced body weight gain and increased the organ weight to body weight ratios of the muscles but not of the liver.

It seems that the increased oxidative capacity usually found with endurance training in rats (Baldwin et al., 1972; Benzi et al., 1975; Fitts et al., 1975; Gollnick et al., 1970; Holloszy, 1967; Molé et al., 1973; Pattengale and Holloszy, 1967) might explain the decrease in LDH, M-LDH% and CPK and the absence of change in AK and high energy compound stores. On the other hand, the absence of adaptation for the oxidative capacity of the heart as found by Baldwin et al. (1977a), Holloszy et al. (1975), Holloszy (1975) and Oscai et al. (1971b), would explain the retention or the increase of LDH, M-LDH and CPK activities. In the soleus, retention of LDH and CPK activities were explained by a higher relative intensity of work for this muscle as compared to other muscles since similar increases in the oxidative capacity of the slow twitch soleus and the fast twitch muscles were also reported after endurance training in rats (Baldwin et al., 1972 and 1973; Holloszy et al., 1975).

Conclusions

Within the limitations of this study, the following conclusions were drawn:

1. Non-oxidative metabolism in heart, liver and slow and fast twitch muscles is affected in a similar manner by high speed intermittent and low speed continuous training in the laboratory rat;

2. Slow twitch and fast twitch muscles appear to adjust their non-oxidative metabolism reciprocally with sprint and endurance training;

3. The large coefficient of variation found with some variables and different results found with the one way and two way (pooled data) analyses of variance indicate the need for larger sample size in future studies to avoid possible statistical artifact.

BIBLIOGRAPHY

Acebal, C., J. Castro, E. Miléndez, and A.M. Municio. "Metabolic Influences on the Lactate Dehydrogenase Isoenzyme Patterns in Several Rabbit Tissues", Int. J. Biochem, 5:23-29, 1974.

Adam, H. "Adenosine -5- Triphosphate. Determination with Phosphoglycerate Kinase", in Bergmeyer, H.U., Methods of Enzymatic Analysis, New York: Academic Press , 1963, p. 539.

Ahlborg, B. "Blood Glucose During Prolonged Physical Exercise in Man", Forsvarsmedicin, 3:3-13, 1967.

Ahlborg, B., J. Bergstrom, L.G. Ekelund, G. Guarmieri, R. Harris, E. Hultman, L.O. Nordesjo. "Muscle Metabolism During Isometric Exercise Performed at Constant Force", J. Appl. Physiol., 33(2): 224-228, 1972.

Altland, P.D., B. Highman. "Effects of Exercise on Serum Enzyme Values and Tissue of Rats", Am. J. Physiol., 201:393-395, 1961.

Amarasingham, C.R. and A. Uong. "An Examination of Reaction Kinetics in Evaluating the Differential Distribution of Lactate Dehydrogenase Isozymes", Ann. N.Y. Acad. Sci., 15(1):424-428, 1968.

Ariano, M.A., R.B. Armstrong, and V.R. Edgerton. "Hindlimb Muscle Fiber Populations of Fine Mammals", J. Histochemistry and Cytochemistry, 21(1):51-55, 1973.

Ashley, C.C. "Calcium and the Activation of the Skeletal Muscle", Endeavour, 30(109):18-25, 1971

Astrand, P.-O., I. Hallbäch, R. Hodman, and B. Saltin. "Blood Lactates after Prolonged Exercise", J. Appl. Physiol., 18:619-629, 1963.

Astrand, P.-O., and K. Rodahl. Textbook of Work Physiology, New-York: McGraw-Hill, 1970.

Baba, N., and H.M. Sharma. "Histochemistry of Lactic Dehydrogenase in Heart and Pectoralis Muscles of Rat", J. Cell. Biol., 51:621-635, 1971.

Bagby, G.J., W.L. Sembrowich, and P.D. Gollnick. "Myosin ATPase and Fiber Composition from Trained and Untrained Rats", Am. J. Physiol., 223(6):1515-1417, 1972.

Bahn, A.K., and J. Scheuer. "Effects of Physical Training on Cardiac Actomyosin Ademosine Triphosphatase Activity", Am. J. Physiol., 223:1486-1490, 1972.

Baldwin, K.M., G.H. Klinkerfuss, R.L. Terjung, P.A. Molé, and J.O. Holloszy. "Respiratory Capacity of White, Red and Intermediate Muscle: Adaptive Response to Exercise", Am. J. Physiol., 222(2): 373-378, 1972a.

Baldwin, K.M., W.W. Winder, P.L. Terjung, and J.O. Holloszy. "Glycolytic Capacity of Red, White, and Intermediate Muscle: Adaptive Response to Running", Med. Sci. Sport, 4(1):50, 1972b.

Baldwin, K.M., W.W. Winder, R.L. Terjung, and J.O. Holloszy. "Glycolytic Enzymes in Different Types of Skeletal Muscle: Adaptation to Exercise", Am. J. Physiol., 225:962-966, 1973.

Baldwin, K.M., D.A. Cooke, and W.G. Cheadle. "Time Course Adaptations in Cardiac and Skeletal Muscle to Different Running Programs", J. Appl. Physiol., 42(2):267-272, 1973a.

Baldwin, K.M., W.G. Cheadle, O.M. Martinez, and D.A. Cooke. "Effect of Functional Overload on Enzyme Lends in Different Types of Skeletal Muscle", J. Appl. Physiol., 42(2):312-317, [redacted]7b.

Baldwin, K.M., P.J. Campbell, and D.A. Cooke. "Glycogen, Lactate, and Alamine Changes in Muscle Fiber Types During Graded Exercise", J. Appl. Physiol., 43(2):288-291, 1977c.

Barany, M. "ATPase Activity of Myosin Correlated with Speed of Muscle Shortening", J. Gen. Physiol., 50:197-220, 1970.

Barengo, E., and J.E. Itoiz. "Rapid Quantitative Evaluation of Serum LDH Isoenzyme Patterns after Gel Etectrophoresis", Experientia, 28(1): 19-20, 1972.

Barnard, R.J., and J.B. Peter. "Effect of Training and Exhaustion on Hexokinase Activity of Skeletal Muscle", J. Appl. Physiol., 27(5):691-695, 1969.

Barnard, R.J., V.R. Edgerton, and J.B. Peter. "Effect of Exercise on Skeletal Muscle, I. Biochemical and Histochemical Properties", J. Appl. Physiol., 28(6):762-766, 1970a.

Barnard, R.J., V.R. Edgerton, and J.B. Peter. "Effect of Exercise on Skeletal Muscle, II. Contractile Properties", J. Appl. Physiol., 28(6):767-770, 1970b.

Barnard, R.J., and J.B. Peter. "Effect of Exercise on Skeletal Muscle, III. Cytochrome Changes", J. Appl. Physiol., 31(6):904-908, 1971.

Beitner, R., and N. Lakat. "Effect of Alloxan Diabetes and Insulin on Lactate Dehydrogenase Isoenzymes in Rat Tissues", Nature New Biology, 241:281-282, 1973.

Ben, M., R.L. Dixon, and R.H.Adamson. "Anesthesia in the Rat", Fed. Proc. , 28(4):1522-1567, 1969.

Benzi, G., P. Panceri, M. De Bernardi, R. Villa, E. Arcelli, L. D'Angello, E. Arrigoni, and F. Bertè. "Mitochondrial Enzymatic Adaptation of Skeletal Muscle to Endurance Training", J. Appl. Physiol., 38(4):565-569, 1975.

Bergström, J., R.C. Harris, E. Huttman, and L.-O. Nordesjö. "Energy rich Phosphagens in Dynamic and Static Work", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New York: Plenum, 1971, pp. 341-355.

Bernstein, L.H., J.M. Horenstein, A.D. Syhers, and P.J. Russell. "Adenylate Kinase in Human Tissue, II. Serum Adenylate Kinase in Myocardial Infarction", J. Mol. Cell. Cardiol., 5:71-85, 1975.

Bevegard, B.S., and J.T. Shepherd. "Regulation of the Circulation During Exercise in Man", Physiol. Rev., 47:178-213, 1967.

Blanchaer, M.C., and M. Van Wijhe. "Isozymes of Lactic Dehydrogenase in Skeletal Muscle", Am. J. Physiol., 202(5):827-829, 1962.

Block, P., M. Van Rijmenant, R. Radjou, A.Y. Van Malsen, and R. Vogeleer. "The Effects of Exhaustive Effort on Serum Enzymes in Man", in Biochemistry of Exercise, Medicine and Sport, edited by J.P. Poortmans, New York: Kaiger, Basel, 1969, Vol. 3, pp. 259-257.

Bloor, C.M., and N.M. Papadopoulos. "Plasma Lactic Dehydrogenase Activity and Myocardial Cellular Changes after Cessation of Training", J. Appl. Physiol., 26(3):371-374, 1969.

Böhmer, D. "Enzymatic Activity in Normal, Trained and Inactivated Muscle", in Biochemistry of Exercise, edited by J.R. Poortmans, New-York/ Basel: Karger, 1969, pp. 249-253.

Booth, M.A. The Effects of Physical Training upon Fat Cell Size and Number in Adipose Tissue of the Rat, Edmonton: Ph. D. Thesis, University of Alberta, 1972.

Borredon, P. "Tracé électrocardiographique et taux de phosphorylcreatine myocardiaque au cours de l'anoxie aigue", C.R. Acad, Sc. Paris, Série D, 265:2066-2069, 1967.

Boström, S., M. Fahlen, A. Hjalmarsson, and R.G. Johansson. "Muscle Enzyme Activities after repeated Ischemia", Int. J. Biochem., 5:359-363, 1974a.

Boström, S.-L., G. Högberg, and R.G. Johansson. "Enzyme Activities in Skeletal Muscle after Contractile Activity in Vitro", Int. J. Biochem., 5:487-494, 1974b.

Boström, S., M. Fahlen, A. Hjalmarsson, and R. Johansson. "Activities of Rat Muscle Enzymes after Acute Exercise", Acta Physiol. Scand., 90:544-554, 1974 c.

Boxer, G.E., and T.M. Devlin. "Pathways of Intracellular Hydrogen Transport", Science, 134(3489):1495-1501, 1961.

Brody, I.A., and W.K. Engel. "Isozyme Histochemistry: the Display of Selective Lactate Dehydrogenase Isozyme in Sections of Skeletal muscle", J. Histochem. Tytochem., 12:687-695, 1964.

Bücher, T., R. Czok, W. Lamprecht, and E. Latzko. "Pyruvate", in Methods of Enzymatic Analysis, edited by Bermeyer, H.U., New York: Academic Press, 1963, p. 253.

Bylund, A.C., T. Bjuro, G. Cederblad, J. Holm, K. Lundholm, M. Sjostiom, K.A. Angquist, and T. Scherstén. "Physical Training in Man, Skeletal Muscle Metabolism in Relation to Muscle Morphology and Running Ability", Europ. J. Appl. Physiol., 36(3):151-170, 1977.

Cahn, R.D., N.O. Kaplan, L. Levine, and E. Zwilling. "Nature and Development of Lactic Dehydrogenase", Science, 136:962-969, 1962.

Cain, D.G., and R.E.Davies. "Breakdown of Adenosine Triphosphate During a Single Contraction of Working Muscle", Biochem. Biophys. Res. Commun., 8(5):361-366, 1962.

Cairella, M., and L. Vecchi. "Osservazioni Sperimentali e Cliniche sull 'Azione Farmocologica e Terapeutica della Fosfocreatina", Clin. Ter., 38:159-170, 1966.

Cartier, P., J.-P. Leroux, and H. Temkine. "Techniques de dosage des intermédiaires de la glycolyse dans les tissus", Ann. Biol. Clin., 25(7-9): 791-813, 1967a.

Cartier, P., J.-P. Leroux, and J.-C. Marchand. "Techniques de dosage des enzymes glycolytiques tissulaires", Ann. Biol. Clin., 25(1-2): 109-136, 1967b

Casten, G.-G. "The Effect of the Aging Process on Acid Soluble Phosphorus Compounds in the Myocardium of Rats", Am. Heart J., 39:353-360, 1950.

Cerretelli, P., and P.E. Di Prampero. "High Energy Phosphate Resynthesis from Anaerobic Glycolysis in Muscle", J. Physiol. (London), 204:115-116, 1969.

Cier, A. "Fatigue neuromusculaire et phosphocreatinate de sodium", Lyon Méd., 213:1517-1520, 1965.

Close, R.I. "Dynamic Properties of Mammalean Skeletal Muscles", Physiol. Rev., 52(1):129-197, 1972.

Codini, M.A., T. Yipintsoi, and J. Scheuer. "Cardiac Responses to Moderate Training in Rats", J. Appl. Physiol., 42(2):262-266, 1977.

Collowick, S.P. "Adenylate Kinase (Myokinase, ADP Phosphomutase)", in Collowick, S.P., and N.O. Kaplan, Methods in Enzymology, New York: Academic Press, 1955, pp. 598-604.

Costill, D.L., J. Daniels, W. Evans, W. Fink, G. Krakenbuhl, and B. Saltin. "Skeletal Muscle Enzymes and Fiber Composition in Male and Female Trach Athletes", J. Appl. Physiol., 40(2):149-154, 1976.

Crews, E.L., K.W. Fuge, L.B. Oscai, J.O. Holloszy, and R.E. Shank. "Weight, Food Intake, and Body Composition, Effects of Exercise and of Protein Deficiency", Am. J. Physiol., 216(2): 359-363, 1969.

Crowley, L.V., and M. Alton. "A Comparison of Four Methods for Measuring Creatine Phosphokinase", Am. J. Clin. Pathology, 53(6):948-955, 1970.

Dart, C.H., and J.O. Holloszy. "Hypertrophied Non-Failing Rat Heart", Circul. Res., 25:245-253, 1969.

Davies, R.E. "Energy-Rich Phosphagens", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New York: Plenum, 1971, pp. 327-339.

Dawson, D.M., T.L. Goodfriend, and N.O. Kaplan. "Lactic Dehydrogenases: Function of the Two Types", Science, 143:929-933, 1964.

Dawson, D.M., and N.O. Kaplan. "Factors Influencing the Concentration of Enzymes in Various Muscle", J. Biol. Chem., 240:3215-3221, 1965.

Degenring, F.H., R. Rubio, and R.M. Berne. "Adenine Nudeotide Metabolism During Cardiac Hypertrophy and Ischemia in Rats", J. Mal. Cell. Cardiol., 7:105-113. 1975.

Dempsey, J.A., and J. Rankin. "Physiologic Adaptations of Gas Transport Systems to Muscular Work in Health and Disease", Am. J Physiol. Med., 46(1):582-647, 1967.

Dieter, M.P. "Enzyme Changes in Red and White Muscle, Liver, and Plasma of Exercised Guinea Pig Maintained on High or Low Levels of Vitamin C", Life Sci., 9(2):301-311, 1970.

Dietz, A.A., and T. Lubrano. "Separation and Quantitation of Lactic Dehydrogenase Isoenzymes by Disc Electrophoresis", Analyt. Biochem., 20:246-257, 1967.

Di Prampero, P.E., P. Cerretelli, and J. Piiper. "Lactic Acid Formation in Gastrocnemius Muscle of the Dog and Its Relation to O_2 Debt Contraction", Respiration Physiol., 8:347-353, 1970.

Don, M., and C.J. Masters. "On the Application of Affinity Chromatography to Turnover Studies on the Lactate Dehydrogenase Isoenzymes", Biochem. Biophys. Acta, 384:25-26, 1975.

Doty, D.H., C.M. Bloor, and B.E. Sobel. "Altered Tissue Lactic Dehydrogenase Activity after Exercise in the Rat", J. Appl. Physiol., 30(4):548-551, 1971.

Dowell, R.T., C.M. Tipton, and R.J. Tomanek. "Cardiac Enlargement Mechanisms with Exercise Training and Pressure Overload", J. Molecular and Cellular Cardiology, 8:407-418, 1976.

Edgerton, V.R., L. Gerchman, and R. Carrow. "Histochemical Changes in Rat Skeletal Muscle after Exercise", Exptl. Neurol., 24:110-123, 1969.

Edgerton, V.R., R.J. Barnard, J.B. Peter, D.R. Simpson, and C.A. Gillespie. "Response of Muscle Glycogen and Phosphorylase to Electrical Stimulation in Trained and Nontrained Guinea Pigs", Exptl. Neurol., 27:46-56, 1970.

Edgerton, V.R., J.L. Smith, and D.R. Simpson. "Muscle Fiber Type Populations of Human Leg Muscles", Histochemical J., 7:259-266, 1975.

Edgerton, V.R. "Neuromuscular Adaptations to Power and Endurance Work", Can. J. Appl. Sport Sci., 1(1):49-58, 1976.

Edington, D.W., and A.C. Cosmas. "Effect of Maturation and Training on Mitochondrial Size Distribution in Rat Hearts", J.Appl. Physiol., 33(6): 715-718, 1972.

Edington, D.W., and V.R. Edgerton. The Biology of Physical Activity, Boston: Houghton Mifflin, 1976.

Edström, L., and B. Nyström. "Histochemical Types and Size of Fibers in Normal Human Muscles", Acta Neural. Scand., 45:257-269, 1969.

Ekblom, B., P.O. Astrand, B. Saltin, J. Stenberg, and B. Wallström. "Effect of Training on Circulatory Response to Exercise", J.Appl. Physiol., 24(4):518-528, 1968.

Engel, W.K. "Fiber-Type Nomenclature of Human Skeletal Muscle for Histochemical Purposes", Neurology, 24:344-248, 1974.

Ennor, A.H., and J.F. Morrison. "Biochemistry of the Phosphagens and Related Guanidines", Physiol. Rev., 38:631-647, 1958.

Eranko, O. "Quenching of Tissues for Freeze-Drying", Acta Anat., 22:331-336, 1954.

Ericksson, B.O., P.D. Gollnick, and B. Saltin. "Muscle Metabolism and Enzyme Activities after Training in Boys 11-13 Years Old", Acta Physiol. Scand., 87:485-497, 1973.

Essen, B., and T. Häggmark. "Lactate Concentration in Type I and II Muscle Fibers During Muscular Contraction in Man", Acta Physiol. Scand., 95:344-346, 1975.

Everse, J., R.L. Berger, and N.O. Kaplan. "Physiological Concentrations of Lactate Dehydrogenases and Substrate Inhibition", Science, 168:1236-1238, 1970.

Everse, J., and N.O. Kaplan. "Lactate Dehydrogenases: Structure and Function", in Master,A., Advances in Enzymology, 37:61-133, 1973.

Exner, G.U., H.W. Staudte, and D. Pette. "Isometric Training of Rats - Effects upon Fast and Slow Muscle and Modification by Anabolic Hormone (Nandrolone Decanoate), I . Female Rats", Pflügers Arch., 345:1-14, 1973a.

Exner, G.U., H.W. Staudte, and D. Pette. "Isometric Training of Rats - Effects upon Fast and Slow Muscle and Modification by Anabolic Hormone (Nandrolone Decanoate), II. Male Rats", Pflügers Arch., 345:15-22, 1973b.

Fabiato, A., and F. Fabiato. "Calcium Release from the Sarcoplasmic Reticulum", Circ. Res., 40(2): 119-129, 1977.

Faupel, R.P., H.J. Seitz, and W. Tarnowski. "The problem of tissue sampling from experimental animals with respect to freezing techniques, anoxia, stress and narcosis. A new method for sampling rat liver tissue and the physiological values of glycolytic intermediates and related compounds", Arch. Biochem. Biophys., 148:509-522, 1972.

Fawaz, E.N., G. Fawaz, and K. von Dahl. "Enzymatic Estimation of Phosphocreatine", Proc. Soc. Exptl. Biol. (N.Y.), 109:38-41, 1962.

Fedak, M.A., B. Pinshow, and K. Schmidt-Nielsen. "Energy Cost of Bipedal Running", Am. J. Physiol., 227(5): 1038-1044, 1974.

Feinstein, M.B. "Effects of Experimental Congestive Heart Facture, Oreabain, and Asphyxia on the High-Energy Phosphate and Creatine Content of the Guinea Pig Heart", Circ. Res., 10:333-346, 1962.

Felig, P., and J. Wahren. "Interrelationship Between Amino Acid and Carbohydrate Metabolism During Exercise: the Glucose Alanine Cycle", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New York: Plenum, 1971, pp. 205-214.

Felig, P. "The Glucose-Alanine Cycle", Metabolism 22:179-207, 1973.

Felig, P., and J. Wahren. "Fuel Homesstasis in Exercise", New Engl. J. Med., 293(21):1078-1084, 1975.

Ferris, N.P. Jr. "The Biochemistry of Anxiety", Sci. Am., 220(2):69-76, 1969.

Fine, I.H., N.O. Kaplan, and D. Kuftinec. "Developmental Changes of Mammalian Lactic Dehydrogenases", Biochemistry, 2:116-121, 1963a.

Fitts, R.H., F.J. Nagle, and R.G. Cassens. "The Adaptation of Myoglobin with Age and Training and Its Relationship to the Three Fiber Types of Skeletal Muscle in Miniature Pig", Europ. J. Appl. Physiol., 33:275-284, 1974.

Fitts, R.H., F.W. Booth, W.W. Winder, and J.O. Holloszy. "Skeletal Muscle Respiratory Capacity Endurance and Glycogen Utilization", Am. J. Physiol., 228(4):1029-1033, 1975.

Fowler, W.M. Jr., S.R. Chowdhury, C.M. Pearson, G. Gardner, and R. Bratton. "Changes in Serum Enzyme Levels after Exercise in Trained and Untrained Subjects", J. Appl. Physiol., 17:943-946, 1962.

Fowler, W.M. Jr., G.W. Gardner, H.H. Kazerunian, and W.A. Lauvstad. "The Effect of Exercise on Serum Enzymes", Arch. Phys. Med. Rehabil., 49:554-565, 1968.

Fox, A.C., and G.E. Reed. "Exchanges of Nucleotide Phosphates in Normal and Facting Canine Hearts", Am. J. Physiol., 210(6):1383-1390, 1966.

Fox, A.C., and G.E. Reed. "Changes in Lactate Dehydrogenase Composition of Hearts with Right Ventricular Hypertrophy", Am. J. Physiol., 216(5):1026-1033, 1969.

Fox, E.L., S. Robinson, and L.Wiegman. "Metabolic Energy Sources During Continuous and Interval Running", J. Appl. Physiol., 27(2):174-178, 1969.

Fredholm, B.B. "The Effect of Lactate in Canine Subcutaneous Adipose Tissue in Situ", Acta Physiol. Scand., 81:110-123, 1970.

Freminet, A., C. Poyart, E. Bursaux, and T. Talbou. "Effect of Physical Training on the Rates of Lactate Turnover and Oxidation in Rats", in Howald, H. and J.R. Poortmans (eds.), Metabolic Adaptation to Prolonged Physical Exercise, Birkhauser Verlag, Basel, 1975.

Fritz, P.J. "Rabbit Muscle Lactate Dehydrogenase 5: Aregulatory Enzyme", Science, New York, 150:364-366, 1965.

Fritz, P.J., E.S. Vesell, E.L. White, and K.M. Pruitt. "The roles of Synthesis and Degradation in Determining Tissue Concentrations of Lactate Dehydrogenase 5", Proc. Nat. Acad. Sci. (U.S.A.), 62:558-565, 1969.

Fritz, P.J., E.L. White, K.M. Pruitt, and E. Vesell. "Lactate Dehydrogenase Isozymes. Turnover in Rat Heart, Skeletal Muscle, and Liver", Biochemistry, 12(21):4034-4039, 1973.

Gale, J.B., and F.J. Nagle. "Changes in ATP and Creatine Phosphate Storage in Skeletal Muscle of Rats Trained at 900 and 7,600 Feet", Nature, 232:342-343, 1971.

Gangloff, E.C., I.L. Hemmings, and R.F. Krause. "Creatine, Creatine and Creatine Phosphate in Normal and Hypertrophied Rat Hearts", Am. J. Physiol., 201(2):363-364, 1961.

Garbus, J., B. Highman, and P.D. Altland. "Serum Enzymes and Lactic Dehydrogenase Isoenzymes after Exercise and Training in Rats", Am. J. Physiol., 207(2):467-472, 1964.

Gardner, G.W., R. Bratton, J.R. Chowdhury, W.M. Fowler Jr., and C.M. Pearson. "Effect of Exercise on Serum Enzyme Level in Trained Subjects", J. Sports Med., 4:103-110, 1964.

Goldberg, A.L., J.D. Ettinger, D.F. Goldspink, and C. Jablecki. "Mechanism of Work - Induced Hypertrophy of Skeletal Muscle ", Med. Sci. Sports, 7(4):248-261, 1975.

Gollnick, P.D., and G.R. Hearn. "Lactic Dehydrogenase Activities of Heart and Skeletal Muscle Exercised Rats", Am. J. Physiol., 201(4): 694-696, 1961.

Gollnick, P.D., P.J. Struck, and T.P. Bogyo. "Lactic Dehydrogenase Activities of Rat Heart and Skeletal Muscle after Exercise and Training", J. Appl. Physiol., 22(4):623-627, 1967.

Gollnick, P.D., R.G. Soule, A.W. Taylor, C. Williams, and C.D. Ianuzzo. "Exercise Induced Glycogenolysis and Lipolysis in the Rat: Hormonal Influence", Am. J. Physiol., 219(3):729-733, 1970.

Gollnick, P.D., R.B. Armstrong, C.W. Saubert IV, K. Piehl, and B. Saltin. "Enzyme Activity and Fiber Composition in Skeletal Muscle of Untrained and Trained Men", J. Appl. Physiol., 33(3):312-319, 1972.

Gollnick, P.D., R.B. Armstrong, W.L. Sembrowick, R.E. Shepherd, and B. Saltin. "Glycogen Depletion Patterns in Human Skeletal Muscle Fibers after Heavy Exercise", J. Appl. Physiol., 34(5):615-618, [illegible]

Gollnick, P.D., R.B. Armstrong, B. Saltin, C.W. Saubert IV, W.L. Sembrowick, and R.E. Shepherd. "Effect of Training on Enzyme Activity and Fiber Composition of Human Skeletal Muscle", J. Appl. Physiol., 34(1):107-111, 1973b.

Gollnick, P.D., and L. Hermansen. "Biochemical Adaptations to Exercise: Anaerobic Metabolism", in Wilmore, J.H. (ed.), Exercise and Sport Sciences Reviews, Vol. 1, pp. 1-45, 1973c.

Gollnick et al., P.D., R.B. Armstrong, C.W. Saubert IV, W.L. Sembrowick, R.E. Shepherd, and B. Saltin. "Glycogen Depletion Patterns in Human Skeletal Muscle Fibers During Prolonged Work", Pflügers Arch., 344:1-12, 1973d.

Gollnick, P.D., and D.W. King. "Energy Release in the Muscle Cell", Med. Sci. Sports, 1:23-31, 1969.

Gollnick, P.D., B. Sjodin, J. Karlsson, E. Hansson, and B. Saltin. "Human Soleus Muscle: A Comparison of Fiber Composition and Enzyme Activities with Other Leg Muscles", Pflügers Arch., 348:247-255, 1974.

Gordon, E.E., K. Kowalski, and M. Fritts. "Protein Changes in Quadriceps Muscle of Rat with Repetitive Exercises", Arch. Phys. Med., 48:296-303, 1967.

Gould, M.K., and W.A. Rawlinson. "Biochemical Adaptation as a Response to Exercise. 1. Effect of Swimming on the Levels of Lactic Dehydrogenase, Malic Dehydrogenase and Phosphorylase in Muscle of 8-, 11- and 15-week-old Rats", Biochem. J., 73:41-44, 1959.

Goss, R.J. "Hypertrophy and Hyperplasia", Science, 153:1615-1620, 1966.

Halonen, P.L., and A. Konttinen. "Effect of Physical Exercise on Some Enzymes in the Serum", Nature, 193:742-744, 1962.

Haralambie, G. "The Adaptation of the Energy Supply in Muscle to Physical Activity", in Keul, J., E. Doll and D. Keppler, Energy Metabolism of Human Muscle, Baltimore: University Park Press, 1972, pp. 244-255.

Haralambie, G. "Neuromuscular Irritability and Serum Creatine Phosphate Kinase in Athletes in Training", Int. Z. Angew. Physiol., 31:279-288, 1973.

Harris, K.C., E. Hultman, and L.-O. Nordesjö. "Glycogen, Glycolytic Intermediates and High Energy Phosphates Determined in Biopsy Samples of Musculus Quadriceps Femoris of Man at Rest. Methods and Variance of Values", Scand. J. Clin. Lab. Invest., 33:109-120, 1974.

Harris, R.C., E. Hultman, L.Kaijser, and L.-O. Nordesjö. "The Effect of Circulatory Occlusion on Isometric Exercise Capacity and Energy Metabolism of the Quadriceps Muscle in Man", Scand. J. Clin. Lab. Invest., 35:87-95, 1975.

Hellung, P.J., M.A. Jensen, and S.C. Sorensen. "Changes in Lactate-Dehydrogenase Isoenzyme Pattern of Human Brains Following Lifelong Exposure to Hypoxia", Acta Physiol. Scand., 87A:15-16, 1973.

Henriksson, J., and J.S. Reitman. "Quantitative Measures of Enzyme Activities in Type I and Type II Muscle Fibers of Man after Training", Acta Physiol. Scand., 97:392-397, 1976.

Hepp, A., M. Hansis, R. Gülch, and R. Jacob. "Left Ventricular Isovolumetric Pressure-Volume Relations, "Diastolic Tone", and contractility in the Rat Heart after Physical Training", Basic Res. Cardiol., 69(5):516-522, 1974.

Hermansen, L., and J.-B. Osnes. "Blood and Muscle pH after Maximal Exercise in Man", J. Appl. Physiol., 32(3):304-308, 1972.

Héroux, O., and N.T. Gridgeman. "The Effect of Cold Acclimation on the Size of Organs and Tissues of the Rat, with Special Reference to Modes of Expression of Results, Can. J. Biochem. Physiol., 36:209-216, 1958.

Hess, B. "Cell and Tissue Disintegration" in Bergmeyer, H.U., Methods of Enzymatic Analysis, New York: Academic Press, 1963, pp. 43-55.

Hickson, R.C., W.W. Heusner, and W.D. Van Huss. "Skeletal Muscle Enzyme Alterations after Sprint and Endurance Training", J. Appl. Physiol., 40(1): 868-872, 1976a.

Hickson, R.C., W.W. Heusner, W.D. Van Huss, J.F. Taylor, and R.E. Carrow. "Effects of an Anabolic Steroid and Sprint Training on Selected Histochemical and Morphological Observations in Rat Skeletal Muscle Types", Europ. J. Appl. Physiol., 35:251-259, 1976b.

Highman, B., and P.D. Altland. "Effect of Exercise and Training on Serum Enzyme and Tissue Change in Rats", Am. J. Physiol., 205:162-165, 1963.

Hirota, Y., E.M. Cohen, and O.H. Bing. "Lactate Dehydrogenase and Isoenzyme Changes in Rats with Experimental Thiamine Deficiency", Metabolism, 25(1):1-8, 1976.

Hohorst, H.J., M. Rein, and H. Bartels. "Studies on the Creatine Kinase Equilibrium in Muscle and the Significance of ATP and ADP Levels", Biochem. Biophys. Res. Commun., 7(2):142-147, 1962.

Holloszy, J.O. "Biochemical Adaptations in Muscle", J. Biol. Chem., 242(9):2278-2282, 1967.

Holloszy, J.O., and L.B. Oscai. "Effect of Exercise on α-Glycerophosphate Dehydrogenase Activity in Skeletal Muscle", Arch. Biochem. Biophys., 130:653-656, 1969.

Holloszy J., L. Oscai, P. Molé, and I. Don. "Biochemical Adaptations to Endurance Exercise in Skeletal Muscle", in Pernow, B and B. Saltin, Muscle Metabolism During Exercise, New York: Plenum Press, 1971, p. 51.

Holloszy, J.O. "Biochemical Adaptations to Exercise: Aerobic Metabolism", in Exercise and Sport Sciences Reviews, edited by J. H. Wilmore, New York: Academic Press, 1973, vol. 1, pp. 45-71.

Holloszy, J.O., P.A. Molé, K.M. Baldwin, and R.L. Terjung. "Exercise Induced Enzymatic Adaptations in Muscle", in Limiting Factors of Physical Performance, edited by J. Keul, Stuttgart: Thieme Publ., 1973, pp. 66-80.

Holloszy, J.O. "Adaptation of Skeletal Muscle to Endurance Exercise", Med. Sci. Sports, 7(3):155-164, 1975.

Holloszy, J.O., F.W. Booth, W.W. Winder, and R.H. Fitts. "Biochemical Adaptation of Skeletal Muscle to Prolonged Exercise", in Metabolic Adaptation to Prolonged Physical Exercise, edited by Howald, H. and J.R. Poortmans, Birkhauser Verlag, Basel, 1975.

Horstman, D., J. Mendez, E.R. Buskirk, R. Boileau, and W.C. Nicholas. "Lipid Metabolism During Heavy and Moderate Exercise", Med. Sci. Sports, 3(1):18-23, 1971.

Hubbard, R.W., J.A. Smooke, W.T. Matthew, J.D. Linduska, and W.D. Bowers. "The Effects of Growth and DNA Content of Rat Soleus, Plantaris and Gastrocnemius Muscles", Growth, 38:171-185, 1974.

Hubbard, R.W., C.D. Iannuzzo, W.T. Matthew, and J.D. Linduska. "Compensatory Adaptations of Skeletal Muscle Composition to a Long-Term Functional Overload", Growth, 39:85-93, 1975.

Hultman, E., J. Bergström, and N. McLennan-Anderson. "Breakdown and Resynthesis of Phosphorylcreatine and Adenosine Triphosphate in Connection with Muscular Work in Man", Scand. J. Clin. Lab. Invest., 19:56-66, 1967a.

Hultman, E. "Physiological Role of Muscle Glycogen in Man, with Special Reference to Exercise", Circulation Research, Suppl. No 1, Vol. 20-21: 1,99-1,114, 1967b.

Hunter, J.B., and J.B. Critz. "Effect of Training on Plasma Enzymes Levels in Man", J. Appl. Physiol., 31(1):20-23, 1971.

Issekutz, B Jr., H.I. Miller, P.Paul, and K. Rodahl. "Effect of Lactic Acid on Free Fatty Acids and Glucose Oxidation in Dogs", Am. J. Physiol., 209(6):1137-[illegible]5.

Issekutz, B. Jr., H.I. Miller, and K. Rodahl. "Lipid and Carbohydrate Metabolism During Exercise", Fed. Proc., 25:1415-1420, 1966.

Jacobs, H, H.W. Heldt, and M. Klingenberg. "High Activity of Creatine Kinase in Mitochondria from Muscle and Brain and Evidence for a Separate Mitochondrial Isoenzyme of Creatine Kinase", Biochem. Biophys. Res. Commun., 16:516-521, 1964.

Jorfeldt, L., and J. Wahren. "Human Forearm Muscle Metabolism During Exercise V. Quantitative Aspects of Glucose Uptake and Lactate Production During Prolonged Exercise", Scand. J. Clin. Lab. Invest., 26:73-81, 1970.

Jorfeldt, L. "Metabolism of L(+)- Lactate in Human Skeletal Muscle During Exercise, Acta Physiol. Scand., Suppl. 338, 1971.

Kaijser, L. "Limiting Factors for Aerobic Muscle Performance", Acta Physiol. Scand., Suppl. 246, 1970.

Kaijser, L. "Oxygen Supply as a Limiting Factor in Physical Performance", in Limiting Factors of Physical Performance, edited by J. Keul, Stuttgart: Thieme, 1973, pp. 145-156.

Kaplan, N.O., M.M. Ciotti, M. Hamolsky, and R.E. Bieber. "Molecular Heterogeneity and Evolution of Enzymes", Science, 131:392-397, 1960.

Kaplan, N.O., and M. Ciotti. "Evolution and Differentiation of Dehydrogenases", Ann. N.Y. Acad. Sci., 94:701-722, 1961.

Kaplan, N.O., and R.D. Cahn. "Lactic Dehydrogenases and Muscular Dystrophy in the Chicken", Proc. Nat. Acad. Sci., 48:2123-2130, 1962.

Kaplan, N.O., J. Everse, and J. Admiral. "Significance of Substrate Inhibition of Dehydrogenases", Ann. N.Y. Acad. Sci., 151(1):400-412, 1968.

Karlsson, J., P.-O. Astrand, B. Ekblom. "Training of the Oxygen Transport System in Man", J. Appl. Physiol., 22(6):1061-1065, 1967.

Karlsson, J., B. Diamant, and B. Saltin. "Lactate Dehydrogenase Activity in Muscle after Prolonged Severe Exercise in Man", J. Appl. Physiol., 25(1):88-91, 1968.

Karlsson, J. "Lactate and Phosphagen Concentrations in Working Muscle of Man", Acta Physiol. Scand., Suppl. 358, 1971a.

Karlsson, J., L.-O. Nordesjö, L. Jorfeldt, and B. Saltin. "Muscle Lactate, ATP and CP Levels During Exercise after Physical Training in Man", J. Appl. Physiol., 33(2):199-203, 1972.

Karlsson, J., K. Frith, B. Sjödin, P.D. Gollnick, and B. Saltin. "Distribution of LDH Isozymes in Human Skeletal Muscle", Scand. J. Clin. Lab. Invest., 33:307-312, 1974a.

Karlsson, J., B. Hultén, and B. Sjödin. "Substrate Activation and Product Inhibition of LDH Activity in Human Skeletal Muscle", Acta Physiol. Scand., 92:21-26, 1974b.

Karlsson, J., B. Sjödin, A. Thorstensson, B. Hultén, and K. Frith. "LDH Isozymes in Skeletal Muscles of Endurance and Strength Trained Athletes", Acta Physiol. Scand., 93:150-156, 1975.

Karsten, U., A. Kössler, E. Janiszewski, and A. Wollenberger. "Influence of variations in pericellular oxygen tension of individual cell growth, muscle-characteristic proteins, and lactate dehydrogenase isoenzyme pattern in cultures of beating rat heart cells", In Vitro, 9(3):139-146, 1973.

Katona, E. "The Influence of Short Ether Anesthesia on Some Liver Enzyme Activities in the Rat", Can. J. Physiol. Pharmacol., 51:604-607, 1973.

Kendrick-Jones, J., and S.V. Perry. "Enzymatic Adaptation to Contractile Activity in Skeletal Muscle", Nature, 208:1068-1070, 1965.

Keul, J., E. Doll, and D. Keppler. "The Substrate Supply of the Human Skeletal Muscle at Rest, During and After Work", Experientia, 23:1-6, 1967.

Keul, J. "Myocardial Metabolism in Athletes", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New York: Plenum Press, 1971, pp. 447-455.

Keul, J., E. Doll, and D. Keppler. Energy Metabolism of Human Muscle, Baltimore: University Park Press, 1972.

Keul, J. "The Relationship Between Circulation and Metabolism During Exercise", Med. Sci. Sport, 5(4):209-219, 1973a.

Keul, J., and E. Doll. "Intermittent Exercise: Metabolites, Po_2, and Acid Base Equilibrium in the Blood", J. Appl. Physiol., 34(2):220-225, 1973b.

Kleine, T., and H. Chlond. "Enzymmuster gesunder Skelett-, Herz- und glatter Muskulatur des Menschen sowie ihrer pathologischen Veränderungen mit besonderer Berücksichtigung der progressinen Muskeldystrophie", Clin. Chim. Acta, 15:19-33, 1967.

Klingenberg, M. "Control Characteristics of the Adenine Nucleotide System", in Control of Energy Metabolism, edited by Chance B., R.W. Estabrook, and J.R. Williamson, New York: Academic, 1965, pp. 149-155.

Knuttgen, H.G., and B. Saltin. "Oxygen Uptake, Muscle High-Energy Phosphates, and Lactate in Exercise under Acute Hypoxic Conditions in Man", Acta Physiol. Scand., 87:368-376, 1973.

Kowalski, K., E.E. Gordon, A. Martinez, and J. Adamek. "Changes in Enzyme Activities of Various Muscle Fiber Types in Rat Induced by Different Exercises", J. Histochem. Cytochem., 17(9):601-607, 1969.

Kraus, H. "Influence of Exercise on Electron Transport Capacity of Heart Mitochondria", in Muscle Metabolism during Exercise, edited by B. Pernow and B. Saltin, New York: Plenum Press, 1971, pp. 63-68.

Lamprecht, W., and I. Trautschold. "Adenosine-5- Triphosphate. Determination with Hexokinase and Glucose-6- Phosphate Dehydrogenase" in Methods of Enzymatic Analysis, edited by Bermeyer, H.U., New York: Academic Press, 1963, p. 543.

Lamprecht, W., and P. Stein. "Creatine Phosphate", in Methods of Enzymatic Analysis, edited by Bermeyer, H.U., New York: Academic Press, 1963, pp. 610-616.

Latner, A.L., S.A. Siddiqui, and A.W. Skillen. "Pyruvate Inhibition of Lactate Dehydrogenase Activity in Human Tissue Extracts", Science, N.Y., 154:527-529, 1966.

Latner, A.L., and A.W. Skillen. Isoenzymes in Biology and Medecine, New York: Academic Press, 1968.

Léger, L. "Etude comparative du lactate sanguin chez le rat et l'humain en course continue et en course intermittente", Communication presented at the Annual Meeting of the Canadian Association of Sport Sciences, Ottawa, 1975.

Leunissen, R.L.A., and D.A. Piatnek-Ieunissen. "A Device Facilitating in Situ Freezing of Rat Heart with Modified Wollenberger Tongs", J. Appl. Physiol., 25(6):769-771, 1968.

Lowry, O.H., J.V. Passonneau, F.X. Haselberger, and D.W. Shulz. "Effect of Isch[redacted]on Known Substrates and Cofactors of the Gly[redacted]tic Pathway in Brain", J. Biol. Chem. 239(1):18-[redacted] 1964.

Lowry, O.H., and J.V. Passonneau. "The Relationship Between Substrates and Enzymes of Glycolysis in Brain", J. Biol. Chem., 239(1):31-42, 1964.

Lowry, O.H., and J.V. Passonneau. A Flexible System of Enzymatic Analysis, New York: Academic Press, 1972.

Mahler, H.R., and E. Cordes. Biological Chemistry, New York: Harper & Row, 1966.

Maréchal, G. "Les sources d'énergie immédiate de la contraction musculaire", J. Physiol. (Paris), 65:5A-50A, 1972.

Margaria, R., H.T. Edwards, and D.B. Dill. "The Possible Mechanism of Contracting and Paying the Oxygen Debt and the Role of Lactic Acid in Muscular Contraction", Am. J. Physiol., 106:689-715, 1933.

Margaria, R. "Capacity and Power of the Energy Processes in Muscle Activity: their Practical Relevance in Athletics", Int. Z. angew. Physiol., 25:352-360, 1968.

Margaria, R. "The Sources of Muscular Energy", Sci. Am., 226:84-92, 1972.

Margreth, A., G. Salviati, and U. Carraro. "Neural Control on the Activity of the Calcium-Transport System in Sarcoplasmic Reticulum of Rat Skeletal Muscle", Nature, 241:285-286, 1973.

Mayer, J., N.B. Marshall, J.J. Vitale, J.H. Christensen, M.B. Mashayekhi, and F.J. Store. "Exercise, Food Intake and Body Weight in Normal Rats and Genetically Obese Adult Mice", Am. J. Physiol., 177:544-548, 1954.

Mayer, J. Overweight, Causes, Cost and Control, Englewood Cliffs: Prentice Hall, 1968.

McGilvery, R.W., and T.W. Murray. "Calculated Equilibria of Phosphocreatine and Adenosine Phosphates During Utilization of High Energy Phosphate by Muscle", J. Biol. Chem., 249(18):5845-5850, 1974.

McMillan, P.J. "Differential Demonstration of Muscle and Heart Type Lactic Dehydrogenase of Rat Liver, Muscle and Kidney", J. Histochem. Cytochem., 15:21-31, 1967.

Meijer, A.E.F.H. "Semipermeable Membranes for Improving the Histochemical Demonstration of Enzyme Activities in Tissue Sections, III. Lactate Dehydrogenase", Histochemie, 35:165-172, 1973.

Millar, D.B. "The quaterny structure of lactate dehydrogenase, II. The Mechanisms, Kinetics and Thermodynamics of Dissociation Denaturation and Hybridization in Ethylene Glycol", Biochem., Biophys. Acta, 359:152-176, 1974.

Molé, P.A., K.M. Baldwin, R.L. Terjung, and J.O. Holloszy. "Enzymatic Pathways of Pyruvate Metabolism in Skeletal Muscle: Adaptations to Exercise", Am. J. Physiol., 224(1):50-54, 1973.

Mommaerts, W.F.H.M. "Energetics of Muscular Contraction", Physiol. Rev., 49(3):427-508, 1969.

Morgan, T., L. Cobb, F. Short, R. Ross, and D. Gunn. "Effects of Long-Term Exercise on Human Muscle Mitochondria", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New York: Plenum Press, 1971.

Müller, W. "Temporal Progress of Muscle Adaptation to Endurance Training in Hind Limb Muscles of Young Rats", Cell. Tiss. Res., 156:61-87, 1974.

Müller, W. "A Method for the Comparison of Morphometrical Data on Skeletal Muscles in Young Rats of Different Ages and Body Weights", Histochemistry, 43:241-248, 1975a.

Müller, W. "Isometric Training of Young Rats - Effects upon Hind Limb Muscles", Cell. Tiss. Res., 161:225-237, 1975b.

Myers, J.L. Fundamentals of Experimental Design, Boston: Allyn and Bacon, 1966.

Nielsen, L., and B. Ludvigsen. "Improved Method for Determination of Creatine Kinase", J. Lab. & Chem. Med., 62:159-168, 1963.

Newsholme, E.A., and C. Start. Regulation in Metabolism, London: Wiley, 1973.

Novasadova, J. "Lactic Dehydrogenase Isoenzymes in Serum and Tissues after Exercise in Rat", in Biochemistry of Exercise, Medecine and Sport, edited by J.R. Poortman, New York/ Basel: Karger, 1969, Vol. 3, pp. 254-258.

Nuttall, F.Q., and B. Jones. "Creatine Kinase and Glutamic Oxaloacetic Transaminase Activity in Serum: Kinetics of Changes with Exercise and Effect of Physical Conditioning", J. Lab. Clin. Med., 71:847-857, 1968.

Oliver, I.T. "A Spectrophotometric Method for Determination of Creatine Phosphokinase and Myokinase", Biochem. J., 61:116-122, 1955.

Oscai, L., and J. Holloszy. "Biochemical Adaptations in Muscle, II. Response of Mitochondrial Adenosine Triphosphatase, Creatine Phosphokinase and Adenylate Kinase Activities in Skeletal Muscle to Exercise", J. Biol. Chem., 246: 6968-6972, 1971a.

Oscai, L.B., P. Molé, and J.O. Holloszy. "Effects of Exercise on Cardiac Weight and Mitochondria in Male and Female Rats", Am. J. Physiol., 220:1944-1948, 1971b.

Osnes, J.-B., and L. Hermansen. "Acid-Base Balance after Maximal Exercise of Short Duration", J. Appl. Physiol., 32(1):59-63, 1972.

Papadopoulos, N.M., A.S. Leon, and C.M. Bloor. "Effect of Exercise on Plasma Lactate Dehydrogenase and Isoenzyme Activities in Trained and Untrained Rats", Proc. Soc. Exptl. Biol. Med., 129: 232-234, 1968.

Pattengale, P.K., and J.O. Holloszy. "Augmentation of Skeletal Muscle Myoglobin by a Program of Treadmill Running", Am. J. Physiol., 213(3):783-785, 1967.

Penney, D.G., L.B. Bugaisky, and J.R. Mieszala. "Lactate Dehydrogenase and Pyruvate Kinase in Rat Heart During Sideropenic Anemia", Biochem. Biophys. Acta, 334:24-30, 1974.

Penpargkul, S., and J. Scheuer. "The Effect of Physical Training of the Mechanical and Metabolic Performance of the Rat Heart", J. Clin. Invest., 49:1859-1868, 1970.

Pernow, B., and B. Saltin (eds.). Muscle Metabolism during Exercise, New York: Plenum Press, 1971.

Peter, J.B. "Histochemical, Biochemical, and Physiological Studies of Skeletal Muscle and Its Adaptation to Exercise", in Contractility of Muscle Cells and Related Processes, edited by R.J. Podolsky, Englewood Cliffs: Prentice-Hall, 1970, pp.151-133.

Peter, J.B., S. Sawaki, R.J. Barnard, V.R. Edgerton, and C.A. Gillespie. "Lactate Dehydrogenase Isoenzymes: Distribution in Fast-Twitch Red, Fast-Twitch White, and Slow-Twitch Intermediate Fibers of Guinea Pig Skeletal Muscle", Arch. Biochem. Biophys., 144(1):304-307, 1971.

Pette, D. "Metabolic Differenciation of Distinct Muscle Types at the Level of Enzymatic Organization", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New-York: Plenum Press, 1971, pp. 39-49

Pette, D., M.E. Smith, H.W. Staudte, and G. Vrbova. "Effects of Long-Term Electrical Stimulation on Some Contractile and Metabolic Characteristics of Fast Rabbit Muscles", Pflügers Archiv. (Europ. J. Physiol.), 338:257-272, 1973.

Pette, D. "Some Aspects of Supramollecular Organization of Glycogenolytic and Glycolytic Enzymes in Muscle", Acta Histochem., 14,S:47-68, 1975.

Pfleiderer, G., and E.D. Wachsmuth. "Alters-und funktionsabhängige Differenzierung der Lactatdehydrogenase menschlicher Organe", Biochem. Z., 334:185, 1961.

Piehl, K. "Glycogen Storage and Depletion in Human Skeletal Muscle Fibers", Acta Physiol. Scand., Suppl. 402, 1974.

Piiper, J., and P. Spiller. "Repayment of O_2 Debt and Resynthesis of High-Energy Phosphates in Gastrocnemius Muscle of the Dog", J. Appl. Physiol., 28(5):657-662, 1970.

Plagemann, P.G.W., K.F. Gregory, and F. Wroblewski. "The Electrophoretically Distinct Forms of Mammalian Lactic Dehydrogenase, I. Distribution of Lactic Dehydrogenases in Rabbit and Human Tissues", J. Biol. Chem., 235(8): 2282-2287, 1960a.

Plagemann, P.G.W., K.F. Gregory, and F.W. Wroblewski. "The Electrophoretically Distinct Forms of Mammalian Lactic Dehydrogenase, II. Properties and Interrelationships of Rabbit and Human Lactic Dehydrogenase Isozymes", J. Biol. Chem., 235(8):2288-2293, 1960b.

Pool, P.E., G.F. Norris, R.M. Lewis, and J.W. Covell. "A Biopsy Drill Permitting Rapid Freezing", J. Appl. Physiol., 24(6):832-833, 1968.

Porter, K.R., and C. Franzini-Armstrong. "The Sarcoplasmic Reticulum", Sci. Am., 212(3):72-80,1965.

Pourel, J. La phosphocreatine du muscle squelettique humain, Paris: Doin, 1968.

Pruett, E.D.R. "Glucose and Insulin During Prolonged Work Stress in Men Living on Different Diets", J. Appl. Physiol., 28(2):199-208, 1970.

Quarto di Palo, F.M., A.M. Bertolini, and L. Gastaldi. "Il metabolismo dei corpi creatinici nell' animale senescente", Giorn. Geront., 8:85-89, 1960.

Rabinowitz, M., and R. Zak. "Mitochondria and Cardiac Hypertrophy", Circ. Res., 36:367-376, 1975.

Ratner, J.H., P. Nitisewojo, S. Hirway, and H.O. Hultin. "A Study of Some Factors Involved in the Interaction Between Lactate Dehydrogenase Isoenzymes and Particulate Fractions of Muscle", Int. J. Biochem., 5:525-533, 1974.

Raven, P.B., T.J. Conners, and E. Evonuk. "Effects of Exercise on Plasma Lactic Dehydrogenase Isoenzymes and Catecholamines", J. Appl. Physiol., 29(3):374-377, 1970.

Rawlinson, W.A., and M.K. Gould. "Biochemical Adaptation as a Response to Exerercise 2. Adenosine Triphosphatase and Creatine Phosphokinase Activity in Muscle of Exercised Rats", Biochem. J., 73:44-48, 1959.

Robinson, S., and P.M. Harmon. "The Lactic Acid Mechanism and Certain Properties of the Blood in Relation to Training", Am. J. Physiol., 132:757-769, 1941.

Rogozkin, V.A. "The Effect of the Number of Daily Training Sessions on Skeletal Muscle Protein Synthesis", Med. Sci. Sports, 8(4):223-225, 1976.

Rosalki, S.B. "An Improved Procedure for Serum Creatine Phosphokinase Determination", J. Clin. Lab. Med., 69:696-705, 1967.

Rosalki, S.B. Diagnostic Enzymology, 2nd Ed. Dade Lit. No. CH-3, (Divison American Hospital Supply Corporation), Miami, Florida U.S.A., 1969.

Rosalki, S.B. "Standardisation of Isoenzyme Assays with Special Reference to Lactate Dehydrogenase Isoenzyme Electrophoresis", Clin. Biochem., 7:29-40, 1974.

Rose, L.I., S.L. Lowe, D.R. Carroll, S. Wolfson, and K.H. Cooper. "Serum Lactate Dehydrogenase Isoenzyme Changes after Muscular Exertion", J. Appl. Physiol., 28(3):279-281, 1970a.

Rose, L.I., J.E. Bousser, and K.H. Cooper. "Serum Enzymes after Maraton Running", J. Appl. Physiol., 29(3):355-357, 1970b.

Rowell, B.L., K.K. Kroning II, T.O. Evans, J.W. Kennedy, J.K. Blackmon, and F. Kussumi. "Splanchic Removal of Lactate and Pyruvate During Prolonged Exercise in Man", J. Appl. Physiol., 21(6):1773-1783, 1966.

Rowell, L.B. Circulation Med. Sci. Sports, 1(1):15-22, 1969.

Rowell, L.B. "The Liver as an Energy Source in Man During Exercise", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New York: Plenum Press, 1971, pp. 127-141.

Rowell, L.B. "Human Cardiovascular Adjustments to Exercise and Thermal Stress", Physiol. Rev., 54(1):75-159, 1974.

Ruhling, R.O., W.D. van Huss, W.W. Heusner, R.E. Carrow, and S.D. Sleight. "Histochemical and Morphological Observations on Rat Myocardium after Exercise", Int. Z. Angew. Physiol., 31:306-313, 1973.

Saks, V.A., G.B. Chernousova, Iu.I. Voronkov, V.N. Smirnov, and E.I. Chazov. "Study of Energy Transport Mechanism in Myocardial Cells", Circul. Res., Suppl. III:138-149, 1974.

Saltin, B., B. Blomqvist, J.H. Mitchell, R.L. Johnson Jr., K. Wildenthal, and C.B. Chapman. "Response to Submaximal and Maximal Exercise after Bed Rest and Training", Circulation 38: Suppl. 7, 1968.

Saltin, B., and J. Karlsson. "Muscle ATP, CP and Lactate During Exercise after Physical Conditioning", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New York: Plenum Press, 1971a, pp. 395-399.

Saltin, B., and B. Essén. "Muscle Glycogen, Lactate, ATP and CP in Intermittent Exercise", in Muscle Metabolism During Exercise, edited by B. Pernow and B. Saltin, New York: Plenum Press, 1971b, pp. 419-424.

Saltin, B. "Metabolic Fundamental in Exercise", Med. Sci. Sports, 5(3):137-146, 1973.

Samaka, F.J., L. Guth, and R.W. Albers. "Differences Between Slow and Fast Muscle Myosen: Adenosine Triphosphatase Activity and Release of Associated Protein by Chloromercuriphenyl-sulfonate", J. Biol. Chem., 245:219-224, 1970.

Sander, T.M., and C.M. Bloor. "Effects of Repeated Endurance on Serum Enzymes Activities in Well-Conditioned Males", Med. Sci. Sports, 3(1):44-47, 1975.

Saubert, C.W. IV, R.B. Armstrong, R.E. Shepherd, and P.D. Gollnick. "Anaerobic Enzyme Adaptations to Sprint Training in Rats", Pflügers Arch., 341:305-312, 1973.

Sawyer, D.C., W.V. Lumb, and H.L. Stone. "Cardiovascular Effects of Halothane, Methopyflurane, Pentobarbital, and Thiamytal", J. Appl. Physiol., 30(1):36-43, 1971.

Scheffé, H.A. The Analysis of Variance, New York: Wiley, 1959.

Scheuer, J., L. Kapner, G.A. Stringfellow, C.L. Armstrong, and S. Penpargkul. "Glycogen, Lipid, and High Energy Phosphate Stores in Hearts from Conditioned Rats", J. Lab. Clin. Med., 75:924-930, 1970.

Schmalbruck, H., and Z. Kamieniecka. "Histochemical Fiber Typing and Staining Intensity in Cat and Rat Muscles", J. Histochem. Cytochem., 23(6):395-401, 1975.

Schmidt, E., and F.W. Schmidt. "Enzyme Modifications During Activity", in Biochemistry of Exercise, Medicine and Sport, edited by J.K. Poortmans, Baltimore (U.S.A.): University Park Press, 1969, Vol. 3, pp. 216-218.

Seraydarian, M.W., L. Artaza, and B.C. Abbott. "Creatine and the Control of Energy Metabolism in Cardiac and Skeletal Muscle Cells in Culture", J. Mol. Cell. Cardiol., 6:405-413, 1974.

Shields, R.P., C.K. Whitehair, R.E. Carrow, N.W. Heusner, and W.D. Van Huss. "Skeletal Muscle Function and Structure after Depletion of Creatine", Lab. Invest., 33(2):151-158, 1975.

Siest, G, and M.M. Galteau. "Variations in Plasmatic Enzymes During Exercise", Enzyme, 17:179-195, 1974.

Sjödin, B., H.T. Thorstensson, K. Frith, and J. Karlsson. "Effect of Physical Training on LDH Activity and LDH Isozyme Pattern in Human Skeletal Muscle, Acta Physiol. Scand., 97(2):150-157, 1976.

Sjödin, B. "Lactate Dehydrogenase in Human Skeletal Muscle", Acta Physiol. Scand., Suppl. 436, 1976.

Sreter, F.A., J.C. Seidel, and J. Gergely. "Studies on Myosin from Red and White Skeletal Muscles of the Rabbit: ATPase Activity", J. Biol. Chem., 241:5772-5776, 1966.

Stainsby, W.N. "Critical Oxygen Tensions in Muscle", in Limiting Factors of Physical Performance, edited by J. Keul, Stuttgart: Thieme, 1973, pp. 137-144.

Stambaugh, R., and D. Post. "Substrate and Product Inhibition of Rabbit Muscle Lactic Dehydrogenase Heart (H_4) and Muscle (M_4) Isozymes", J. Biol. Chem., 241:1462-1467, 1966a.

Stambaugh, R., and D. Post. "Effects of Tissue Extracts and Temperature on Lactate Dehydrogenase Isozymes", Biochem. Biophys. Acta, 122:541-543, 1966b.

Staudte, H.W., G.U. Exner, and D. Pett[illegible] Effects of Short-Term, High Intensity [illegible]t) Training on Some Contractile and Meta[illegible]ic Characteristics of Fast and Slow Muscles of the Rat", Pflügers Arch., 344:159-168, 1973.

Suominen, H., and E. Heikkinen. "Enzyme Activities in Muscle and Connective Tissue of M. Vastus Lateralis in Habitually Training and Sedentary 33 to 70 Year-Old Men", Europ. J. Appl. Physiol., 34:249-254, 1975.

Swynghedauw, B., A. Corsin, P.-Y. Hatt, and A. Sag. "Le prélèvement "in situ" du tissu myocardique par congélation instantanée en expérimentation animale", Path.-Biol., 15:853-856, 1967.

Tanner, J.M. "Fallacy of Per-Weight and Per-Surface Area Standards and their Relation to Spurious Correlations", J. Appl. Physiol., 2:1-15, 1949.

Taylor, A.W., R. Lappage, and S. Rao. "Skeletal Muscle Glycogen Stores after Submaximal and Maximal Work", Med. Sci. Sports, 3(2):75-78, 1971.

Taylor, A.W., M.A. Booth, and S. Rao. "Human Skeletal Muscle Phosphorylase Activities with Exercise and Training", Can. J. Physiol. Pharmacol., 50:1038-1042, 1972.

Thorling, E.B., and K. Jensen. "The Lactate Dehydrogenase Isoenzymes in Various Organs of the Rabbit in Anaemia, Hypoxia, and after Cobalt Administration", Acta Path. Microbiol. Scand., 66:426-436, 1966.

Thorstensson, A., and J. Karlsson. "The Effect of Strength Training on Muscle Enzymes to High Energy Phosphate Metabolism", Acta Physiol. Scand., 91(3):21A, 1974.

Thorstensson, A., B. Sjödin, and J. Karlsson. "Enzyme Activities and Muscle Strength after "Sprint Training" in Man", Acta Physiol. Scand., 94:313-318, 1975.

Thorstensson, A., J. Karlsson, J.H.T. Viitasalo, P. Luhtanen, and P.V. Komi. "Effect of Strength Training on EMG of Human Skeletal Muscle", Acta Physiol. Scand., 98:232-236, 1976a.

Thorstensson, A., B. Hultén, W. Von Döbeln, and J. Karlsson. "Effect of Strength Training on Enzyme Activities and Fiber Characteristics in Human Skeletal Muscle", Acta Physiol. Scand., 96:392398, 1976b.

Trangott, C., and E.J. Massaro. "The Molecular Basis of the Heterogeneity of the MM Isozyme of Rabbit Muscle Creatine Phosphokinase", Biochem. Biophys. Acta, 295(2):549-554, 1973a.

Van Wijhe, M., M.C. Blanchaer, and S. St. George-Stubbs. "The Distribution of Lactate Dehydrogenase Isozymes in Human Skeletal Muscle Fibers" J. Histochem. Cytochem., 12:608-614, 1964.

Vesell, E.S., and P.E. Pool. "Lactate and Pyruvate Concentrations in Exercised Ischemic Canine Muscle: Relationship of Tissue Substrate Level to Lactate Dehydrogenase Isozyme Pattern", Proc. Nat. Acad. Sci., 55:756-762, 1966.

Wagner, J.A., and J.B. Critz. "The Effect of Physical Activity on Creatine Phosphokinase and Glutamic-Oxalacetic Transaminase Levels in Muscle and Blood Plasma of Rats", (Abstract) Physiologist, 13:332, 1970.

Walpurger, VonG., and H. Anger. "Die enzymatische organisation des energiesloffwechsels in rattenherzen nach schewimm-und lauftraining", Zeitsch. Kreislaufforch., 59(5):438-449, 1970.

Wahren, J. "Human Forearm Muscle Metabolism During Exercise, IV. Glucose Uptake at Different Work Intensities", Scand. J. Clin. Lab. Invest., 25:129-135, 1970.

Weil, R., P.-P. Ho, and N. Altszuler. "Effect of Free Fatty Acids on Metabolism of Pyruvic and Lactic Acids", Am. J. Physiol., 208(5): 887-891, 1965.

Wenger, H.A., and A.T. Reed. "Metabolic Factors Associated with Muscular Fatigue During Aerobic and Anaerobic Work", Can. J. Appl. Sport Sci., 1(1):43-48, 1976.

Wilkinson, J.H. Isoenzymes, Philadelphia: J.B. Lippincott Co., 1970.

Wilson, A.C., R.D. Cahn, N.O. Kaplan. "Functions of the Two Forms of Lactic Dehydrogenase in the Brease Muscle of Birds", Nature, 197:331-334, 1963.

Winer, B.J. Statistical Principles in Experimental Design, New York: McGraw Hill, 1971.

Wolfson, S., L.I. Rose, J.E. Bousser, A.F. Parisi, A.E. Acosta, K.H. Cooper, and E: Schechter. "Serum Enzyme Levels During Exercise in Patients with Coronary Heart Disease: Effects of Training", Am. Heart J., 84(4):478-483, 1972.

Wollenberger, A., E.-G. Krause, and B.E. Wahler. "uber den tatsächichen orthophosphat- und N-phosphorylkreatingehalt des saugetierherzens", Pflügers Archiv., 270:413-427, 1960.

Wuntch, T., R.F. Chen, and E.S. Vesell. "Lactate Dehydrogenase Isozymes: Kinetic Properties at High Enzyme Concentrations", Science, 167:63-65, 1970a.

Wuntch, T., R.F. Chen, and E.S. Vesell. "Lactate Dehydrogenase Isozymes: Further Kinetic Studies at High Enzyme Concentrations", Science, 169:480-481, 1970b.

Yakolev, N.N. "L'influence du travail musculaire systématique sur l'activité des ferments du métabolisme du glycogène et de G-6-P dans les muscles et dans le foie", in Biochemistry of Exercise, Medecine and Sport, edited by J.R. Poortmans, New York/Basel: Karger, 1969, Vol. 3, pp. 245-248.

York, J.W., L.B. Oscai, and D.V. Penney. "Alterations in Skeletal Muscle Lactate Dehydrogenase Isozymes Following Exercise Training", Biochem. Biophys. Res. Commun., 61:1387-1393, 1974.

York, J.W., D.G. Penney, and L.B. Oscai. "Effects of Physical Training on Several Glycolytic Enzymes in Rat Heart", Biochem. Biophys. Acta, 381:22-27, 1975.

York, J.W., D.G. Penney, T.A. Weeks, and P.A. Stagno. "Lactate Dehydrogenase Changes Following Several Cardiac Hypertrophic Stress", J. Appl. Physiol., 40(6):923-926, 1976.

Zika, K., A. Lojda, and M. Kucera. "Activities of Some Oxidative and Hydrolytic Enzymes in Musculus Biceps Brachii of Rats after Tonic Stress", Histochemie, 35:153-164, 1973.

York, J.W., D.G. Penney, and L.B. Oscai. "Effects of Physical Training on Several Glycolytic Enzymes in Rat Heart", Biochem. Biophys. Acta, 381:22-27, 1975.

York, J.W., D.G. Penney, T.A. Weeks, and P.A. Stagno. "Lactate Dehydrogenase Changes Following Several Cardiac Hypertrophic Stress", J. Appl. Physiol., 40(6):923-926, 1976.

Zika, K., A. Lojda, and M. Kucera. "Activities of Some Oxidative and Hydrolytic Enzymes in Musculus Biceps Brachii of Rats after Tonic Stress", Histochemie, 35:153-164, 1973.

APPENDIX A

LDH ELECTROPHORESIS

Appendix A contains an illustration of LDH isoenzyme separation with polyacrylamide gel electrophoresis (plate 2). From such a separation, M_4 and H_4 LDH were analysed at different pyruvate concentrations to find the respective optimal pyruvate concentration (Table 21 and Figure 6). For further details, see Chapter III, Methods and Procedures.

PLATE 2. LDH ISOENZYME SEPARATION WITH POLYACRYLAMIDE GEL ELECTROPHORESIS. From the 5 isoenzymes, H_4 is the fastest moving one toward the anode (bottom of the gels). From left to right: 1. LDH from beef heart (Sigma L 2625), 2. LDH from rabbit muscle (Sigma L 2500), 3, 4 and 5. Rat muscle homogenates, 6. H_4 (Sigma L3125), 7. Mixture of H_4 and M_4, 8. M_4 (Sigma L 2875).

TABLE 21 Optimal Pyruvate Concentrations for M_4 and H_4 LDH in the Rat.*

PA ($X10^{-4}M$)	H_4 LDH** ($\Delta A/mn$)	(%)	M_4 LDH** ($\Delta A/mn$)	(%)
0	0	0	0	0
.5	0.065	29.5	0.010	25
1	0.130	69.2	0.015	37.5
3	0.220	100	0.031	77.5
5	0.217	98.4	0.036	90
10	0.220	100	0.040	100
20	0.182	82.5	0.040	100
40	0.111	50.5	0.029	72.7
100	0.065	29.6	0.020	50

* M_4 and H_4 were electrophoretically separated from heart and skeletal muscles of the rat.

** Average of three values.

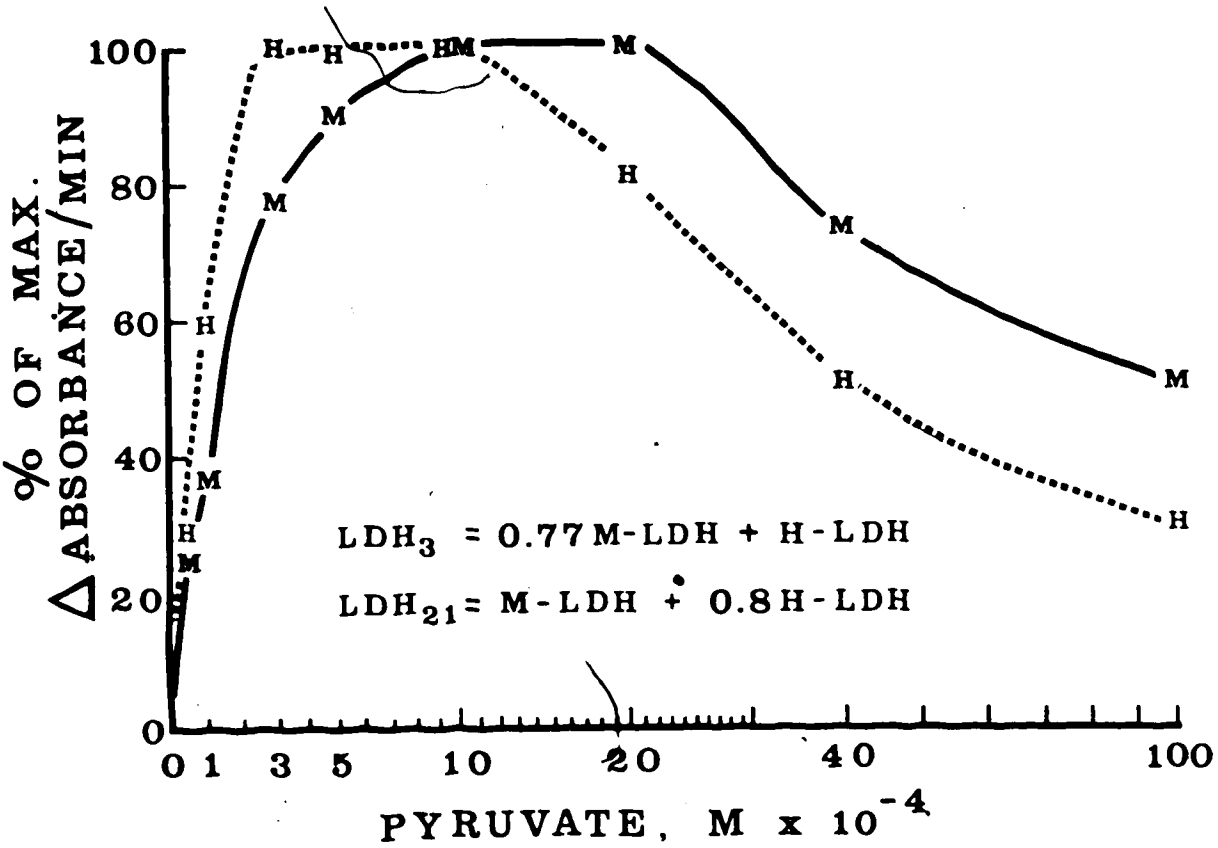

FIGURE 6. OPTIMAL PYRUVATE CONCENTRATIONS FOR M-LDH AND H-LDH IN RAT TISSUE

APPENDIX B

BODY WEIGHT PROGRESS FOR TRAINING GROUPS

TABLE 22 Body Weight Progress for Training Groups: Means and Standard Deviations.

AGE (week)	Sgr $\bar{X}$	Sgr SD	Cgr $\bar{X}$	Cgr SD	Igr $\bar{X}$	Igr SD
ARRIVAL[a]						
6	158.5	17.8	157.4	20.3	157.4	16.8
RUNNING EDUCATION						
7	204.5	13.9	203.4	17.4	205.7	14.6
8	255.6	19.2	258.4	19.2	256.8	16.5
9	290.7	20.1	293.3	18.2	293.5	19.5
10	305.0	26.5	317.5	18.0	311.7	20.4
TRAINING PERIOD						
11	341.8	25.4	343.0	20.2	330.5	37.4
12	358.8	26.5	362.0	20.7	361.7	26.9
13	390.0	28.6	383.0	26.9	375.1	27.8
14	410.5	32.5	384.5	37.8	386.5	25.6
15	433.9	31.7	395.5	36.9	399.4	26.8
16	459.3	38.0	420.5	35.2	419.6	26.6
17	476.0	36.1	430.4	41.6	426.5	26.2
18	489.2	40.4	438.5	40.6	432.2	28.0
19	504.9	40.0	457.5	36.4	439.8	25.5
20	507.7	48.4	459.0	29.2	453.3	30.0
21	527.1	45.1	475.1[b]	29.5[b]	460.2	32.2
22	535.9	41.4	478.6[b]	31.4[b]	465.2	35.0
23	553.7[b]	50.9[b]	487.2	34.3	471.9	33.1
24	558.8[b]	50.5[b]	495.6	33.4	478.2[b]	35.7[b]
25	568.9	50.9	503.0	35.7	473.4[b]	31.0[b]
26	572.6	52.4	513.6	37.1	481.9	28.0
27	571.7	54.4	516.0	37.3	488.5	36.7
33[d]	598.9	59.9	537.5[c]	37.7[c]	537.6[c]	54.2[c]
34	613.2	60.6	525.4	35.9	511.4	40.9
35	624.6[c]	69.7[c]	534.0	41.7	534.1	44.0

a. Groups: n = 10.
b. From that time and on, n = 9.
c. From that time and on, n = 8.
d. Subtitute trainer forgot to weigh rats.

APPENDIX C

RAW DATA

This appendix gives the raw data for each group, each organ and each variable in the following order:

1. Body weight
2. W_{abs}
3. W_{rel}
4. ATP
5. PC
6. ATP + PC
7. CPK
8. AK
9. LDH_{21}
10. LDH_3
11. LDH_{21}/LDH_3
12. M-LDH
13. H-LDH
14. TOTAL LDH

FINAL BODY WEIGHT (g)

Rat #	Igr	Cgr	Sgr
1	510	623	655
2	610	565	597
3	535	515	630
4	475	520	507
5	535	490	753
6	505	517	650
7	585	530	615
8	518	512	590

	W_{abs} (mg)		
	Igr	Cgr	Sgr
TA	870.600	1106.000	1006.600
	878.600	995.400	935.500
	1065.600	1023.600	789.800
	950.600	992.600	969.000
	1072.000	979.800	921.200
	1047.600	965.600	1104.200
	983.800	953.800	1080.000
	874.800	1010.200	1084.600
P	491.200	670.400	553.200
	464.600	620.800	652.800
	539.200	564.800	476.800
	518.600	566.800	583.800
	627.400	499.200	475.600
	616.400	623.400	626.600
	582.200	554.200	549.000
	565.200	556.400	575.200
GM	1209.400	1421.000	1335.800
	1280.000	1329.400	1180.700
	1177.600	1257.800	1079.000
	1040.400	1123.800	1172.800
	1357.800	1131.600	1026.000
	1298.200	1211.000	1327.200
	1238.200	1283.800	1186.000
	1170.200	1197.200	1244.000
GL	1696.000	1484.400	1508.200
	1477.800	1576.800	1313.300
	1564.000	1294.800	1174.200
	1178.200	1401.400	1607.400
	1607.400	1332.400	1234.000
	1402.600	1435.200	1576.000
	1492.400	1404.000	1590.400
	1182.000	1314.600	1514.200
S	0.000	294.600	282.800
	244.400	301.000	263.400
	201.000	244.000	192.800
	246.200	244.400	261.200
	239.900	225.600	167.800
	256.800	236.600	249.200
	304.800	253.200	317.600
	265.200	268.600	267.800
H	1602.800	1639.200	1496.000
	1416.000	1399.600	1468.500
	1558.000	0.000	1270.200
	1384.200	1707.000	0.000
	1497.000	1302.600	1745.100
	1500.000	1510.000	1469.000
	1533.800	1267.800	0.000
	0.000	0.000	1534.500
L	16472.300	15706.900	16564.300
	15615.000	15319.800	17400.000
	13950.000	11800.700	14773.900
	12025.300	10086.500	17823.400
	12788.500	11496.300	16968.200
	10940.000	14772.500	15188.000
	13583.600	10567.500	14100.700
	15246.700	13903.600	15153.900

W_{rel} (mg/g)

	Igr	Cgr	Sgr
TA	1.707	1.775	1.537
	1.440	1.762	1.567
	1.992	1.984	1.254
	2.001	1.969	1.611
	2.004	2.000	1.223
	2.074	1.868	1.837
	1.682	1.800	1.756
	1.689	1.973	1.438
P	.963	1.076	.845
	.762	1.097	1.093
	1.008	1.096	.757
	1.092	1.089	1.152
	1.173	1.019	.632
	1.221	1.206	.964
	.995	1.046	.893
	1.091	1.087	.975
GM	2.371	2.281	2.039
	2.098	2.353	1.976
	2.201	2.402	1.713
	2.190	2.161	2.515
	2.538	2.309	1.363
	2.571	2.382	2.042
	2.117	2.422	1.929
	2.259	2.338	2.109
GL	3.326	2.383	2.303
	2.823	2.791	2.200
	2.924	2.514	1.864
	2.480	2.695	3.170
	3.004	2.719	1.639
	2.777	2.776	2.425
	2.551	2.649	2.586
	2.282	2.568	2.566
S	0.000	.473	.432
	.401	.533	.441
	.376	.474	.306
	.518	.470	.515
	.448	.460	.223
	.508	.458	.383
	.521	.478	.516
	.512	.525	.454
H	3.143	2.631	2.284
	2.305	2.477	2.406
	2.912	0.000	2.016
	2.914	3.283	0.000
	2.798	2.658	2.318
	2.970	2.931	2.260
	2.622	2.392	0.000
	0.000	0.000	2.601
L	32.299	25.212	25.289
	25.598	27.115	29.146
	26.075	22.914	23.451
	25.316	27.089	35.155
	23.842	23.462	22.534
	21.663	28.574	23.366
	23.220	27.486	22.928
	29.433	27.155	25.685

ATP (mmoles/g)

	Igr	Cgr	Sgr
TA	4.770	4.610	6.000
	4.870	4.630	5.750
	4.700	6.250	5.430
	5.280	5.020	5.260
	5.420	7.300	4.690
	4.960	5.250	5.540
	4.210	5.280	6.200
	4.700	6.410	5.410
P	5.210	4.830	4.630
	4.360	4.430	4.860
	5.020	6.150	5.430
	4.440	4.710	4.620
	4.520	6.790	4.600
	4.120	0.000	4.710
	5.160	4.830	6.570
	3.850	4.990	5.420
GM	4.500	4.980	4.740
	4.570	5.150	4.950
	5.480	5.400	4.900
	4.890	6.460	4.340
	4.270	7.300	4.290
	3.400	5.790	5.850
	5.480	4.760	7.740
	3.260	6.560	4.840
GL	4.810	4.660	4.700
	4.140	4.950	4.580
	4.450	7.460	4.950
	4.220	5.300	4.790
	4.020	5.770	6.360
	4.570	4.550	4.560
	4.630	4.390	5.980
	3.870	4.980	4.910
S	3.430	3.770	3.120
	2.110	4.130	3.510
	3.350	4.040	3.540
	3.500	3.760	3.060
	3.410	4.930	3.560
	3.410	3.450	3.370
	2.700	3.720	3.150
	3.550	3.470	3.020
H	3.970	4.860	3.920
	4.170	4.520	3.550
	4.210	4.950	3.870
	4.020	4.790	0.000
	4.310	4.360	3.980
	3.960	3.510	3.640
	4.580	4.210	4.750
	5.020	0.000	4.100

PC (mmoles/g)

	Igr	Cgr	Sgr
TA	18.390	17.810	22.470
	19.330	18.700	25.430
	18.830	17.600	16.910
	0.000	22.700	18.890
	15.750	16.740	13.870
	0.000	19.670	14.600
	18.700	19.260	20.280
	14.150	0.000	0.000
P	11.950	14.530	12.670
	15.060	16.830	30.130
	19.390	14.910	14.990
	0.000	11.490	15.420
	14.560	13.880	11.830
	0.000	11.670	9.260
	18.530	9.180	17.070
	14.360	0.000	0.000
M	17.420	12.780	[illegible]
	17.350	17.650	[illegible]
	19.730	13.600	[illegible]
	0.000	23.120	[illegible]
	14.370	17.700	[illegible]
	0.000	18.390	[illegible]
	20.390	14.330	[illegible]
	11.[illegible]	0.000	0.000
GL	10.[illegible]	14.670	16.680
	12.[illegible]	16.100	18.610
	13.[illegible]	14.850	13.060
	0.[illegible]	15.870	0.000
	11.[illegible]	16.340	15.160
	0.000	14.740	13.200
	13.490	8.440	11.740
	12.250	0.000	0.000
S	8.940	8.680	7.220
	6.610	12.720	11.940
	8.970	8.[illegible]	8.370
	0.000	5.[illegible]	8.550
	7.830	12.880	8.530
	0.000	13.[illegible]	10.540
	7.680	6.330	6.960
	0.000	0.000	0.000
H	2.770	6.460	3.730
	6.870	5.720	7.410
	4.370	4.970	4.560
	0.000	6.400	0.000
	6.020	5.440	4.290
	0.000	0.[illegible]	6.650
	3.750	6.650	4.910
	7.320	0.000	0.000

ATP & PC (mmoles/g)

	Igr	Cgr	Sgr
TA	19.160	24.420	28.470
	24.200	23.330	31.180
	23.530	23.850	22.340
	19.960	27.810	24.150
	22.910	24.040	20.560
	18.450	24.920	20.800
	0.000	24.540	25.690
	0.000	0.000	0.000
P	17.160	19.360	17.300
	19.820	21.660	18.990
	24.410	21.000	20.420
	19.080	16.200	20.040
	23.090	20.870	16.430
	15.210	14.530	15.830
	0.000	14.010	22.490
	0.000	0.000	0.000
GM	21.920	17.760	20.390
	21.920	22.800	24.740
	25.210	19.000	20.340
	18.640	29.640	18.680
	25.870	25.000	15.740
	14.260	24.180	22.890
	0.000	19.000	21.340
	0.000	0.000	0.000
GL	14.090	19.330	21.380
	18.180	21.050	23.190
	23.540	22.310	17.530
	15.790	21.210	0.000
	18.120	22.150	20.060
	16.120	19.270	19.180
	0.000	12.830	16.650
	0.000	0.000	0.000
S	12.370	12.850	10.340
	8.720	16.850	15.450
	12.320	12.310	11.910
	11.240	9.720	11.610
	10.380	17.820	12.090
	0.000	16.680	13.690
	0.000	10.050	9.980
	0.000	0.000	0.000
H	6.740	11.320	7.650
	11.040	10.240	10.960
	8.580	9.920	8.430
	10.330	11.200	0.000
	8.330	9.800	8.270
	12.340	6.870	11.400
	0.000	10.860	9.010
	0.000	0.000	0.000

AK (IU/g)

	Igr	Cgr	Sgr
TA	117.290	143.200	231.800
	133.400	206.840	0.000
	63.380	50.940	108.070
	141.710	40.760	79.990
	178.800	37.360	154.060
	138.940	174.020	0.000
	153.440	116.340	60.440
	0.000	201.930	132.350
P	115.040	131.400	204.000
	151.000	212.150	0.000
	49.520	56.370	113.910
	172.740	45.130	84.360
	138.000	40.810	121.230
	118.340	168.308	0.000
	132.450	120.610	49.660
	0.000	218.890	201.340
GM	113.070	138.200	179.800
	143.000	210.750	0.000
	40.570	47.800	97.830
	179.130	36.670	98.610
	143.800	30.740	173.820
	151.600	203.620	0.000
	109.410	125.560	43.990
	0.000	176.530	180.000
GL	114.640	101.200	189.600
	117.400	0.000	0.000
	38.690	46.430	95.230
	163.860	36.070	101.840
	134.650	32.990	112.180
	102.000	143.460	0.000
	117.600	114.560	45.810
	0.000	196.140	166.340
S	0.000	71.490	92.700
	70.630	0.000	0.000
	23.340	24.480	55.410
	112.110	34.910	65.060
	60.020	23.610	56.640
	58.980	43.800	0.000
	62.310	57.080	21.110
	0.000	75.510	80.030
H	69.240	74.430	83.100
	55.920	90.520	0.000
	34.550	30.600	58.530
	77.520	32.190	47.840
	63.940	32.350	69.040
	106.970	117.350	0.000
	90.460	57.880	33.900
	0.000	81.690	71.490
L	19.080	22.160	22.700
	15.080	0.000	0.000
	17.240	21.450	16.890
	17.750	20.140	18.250
	12.650	20.410	16.730
	29.770	21.920	0.000
	27.320	12.850	18.550
	0.000	13.730	24.810

CPK (IU/g)

	Igr	Cgr	Sgr
TA	2275.200	0.000	0.000
	2614.600	3226.500	0.000
	2066.200	2978.000	3288.400
	2910.200	2039.700	2795.500
	2020.300	2750.300	2486.200
	2170.500	2499.500	3000.900
	2701.600	2500.400	2344.200
	2569.500	2706.800	3295.200
P	2088.900	0.000	2890.600
	3018.500	2074.100	2412.500
	1829.900	3048.200	2781.900
	2111.300	1962.800	2228.300
	1628.000	2200.900	3085.300
	2165.900	2192.300	2706.700
	2377.900	2715.900	2885.900
	2638.100	2873.500	3265.800
GM	3147.700	0.000	2470.600
	2945.100	2246.200	3060.000
	2034.100	2551.900	2607.400
	2384.300	1797.400	2121.600
	2216.400	2105.900	3810.400
	2881.700	1916.700	3020.600
	1855.800	2947.200	3118.900
	2216.400	2804.800	3491.300
GL	1990.800	2020.200	2486.600
	1837.700	2020.200	3177.000
	1309.400	2001.500	2555.600
	2485.500	1728.800	2605.400
	1431.900	1846.300	2963.900
	2128.900	1374.800	3599.400
	2167.400	2087.500	2552.200
	1853.500	2608.700	3226.500
S	0.000	1108.000	1197.600
	1130.700	1196.500	549.200
	1028.900	1192.800	1105.400
	1314.400	954.900	987.700
	1039.500	1116.200	1228.900
	1174.100	816.100	1039.500
	1254.900	1247.200	1026.400
	1235.700	843.400	1167.000
H	855.200	1159.200	868.800
	776.500	767.900	669.900
	859.600	879.000	943.000
	817.800	689.800	702.400
	826.700	1014.500	1053.100
	1070.100	1061.900	636.500
	1073.000	692.900	958.800
	810.100	1039.500	660.800
L	9.020	15.360	12.490
	10.060	39.230	7.190
	7.250	14.820	9.110
	11.150	13.610	8.490
	13.640	6.870	14.420
	11.100	17.880	9.810
	10.550	14.590	10.030
	40.550	16.970	0.000

LDH_{21} (IU/g)

	Igr	Cgr	Sgr
TA	424.600	339.300	850.000
	654.400	525.700	636.500
	468.300	615.500	717.900
	636.000	415.100	642.200
	241.300	576.000	612.200
	517.700	248.700	358.900
	433.800	480.100	455.600
	375.600	413.900	423.700
P	899.200	447.200	702.900
	682.500	486.400	521.700
	553.400	752.200	0.000
	545.300	459.700	722.300
	472.700	617.400	635.400
	401.800	228.000	510.000
	454.100	451.700	668.300
	417.800	559.000	431.500
GM	0.000	424.700	489.900
	514.800	434.500	555.100
	488.000	541.200	0.000
	454.500	447.600	0.000
	457.100	470.000	625.800
	587.800	197.300	398.200
	280.600	439.500	488.200
	312.800	561.900	333.400
GL	496.200	277.600	494.900
	376.900	392.300	490.400
	368.000	415.100	611.600
	573.900	393.800	682.000
	0.000	384.400	515.800
	465.300	175.800	457.000
	385.900	366.700	447.600
	192.200	490.400	342.300
S	0.000	215.800	115.500
	109.900	0.000	196.100
	107.200	159.300	122.800
	167.800	115.900	178.000
	98.100	138.500	149.800
	107.200	61.300	100.000
	128.700	0.000	108.300
	97.100	10.000	76.500
H	325.600	545.300	209.000
	273.900	196.100	359.600
	273.900	310.900	254.000
	200.500	246.100	0.000
	211.800	343.800	383.500
	356.500	0.000	0.000
	284.400	195.100	240.400
	143.200	182.400	235.400
L	260.900	0.000	314.300
	403.900	0.000	260.900
	318.600	347.800	314.800
	268.400	261.800	297.700
	0.000	339.100	484.900
	0.000	148.200	353.100
	305.300	145.700	248.200
	244.200	214.800	0.000

LDH_3 (IU/g)

	Igr	Cgr	Sgr.o
TA	361.900	286.400	758.600
	573.000	473.700	555.000
	452.700	517.700	534.000
	498.700	355.000	494.100
	196.100	420.700	506.600
	399.700	176.900	325.600
	315.000	345.900	357.600
	300.100	332.500	358.900
P	457.000	329.500	615.000
	582.800	440.300	490.000
	509.100	628.000	0.000
	454.000	403.700	539.400
	375.600	531.200	594.100
	553.900	167.900	424.600
	343.300	379.200	511.500
	323.600	476.600	395.200
GM	0.000	374.700	468.400
	480.100	312.400	521.700
	461.500	480.000	0.000
	376.300	376.500	0.000
	432.800	369.000	482.800
	432.900	166.100	368.700
	229.700	313.900	394.800
	294.200	440.300	300.[illegible]
GL	454.100	254.000	45[illegible]
	350.100	492.300	425.700
	337.900	377.700	538.400
	439.400	321.100	505.800
	0.000	328.400	465.200
	391.600	136.700	408.000
	293.900	282.000	355.800
	189.300	430.500	297.200
S	0.000	196.100	191.300
	104.500	0.000	196.100
	99.900	207.000	185.200
	185.900	131.100	158.900
	98.100	159.300	170.100
	129.100	60.400	100.000
	132.100	0.000	110.800
	94.100	0.000	90.200
H	450.100	608.000	211.700
	346.300	215.700	455.400
	370.900	349.600	339.900
	329.600	281.300	0.000
	260.900	402.700	440.900
	426.200	0.000	0.000
	321.200	201.300	263.100
	162.800	196.100	274.600
L	227.500	0.000	276.200
	387.900	0.000	227.500
	276.500	313.900	272.100
	221.900	232.900	214.800
	0.000	282.900	451.000
	0.000	119.200	309.900
	230.300	123.300	202.400
	202.000	109.700	0.000

LDH_{21}/LDH_3

	Igr	Cgr	Sgr
TA	1.170	1.180	1.120
	1.140	1.110	1.150
	1.030	1.190	1.340
	1.280	1.170	1.300
	1.230	1.370	1.210
	1.300	1.400	1.100
	1.380	1.390	1.270
	1.250	1.240	1.180
P	1.090	1.360	1.140
	1.170	1.100	1.060
	1.090	1.200	0.000
	1.200	1.140	1.340
	1.260	1.160	1.070
	1.140	1.360	1.200
	1.320	1.190	1.310
	1.290	1.170	1.090
GM	0.000	1.130	1.050
	1.070	1.390	1.060
	1.060	1.150	0.000
	1.210	1.190	0.000
	1.060	1.270	1.300
	1.360	1.190	1.080
	1.220	1.400	1.240
	1.060	1.280	1.110
GL	1.090	1.090	1.090
	1.080	1.000	1.160
	1.090	1.100	1.140
	1.310	1.230	1.340
	0.000	1.170	1.110
	1.190	1.310	1.120
	1.310	1.300	1.200
	1.020	1.140	1.150
S	0.000	1.100	.720
	1.050	0.000	1.000
	1.070	.770	.790
	.900	.880	1.120
	1.000	.870	.880
	.830	1.010	1.000
	.970	0.000	.980
	1.030	0.000	.870
H	.720	.900	.990
	.790	.910	.830
	.740	.890	.750
	.880	.870	0.000
	.810	.850	.870
	.840	0.000	0.000
	.890	.970	.910
	.880	.930	.860
L	1.150	0.000	1.140
	1.040	0.000	1.150
	1.050	1.110	1.160
	1.210	1.210	1.590
	0.000	1.200	1.080
	0.000	1.240	1.140
	1.330	1.180	1.230
	1.210	1.270	0.000

M - LDH (IU/g)

	Igr	Cgr	Sgr
TA	351.800	286.900	633.100
	510.900	382.100	501.300
	276.400	528.300	757.000
	617.500	340.800	645.000
	219.800	624.800	538.900
	515.500	279.100	256.300
	473.400	529.600	401.500
	352.900	385.200	355.700
P	347.900	478.100	549.200
	563.700	309.400	516.900
	380.500	650.500	0.000
	474.200	356.100	757.200
	848.500	501.100	417.000
	309.100	248.000	443.500
	467.300	386.300	674.700
	413.900	462.800	300.400
GM	0.000	325.400	299.900
	340.400	479.800	358.700
	309.400	409.400	0.000
	399.600	381.300	0.000
	288.700	455.200	623.900
	628.500	167.800	288.900
	252.200	490.600	448.900
	201.700	546.000	243.000
GL	346.100	193.800	341.900
	252.100	204.300	394.400
	254.400	294.100	471.000
	579.100	356.600	716.000
	0.000	310.900	374.100
	395.900	180.800	300.100
	392.700	367.400	424.400
	106.100	380.200	272.200
S	0.000	153.400	-35.300
	68.500	0.000	102.100
	71.000	-41.000	-3.500
	49.700	28.700	132.500
	51.100	28.800	35.700
	10.200	33.800	52.100
	59.900	0.000	51.200
	56.800	0.000	16.500
H	-89.800	153.400	103.200
	-8.200	61.500	55.500
	-59.400	84.300	-46.700
	69.800	54.800	0.000
	8.000	56.400	80.400
	44.500	0.000	0.000
	71.600	88.700	77.900
	33.700	66.500	40.900
L	205.500	0.000	243.100
	243.700	0.000	205.500
	253.600	251.800	252.900
	236.700	248.600	327.800
	0.000	293.700	323.200
	0.000	137.600	273.900
	315.300	122.600	224.700
	215.100	205.800	0.000

H - LDH (IU/g)

	Lgr	[illegible]	Sgr
TA	91.000	93.500	271.100
	170.000	170.500	160.000
	239.400	114.000	240.000
	23.800	93.200	-1.000
	26.800	-61.000	91.700
	2.800	-36.800	120.300
	-84.500	-61.900	17.700
	28.400	35.000	85.000
P	189.100	-38.700	192.100
	149.200	171.500	231.000
	216.100	127.100	0.000
	88.900	129.500	-43.700
	30.300	145.300	273.000
	115.900	-19.900	83.100
	-16.600	81.700	-8.000
	8.930	124.200	163.900
GM	0.000	124.200	237.400
	218.000	-56.700	245.500
	223.300	164.800	0.000
	68.000	82.900	0.000
	210.500	18.500	2.400
	-51.300	36.900	161.700
	35.500	-63.800	49.200
	138.900	19.900	113.000
GL	187.600	104.800	191.200
	156.000	235.800	120.000
	142.000	151.200	175.700
	-6.500	46.500	-42.600
	0.000	84.400	177.200
	36.800	-2.500	146.100
	-8.400	-.900	29.000
	107.600	137.700	87.600
S	0.000	78.000	188.400
	51.800	0.000	117.500
	45.200	207.200	157.900
	147.600	109.800	56.900
	58.800	137.100	142.800
	121.200	34.400	59.900
	85.900	0.000	71.400
	50.300	0.000	77.500
H	519.200	439.900	132.200
	352.800	168.500	407.600
	416.700	287.000	375.800
	275.800	239.100	0.000
	254.700	359.300	378.900
	345.000	0.000	0.000
	266.200	133.000	203.100
	136.800	144.900	243.100
D	69.300	0.000	39.000
	200.300	0.000	69.300
	81.200	120.000	77.400
	39.700	41.400	-37.600
	0.000	56.800	202.200
	0.000	13.200	99.000
	-12.450	28.900	29.400
	36.400	11.200	0.000

TOTAL LDH (IU/g)

	Igr	Cgr	Sgr
TA	442.800	352.400	904.200
	666.500	561.600	670.300
	516.300	638.300	768.100
	640.700	433.700	647.000
	219.600	563.800	630.500
	518.300	241.100	364.500
	423.900	467.700	459.100
	381.300	421.100	440.700
P	537.000	439.500	741.300
	512.300	520.700	567.000
	596.800	777.600	0.000
	563.100	435.600	713.600
	478.800	646.400	690.000
	425.000	224.000	526.600
	450.800	468.000	666.700
	418.800	583.000	464.300
GM	0.000	449.500	537.400
	558.400	425.200	604.200
	532.700	574.200	0.000
	468.200	464.200	0.000
	499.200	473.700	626.300
	577.500	204.700	430.500
	287.700	426.700	408.000
	340.600	565.900	356.000
GL	533.700	298.600	533.100
	408.100	439.300	514.400
	396.400	445.300	646.700
	572.600	405.100	673.500
	0.000	401.300	551.200
	432.700	178.300	486.200
	384.200	360.500	453.400
	213.700	517.900	272.200
S	0.000	231.400	153.200
	120.300	0.000	219.600
	116.200	165.800	154.400
	167.300	137.700	130.400
	110.900	165.900	178.300
	131.400	68.200	112.000
	145.900	0.000	122.600
	107.200	0.000	94.000
H	429.400	643.300	235.400
	344.400	229.800	441.100
	357.200	368.300	329.200
	345.700	393.900	0.000
	262.700	415.700	459.300
	435.600	0.000	0.000
	337.600	221.700	281.000
	170.600	211.400	284.000
L	274.800	0.000	332.100
	444.000	0.000	274.800
	334.800	371.400	330.300
	376.300	290.100	290.200
	0.000	350.500	525.300
	0.000	150.800	372.900
	302.800	151.500	254.100
	251.500	217.000	0.000

APPENDIX D

TRAINING GROUP COMPARISONS FOR EACH DEPENDANT VARIABLE AND TISSUE: ONE WAY ANALYSES OF VARIANCE AND OTHER RELATED STATISTICS

This appendix contains only those among the 89 one way analyses of variance, that revealed significant differences between groups. Scheffe's contrasts are also shown. ANOVA Tables appears in the following order:

1. W_{abs} (BW)
2. W_{rel} (TA, P, GM, H)
3. ATP (TA, GM, GL, S)
4. CPK (P, GM, GL)
5. $LDH_{2,1}$ (GL)
6. LDH_3 (GL)
7. M-LDH (H)

On the computer print-out, Sgr, Cgr and Igr are represented by No 3, 2, 1 respectively. "Moyenne" and "Ecart-type" are the mean and standard deviation. Scheffe's contrasts (alpha (j) - alpha (i)) are significant at $P < 0.05$ when their confidence interval are both positive or negative in which case group j is larger or smaller than group i respectively.

[redacted]

SOURCE	SS	DL	S	F
ENTRE LES GROUPES	.03742E+05	2	.21471E+05	7,498
ERREUR	.59605E+05	21	.28411E+04	
TOTAL	.10340E+06	23		

LA VALEUR CALCULEE DE PHI EST 2,205

[redacted]

LES W(I)= 1.0 1.0 1.0

NB DE LES Y DU GROUPE SONT
1.0 1.0 2.0 2.0 2.0 2.0 1.0 1.0

NB DE LES Y DU GROUPE SONT
1.0 1.0 2.0 1.0 2.0 1.0 1.0 2.0

NB DE LES Y DU GROUPE SONT
1.0 1.0 1.0 1.0 1.0 1.0 1.0 1.0

GROUPE NUMERO 1 MOYENNE= 1.000 ECART-TYPE= .535
GROUPE NUMERO 2 MOYENNE= 1.286 ECART-TYPE= .756
GROUPE NUMERO 3 MOYENNE= 1.615 ECART-TYPE= .767
MOYENNE GENERALE= 1.379

ALPHA(1)= .000
ALPHA(2)= .110
ALPHA(3)= .150

NB DE LES RESIDUS DU GROUPE SONT
-.117 -.344 .104 .177 .189 .290 -.102 -.135

NB DE LES RESIDUS DU GROUPE SONT
-.109 -.122 .104 .025 .114 -.010 -.088 .089

NB DE LES RESIDUS DU GROUPE SONT
-.078 -.044 -.364 .296 -.392 .222 .141 .223

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.31045E+00	2	.15523E+00	1.635
ERREUR	.91476E+00	21	.43798E-01	
TOTAL	.12382E+01	23		

LA VALEUR CALCULEE DE PHI EST 1.557

CALCUL DES CONTRASTES DE SCHEFFE PAR L'HYPOTHESE NO EST REJETEE AVEC COMME FRF 3.4668

ALPH(2) - ALPH(1) ∈ (-.2184, .3383)

ALPH(3) - ALPH(1) ∈ (-.0834, .0673)
ALPH(3) - ALPH(2) ∈ (-.5085, .0065)

LES MOYENNES A B B B

NB A LES Y DU GROUPE SONT
1,0 1,0 1,0 1,1 1,2 1,2 1,0 1,1

NB A LES Y DU GROUPE SONT
1,1 1,1 1,1 1,1 1,0 1,2 1,0 1,1

NB A LES Y DU GROUPE SONT
,0 1,0 ,0 1,0 ,0 1,0 ,0 1,0

GROUPE NUMERO 1 MOYENNE= 1,038 ECART-TYPE= ,102
GROUPE NUMERO 2 MOYENNE= 1,041 ECART-TYPE= ,088
GROUPE NUMERO 3 MOYENNE= ,918 ECART-TYPE= ,111
MOYENNE GENERALE= 1,010

ALPHA(1)= ,000
ALPHA(2)= ,078
ALPHA(3)= -,108

NB A LES RESIDUS DU GROUPE SONT
,075 -,276 -,030 ,054 ,135 ,103 -,083 ,053

NB A LES RESIDUS DU GROUPE SONT
-,013 ,008 ,007 -,000 -,070 ,117 ,003 -,002

NB A LES RESIDUS DU GROUPE SONT
-,048 ,170 -,157 ,238 -,202 ,050 -,021 ,001

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,13040E+00	2	,65229E-01	3,853
ERREUR	,36099E+00	21	,17076E-01	
TOTAL	,49139E+00	23		

LA VALEUR CALCULEE DE PHI EST 1,577

CALCUL DES CONTRASTES DE SCHEFFE CAR L'HYPOTHESE H0 EST REJETEE AVEC COMME FFF 3,4668

ALPH(2) - ALPH(1) = (-,1227, ,2256)

ALPH(3) - ALPH(1) = (-,2083, ,0868)
ALPH(3) - ALPH(2) = (-,3697, -,0010)

[redacted]

Y=
LES MESURES A A A A

NB A LES Y DU GROUPE SONT
2.0 2.1 2.2 2.2 2.5 2.3

NB A LES Y DU GROUPE SONT
2.5 2.0 2.0 2.2 2.3

NB A LES Y DU GROUPE SONT
2.0 2.0 1.7 2.3 1.0 2.0 2.1

GROUPE NUMERO 1 MOYENNE= 2.293 ECART-TYPE= .182
GROUPE NUMERO 2 MOYENNE= 2.111 ECART-TYPE= .087
GROUPE NUMERO 3 MOYENNE= 1.036 ECART-TYPE= .286
MOYENNE GENERALE= 2.187

ALPHA(1)= .107
ALPHA(2)= .188
ALPHA(3)= -.295

NB A LES RESIDUS DU GROUPE SONT
.078 -.195 -.092 -.103 .245 .278 -.176 -.038

NB A LES RESIDUS DU GROUPE SONT
-.050 .022 .111 -.170 -.022 .011 .091 -.007

NB A LES RESIDUS DU GROUPE SONT
.103 .042 -.223 .377 -.573 .106 -.007 .173

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.76100E+00	2	.38050E+00	9.296
ERREUR	.85954E+00	21	.40930E-01	
TOTAL	.16205E+01	23		

LA VALEUR CALCULEE DE PHI EST 2.489

CALCUL DES CONTRASTES DE SCHEFFE PAR L'HYPOTHESE H0 EST REJETEE AVEC COMME FFF 3.4668

ALPH(2) - ALPH(1) = (-.2285, .3062)

ALPH(3) - ALPH(1) = (-.6237, -.0910)
ALPH(3) - ALPH(2) = (-.6418, -.1289)

M_rci = N

To 1
LES N(I)= 7 6 6

NO 7 LES Y DU GROUPE SONT
3,1 2,3 2,9 2,0 2,0 3,0 2,0

NO 6 LES Y DU GROUPE SONT
2,0 2,5 3,3 2,7 2,0 2,0

NO 6 LES Y DU GROUPE SONT
2,3 2,0 2,0 2,3 2,3 2,0

GROUPE NUMERO 1 MOYENNE= 2,800 ECART-TYPE= ,271
GROUPE NUMERO 2 MOYENNE= 2,727 ECART-TYPE= ,327
GROUPE NUMERO 3 MOYENNE= 2,327 ECART-TYPE= ,196
MOYENNE GENERALE= 2,629

ALPHA(1)= ,189
ALPHA(2)= ,098
ALPHA(3)= -,508

NO 7 LES RESIDUS DU GROUPE SONT
,334 -,504 ,103 ,105 -,011 ,101 -,187

NO 6 LES RESIDUS DU GROUPE SONT
-,098 -,250 ,558 -,069 ,104 -,335

NO 6 LES RESIDUS DU GROUPE SONT
-,037 ,125 -,305 -,003 -,061 ,288

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,85458E+00	2	,42729E+00	5,81#
ERREUR	,11758E+01	16	,73490E-01	
TOTAL	,20304E+01	18		

LA VALEUR CALCULEE DE PHI EST 1,909

CALCUL DES CONTRASTES DE SCHEFFE CAR L'HYPOTHESE HO EST REJETEE AVEC COMME FFF 3,6337

ALPH(2) - ALPH(1) = (-,8988, ,3268)

ALPH(3) - ALPH(1) = (-,8969, -,0817)
ALPH(3) - ALPH(2) = (-,8281, ,0158)

ACP - TA

TA 3
LES MESURES A A A

NB A LES Y DU GROUPE SONT
A,A A,A A,A 5,1 5,4 5,8 6,2 6,7

NB A LES Y DU GROUPE SONT
A,A A,A A,A 5,8 7,3 5,3 5,3 6,8

NB A LES Y DU GROUPE SONT
A,A 5,A 5,8 5,3 6,7 5,5 6,2 5,8

GROUPE NUMERO 1 MOYENNE= 6,866 ECART-TYPE= ,375
GROUPE NUMERO 2 MOYENNE= 5,444 ECART-TYPE= ,827
GROUPE NUMERO 3 MOYENNE= 5,745 ECART-TYPE= ,644
MOYENNE GENERALE= 5,697

ALPHA(1)= -,638
ALPHA(2)= ,146
ALPHA(3)= ,268

NB A LES RESIDUS DU GROUPE SONT
-,000 ,000 -,144 ,416 ,556 ,096 -,656 -,164

NB A LES RESIDUS DU GROUPE SONT
,766 -1,214 ,000 -,824 1,456 -,594 -,566 ,566

NB A LES RESIDUS DU GROUPE SONT
,215 -,035 -,355 -,525 ,905 -,245 ,415 -,375

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,48335E+01	2	,24167E+01	5,875
ERREUR	,86384E+01	21	,41135E+00	
TOTAL	,13072E+02	23		

LA VALEUR CALCULEE DE PHI EST 1,979

CALCUL DES CONTRASTES DE SCHEFFE CAR L'HYPOTHESE HO EST REJETEE AVEC COMME RSE 3,4668

ALPH(2) = ALPH(1) + (,1356, 1,8288)

ALPH(3) = ALPH(1) + (,0768, 1,7657)
ALPH(3) = ALPH(2) + (-,9032, ,7857)

ATE = OM

LES [illegible] A B A

NB A LES Y DU GROUPE SONT 5,5 6,0 6,3 3,8 5,9 3,8
6,0 6,0

NB A LES Y DU GROUPE SONT 5,0 6,5 7,3 5,8 6,8 6,6
5,8 5,2

NB A LES Y DU GROUPE SONT 6,0 6,3 6,3 5,9 7,7 6,8
6,7 5,0

GROUPE NUMERO 1 MOYENNE= 6,681 ECART-TYPE= ,835
GROUPE NUMERO 2 MOYENNE= 5,230 ECART-TYPE= ,446
GROUPE NUMERO 3 MOYENNE= 5,236 ECART-TYPE= 1,130
MOYENNE GENERALE= 5,183

ALPHA(1)= -,681
ALPHA(2)= ,014
ALPHA(3)= ,666

NB A LES RESIDUS DU GROUPE SONT ,009 -,211 -1,081 ,300 -1,221
,010 ,040 ,000

NB A LES RESIDUS DU GROUPE SONT ,000 1,500 -,810 -1,000 ,700
-,020 -,050 -,000

NB A LES RESIDUS DU GROUPE SONT -,000 -,916 ,666 2,534 -,366
-,000 -,250 ,300

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,69790E+01	2	,34897E+01	3,775
ERREUR	,19410E+02	21	,92431E+00	
TOTAL	,26390E+02	23		

LA VALEUR CALCULEE DE PHI EST 1,586

CALCUL DES CONTRASTES DE SCHEFFE CAR L'HYPOTHESE H0 EST REJETEE AVEC COMME FFF 3,4668

ALPH(2) - ALPH(1) = (,0530, 2,5885)

ALPH(3) - ALPH(1) = (-,5008, 1,9908)
ALPH(3) - ALPH(2) = (-1,8525, ,6720)

ATB: - 01

LES VALEURS DU GROUPE SONT
0,0 0,1 0,2 0,6 0,8 0,8 0,8 0,9

NB DE LES V DU GROUPE SONT
0,0 0,0 0,5 0,3 0,0 0,5 0,0 0,0

NB DE LES V DU GROUPE SONT
0,0 0,0 0,0 0,0 0,0 0,0 0,0 0,0

GROUPE NUMERO 1 MOYENNE= 0,275 ECART-TYPE= ,121
GROUPE NUMERO 2 MOYENNE= 0,261 ECART-TYPE= ,064
GROUPE NUMERO 3 MOYENNE= 0,829 ECART-TYPE= ,510
MOYENNE GENERALE= 0,788

ALPHA(1)= -,513
ALPHA(2)= ,513
ALPHA(3)= ,000

NB DE LES RESIDUS DU GROUPE SONT
-,345 -,135 ,305 -,035 -,255 ,295 ,355 -,405

NB DE LES RESIDUS DU GROUPE SONT
-,001 -,511 2,100 ,070 ,500 -,731 -,071 -,271

NB DE LES RESIDUS DU GROUPE SONT
-,129 -,249 ,121 -,039 -,469 -,069 1,151 ,001

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,39104E+01	2	,19552E+01	4,312
ERREUR	,95212E+01	21	,45339E+00	
TOTAL	,13432E+02	23		

LA VALEUR CALCULEE DE PHI EST 1,696

CALCUL DES CONTRASTES DE SCHEFFE CAR L'HYPOTHESE HO EST REJETEE AVEC COMME FFF 3,0668

ALPHE 2) - ALPH(1) E (,0007, 1,0728)

ALPH(3) - ALPH(1) E (-,1328, 1,4803)
ALPH(3) - ALPH(2) E (-1,3198, ,0580)

LES MOYENNES DES GROUPES SONT

3,9 4,1 3,8 3,2 3,8 3,8 3,7 3,8

LES Y DU GROUPE SONT

3,8 3,1 3,1 3,0 3,0 3,0 3,7 3,8

LES Y DU GROUPE SONT

3,1 3,9 3,9 3,1 3,4 3,0 3,2 3,0

GROUPE NUMERO 1 VARIANCE 1,107 ECART-TYPE ,900
GROUPE NUMERO 2 VARIANCE ,811 ECART-TYPE ,111
GROUPE NUMERO 3 VARIANCE ,241 ECART-TYPE ,200
MOYENNE GENERALE 3,663

ALPHA(1)= -,200
ALPHA(2)= ,051
ALPHA(3)= ,171

LES RESIDUS DU GROUPE SONT

,288 -1,071 ,188 ,318 ,220 ,220 -,082 ,300

LES RESIDUS DU GROUPE SONT

-,144 ,218 ,188 -,158 1,018 -,080 -,198 -,080

LES RESIDUS DU GROUPE SONT

-,771 ,218 ,200 -,231 ,260 ,079 -,181 -,071

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,899988E+01	2	,129568+01	6,920
ERREUR	,37772E+01	21	,17987E+00	
TOTAL	,62681E+01	23		

LA VALEUR CALCULEE DE PHI EST = 2,188

CALCUL DES CONTRASTES DE SCHEFFE CAR L'HYPOTHESE HO EST REJETEE AVEC COMME FFF 3,0088

ALPH(2) - ALPH(1) E (,1726, 1,2486)

ALPH(3) - ALPH(1) E (-,0896, ,0671)
ALPH(3) - ALPH(2) E (-1,1809, -,0061)

[redacted]

SOURCE	DF	SC	MC	
ENTRE LES GROUPES	.1231E+07	3	.4103E+06	3.797
GROUPES	.3270E+07	30	.1089E+06	
TOTAL	.4501E+07	33		

LA VALEUR CALCULEE DE PHI EST 1.383

CALCUL DES CONTRASTES DE SCHEFFE SUR L'HYPOTHESE HO EST REJETEE AVEC COMME PPP 3.0920

[redacted]

CPK - CM

TR 3
LES N(I)= 8 7 8

NB 8 LES Y DU GROUPE SONT
2197.7 2945.1 2034.1 2384.3 2216.8 2981.7 1855.8 2216.8

NB 7 LES Y DU GROUPE SONT
2240.2 2551.9 1797.8 2105.9 1918.7 2987.2 2804.8

NB 8 LES Y DU GROUPE SONT
2470.8 3040.0 2007.8 2121.8 3818.4 3020.8 3118.9 3691.3

GROUPE NUMERO 1 MOYENNE= 2387.688 ECART-TYPE= 409.699
GROUPE NUMERO 2 MOYENNE= 2338.586 ECART-TYPE= 440.987
GROUPE NUMERO 3 MOYENNE= 2862.600 ECART-TYPE= 549.168
MOYENNE GENERALE= 2558.800

ALPHA(1)= -211.112
ALPHA(2)= -220.214
ALPHA(3)= 883.408

NB 8 LES RESIDUS DU GROUPE SONT
-189.887 557.413 -313.587 -34.013 -131.287 634.013 -491.87 -131.287

NB 7 LES RESIDUS DU GROUPE SONT
-97.386 213.314 -541.186 -232.686 -421.886 608.814 466.214

NB 8 LES RESIDUS DU GROUPE SONT
-692.000 97.800 -355.200 -841.000 847.800 58.000 156.300 528.700

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.20004E+07	2	.10002E+07	4.492
ERREUR	.44529E+07	20	.22264E+06	
TOTAL	.64533E+07	22		

LA VALEUR CALCULEE DE PHI EST 1.731

CALCUL DES CONTRASTES DE SCHEFFE PAR L'HYPOTHESE H0 EST REJETEE AVEC COMME FFF 3.8928

ALPH(2) - ALPH(1) = (-454.5083, 636.3408)

ALPH(3) - ALPH(1) = (4.6881, 1238.8731)
ALPH(3) - ALPH(2) = (21.4523, 1269.4008)

```
                                        CPK = GL

TR  1
LES N(I)=   8   8   8

NB  8 LES Y DU GROUPE SONT
  1900,8    1837,7    1349,6    2685,5    1431,0    2124,0    2147,8    1853,5

NB  8 LES Y DU GROUPE SONT
  2020,2    2020,2    2001,5    1728,8    1846,3    1376,8    2087,5    2608,7

NB  8 LES Y DU GROUPE SONT
  2486,6    2177,0    2555,6    2605,8    2963,0    3589,4    2552,2    3226,5

GROUPE NUMERO  1      MOYENNE=  1900,638      ECART-TYPE=  387,099
GROUPE NUMERO  2      MOYENNE=  1941,038      ECART-TYPE=  309,443
GROUPE NUMERO  3      MOYENNE=  2780,575      ECART-TYPE=  458,237
   MOYENNE GENERALE=   2210,404

ALPHA( 1)=   -309,767
ALPHA( 2)=   -269,404
ALPHA( 3)=    570,171

NB  8 LES RESIDUS DU GROUPE SONT
   90,163   -82,937  -550,237   584,863  -888,737   228,263   206,763   -87,137

NB  8 LES RESIDUS DU GROUPE SONT
   59,200    59,200    80,500  -232,200  -116,700  -586,200   126,500   887,700

NB  8 LES RESIDUS DU GROUPE SONT
 -242,975  -592,575  -213,975  -164,175   194,325   810,825  -217,375   425,925

                                SOURCE      SS          DL        MS          F
**********************************************************************************
                                ENTRE LES   .37606E+07     2     .18833E+07   11,723
                                GROUPES
                                ERREUR      .33737E+07    21     .16065E+06
**********************************************************************************
                                TOTAL       .71403E+07    23

LA VALEUR CALCULEE DE PHI EST      2,796

CALCUL DES CONTRASTES DE SCHEFFE CAR L'HYPOTHESE H0 EST REJETEE AVEC COMME FFF      3,4668

ALPH( 2) - ALPH( 1) = (-269,404, 341,2312, 588,0662)

ALPH( 3) - ALPH( 1) = ( 341,2312, 1196,6412)
ALPH( 3) - ALPH( 2) = ( 270,8713, 1339,2787)
```

LDH$_{21}$ = 61

T= 3
LES N(I)= 7 8 8

N= 7 LES Y DU GROUPE SONT
490,2 378,9 388,0 573,9 465,3 385,9 192,2

N= 8 LES Y DU GROUPE SONT
277,6 392,3 415,1 393,8 384,8 178,8 366,7 490,8

N= 8 LES Y DU GROUPE SONT
404,9 490,8 611,6 682,0 515,8 457,0 487,6 342,3

GROUPE NUMERO 1 MOYENNE= 408,343 ECART-TYPE= 121,250
GROUPE NUMERO 2 MOYENNE= 362,338 ECART-TYPE= 84,489
GROUPE NUMERO 3 MOYENNE= 505,200 ECART-TYPE= 103,604
MOYENNE GENERALE= 426,048

ALPHA(1)= -17,705
ALPHA(2)= -41,660
ALPHA(3)= 79,152

N= 7 LES RESIDUS DU GROUPE SONT
-17,857 -31,443 -40,343 165,557 56,957 -22,443 -216,143

N= 8 LES RESIDUS DU GROUPE SONT
-44,737 29,013 52,713 31,013 22,013 -183,588 4,313 128,013

N= 8 LES RESIDUS DU GROUPE SONT
-18,300 -18,800 100,400 176,800 10,600 -88,200 -57,600 -162,900

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,84736E+05	2	,42368E+05	3,752
ERREUR	,22585E+06	20	,11292E+05	
TOTAL	,31058E+06	22		

LA VALEUR CALCULEE DE PHI EST 1,582

CALCUL DES CONTRASTES DE SCHEFFE PAR L'HYPOTHESE HO EST REJETEE AVEC COMME FEF 3,4928

ALPH(2) - ALPH(1) = (-101,3164, 99,4057)

ALPH(3) - ALPH(1) = (-48,5039, 242,2182)
ALPH(3) - ALPH(2) = (2,3604, 243,2486)

LDH$_3$ = OL

T= 1
LES N(I)= 7 8 9

N= 7 LES Y DU GROUPE SONT
954,1 350,1 337,9 439,4 391,8 293,9 189,3

N= 8 LES Y DU GROUPE SONT
254,0 392,3 377,7 321,1 328,8 136,7 282,0 430,5

N= 9 LES Y DU GROUPE SONT
854,5 823,7 538,4 505,8 465,2 408,0 355,8 297,2

GROUPE NUMERO 1 MOYENNE= 350,900 ECART-TYPE= 90,987
GROUPE NUMERO 2 MOYENNE= 315,338 ECART-TYPE= 92,631
GROUPE NUMERO 3 MOYENNE= 431,075 ECART-TYPE= 78,369
MOYENNE GENERALE= 366,417

ALPHA(1)= -15,517
ALPHA(2)= -51,080
ALPHA(3)= 66,658

N= 7 LES RESIDUS DU GROUPE SONT
103,200 ,800 -13,000 88,500 40,700 -57,000 -101,600

N= 8 LES RESIDUS DU GROUPE SONT
-61,337 76,963 62,363 5,763 13,063 -178,638 -33,337 115,163

N= 8 LES RESIDUS DU GROUPE SONT
23,425 -7,375 107,325 74,725 34,125 -23,075 -75,275 -133,875

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,56004E+05	2	,28002E+05	3,667
ERREUR	,15273E+06	20	,76364E+04	
TOTAL	,20873E+06	22		

LA VALEUR CALCULEE DE PHI EST 1,568

CALCUL DES CONTRASTES DE SCHEFFE PAR L'HYPOTHESE HO EST REJETEE AVEC COMME FFF 3,8928

ALPH(2) - ALPH(1) = (-155,0944, 83,9738)

ALPH(3) - ALPH(1) = (-39,3609, 199,7109)
ALPH(3) - ALPH(2) = (,2588, 231,2202)

13.

M = 10N = M

Ts 3
LES N(I)= 8 7 6

NB A LES Y DU GROUPE SONT
=89,8 =8,2 =59,8 A9,8 8,8 89,5 71,5 33,7

NB 7 LES Y DU GROUPE SONT
143,8 61,3 81,3 96,8 96,8 88,7 66,5

NB 6 LES Y DU GROUPE SONT
103,8 33,5 =86,7 88,4 77,9 88,9

GROUPE NUMERO 1 MOYENNE= 8,263 ECART-TYPE= 58,478
GROUPE NUMERO 2 MOYENNE= 88,363 ECART-TYPE= 19,813
GROUPE NUMERO 3 MOYENNE= 88,200 ECART-TYPE= 53,328
MOYENNE GENERALE= 83,900

ALPHA(1)= =35,638
ALPHA(2)= 36,863
ALPHA(3)= 4,500

NB 8 LES RESIDUS DU GROUPE SONT
=88,363 =18,863 =87,863 61,538 =,263 32,238 63,238 25,438

NB 7 LES RESIDUS DU GROUPE SONT
73,857 =19,843 ,957 =25,943 =23,943 8,357 =13,843

NB 6 LES RESIDUS DU GROUPE SONT
55,800 =18,900 =84,900 32,200 29,700 =7,300

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	,19567E+05	2	,97835E+04	3,884
ERREUR	,45345E+05	18	,25192E+04	
TOTAL	,64912E+05	20		

LA VALEUR CALCULEE DE PHI EST 1,609

CALCUL DES CONTRASTES DE SCHEFFE CAR L'HYPOTHESE HO EST REJETEE AVEC COMME FFF 3,5546

ALPH(2) - ALPH(1) E (2,8196, 141,3813)

ALPH(3) - ALPH(1) E (=32,5362, 112,2112)
ALPH(3) - ALPH(2) E (=106,5981, 42,3106)

APPENDIX E

ORGAN COMPARISONS FOR EACH DEPENDANT VARIABLE ON COMBINED DATA FROM ALL TRAINING GROUPS: ONE WAY ANALYSES OF VARIANCE AND OTHER RELATED STATISTICS

This appendix contains organ comparisons: one way analysis of variance and Scheffé's contrasts. The computer print-outs appear in the following order:

1. W_{abs}
2. W_{rel}
3. ATP
4. PC
5. ATP + PC
6. CPK
7. AK
8. LDH_{21}
9. LDH_3
10. LDH_{21}/LDH_3
11. M-LDH
12. H-LDH
13. TOTAL LDH

Legend for the computer print outs:

Group No.	Organ
1	TA
2	P
3	GM
4	GL
5	S
6	H
7	L

Moyenne = Mean
Ecart-type = Standard deviation

For the Scheffé's contrasts (alpha (j) - alpha (i)), two positive (or negative) limits of the confidence interval ($P < 0.05$) indicates that organ j is bigger (or smaller) than organ i.

W_{abs}

[illegible] NUMERO	1	MOYENNE=	909.645	ECART-TYPE=	83.950
[illegible] NUMERO	2	MOYENNE=	984.567	ECART-TYPE=	57.218
[illegible] NUMERO	3	MOYENNE=	1219.954	ECART-TYPE=	99.813
[illegible] NUMERO	4	MOYENNE=	1421.754	ECART-TYPE=	151.571
[illegible] NUMERO	5	MOYENNE=	253.430	ECART-TYPE=	35.445
[illegible] NUMERO	6	MOYENNE=	1409.121	ECART-TYPE=	132.329
[illegible] NUMERO	7	MOYENNE=	14114.060	ECART-TYPE=	3276.097

MOYENNE GENERALE= 2924.717

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.35540E+10	6	.59240E+09	359.996
ERREUR	.24021E+09	155	.15014E+07	
TOTAL	.33031E+10	161		

ALPH(2) - ALPH(1) = (-1739.3213, 889.3538)

ALPH(3) - ALPH(1) = (-1084.0338, 1544.6505)
ALPH(3) - ALPH(2) = (-659.0347, 1969.6297)

ALPH(4) - ALPH(1) = (-872.2338, 1756.4505)
ALPH(4) - ALPH(2) = (-447.2547, 2181.4297)
ALPH(4) - ALPH(3) = (-1102.5422, 1526.1422)

ALPH(5) - ALPH(1) = (-2064.7671, 592.3363)
ALPH(5) - ALPH(2) = (-1639.7879, 1017.3155)
ALPH(5) - ALPH(3) = (-2295.0754, 352.0280)
ALPH(5) - ALPH(4) = (-2566.8754, 150.2280)

ALPH(6) - ALPH(1) = (-800.6635, 1897.6159)
ALPH(6) - ALPH(2) = (-473.6838, 2322.6951)
ALPH(6) - ALPH(3) = (-1128.9723, 1567.2076)
ALPH(6) - ALPH(4) = (-1340.7728, 1455.5076)
ALPH(6) - ALPH(5) = (-175.8162, 2647.1075)

ALPH(7) - ALPH(1) = (11810.0720, 14439.7563)
ALPH(7) - ALPH(2) = (12235.0512, 14863.7355)
ALPH(7) - ALPH(3) = (11579.7937, 14208.4480)
ALPH(7) - ALPH(4) = (11367.9937, 13995.6480)
ALPH(7) - ALPH(5) = (12533.0779, 15160.1313)
ALPH(7) - ALPH(6) = (11283.7083, 14023.0795)

W_{rel}

GROUPE NUMERO	1	MOYENNE=	1.774	ECART-TYPE=	.222
GROUPE NUMERO	2	MOYENNE=	1.014	ECART-TYPE=	.147
GROUPE NUMERO	3	MOYENNE=	4.107	ECART-TYPE=	.263
GROUPE NUMERO	4	MOYENNE=	2.567	ECART-TYPE=	.371
GROUPE NUMERO	5	MOYENNE=	.453	ECART-TYPE=	.075
GROUPE NUMERO	6	MOYENNE=	2.629	ECART-TYPE=	.235
GROUPE NUMERO	7	MOYENNE=	26.000	ECART-TYPE=	3.242

MOYENNE GENERALE= 5.342

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.12111E+05	6	.20184E+04	1249.647
ERREUR	.25035E+03	155	.16152E+01	
TOTAL	.12361E+05	161		

ALPH(2) - ALPH(1) = (-2.0806, .5594)

ALPH(3) - ALPH(1) = (-.9078, 1.7322)

ALPH(3) - ALPH(2) = (-.1472, 2.4928)

ALPH(4) - ALPH(1) = (-.5272, 2.1128)

ALPH(4) - ALPH(2) = (.2334, 2.8734)

ALPH(4) - ALPH(3) = (-.9393, 1.7007)

ALPH(5) - ALPH(1) = (-2.6555, .0131)

ALPH(5) - ALPH(2) = (-1.0949, .7737)

ALPH(5) - ALPH(3) = (-3.0676, -.3991)

ALPH(5) - ALPH(4) = (-3.4493, -.7797)

ALPH(6) - ALPH(1) = (-.5496, 2.2587)

ALPH(6) - ALPH(2) = (.2110, 3.0193)

ALPH(6) - ALPH(3) = (-.9518, 1.8469)

ALPH(6) - ALPH(4) = (-1.3425, 1.4659)

ALPH(6) - ALPH(5) = (.7551, 3.5933)

ALPH(7) - ALPH(1) = (22.9058, 25.5458)

ALPH(7) - ALPH(2) = (23.6564, 26.3065)

ALPH(7) - ALPH(3) = (22.4937, 25.1237)

ALPH(7) - ALPH(4) = (22.1130, 24.7530)

ALPH(7) - ALPH(5) = (24.2127, 26.8413)

ALPH(7) - ALPH(6) = (21.4671, 24.7755)

ATP

GROUPE NUMERO	1	MOYENNE=	5.497	ECART-TYPE=	.765
GROUPE NUMERO	2	MOYENNE=	4.095	ECART-TYPE=	.713
GROUPE NUMERO	3	MOYENNE=	5.162	ECART-TYPE=	1.071
GROUPE NUMERO	4	MOYENNE=	4.700	ECART-TYPE=	.734
GROUPE NUMERO	5	MOYENNE=	3.453	ECART-TYPE=	.522
GROUPE NUMERO	6	MOYENNE=	4.239	ECART-TYPE=	.445
MOYENNE GENERALE=		4.693			

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.63331E+02	5	.12775E+02	23.022
ERREUR	.74920E+02	135	.55495E+00	
TOTAL	.13825E+03	140		

ALPH(2) - ALPH(1) = (	-1.2469,	.2214)
ALPH(3) - ALPH(1) = (	-1.0513,	.3913)
ALPH(3) - ALPH(2) = (	-.5564,	.9119)
ALPH(4) - ALPH(1) = (	-1.4055,	.0171)
ALPH(4) - ALPH(2) = (	-.9306,	.5377)
ALPH(4) - ALPH(3) = (	-1.1005,	.3521)
ALPH(5) - ALPH(1) = (	-2.7613,	-1.3097)
ALPH(5) - ALPH(2) = (	-2.2564,	-.7031)
ALPH(5) - ALPH(3) = (	-2.4263,	-.9737)
ALPH(5) - ALPH(4) = (	-2.0521,	-.5995)
ALPH(6) - ALPH(1) = (	-2.0015,	-.5162)
ALPH(6) - ALPH(2) = (	-1.4965,	.0042)
ALPH(6) - ALPH(3) = (	-1.6665,	-.1812)
ALPH(6) - ALPH(4) = (	-1.2923,	.1529)
ALPH(6) - ALPH(5) = (	.0335,	1.5188)

PC

GROUPE NUMERO	1	MOYENNE=	10.309	ECART-TYPE=	3.031
GROUPE NUMERO	2	MOYENNE=	14.705	ECART-TYPE=	4.571
GROUPE NUMERO	3	MOYENNE=	13.311	ECART-TYPE=	3.110
GROUPE NUMERO	4	MOYENNE=	14.203	ECART-TYPE=	2.574
GROUPE NUMERO	5	MOYENNE=	9.986	ECART-TYPE=	2.253
GROUPE NUMERO	6	MOYENNE=	5.461	ECART-TYPE=	1.359
MOYENNE GENERALE=		12.151			

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.220205E+04	5	.440409E+03	48.096
ERREUR	.100722E+04	110	.915659E+01	
TOTAL	.320927E+04	115		

ALPH(2) - ALPH(1) = (-6.8158, -.3202)

ALPH(3) - ALPH(1) = (-5.2408, 1.2448)
ALPH(3) - ALPH(2) = (-1.6679, 4.3178)

ALPH(4) - ALPH(1) = (-7.3858, -.8154)
ALPH(4) - ALPH(2) = (-3.0128, 2.7576)
ALPH(4) - ALPH(3) = (-5.2378, 1.1825)

ALPH(5) - ALPH(1) = (-12.6374, -6.0570)
ALPH(5) - ALPH(2) = (-9.0544, -2.4940)
ALPH(5) - ALPH(3) = (-10.6204, -4.0590)
ALPH(5) - ALPH(4) = (-8.5786, -1.9245)

ALPH(6) - ALPH(1) = (-16.1796, -9.5163)
ALPH(6) - ALPH(2) = (-12.6056, -5.9433)
ALPH(6) - ALPH(3) = (-14.1016, -7.5133)
ALPH(6) - ALPH(4) = (-12.1203, -5.3744)
ALPH(6) - ALPH(5) = (-6.0637, -.1228)

ATP + PC

GROUPE NUMERO	1	MOYENNE=	23.735	ECART-TYPE=	3.094
GROUPE NUMERO	2	MOYENNE=	19.693	ECART-TYPE=	4.634
GROUPE NUMERO	3	MOYENNE=	21.471	ECART-TYPE=	3.723
GROUPE NUMERO	4	MOYENNE=	19.052	ECART-TYPE=	3.033
GROUPE NUMERO	5	MOYENNE=	12.420	ECART-TYPE=	2.595
GROUPE NUMERO	6	MOYENNE=	9.647	ECART-TYPE=	1.640
MOYENNE GENERALE=		17.772			

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.28037E+04	5	.57774E+03	53.585
ERREUR	.11953E+04	111	.10782E+02	
TOTAL	.40355E+04	116		

ALPH(2) - ALPH(1) = (-7.5557, -.5193)

ALPH(3) - ALPH(1) = (-5.7832, 1.2532)
ALPH(3) - ALPH(2) = (-1.7457, 5.2907)

ALPH(4) - ALPH(1) = (-8.2481, -1.1197)
ALPH(4) - ALPH(2) = (-4.2106, 2.9178)
ALPH(4) - ALPH(3) = (-5.9831, 1.1453)

ALPH(5) - ALPH(1) = (-14.8797, -7.7513)
ALPH(5) - ALPH(2) = (-10.8422, -3.7138)
ALPH(5) - ALPH(3) = (-12.6147, -5.4853)
ALPH(5) - ALPH(4) = (-10.2412, -3.0220)

ALPH(6) - ALPH(1) = (-17.6529, -10.5245)
ALPH(6) - ALPH(2) = (-13.6154, -6.4870)
ALPH(6) - ALPH(3) = (-15.3879, -8.2595)
ALPH(6) - ALPH(4) = (-13.0143, -5.7259)
ALPH(6) - ALPH(5) = (-6.3828, .8055)

CPK

GROUPE NUMERO	1	MOYENNE=	2589.319	ECART-TYPE=	405.879
GROUPE NUMERO	2	MOYENNE=	2400.443	ECART-TYPE=	432.791
GROUPE NUMERO	3	MOYENNE=	2000.000	ECART-TYPE=	541.602
GROUPE NUMERO	4	MOYENNE=	2210.404	ECART-TYPE=	567.173
GROUPE NUMERO	5	MOYENNE=	1076.729	ECART-TYPE=	155.012
GROUPE NUMERO	6	MOYENNE=	870.250	ECART-TYPE=	155.542
GROUPE NUMERO	7	MOYENNE=	14.252	ECART-TYPE=	3.608
MOYENNE GENERALE=		1670.570			

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.14473E+09	6	.24125E+03	164.555
ERREUR	.22553E+03	154	.14651E+06	
TOTAL	.16732E+09	160		

ALPH(2) - ALPH(1) = (-518.5810, 312.3293)

ALPH(3) - ALPH(1) = (-446.2244, 385.1864)
ALPH(3) - ALPH(2) = (-333.7912, 478.5042)

ALPH(4) - ALPH(1) = (-790.4561, 32.6363)
ALPH(4) - ALPH(2) = (-677.9340, 125.0554)
ALPH(4) - ALPH(3) = (-750.2905, 53.4989)

ALPH(5) - ALPH(1) = (-1929.2853, -1096.0745)
ALPH(5) - ALPH(2) = (-1815.8520, -1003.5557)
ALPH(5) - ALPH(3) = (-1858.2035, -1075.9132)
ALPH(5) - ALPH(4) = (-1535.5597, -731.7703)

ALPH(6) - ALPH(1) = (-2130.6203, -1307.5178)
ALPH(6) - ALPH(2) = (-2018.0892, -1214.2938)
ALPH(6) - ALPH(3) = (-2090.4447, -1235.6533)
ALPH(6) - ALPH(4) = (-1787.7584, -942.5579)
ALPH(6) - ALPH(5) = (-608.3938, 195.4056)

ALPH(7) - ALPH(1) = (-2990.7727, -2159.3619)
ALPH(7) - ALPH(2) = (-2878.3394, -2065.8441)
ALPH(7) - ALPH(3) = (-2950.6959, -2138.4006)
ALPH(7) - ALPH(4) = (-2598.0471, -1794.2577)
ALPH(7) - ALPH(5) = (-1468.5351, -386.3397)
ALPH(7) - ALPH(6) = (-1257.8930, -454.1036)

AK

GROUPE NUMERO	1	MOYENNE=	120.455	ECART-TYPE=	55.570
GROUPE NUMERO	2	MOYENNE=	125.005	ECART-TYPE=	57.042
GROUPE NUMERO	3	MOYENNE=	122.931	ECART-TYPE=	55.405
GROUPE NUMERO	4	MOYENNE=	103.535	ECART-TYPE=	43.301
GROUPE NUMERO	5	MOYENNE=	53.906	ECART-TYPE=	25.779
GROUPE NUMERO	6	MOYENNE=	65.601	ECART-TYPE=	24.910
GROUPE NUMERO	7	MOYENNE=	19.473	ECART-TYPE=	4.544
MOYENNE GENERALE=		90.802			

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.218145E+05	6	.36357E+05	18.874
ERREUR	.261955E+05	136	.19259E+04	
TOTAL	.480100E+06	142		

ALPH(2) - ALPH(1) = (	-51.3861,	46.3499)
ALPH(3) - ALPH(1) = (	-54.3809,	43.2742)
ALPH(3) - ALPH(2) = (	-51.9023,	45.7527)
ALPH(4) - ALPH(1) = (	-69.3843,	29.4838)
ALPH(4) - ALPH(2) = (	-66.9058,	31.9624)
ALPH(4) - ALPH(3) = (	-63.8218,	35.0471)
ALPH(5) - ALPH(1) = (	-119.6764,	-19.4825)
ALPH(5) - ALPH(2) = (	-117.1950,	-17.0039)
ALPH(5) - ALPH(3) = (	-114.1281,	-13.9292)
ALPH(5) - ALPH(4) = (	-100.3198,	1.0592)
ALPH(6) - ALPH(1) = (	-111.0819,	-13.9653)
ALPH(6) - ALPH(2) = (	-109.1427,	-11.9977)
ALPH(6) - ALPH(3) = (	-106.0658,	-8.9130)
ALPH(6) - ALPH(4) = (	-92.2778,	5.5965)
ALPH(6) - ALPH(5) = (	-83.3113,	14.3806)
ALPH(7) - ALPH(1) = (	-159.4438,	-99.5777)
ALPH(7) - ALPH(2) = (	-157.5673,	-97.6991)
ALPH(7) - ALPH(3) = (	-154.5025,	-94.6244)
ALPH(7) - ALPH(4) = (	-139.0948,	-79.0282)
ALPH(7) - ALPH(5) = (	-130.1289,	-69.2540)
ALPH(7) - ALPH(6) = (	-126.0520,	-65.2161)

LDH_{21}

GROUPE NUMERO	1	MOYENNE=	502.000	ECART-TYPE=	143.750
GROUPE NUMERO	2	MOYENNE=	501.525	ECART-TYPE=	124.157
GROUPE NUMERO	3	MOYENNE=	452.519	ECART-TYPE=	101.753
GROUPE NUMERO	4	MOYENNE=	426.048	ECART-TYPE=	110.917
GROUPE NUMERO	5	MOYENNE=	127.700	ECART-TYPE=	39.252
GROUPE NUMERO	6	MOYENNE=	279.114	ECART-TYPE=	80.931
GROUPE NUMERO	7	MOYENNE=	292.242	ECART-TYPE=	80.1[illegible]

MOYENNE GENERALE= 381.162

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.25720E+07	6	.44549E+06	38.241
ERREUR	.16813E+07	144	.11657E+05	
TOTAL	.42502E+07	150		

ALPH(2) - ALPH(1) = (-84.0690, 142.2565)

ALPH(3) - ALPH(1) = (-166.4046, 66.1750)
ALPH(3) - ALPH(2) = (-186.2712, 38.6571)

ALPH(4) - ALPH(1) = (-190.1472, 36.9752)
ALPH(4) - ALPH(2) = (-220.0417, 9.4852)
ALPH(4) - ALPH(3) = (-143.9354, 90.9929)

ALPH(5) - ALPH(1) = (-492.6740, -257.0127)
ALPH(5) - ALPH(2) = (-522.5254, -234.5469)
ALPH(5) - ALPH(3) = (-446.3252, -203.1329)
ALPH(5) - ALPH(4) = (-417.2471, -179.2635)

ALPH(6) - ALPH(1) = (-339.0094, -107.2287)
ALPH(6) - ALPH(2) = (-369.6760, -134.7476)
ALPH(6) - ALPH(3) = (-293.5039, -53.3006)
ALPH(6) - ALPH(4) = (-264.3977, -29.4504)
ALPH(6) - ALPH(5) = (29.7231, 272.9204)

ALPH(7) - ALPH(1) = (-329.9013, -90.8812)
ALPH(7) - ALPH(2) = (-359.7365, -118.4314)
ALPH(7) - ALPH(3) = (-283.5012, -37.0527)
ALPH(7) - ALPH(4) = (-254.4503, -13.1522)
ALPH(7) - ALPH(5) = (39.7722, 289.1310)
ALPH(7) - ALPH(6) = (-110.0964, 136.3521)

LDH_3

[illegible] NUMERO	1	MOYENNE=	412.342	ECART-TYPE=	130.331
[illegible]	2	MOYENNE=	448.952	ECART-TYPE=	110.512
[illegible]	3	MOYENNE=	324.033	ECART-TYPE=	90.027
[illegible]	4	MOYENNE=	386.417	ECART-TYPE=	97.405
[illegible]	5	MOYENNE=	127.010	ECART-TYPE=	41.939
[illegible]	6	MOYENNE=	227.914	ECART-TYPE=	103.507
[illegible]	7	MOYENNE=	249.674	ECART-TYPE=	80.949
MOYENNE GENERALE=		300.379			

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.14335E+07	6	.23977E+06	24.306
ERREUR	.14205E+07	144	.98649E+04	
TOTAL	.28592E+07	150		

ALPH(2) - ALPH(1) = (-67.7745, 140.9955)

ALPH(3) - ALPH(1) = (-134.6014, 79.1847)
ALPH(3) - ALPH(2) = (-172.2909, 43.6532)

ALPH(4) - ALPH(1) = (+150.3092, 58.4607)
ALPH(4) - ALPH(2) = (+188.9244, 22.9548)
ALPH(4) - ALPH(3) = (-126.1030, 89.7561)

ALPH(5) - ALPH(1) = (-388.6406, -167.0227)
ALPH(5) - ALPH(2) = (-421.3161, -202.5532)
ALPH(5) - ALPH(3) = (-259.3925, -135.3332)
ALPH(5) - ALPH(4) = (-330.7013, -120.0334)

ALPH(6) - ALPH(1) = (-191.3205, 22.4557)
ALPH(6) - ALPH(2) = (-229.0009, -13.0558)
ALPH(6) - ALPH(3) = (-167.1177, 53.6795)
ALPH(6) - ALPH(4) = (-146.4752, 69.4689)
ALPH(6) - ALPH(5) = (79.1341, 302.6744)

ALPH(7) - ALPH(1) = (-272.6206, -52.9154)
ALPH(7) - ALPH(2) = (-310.2913, -89.4757)
ALPH(7) - ALPH(3) = (-248.3253, -21.7930)
ALPH(7) - ALPH(4) = (-227.7465, -5.9409)
ALPH(7) - ALPH(5) = (-2.9481, 227.1675)
ALPH(7) - ALPH(6) = (-191.6073, 34.9251)

LDH_{21}/LDH_3

GROUPE NUMERO	1	MOYENNE=	1.229	ECART-TYPE=	.101
GROUPE NUMERO	2	MOYENNE=	1.153	ECART-TYPE=	.097
GROUPE NUMERO	3	MOYENNE=	1.184	ECART-TYPE=	.115
GROUPE NUMERO	4	MOYENNE=	1.155	ECART-TYPE=	.099
GROUPE NUMERO	5	MOYENNE=	.942	ECART-TYPE=	.113
GROUPE NUMERO	6	MOYENNE=	.851	ECART-TYPE=	.070
GROUPE NUMERO	7	MOYENNE=	1.109	ECART-TYPE=	.083

MOYENNE GENERALE= 1.113

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.26503E+01	6	.44409E+00	45.094
ERREUR	.13800E+01	144	.95510E-02	
TOTAL	.40592E+01	150		

ALPH(2) - ALPH(1) = (-.1309, .0676)

ALPH(3) - ALPH(1) = (-.1511, .0604)

ALPH(3) - ALPH(2) = (-.1165, .0971)

ALPH(4) - ALPH(1) = (-.1672, .0393)

ALPH(4) - ALPH(2) = (-.1326, .0761)

ALPH(4) - ALPH(3) = (-.1254, .0802)

ALPH(5) - ALPH(1) = (-.3943, -.1800)

ALPH(5) - ALPH(2) = (-.3597, -.1433)

ALPH(5) - ALPH(3) = (-.3524, -.1313)

ALPH(5) - ALPH(4) = (-.3314, -.1150)

ALPH(6) - ALPH(1) = (-.4739, -.2525)

ALPH(6) - ALPH(2) = (-.4393, -.2257)

ALPH(6) - ALPH(3) = (-.4321, -.2137)

ALPH(6) - ALPH(4) = (-.4111, -.1975)

ALPH(6) - ALPH(5) = (-.1916, .0295)

ALPH(7) - ALPH(1) = (-.1409, .0504)

ALPH(7) - ALPH(2) = (-.1142, .1052)

ALPH(7) - ALPH(3) = (-.1069, .1172)

ALPH(7) - ALPH(4) = (-.0830, .1334)

ALPH(7) - ALPH(5) = (.1006, .3503)

ALPH(7) - ALPH(6) = (.2130, .4403)

M - LDH

GROUPE NUMERO	1	MOYENNE=	449.092	ECART-TYPE=	144.429
GROUPE NUMERO	2	MOYENNE=	443.339	ECART-TYPE=	135.175
GROUPE NUMERO	3	MOYENNE=	377.110	ECART-TYPE=	123.733
GROUPE NUMERO	4	MOYENNE=	345.052	ECART-TYPE=	130.305
GROUPE NUMERO	5	MOYENNE=	45.110	ECART-TYPE=	47.760
GROUPE NUMERO	6	MOYENNE=	43.700	ECART-TYPE=	56.970
GROUPE NUMERO	7	MOYENNE=	241.111	ECART-TYPE=	54.550
MOYENNE GENERALE=		287.444			

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.39293E+07	6	.65489E+06	55.455
ERREUR	.17002E+07	144	.11807E+05	
TOTAL	.56295E+07	150		

ALPH(2) - ALPH(1) = (-115.7516, 112.6465)

ALPH(3) - ALPH(1) = (-189.7251, 44.1608)

ALPH(3) - ALPH(2) = (-189.3530, 45.8030)

ALPH(4) - ALPH(1) = (-218.2305, 10.1506)

ALPH(4) - ALPH(2) = (-217.8945, 12.9206)

ALPH(4) - ALPH(3) = (-149.3007, 85.3560)

ALPH(5) - ALPH(1) = (-522.2726, -285.2997)

ALPH(5) - ALPH(2) = (-521.8362, -282.5721)

ALPH(5) - ALPH(3) = (-453.2701, -205.7210)

ALPH(5) - ALPH(4) = (-419.3993, -180.0951)

ALPH(6) - ALPH(1) = (-523.1348, -289.2407)

ALPH(6) - ALPH(2) = (-522.7625, -285.5158)

ALPH(6) - ALPH(3) = (-454.1077, -212.6314)

ALPH(6) - ALPH(4) = (-420.2755, -184.0283)

ALPH(6) - ALPH(5) = (-124.6006, 119.0505)

ALPH(7) - ALPH(1) = (-329.9619, -88.5004)

ALPH(7) - ALPH(2) = (-329.5533, -85.8039)

ALPH(7) - ALPH(3) = (-250.9143, -12.0332)

ALPH(7) - ALPH(4) = (-226.0713, 16.5030)

ALPH(7) - ALPH(5) = (69.0010, 320.0702)

ALPH(7) - ALPH(6) = (73.4017, 321.2253)

H - LDH

GROUPE NUMERO	1	MOYENNE=	65.937	ECART-TYPE=	94.093
GROUPE NUMERO	2	MOYENNE=	103.727	ECART-TYPE=	92.131
GROUPE NUMERO	3	MOYENNE=	94.257	ECART-TYPE=	99.957
GROUPE NUMERO	4	MOYENNE=	99.065	ECART-TYPE=	77.990
GROUPE NUMERO	5	MOYENNE=	99.930	ECART-TYPE=	50.079
GROUPE NUMERO	6	MOYENNE=	291.875	ECART-TYPE=	114.579
GROUPE NUMERO	7	MOYENNE=	61.297	ECART-TYPE=	61.695

MOYENNE GENERALE= 135.066

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.70234E+06	6	.13209E+06	17.227
ERREUR	.11041E+07	144	.76575E+04	
TOTAL	.18967E+07	150		

ALPHI(2) - ALPHI(1) = (-54.2386, 129.8184)

ALPHI(3) - ALPHI(1) = (-65.9200, 122.5593)
ALPHI(3) - ALPHI(2) = (-104.6612, 85.7207)

ALPHI(4) - ALPHI(1) = (-59.9007, 124.1562)
ALPHI(4) - ALPHI(2) = (-98.6645, 87.3402)
ALPHI(4) - ALPHI(3) = (-91.3023, 98.9990)

ALPHI(5) - ALPHI(1) = (-61.4954, 129.4804)
ALPHI(5) - ALPHI(2) = (-100.2243, 92.6295)
ALPHI(5) - ALPHI(3) = (-92.8566, 104.2123)
ALPHI(5) - ALPHI(4) = (-94.5621, 98.2917)

ALPHI(6) - ALPHI(1) = (131.6000, 320.1984)
ALPHI(6) - ALPHI(2) = (92.9579, 203.3397)
ALPHI(6) - ALPHI(3) = (100.2087, 294.9494)
ALPHI(6) - ALPHI(4) = (98.6200, 289.0019)
ALPHI(6) - ALPHI(5) = (93.4060, 290.4856)

ALPHI(7) - ALPHI(1) = (-101.4390, 92.2087)
ALPHI(7) - ALPHI(2) = (-140.2048, 53.3447)
ALPHI(7) - ALPHI(3) = (-132.8136, 65.8390)
ALPHI(7) - ALPHI(4) = (-134.5125, 61.0059)
ALPHI(7) - ALPHI(5) = (-139.8703, 62.4050)
ALPHI(7) - ALPHI(6) = (-330.4378, -130.7200)

TOTAL LDH

GROUPE NUMERO	1	MOYENNE=	513.021	ECART-TYPE=	105.745
GROUPE NUMERO	2	MOYENNE=	541.200	ECART-TYPE=	127.214
GROUPE NUMERO	3	MOYENNE=	471.371	ECART-TYPE=	107.552
GROUPE NUMERO	4	MOYENNE=	440.104	ECART-TYPE=	144.430
GROUPE NUMERO	5	MOYENNE=	143.595	ECART-TYPE=	41.200
GROUPE NUMERO	6	MOYENNE=	342.729	ECART-TYPE=	109.141
GROUPE NUMERO	7	MOYENNE=	305.032	ECART-TYPE=	89.716

MOYENNE GENERALE= 401.756

SOURCE	SS	DL	MS	F
ENTRE LES GROUPES	.24502E+07	6	.41137E+06	31.035
ERREUR	.19087E+07	144	.13255E+05	
TOTAL	.43769E+07	150		

ALPH(2) - ALPH(1) = (-93.8182, 148.3766)

ALPH(3) - ALPH(1) = (-166.3541, 81.4553)
ALPH(3) - ALPH(2) = (-194.9039, 55.3268)

ALPH(4) - ALPH(1) = (-194.7139, 47.2809)
ALPH(4) - ALPH(2) = (-223.3735, 21.1822)
ALPH(4) - ALPH(3) = (-156.4224, 93.8833)

ALPH(5) - ALPH(1) = (-493.7817, -244.6980)
ALPH(5) - ALPH(2) = (-524.3934, -270.8346)
ALPH(5) - ALPH(3) = (-457.3444, -189.2235)
ALPH(5) - ALPH(4) = (-423.2997, -169.7200)

ALPH(6) - ALPH(1) = (-294.9969, -47.1876)
ALPH(6) - ALPH(2) = (-323.6268, -73.3161)
ALPH(6) - ALPH(3) = (-256.6111, -.6747)
ALPH(6) - ALPH(4) = (-222.5311, 27.7795)
ALPH(6) - ALPH(5) = (69.5056, 328.7015)

ALPH(7) - ALPH(1) = (-336.1245, -81.4548)
ALPH(7) - ALPH(2) = (-364.7209, -107.6159)
ALPH(7) - ALPH(3) = (-297.6224, -35.0473)
ALPH(7) - ALPH(4) = (-263.5233, -5.5202)
ALPH(7) - ALPH(5) = (28.6040, 294.2231)
ALPH(7) - ALPH(6) = (-100.8006, 93.5056)

APPENDIX F

TRAINING GROUP AND ORGAN COMPARISONS FOR EACH DEPENDANT VARIABLE: TWO WAY ANALYSES OF VARIANCE AND OTHER RELATED STATISTICS

This appendix contains the two by two factors analysis of variance in the usual order:

1. W_{abs}
2. W_{rel}
3. ATP
4. PC
5. ATP + PC
6. CPK
7. AK
8. LDH_{21}
9. LDH_3
10. LDH_{21}/LDH_3
11. M-LDH
12. M-LDH
13. TOTAL LDH

MUSCLE VS ANIMAL (2). W_{ABS} 30/03/77 PAGE

FILE NONAME (CREATION DATE = 30/03/77)

* * * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * * *

MESURE
BY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	3562844211.630	8	445330776.379	320.656	.001
MUSCLE	3555520035.251	6	592587005.875	426.687	.001
ANIMAL	7744340.488	2	3872170.244	2.788	.065
2-WAY INTERACTIONS	44645100.319	12	3720425.027	2.679	.003
MUSCLE ANIMAL	44645100.319	12	3720425.027	2.679	.003
EXPLAINED	3607291312.149	20	180364565.607	129.870	.001
RESIDUAL	195822410.584	141	1388811.451		
TOTAL	3803113726.733	161	23621824.390		

168 CASES WERE PROCESSED.
6 CASES (3.6 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	
		.473E+06	2924.2169	4860.2288	.236E+08	(162)
1		23751.5000	989.6458	88.8807	7896.2661	(24)
2		13552.0000	564.6667	57.2177	3273.8632	(24)
3		29278.0000	1219.9582	99.8135	9962.7339	(24)
4		34362.1000	1431.7542	151.5744	22973.6417	(24)
5		5824.9000	253.2304	35.4477	1256.5395	(23)
6		28293.3000	1489.1211	132.3294	17511.0662	(19)
7		.338E+06	14114.0600	3276.2966	.107E+08	(24)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	
		.473E+06	2924.2169	4860.2288	.236E+08	(162)
1		.144E+06	2883.0063	4470.5066	.199E+08	(50)
2		.156E+06	2896.8296	4707.4041	.221E+08	(54)
3		.172E+06	3194.3148	5428.9632	.294E+08	(54)

MUSCLE VS ANIMAL (3). W rel

FILE NONAME (CREATION DATE = 30/03/77) 30/03/77 PAGE

* * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * *

MEASURE
MY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	12112.719	8	1514.090	862.737	.001
MUSCLE	12110.342	6	2018.39	150.09	.001
ANIMAL	2.038	2	1.019	.581	.561
2-WAY INTERACTIONS	.807	12	.072	.041	.999
MUSCLE ANIMAL	.807	12	.072	.041	.999
EXPLAINED	12113.526	20	605.679	345.120	.001
RESIDUAL	247.453	141	1.755		
TOTAL	12361.039	161	76.777		

160 CASES WERE PROCESSED.
0 CASES (.0 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		865.3960	5.3420	8.7622	76.7766	(162)
1		42.5470	1.7727	.2520	.0634	(24)
2		24.5520	1.0230	.1471	.0216	(24)
3		52.1700	2.1738	.2654	.1745	(24)
4		61.2150	2.5506	.3712	.1378	(24)
5		19.8250	.8594	.1758	.0309	(23)
6		49.9510	2.6290	.3359	.1128	(19)
7		624.0870	26.0036	3.2416	10.5077	(24)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		865.3960	5.3420	8.7622	76.7766	(162)
1		293.4000	5.4333	8.7583	76.6736	(54)
2		292.7740	5.4217	8.7859	77.1000	(54)
3		279.2220	5.1708	8.6119	74.1467	(54)

MUSCLE VS ANIMAL (4).

FILE NONAME (CREATION DATE = 30/03/77) 30/03/77 PAGE

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

MESURE
BY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	79.887	7	11.412	26.114	.001
MUSCLE	63.747	5	12.749	29.173	.001
ANIMAL	16.006	2	8.003	18.312	.001
2-WAY INTERACTIONS	5.159	10	.516	1.180	.310
MUSCLE ANIMAL	5.159	10	.516	1.180	.310
EXPLAINED	85.046	17	5.003	11.447	.001
RESIDUAL	53.755	123	.437		
TOTAL	138.801	140	.991		

[illegible] CASES WERE PROCESSED.
3 CASES (2.1 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		661.7600	4.6933	.9957	.9914	(141)
1		131.9400	5.4975	.7653	.5857	(24)
2		114.6500	4.9848	.7130	.5084	(23)
3		123.0000	5.1250	1.0712	1.1474	(24)
4		114.9200	4.7883	.7642	.5840	(24)
5		83.1000	3.4625	.5220	.2725	(24)
6		93.2500	4.2386	.4459	.1988	(22)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		661.7600	4.6933	.9957	.9914	(141)
1		205.5400	4.2779	.7219	.5211	(48)
2		234.8800	5.1061	1.0400	1.0816	(46)
3		221.5400	4.7136	1.0373	1.0760	(47)

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

MEASURE
BY MUSCLE
ANIMAL

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	2211.308	7	315.901	33.000	.001
MUSCLE	2200.249	5	440.050	45.969	.001
ANIMAL	9.342	2	4.671	.488	.615
2-WAY INTERACTIONS	59.744	10	5.974	.624	.790
MUSCLE ANIMAL	59.744	10	5.974	.624	.790
EXPLAINED	2271.053	17	133.591	13.955	.001
RESIDUAL	938.138	98	9.573		
TOTAL	3209.190	115	27.906		

144 CASES WERE PROCESSED.
29 CASES (19.4 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		1525.5000	13.1509	5.2826	27.9060	(116)
1		366.1700	18.3085	3.0309	9.1862	(20)
2		294.7100	14.7355	4.5711	20.8948	(20)
3		326.2100	16.3105	3.1101	9.6725	(20)
4		239.9500	14.2079	2.2734	7.1496	(17)
5		170.1700	8.9563	2.2584	5.1005	(19)
6		98.2900	5.4606	1.3593	1.8477	(18)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		1525.5000	13.1509	5.2826	27.9060	(116)
1		443.8400	12.6811	5.1380	26.3990	(35)
2		547.4100	13.3515	4.9499	24.5020	(41)
3		534.2500	13.3563	5.8188	33.8580	(40)

MUSCLE VS ANIMAL (6).

FILE NONAME (CREATION DATE = 30/03/77) 30/03/77 PAGE

* * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * *

MEASURE BY MUSCLE ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	2926.672	7	418.096	38.741	.001
MUSCLE	2893.747	5	578.749	53.627	.001
ANIMAL	37.949	2	18.975	1.758	.178
2-WAY INTERACTIONS	90.389	10	9.039	.838	.594
MUSCLE ANIMAL	90.389	10	9.039	.838	.594
EXPLAINED	3017.061	17	177.474	16.445	.001
RESIDUAL	1068.416	99	10.792		
TOTAL	4085.477	116	35.220		

144 CASES WERE PROCESSED.
27 CASES (18.7 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		2079.3300	17.7721	5.9346	35.2196	(117)
1		474.7100	23.7355	3.0938	9.5718	(20)
2		393.9600	19.6980	4.6043	21.2024	(20)
3		429.4100	21.4705	3.7232	13.8621	(20)
4		381.9800	19.0516	3.1276	9.7841	(20)
5		255.9800	12.4200	2.5950	6.7338	(20)
6		143.2900	9.5527	1.8397	3.3856	(15)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		2079.3300	17.7721	5.9346	35.2196	(117)
1		593.6300	16.9609	5.5990	31.3370	(35)
2		762.1100	18.1455	5.8000	33.6408	(42)
3		723.5900	18.0898	5.4276	41.3138	(40)

MUSCLE VS ANIMAL (7).

FILE NONAME (CREATION DATE = 30/03/77)

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

MESURE
BY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	14A203692.524	8	18525449.066	175.594	.001
MUSCLE	1444A2A79.AA2	6	24004079.980	227.248	.001
ANIMAL	344A123.851	2	1724061.926	16.342	.001
2-WAY INTERACTIONS	4388A81.A10	12	362073.484	3.432	.001
MUSCLE ANIMAL	4344881.810	12	362073.484	3.432	.001
EXPLAINED	152548474.335	20	7627423.717	72.297	.001
RESIDUAL	14770218.208	140	105501.559		
TOTAL	167318692.543	160	1045741.828		

168 CASES WERE PROCESSED.
7 CASES (8.2 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		.269E+06	1673.5701	1022.8152	.104E+07	(161)
1		54375.7000	2589.3190	485.A792	.104E+06	(21)
2		57188.2000	2446.8435	452.7904	.205E+06	(23)
3		58852.4000	2558.8000	541.6014	.293E+06	(23)
4		53049.7000	2210.4042	557.1762	.31[illegible]E+06	(24)
5		24705.0000	1076.7391	166.0117	27559.[illegible]52	(23)
6		20[illegible].0000	870.2500	155.5426	24193.[illegible]0a	(24)
7		327.7900	14.2517	9.8575	75.[illegible]761	(23)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		.269E+06	1673.5701	1022.8152	.104E+07	(161)
1		86397.5200	1570.8[illegible]0	901.A166	.813E+06	(55)
2		82849.1300	1563.1911	987.2194	.A97E+06	(53)
3		.100E+06	1890.5309	1185.0475	.140E+07	(53)

MUSCLE VS ANIMAL (B).

FILE NONAME (CREATION DATE = 30/03/77)

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

MESURE
BY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	219719.000	8	27464.876	12.967	.001
MUSCLE	218062.811	6	36343.802	17.163	.001
ANIMAL	1578.294	2	789.147	.373	.690
2-WAY INTERACTIONS	2000.367	12	166.697	.079	.999
MUSCLE ANIMAL	2000.367	12	166.697	.079	.999
EXPLAINED	221719.376	20	11085.969	5.234	.001
RESIDUAL	258403.028	122	2118.058		
TOTAL	480122.404	142	3381.144		

100 CASES WERE PROCESSED.
25 CASES (14.9 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		12984.7500	90.8024	58.1476	3381.1437	(143)
1		2698.1800	128.4848	55.5498	3084.0009	(21)
2		2646.1300	126.0062	57.1023	3263.8205	(21)
3		2561.5800	122.0276	58.0878	3374.2218	(21)
4		2174.6900	103.5567	49.8016	2480.2105	(21)
5		1119.2200	58.9063	25.7786	664.5387	(19)
6		1373.5100	65.4310	24.9104	620.5301	(21)
7		389.4600	19.4730	4.5443	20.6505	(20)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		12984.7500	90.8024	58.1476	3381.1437	(143)
1		4699.2300	93.7340	50.3264	2532.7085	(48)
2		4926.3000	87.2087	45.0227	4227.9057	(53)
3		3359.2200	91.8962	58.5444	3417.3981	(42)

MUSCLE VS ANIMAL (9). 30/03/77 PAGE

FILE NONAME (CREATION DATE = 30/03/77)

* * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * *

MEASURE
BY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	2831604.179	8	353950.522	32.105	.001
MUSCLE	2734886.848	6	456347.808	41.393	.001
ANIMAL	152713.101	2	76356.551	6.926	.001
2-WAY INTERACTIONS	95359.430	12	7946.619	.721	.729
MUSCLE ANIMAL	95359.430	12	7946.619	.721	.729
EXPLAINED	2926963.608	20	146348.180	13.275	.001
RESIDUAL	1433216.447	130	11024.742		
TOTAL	4360180.055	150	29067.867		

168 CASES WERE PROCESSED.
17 CASES (10.1 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		57555.5000	381.1623	170.4930	29067.8670	(151)
1		12063.2100	502.6333	148.7502	22126.6223	(24)
2		12226.5000	531.3261	124.1666	15417.3029	(23)
3		9542.9000	452.5190	110.7541	12973.1946	(21)
4		9799.1000	426.0478	118.8168	14117.0329	(23)
5		2555.8000	127.7900	39.3620	1549.3621	(20)
6		5861.4000	279.1143	88.9210	7906.9453	(21)
7		5552.6000	292.2421	86.1267	8420.6059	(19)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		57555.5000	381.1623	170.4930	29067.8670	(151)
1		18509.8000	362.9373	154.2590	24016.8816	(51)
2		18219.8000	364.3920	154.8302	24533.0234	(50)
3		20826.1000	416.5220	193.8756	37624.5005	(50)

LDH$_3$

MUSCLE VS ANIMAL (10). 30/03/77 PAGE

FILE NONAME (CREATION DATE = 30/03/77)

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * * * *

MESURE
BY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	1571238.100	8	196404.763	20.897	.001
MUSCLE	1493238.227	6	248873.038	26.479	.001
ANIMAL	132598.150	2	66299.075	7.054	.001
2-WAY INTERACTIONS	66097.129	12	5508.094	.586	.850
MUSCLE ANIMAL	66097.129	12	5508.094	.586	.850
EXPLAINED	1637335.229	20	81866.761	8.710	.001
RESIDUAL	1221847.537	130	9398.827		
TOTAL	2859182.766	150	19061.218		

168 CASES WERE PROCESSED.
17 CASES (10.1 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		51095.3000	338.3795	138.0624	19061.2184	(151)
1		9896.2000	412.3417	130.3306	16986.0756	(24)
2		10325.9000	448.9522	110.5116	12212.8035	(23)
3		8077.3000	384.6333	90.9370	8269.5013	(21)
[illegible]		8427.6000	366.4174	97.4052	9487.7035	(23)
[illegible]		2740.2000	137.0100	41.8593	1752.2009	(20)
[illegible]		6886.2000	327.9143	108.5869	11791.1123	(21)
7		4741.9000	249.5737	80.9478	6552.5398	(1[illegible])

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		51095.3000	338.3795	138.0624	19061.2184	(151)
1		16718.1000	327.8059	127.3941	16229.2550	(51)
2		15871.5000	317.4300	131.0128	17164.3479	(50)
3		18505.7000	370.1140	151.7696	23034.0225	(50)

MUSCLE VS ANIMAL (11). 30/03/77 PAGE

FILE NONAME (CREATION DATE = 30/03/77)

* * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

MESURE BY MUSCLE ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	2.711	8	.339	34.009	.001
MUSCLE	2.640	6	.440	44.728	.001
ANIMAL	.041	2	.021	2.105	.126
2-WAY INTERACTIONS	.070	12	.006	.591	.846
MUSCLE ANIMAL	.070	12	.006	.591	.846
EXPLAINED	2.781	20	.139	14.154	.001
RESIDUAL	1.279	130	.010		
TOTAL	4.059	150	.027		

168 CASES WERE PROCESSED.
17 CASES (10.1 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		168.1200	1.1134	.1645	.0271	(151)
1		29.5000	1.2292	.1011	.0102	(24)
2		27.4500	1.1935	.0968	.0093	(23)
3		26.8000	1.1818	.1102	.0135	(21)
4		26.8000	1.1652	.0988	.0098	(23)
5		18.8400	.9420	.1153	.0129	(20)
6		18.0800	.8810	.0731	.0049	(21)
7		22.5900	1.1809	.0827	.0068	(19)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		168.1200	1.1134	.1645	.0271	(151)
1		55.9600	1.0973	.1690	.0286	(51)
2		57.2100	1.1442	.1629	.0265	(50)
3		54.9500	1.0990	.1602	.0257	(50)

MUSCLE VS ANIMAL (13). 30/03/77 PAGE

FILE NONAME (CREATION DATE = 30/03/77)

* * * * * * * * * * * A N A L Y S I S   O F   V A R I A N C E * * * * * * * * * * *

MESURE
BY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	4006725.374	8	500840.670	43.090	.001
MUSCLE	3940315.4[illegible]	6	656719.2[illegible]	56.50[illegible]	.001
ANIMAL	77409.169	2	38704.583	3.330	.039
2-WAY INTERACTIONS	111937.884	12	9328.157	.803	.647
MUSCLE ANIMAL	111937.884	12	9328.157	.803	.647
EXPLAINED	4118[illegible]3.278	20	205933.164	17.719	.001
RESIDUAL	1610866.055	130	11622.047		
TOTAL	5629529.333	150	37530.196		

168 CASES WERE PROCESSED.
17 CASES (10.1 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		43404.1000	287.4444	193.7271	37530.1956	(151)
1		10797.4000	449.8917	144.4295	20859.8756	(24)
2		10311.8000	448.3391	126.1764	15920.4730	(23)
3		7919.3000	377.1095	128.7379	16573.4359	(21)
4		7954.6000	345.8522	130.8866	17131.6335	(23)
5		922.2000	46.1100	47.7603	2281.0483	(20)
6		917.7000	43.7000	56.9702	3245.6010	(21)
7		4581.1000	241.1105	54.5599	2976.7865	(19)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		43404.1000	287.4444	193.7271	37530.1956	(151)
1		13372.7000	262.2098	187.9439	35322.9133	(51)
2		14356.7000	287.1340	172.9537	29912.9651	(50)
3		15674.7000	313.4940	218.1199	47576.3949	(50)

MUSCLE VS ANIMAL (T4). 30/03/77 PAGE

FILE NONAME (CREATION DATE = 30/03/77)

* * * * * * * * * ANALYSIS OF VARIANCE * * * * * * * * *

MESURE BY MUSCLE ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	830116.723	8	103764.590	13.195	.001
MUSCLE	796452.352	6	132742.059	16.880	.001
ANIMAL	37574.061	2	18784.031	2.389	.096
2-WAY INTERACTIONS	44247.114	12	3687.260	.469	.930
MUSCLE ANIMAL	44247.114	12	3687.260	.469	.930
EXPLAINED	874363.838	20	43718.192	5.559	.001
RESIDUAL	1022311.292	130	7863.933		
TOTAL	1896675.130	150	12644.501		

168 CASES WERE PROCESSED.
17 CASES (10.1 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		17495.7800	115.8661	112.4478	12644.5009	(151)
1		1582.5000	65.9375	94.0879	8852.5294	(24)
2		2385.7500	103.7278	92.1311	8488.1398	(23)
3		1979.4000	90.2571	99.9574	9991.4626	(21)
4		2255.5000	98.0652	77.9797	6080.8360	(23)
5		1998.8000	99.9400	50.3766	2538.0095	(20)
6		6129.4000	291.8762	114.6747	13151.2009	(21)
7		1164.6500	61.2974	61.8869	3829.7480	(19)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		17495.7800	115.8661	112.4478	12644.5009	(151)
1		6371.0800	124.9231	123.2517	15190.7473	(51)
2		4735.5000	94.7100	104.3089	10879.9393	(50)
3		6389.2000	127.7840	102.8485	11630.9259	(50)

TOTAL LDH

MUSCLE VS ANIMAL (12).

FILE NONAME (CREATION DATE = 30/03/77) 30/03/77 PAGE

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

MEASURE
BY MUSCLE
ANIMAL

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	2643681.550	8	330460.194	26.505	.001
MUSCLE	2537002.775	6	422833.796	33.915	.001
ANIMAL	175463.319	2	87741.659	7.038	.001
2-WAY INTERACTIONS	112395.751	12	9366.313	.751	.699
MUSCLE ANIMAL	112395.751	12	9366.313	.751	.699
EXPLAINED	2756077.301	20	137803.865	11.053	.001
RESIDUAL	1620789.792	130	12467.614		
TOTAL	4376867.093	150	29179.114		

168 CASES WERE PROCESSED.
17 CASES (10.1 PCT) WERE MISSING.

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		60665.1000	401.7556	170.8190	29179.1140	(151)
1		12331.7000	513.8208	156.7451	24569.0450	(24)
2		12447.6000	541.2000	127.2136	16183.3036	(23)
3		9898.8000	471.3714	107.5615	11560.4601	(21)
4		10122.4000	440.1043	124.4596	15490.2350	(23)
5		2871.7000	143.5850	41.1997	1697.4161	(20)
6		7197.3000	342.7286	109.1406	11911.6671	(21)
7		5795.6000	305.0316	89.7144	8049.3956	(19)

CODE	VALUE LABEL	SUM	MEAN	STD DEV	VARIANCE	N
		60665.1000	401.7556	170.8190	29179.1140	(151)
1		19497.3000	382.3000	150.4657	22645.3496	(51)
2		19191.7000	383.8340	159.9682	25589.8211	(50)
3		21976.1000	439.5220	190.0851	36449.1797	(50)

APPENDIX G

CHEMICALS USED IN THE STUDY

CHEMICALS	SOURCE	ASSAY(S)
Acrylamide	Fisher, 5521	LDH electrophoresis
ADP, trisodium salt	Sigma A-0127	AK & PC
Adenylate Kinase, grade III, from rabbit muscle in sulfate suspension	Sigma M-3003	AK as a Std
ATP, crystalline disodium salt	Sigma A-3127	ATP as a Std
ATP Assay Kit, Calbiochem ATP - Stat Pack	Calbiochem 869206	ATP
Ammonium persulfate	Fisher A-682	LDH electrophoresis
Bromophenol blue	Fisher B-392	LDH electrophoresis
Creatine Phosphokinase, type 1, from rabbit muscle, lyophilized salt-free powder	Sigma C-3755	PC
CPK Control (Dade)	Canlab B-5126	CPK as a Std
CPK Assays Kit, Dade UV-1-CPK or UV-10-CPK	Canlab B-5329	CPK
Dextrose	Canlab 1916	AK
G-6-P de H, type XV-Baker yeast, crystallized and lyophilized sulfate free	Sigma G-6378	AK
Glycine	Fisher G-46	LDH electrophoresis
Hexokinase, type F-300 sulfate free	Sigma H-4502	AK
Hydrochloric acid	Fisher A-144	LDH electrophoresis
Lactate, Lithium Salt	Sigma L-2250	LDH electrophoresis staining solution
Lactate Dehydrogenase, type II, crystalline from rabbit muscle, ammonium sulfate suspension	Sigma L-2500	LDH and LDH electrophoresis as a Std

CHEMICALS	SOURCE	ASSAY(S)
LDH, type III, from beef heart, ammonium sulfate suspension	Sigma L-2625	LDH and LDH electrophoresis as a Std
LDH, type V, LDH-5 (M_4), isoenzyme, crystalline, rabbit muscle, ammonium sulfate suspension	Sigma L-2875	"
LDH, type VII, LDH-1 (H_4), isoenzyme, crystalline, pig heart, ammonium sulfate suspension	Sigma L-3125	"
Magnesium chloride	Anachemia AC-5538	Ak LDH electrophoresis staining solution
N, N'-Methylenebisacrylamide	Fisher 8383	LDH electrophoresis
Methyl Orange, sodium salt	Canlab 2694 (Baker)	ATP & PC (neutralization)
Nembutal, 50mg/ml	Abbott 3778	Anaesthesia
NAD, free acid	Sigma N-7004	LDH electrophoresis staining solution
NADH, disodium salt, grade III	Sigma N-8129	LDH
NADP, monosodium salt	Sigma N-0505	AK
Nitroblue Tetrazolium	Sigma N-6876	LDH electrophoresis staining solution
Nitrogen (liquid)	University of Montreal (Liquid Air)	ATP & PC (deproteinization)
Perchloric acid, 70% W/V	Canlab 1-9652	ATP & PC (deproteinization)
Phenazine methosulfate	Sigma P-9265	LDH electrophoresis staining solution
Phosphorylcreatine, disodium salt	Sigma P-6502	PC as a Std

CHEMICALS	SOURCE	ASSAY(S)
Potassium acid phosphate	Fisher P-285	LDH buffer, LDH electrophoresis staining solution buffer
Potassium carbonate	University of Montreal 4-5469 (BDH-29591)	ATP & PC (neutralization)
Potassium dichromate	Fisher P-188	AK & CPK (blank)
Pyruvate, sodium salt	Sigma P-2256	LDH
Sodium chloride	Canlab 1-3624 (BDH-10241)	LDH electrophoresis staining solution
Sodium phosphate (dibasic)	Fisher 9-374	LDH buffer LDH electrophoresis staining solution buffer
Sucrose	Canlab 4072	AK, CPK, LDH, LDH electrophoresis (homogenization)
N, N, N', N' - tetramethylethylene diamine	Sigma T-8183	LDH electrophoresis
TRIS (TRIZMA)	Sigma T 1503 (Base) T 3253 (HCL)	AK. LDH electrophoresis