ALTERATIONS IN THE POST EXERCISE PLASMA LACTATE RESPONSE FOLLOWING SWIM TRAINING

by

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ABSTRACT

Ten collegiate varsity swimmers were studied in an attempt to determine changes in plasma lactate values for standardized swimming velocities following swim training. The swimmers trained six days per week for a total of ten weeks. Each subject performed three submaximal and one maximal swim test during weeks one (W1), two (W2), six (W6), and ten (W10) of the training program. The submaximal swim tests consisted of 500-yard front crawl swims at intensities corresponding to approximately 80, 86, and 92% of the subject's best time recorded for that distance within the previous 12 months. The maximal swim test consisted of an all-out 200-yard front crawl swim. Peak plasma lactate concentrations were determined following each swim test. Multiple regression analyses were performed using W1-2, W6, and W10 lactates as the dependent variables and the swim velocity for each of these weeks as the independent variables. The plasma lactate values following the 80 and 86% swim tests decreased significantly ($p < 0.05$) by W10 and W6, respectively. No change was observed in the lactate response to the maximal swim tests after training. Evaluation of the mean regression analyses indicated no difference in the slopes, while the intercepts of W6 and W10 were significantly altered. This shift in the lactate response curve was further substantiated after comparing the mean

velocities, corresponding to a delta lactate of 100 mg/100 ml, for W1 and W10. It was therefore concluded that an intense training program of greater than six weeks is necessary for any significant alterations to occur in the lactate/swimming velocity curve in elite swimmers.

CHAPTER I

THE PROBLEM

Each year an increasing number of young people are joining the sport of competitive swimming. There are currently over 150,000 registered amateur competitive swimmers in the United States (Essick, 1980). Swim training for competition requires a tremendous amount of time and dedication. Many of these competitive swimmers train six hours or more per day. With such heavy training schedules comes an increased risk that these athletes will become overtrained, possibly even to the point of being detrimental to their health and performance. Thus, there is a distinct need to determine a technique of optimizing swim training to avoid overtraining, to produce better competitive swimmers, and, in addition, to allow the development of a sound exercise prescription for the adult population.

Seventy-five percent of all swimming races for both men and women are completed in less than two minutes, and of these, 50% are completed in less than 60 seconds. Åstrand and Rodahl (1977) state that 50% of the energy supplied to support the work of maximum effort lasting for two minutes is from anaerobic metabolism. The percentage contribution of anaerobic metabolism increased to 65% and greater in work completed in 60 seconds or less. These facts lead to the conclusion that most swimming races are performed under predominantly anaerobic conditions.

Lactic acid, a product of anaerobic metabolism, is frequently used as an index of the extent of involvement of the anaerobic pathways. A number of studies have shown that once a certain level of exercise has been reached, i.e. ∿50-70% of capacity, plasma lactate increases linearly above resting levels with further increases in exercise intensity (Åstrand and Rodahl, 1977; and Skinner and McLellan, 1980). Following endurance training, the plasma lactate response to exercise is altered. Saltin and Karlsson (1971) have shown that plasma lactate accumulation is less pronounced at standardized submaximal workloads following training, i.e. for the same level of work the plasma lactate level is reduced. The linearity of the relationship between plasma lactate level and exercise intensity, coupled with the observed reduction in plasma lactate response to standardized submaximal levels of work following training, should provide the basis for the development of a model to predict performance potential and its alteration consequent to training, overtraining, and the cessation of training.

The maximal plasma lactate response to exercise has also been shown to be altered by training. Ekblom (1969) has shown that at maximal work loads the plasma lactate concentration was significantly higher after training. In addition, the highest plasma lactate values reported thus far are from well-conditioned athletes at the end of competitive events of one to two-minute duration (Åstrand and Rodahl, 1977). It would, therefore, be expected that well-conditioned athletes would produce higher concentrations of plasma lactate following a

maximal workout than non-athletes, or athletes who were not as well conditioned.

The relationship between plasma lactate levels and the overtrained syndrome has never been investigated. Figure 1 illustrates the theoretical linear increase in plasma lactate with increases in swimming velocity, and the shift towards the right in the concentration of plasma lactate following training as illustrated by weeks 5 and 15. It is hypothesized that there is no change in the slope of the relationship following training. In addition, it is hypothesized that overtraining causes the plasma lactate curve to shift back towards the untrained state, as illustrated by week 10 in Figure 1. If this backward shift in the plasma lactate curve were to occur, the results would be detrimental to the trained athlete's swimming performance.

Statement of Problem

The purpose of the present study was to determine the changes in plasma lactate values for standardized swimming velocities following swim training in male swimmers, and to determine if these changes could be used as a biochemical marker of overtraining.

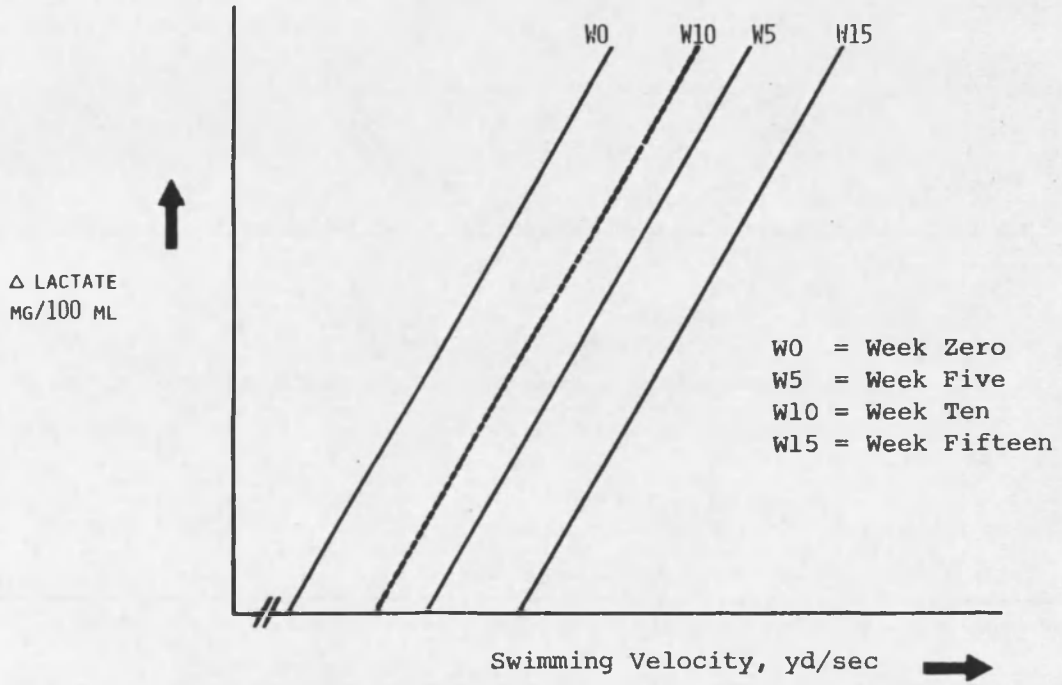


Figure 1. Theoretical Model of the Plasma Lactate Response to Swim Training

CHAPTER II

REVIEW OF RELATED LITERATURE

Over the years the desire to improve competitive performance has manifested itself in new and improved methods for optimizing training. Developments in swim training over the past 15 years have placed increased demands on the athlete in terms of intensity, duration, and frequency of training. These added demands have been associated with the overtrained syndrome recognized today in many competitive swimmers. When considering that 75% of all swimming races are completed in less than two minutes, and, of these, 50% are completed in less than 60 seconds, it is inconceivable why competitive swimmers would need to spend up to six hours per day training, swimming up to 12 miles per day.

Energy Yielding Processes

Man's ability to do physical work is powered by the breakdown of adenosine triphosphate (ATP). While the concentrations of ATP present in resting muscle are relatively low, i.e. 4-6 m moles x kg^{-1} wet weight (Karlsson, Diamant, and Saltin, 1971), these concentrations do not fall below 60% of the resting values no matter how intense or prolonged the exercise (Sahlin, Harris, and Hultman, 1975; and Hultman, Karlsson, Diamant, and Saltin, 1967). It is, therefore, obvious that the levels of ATP are rapidly replenished even within a few seconds

following the completion of work. There are essentially three pathways through which ATP levels may be replenished. The first is via the oxidative pathway, i.e. aerobic metabolism. Aerobic metabolism involves oxygen utilization in the mitochondria. The fuels that are oxidized during aerobic metabolism include carbohydrate-derived pyruvate, fatty acids, and ketone bodies. The remaining two pathways for the replenishment of ATP can function for short periods of time without oxygen, i.e. glycolysis and high energy phosphogens. The processes of the anaerobic system operate essentially in the sarcoplasm of the muscle cell. For the remaining discussion only anaerobic metabolism will be reviewed. For the purposes of this discussion anaerobic metabolism will be defined as that energy delivering process which is not dependent upon the immediate consumption of oxygen.

Anaerobic Metabolism

ATP and creatine phosphate (CP) are supplied anaerobically to working muscle by the degradation of glycogen to lactate through glycolysis. Lactate is a small, diffusible molecule that may be located in any of the body's water compartments except the cerebrospinal fluid (Hermansen, 1971). The concentration present in the water compartments is related not only to the rate and amount of lactate produced in working muscle, but also to the amount of lactate that diffuses from the muscle and the amount of lactate that is utilized by various organs. Lactate concentrations during exercise in both the muscle and the blood are regarded as an index of the energy produced anaerobically (Gollnick and Hermansen, 1973).

There are important considerations that must be kept in mind when interpreting plasma lactate concentrations. First, plasma lactate concentrations are an indication of the lactate production in the muscle and the diffusion of lactate from the muscle to various water compartments. Diamant, Karlsson, and Saltin (1968) demonstrated that following exercise there is a rapid ($\sim$3-5 minute) distribution of lactate from the working muscle to all water compartments. They noted, however, that immediately following maximal exercise of about three minutes in duration, the lactate concentration in the skeletal muscle was much higher than that of the plasma. At five minutes following the cessation of exercise, there was a peak in plasma lactate concentration which was shown to be in equilibrium with the lactate concentration in the muscle at that time. Therefore, peak plasma lactate concentrations can be used to indicate the extent of lactate production in the working muscle.

Margaria, et al. (1963), has stated that there is no lactate produced during any submaximal work once a steady state oxygen consumption, below 50% of maximum capacity, is reached. These investigators explained any increase in lactate produced above resting levels as limited to the initial phase of the exercise, i.e. the period of oxygen deficit. In relation to work load, Hermansen (1971) has illustrated that the amount of plasma lactate remains unchanged until the work load represents approximately 50 to 60% of the individual's maximal oxygen consumption. Following this critical work intensity,

Hermansen (1971) found that plasma lactate values rose exponentially as work loads approached 100% of the maximal oxygen consumption.

The fate of lactate must also be considered when interpreting plasma lactate concentrations. To begin with, lactate is metabolized by various tissues which would reduce plasma lactate concentrations. Rowell, et al. (1966), demonstrated that lactate is primarily metabolized by the liver, while Jorfeldt (1970) demonstrated that lactate is also metabolized by skeletal muscle. Secondly, there have been several studies that have demonstrated that there is an ongoing turnover of lactate between skeletal muscle and the blood, and that during exercise this rate of turnover increases (Stainsby and Welch, 1966; Freyschuss and Strandell, 1967; Issekutz, Shaw, and Issekutz, 1975; and Kreisberg, 1980). Therefore, it can be concluded that the appearance of lactate in the plasma is not an indication that the muscle is at the onset of anaerobiosis since it appears that the muscle is constantly deriving part of its energy requirement from anaerobic pathways, even at rest. A more accurate interpretation of an increase in lactate is that the concentration of lactate in the muscle has become large enough to overcome the concentration gradient that exists between skeletal muscle and blood. Diamant et al. (1968), have demonstrated that there is a concentration gradient between skeletal muscle and blood at rest and the gradient increases with exercise. This simply means that the concentration of lactate in the muscle must be large enough before an appreciable amount of lactate will diffuse into the plasma.

Ekblom (1969) demonstrated that the concentration of plasma lactate during exercise at a constant submaximal work load decreases following physical training. This decrease following training is felt to be the result of an increased oxygen transport, i.e. increased $\dot{V}O_2$ max. However, Hermansen (1971) demonstrated a reduction in plasma lactate following training even when the work loads were expressed relative to the individual's $\dot{V}O_2$ max. It can therefore be concluded that physical training not only elicits changes in the oxygen transport system, but also changes within the muscle cell.

It has been demonstrated for many years that the highest lactate values come from well-conditioned athletes (Robinson and Harmon, 1941; and Åstrand and Rodahl, 1977). Ekblom (1969) also demonstrated that maximal plasma lactate concentrations increase significantly with training. The attainment of higher levels of lactate in trained athletes is the result of several factors. To begin with, the well-trained athlete is probably able to push himself to a higher maximal effort than the untrained individual. This ability is most likely related to the athlete's competitive drive. The second reason for the higher lactate values has to do with changes in the ability of the muscle to regulate the process of anaerobic metabolism. Most likely there are changes in the amount and effectiveness of the glycolytic enzymes, such as phosphorylase, hexokinase, and phosphofructokinase. Changes in these enzymes may produce a damping effect on the processes of anaerobic metabolism, therefore, the anaerobic pathways would be regulated in a manner different from that in untrained individuals.

The relative importance of the aerobic and anaerobic metabolic reactions for total energy yield during maximal work is fairly well understood (Hermansen and Karlsson, 1967; and Hermansen, 1969). Recently, Houston (1978) devised estimates of the total energy cost and the proportional contribution of aerobic and anaerobic metabolism during front crawl swimming at world record speeds. The estimates (Table 1) were based upon a male swimmer having a $\dot{V}O_2$ max of 5.0 liters per minute and a maximal oxygen debt of 12.0 liters. These estimates illustrate the heavy reliance upon anaerobic metabolism for races of 200 meters or less (75% of all swimming races). Based upon this fact it is easy to see why such great emphasis should be placed on training the anaerobic component of metabolism.

Overtraining

To date, there has been little or no research performed to determine the potential detrimental effects of an excessive volume of continuous, high intensity training, i.e. overtraining, upon the athlete's health or athletic performance. The biochemical variables associated with overtraining remain speculative at best. In fact, the entire concept of overtraining is one that is in need of precise scientific definition.

It may be possible to use various enzymes and blood borne metabolites as biochemical markers in an attempt to better define overtraining. Many of these variables have been studied extensively but never throughout an intense training period with highly trained athletes, nor with the sole purpose of quantifying the overtraining syndrome.

Table 1. Relative contribution of anaerobic and aerobic metabolism to total energy requirements during front crawl swimming at world record speeds* for men (Houston, 1978).

Front Crawl Distance (m)	Swimming Speed (percent of 100-m speed)	Total Energy Requirements Liters of O_2, (liters)	Energy Output (Liters of O_2)		Relative Contribution (percent)	
			anaerobic metabolism	aerobic metabolism	anaerobic metabolism	aerobic metabolism
100	100.0	15.0	12.0	3.0	80	20
200	90.7	20.0	12.0	8.0	60	40
400	86.2	30.0	12.0	18.0	40	60
800	83.1	47.0	8.0	39.0	17	83
1500	83.1	82.0	8.0	74.0	10	90

*World record speeds as of July, 1976.

The concentrations of various intramuscular enzymes, i.e. creatine phosphokinase (CPK), lactate dehydrogenase (LDH), and the presence of myoglobin in the blood or urine, have been used to determine damage to working muscle or alterations in the permeability of the muscular membrane, and are possibly related to overtraining. CPK may be the most sensitive to exercise of the serum muscle enzymes (Bunch, 1980). Block, et al. (1971), Magazanik, et al. (1974), Sanders and Bloor (1975), and Bunch (1980) have demonstrated that following a single bout of exhaustive exercise the plasma concentrations of CPK and LDH are elevated. Bunch (1980) reported that it appears that the degree of exhaustion and not the duration of exercise is responsible for the amount of increase in CPK levels. Serum concentrations of CPK and LDH have also been shown to remain elevated at rest in trained individuals (Hunter and Critz, 1971; and Martin, Haskell, and Wood, 1977). Martin, Haskell, and Wood (1977) have also reported that the better the distance trained runner, the higher the concentration of serum LDH at rest. They reported the concentration of serum LDH to be above normal limits for world class runners. Bank (1977) and Castenfors (1977) have reported the presence of elevated myoglobin in the blood and urine following exercise. The presence of myoglobin in the urine is cause for alarm as reabsorption of myoglobin is toxic to the convulated tubules and is a cause of kidney failure (Bank, 1977). It is unlikely, however, that the appearance of CPK, LDH, or myoglobin is the result of muscular damage, but is more likely the result of a change in the muscle membrane permeability.

Elevated concentrations of liver enzymes found in the plasma, i.e. serum glutamic oxalacetic transaminase (SGOT) and serum pyruvic glutamic transaminase (SPGT), and blood borne metabolites such as bilirubin, have been used to indicate liver damage or possible alterations in the permeability of the liver membrane. Block, et al. (1971), and Sanders and Bloor (1975) have exhibited elevated concentrations of SGOT and bilirubin in trained individuals both at rest and following exercise. It has been postulated that the increased concentration of SGOT is the result of an increase in liver permeability with exercise, and not tissue damage (Sanders and Bloor, 1975). The increased concentration of bilirubin in the trained runner is felt to be the result of increased erythrocyte destruction produced by the trauma of running (Block, et al., 1971).

Sulman, et al. (1970), described an exhaustive syndrome in persons chronically exposed to a hot and windy environment. The subjects demonstrated the classic characteristic of stress, i.e. irritability, fatigue, sleeplessness, apathy, and exhaustion. Many of these characteristics are similar to those exhibited by the highly trained athlete who may be suffering from overtraining fatigue. Sulman, et al., felt that these subjects suffered from either a decrease in or a lack of adrenal medullary hormones, epinephrine and norepinephrine, and an exhaustion of the adrenal cortical hormones, the adrenocorticosteroids and the glucocorticosteroids. Weller and Sulman (1969) reported similar changes in the activity of the adrenal hormones for long distance runners. They postulated that the exhaustive syndrome was a possible cause for the "staleness" often encountered in long distance runners.

It is obvious that the concept of overtraining is in need of a more precise definition and extensive research. It may prove possible to use the biochemical markers mentioned previously, or additional markers, e.g. lactate, to define overtraining. To be able to accurately identify overtraining would be of great benefit to endurance trained athletes.

CHAPTER III

EXPERIMENTAL DESIGN

Subject Characteristics

Ten members of the University of Arizona Men's Swimming Team volunteered to participate in this study. The study and its design were explained to each subject, a medical history form was completed, and informed consent was obtained (Appendix A). Table 2 summarizes the subjects' physical characteristics.

Body Composition

Body composition was determined by body density assessment using the hydrostatic weighing technique according to the procedures outlined by Behnke and Wilmore (1974). A Chatillion scale (9-kg capacity) was used to measure underwater weight to within ±0.01 kg. Ten trials were conducted per subject and the highest weight if it was reproduced three times was taken as the true weight in water. If the highest weight did not occur three times then the highest weight which occurred two times was taken as the true weight in water. Residual lung volume was determined by the nitrogen dilution technique (Wilmore, 1969). If two trials differed by more than ±0.1 L, a third trial was conducted and the two closest trials were averaged. The Siri (1956) equation was used to estimate relative fat from total body density.

Table 2. Characteristics of the Subjects

Subject	Age, yr.	Ht., cm.	Weight, kg pre	Weight, kg post	Skinfolds, mm pre	Skinfolds, mm post	Relative Body Fat, % pre	Relative Body Fat, % post
01	18	188	81.4	81.7	57.0	45.6	12.9	12.9
02	18	188	82.7	83.8	36.6	36.5	13.3	12.6
03	18	193	82.9	80.0	94.1	60.7	17.3	14.6
04	18	178	71.4	69.3	41.1	39.4	16.0	12.5
05	19	188	74.4	73.0	35.2	36.2	11.7	9.8
06	20	183	71.0	69.7	48.7	42.5	12.9	9.2
07	18	190	77.9	77.1	40.7	39.5	8.7	7.3
08	18	182	78.5	79.2	61.7	48.3	11.3	10.7
09	19	182	72.2	76.4	44.0	42.5	15.9	15.5
10	19	180	73.3	73.9	33.2	41.0	9.7	10.8
Mean	18.5	185.2	76.6	76.4	49.2	43.2	13.0	11.6
S. D.	0.7	4.8	4.7	4.9	18.3	7.2	2.8	2.5
Δ (pre-post)			0.2		6.0		1.4[1]	
%Δ [(pre-post)/pre]x100			0.3		12.2		10.8	

[1]significant at $p < 0.05$

Skinfold measurements were determined in triplicate, to assure reliability, at six sites: abdomen, chest, front thigh, subscapular, suprailiac, and triceps, as described by Behnke and Wilmore (1974). The mean of the closest two trials was used in subsequent analysis, and in addition, the skinfold measurements for each individual were totaled. Body composition and skinfold measurements were determined both prior to and upon completion of ten weeks of swim training.

Maximal and Submaximal Swim Testing

Each subject performed three submaximal swim tests and one maximal swim test during the first (W1), second (W2), sixth (W6), and the tenth week (W10) of an intense swim training program. Testing during the first and second week was conducted to determine the test reliability, and further analyses were conducted on the average of W1 and W2, i.e. W1-2. The submaximal swim test consisted of a 500-yard front crawl swim at intensities corresponding to 80, 86, and 92% of the subject's best time recorded for that distance within the previous 12 months. These submaximal intensities were chosen so as to exceed the subject's anaerobic threshold (Skinner and McLellan, 1980).

The swim tests were conducted on three separate days during each week of testing. The first day of testing consisted of two 500-yard submaximal swims at intensities corresponding to 80% and 92% of the subject's best time. There was a rest period of at least 30 minutes to allow lactate levels to return to resting values (Hermansen and Stensvold, 1972). The second day of testing consisted of the 500-yard submaximal swim at an intensity corresponding to 86% of the subject's

best time. On the final day of testing, the subjects performed a maximal swim test, swimming an all-out 200-yard swim. All subjects were allowed to warm up on their own for 15 minutes prior to all swim tests.

Upon reporting to the laboratory after a 2-3 hour fast, a venous blood sample (2.0 ml) was obtained without stasis from a prominent forearm vein and immediately pipetted into 4 ml cold 8% perchloric acid (.6N $HC1O_4$). The sample was then refrigerated ($\sim 5^{o}$ C) until the lactate analysis could be performed. All samples were analyzed within seven days after collection. Immediately following the submaximal swim tests corresponding to intensities of 80% and 86%, a second venous blood sample (2.0 ml) was drawn. A pilot study had been previously conducted and it was found that following submaximal swim tests at 80% and 86% intensities the concentration of plasma lactate was at peak values immediately following the swim. However, it was found that following a submaximal swim test at 92% intensity a delay of between 3 to 5 minutes was necessary for the concentration of plasma lactate to reach peak values. On the other hand, Diamant, Karlsson, and Saltin (1968) have shown that plasma lactate concentrations peak five minutes following maximal exercise. Therefore the venous blood sample (2.0 ml) following the submaximal swim test at an intensity corresponding to 92% of a subject's maximal effort was taken 3-5 minutes following completion of the swim and the venous blood sample (2.0 ml) following the all-out 200-yard swim was taken at least five minutes following completion of the swim.

Plasma Lactate Determinations

All plasma lactate values are expressed as the difference between the pre-exercise and the post-exercise values, in an attempt to better estimate the change in lactate which resulted from exercise, controlling for normal daily fluctuations in resting values. All lactate concentrations are expressed in mg/100 ml. Plasma lactates were determined in duplicate by a standard enzymatic method (Sigma Technical Bulletin No. 826-uv, 1977). The first 100 duplicate samples were highly correlated (r = .91). The amount of NADH found was determined at 340 nm on a Beckman Model 35 narrow band spectrophotometer. A standard calibration curve was established one week prior to each week of testing (Appendix D).

Swim Training Program

All subjects were members of the University of Arizona Men's Varsity Swimming Team and were required to attend all practices under the observation of the head coach. In addition to the swim workout, all subjects were required to attend a weight lifting workout. The weight training program utilized both free weights and Nautilus weight training equipment. The weight workouts were under the supervision of the assistant coach. The training regime is illustrated in Table 3.

Table 3. Training Regimen

Week Number	Swim Workout Per Week	Swim Workout Per Day	Yardage Swam Per Workout	Total Yardage Swam Per Day	Weight Workout Per Week
1	5	1	5,000	5,000	5
2	5	1	5,000	5,000	5
3	10	2	6,000	12,000	3
4	11	2	7,000	14,000	3
5	11	2	7,500	15,000	3
6	12	2	8,000	16,000	3
7	12	2	8,000	16,000	3
8	12	2	8,000	16,000	3
9	12	2	8,000	16,000	3
10	12	2	8,000	16,000	3

Statistical Analysis

Multiple regression analyses were done by computer using the Statistical Package for the Social Sciences (1975). The slopes and the intercepts from the multiple regression analyses were analyzed for significance by the Student t-test and a repeated measures ANOVA. Where indicated, Tukey post hoc tests were employed to determine the location of significance. A 0.05 level of significance was chosen for all analyses.

CHAPTER IV

RESULTS

The physical characteristics of the subjects prior to, and upon completion of this study are presented in Table 1. The changes in weight (WGT), total skinfold (SF), and in relative body fat (RBF) were each tested for statistical significance using a Student t-test, with significance established at $p < 0.05$ (Table 1).

The values (mean and standard deviations) for the submaximal and maximal swimming lactate responses for each of the four test weeks (W1, W2, W6, W10) and for the average of W1 and W2, i.e. W1-2, appear in Table 4. The submaximal lactate values for the first two weeks were correlated to establish test reliability. The correlations were $r = 0.55$, $r = 0.72$, $r = 0.88$, and $r = 0.48$ for the 80%, 86%, 92%, and 100% swimming velocities, respectively. Following the establishment of test reliability, the lactate values and swim velocities for the first two weeks were averaged (W1-2 La and W1-2 V, respectively).

A multiple regression analysis was performed using W1-2, W6, and W10 lactates as the dependent variable and swim velocity for each of these weeks as the independent variable. The plotted regression lines and their corresponding correlations are illustrated in Figure 2. Individual regression analyses appear in Appendix B.

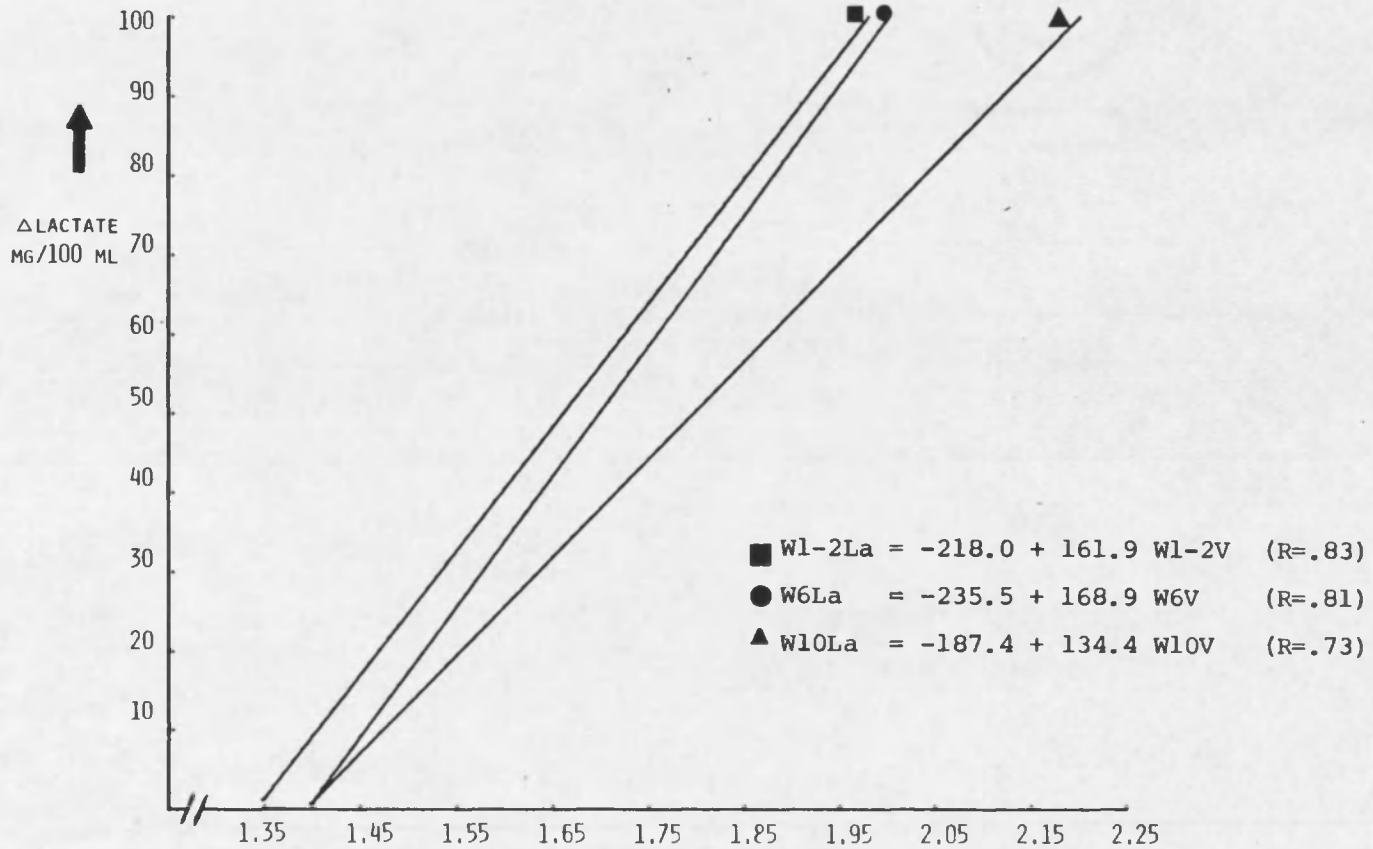


Figure 2. Mean Regression Analyses

There were no significant differences ($p < 0.05$) between the slopes for any of the weeks of testing. The only significant difference ($p < 0.05$) occurred between the intercepts for W6 and W10.

The velocity corresponding to a delta lactate value of 100 mg/100 ml was determined for each individual for each of the test weeks. The values (mean and standard deviation) are 1.87 ± 0.20, 2.01 ± 0.13, and 2.20 ± 0.34, for W1-2, W6, and W10, respectively.

In addition, a velocity was estimated for W10 by using the slope from the W1-2 mean regression analysis and the lactate value following the 86% swim test (2.10 ± 0.27). The two velocities determined for W10 were not significantly different ($p < 0.05$). However, both velocities were found to be significantly greater than the W1-2 velocity ($p < 0.05$).

Table 4. Values for Lactates.[1]

Percentage of Maximal Effort	Weeks of Testing		
	W1-2	W6	W10
80%	7.9 ± 4.5	3.9 ± 2.4	2.3 ± 3.8[2]
86%	28.3 ± 11.9	12.7 ± 6.6[3]	12.6 ± 8.7[2]
92%	50.1 ± 15.0	47 ± 21.8	31.8 ± 22.4
100%	74.5 ± 16.7	70.2 ± 13.5	70.6 ± 17.1

[1]All values expressed as mean ± S.D., in mg/100 ml.

[2]W1-2 significantly different from W10.

[3]W1-2 significantly different from W6.

CHAPTER V

DISCUSSION

The subjects in the present study represented nationally-ranked varsity swimmers. The submaximal plasma lactate concentrations obtained compared favorably with other submaximal values reported for swimming at similar relative velocities (Holmer and Åstrand, 1972; and Holmer, 1972). The peak plasma lactate concentrations, however, were lower than values reported following competitive swim races (Sawka et al., 1979) and following maximal effort noncompetitive swimming in a flume (Holmer, 1972; and Nadel et al., 1974) and free swimming (Sawka et al., 1979). Plasma lactate concentrations of 18 mM/L after competitive races (Sawka et al., 1979) and concentrations between 9-15 mM/L after maximal noncompetitive swimming (Holmer, 1972; Nadel et al., 1974; and Sawka et al., 1979) have been previously reported. The present peak plasma lactate values were 71.7 ± 2.4, or approximately 8 mM/L, and there were no significant differences ($p < 0.05$) between any of the weeks of testing (Table 4). It is important to note that the lactate concentrations following competition reported previously are significantly higher than that of the lactate concentrations obtained in the present study following noncompetitive all-out swimming. The present peak lactate concentrations are also lower than peak values reported for a variety of exercise modes (Hermansen, 1971; and Osnes, 1972). As a possible explanation for the

low peak lactate values obtained in this study, the subjects may not have been highly motivated and therefore did not give a maximal effort for the 200-yard swim. However, an attempt was made to have the subjects compete against one another to insure maximal efforts. In actual competitive races, emotions, anxiety, and other psychological factors influence performance which were impossible to duplicate in the testing situation. A second possible explanation for the low peak lactate values was that if the subjects did not perform maximal efforts for the 200-yard swims then the concentration of plasma lactate would have peaked prior to the drawing of the blood sample at five minutes post swimming. Diamant, Karlsson, and Saltin (1968) have shown that the plasma lactate concentrations peak and then decline five minutes following maximal exercise. Therefore, had the blood samples been taken earlier, approximately one to two minutes, then higher peak lactate values may have been obtained which would have compared more favorably with maximal values reported previously.

The major purpose of the present investigation was to determine the extent of change in the plasma lactate values for standardized swimming velocities following swim training in male swimmers, and to determine if these changes could be used in the development of a model to predict overtraining. It was not the intention of this study to quantify the overtraining syndrome, since there was no attempt to define overtraining either psychologically, e.g. irritability and sleeplessness, or physiologically, e.g. alterations in serum enzyme and hormone levels. It was, however, important to determine initial basic levels of

plasma lactate for standardized workloads of increasing intensity and to observe the subsequent change in plasma lactate concentrations for these standardized workloads as the result of swim training. The subjects in the present investigation had been doing no training for a minimum of one month and in most cases three months prior to this study; however, the subjects could not be classified as unfit as most subjects had been training for ten months a year for a number of years. It was felt, therefore, that if these subjects experienced significant alterations in the concentration of plasma lactate for standardized workloads as a result of training, then the plasma lactate/swimming intensity relationship could be used as a possible marker of training and overtraining, as illustrated previously in Figure 1.

The alteration in the relative body fat (RBF) subsequent to training, was similar to that reported by Pollock (1973). There was a significant ($p < 0.05$) decrease in mean RBF of 10.8% with only two subjects failing to demonstrate losses in RBF. Subject 01 demonstrated no change in RBF, while Subject 10 exhibited an 11.7% increase in RBF. A possible explanation for the increase in RBF for Subject 10 was his expressed desire to gain weight, as he considered himself too thin prior to training. However, Subject 10 demonstrated only a slight gain in weight (+ .6 kg) which can only partially explain his change in RBF. Possibly the low RBF of Subject 10 prior to training can explain the increase in RBF after training. Only Subject 07 had a lower RBF prior to training than did Subject 10, and following training, the RBF for Subject 10 was still lower than 50% of the subjects.

The submaximal plasma lactate values decreased throughout the ten weeks of training (Table 4). The lactate values following the 80% and 86% swim tests decreased significantly ($p < 0.05$) by W10 and W6, respectively. The values following the 92% swim test decreased, yet the decreases were not statistically significant ($p < 0.05$) between any of the weeks of testing. This may be due to the relatively high work load represented by the 92% swim test and to the high standard deviation. The decreases in the submaximal plasma lactate concentrations following training were similar to those reported by Ekblom (1969) and Hermansen (1971). Ekblom (1969) demonstrated that the concentration of plasma lactate at a given absolute submaximal work load decreases with training. Hermansen (1971) demonstrated the same reduction in submaximal plasma lactate concentrations following training even when the work loads were expressed as a percentage of the subject's $\dot{V}O_2$ max, i.e. the same relative work load. Hermansen concluded that training caused alterations within the muscle itself in addition to those changes elicited in the oxygen transport system, i.e. increased $\dot{V}O_2$ max. The subjects in the present study probably did not demonstrate a great increase in the capacity of the oxygen transport system ($\dot{V}O_2$ max) due to their relatively high initial level of fitness. Therefore, the reduction in submaximal plasma lactate concentrations in these subjects was probably a reflection of alterations within the working muscle which might include changes in the amount and effectiveness of the glycolytic regulatory enzymes, such as phosphorylase, hexokinase, and phosphofructokinase.

The decrease in submaximal plasma lactate concentrations at the same absolute swimming velocities in the present study indicate that training caused the plasma lactate/swimming velocity curve to shift towards the right. To determine when during the training program the shift in the lactate curve occurred the intercepts for the mean regression analyses of the lactate/swimming velocity relationship were tested for significant differences ($p < 0.05$). The only significant differences occurred between W6 and W10, with the intercept at W10 shifting to the right, indicating that training shifted the entire curve towards the right after six weeks. To further substantiate when the shift in the lactate curve occurred, a velocity corresponding to a delta lactate of 100 mg/100 ml was determined for each individual for each test week. Only the W10 velocity was found to be significantly greater than the W1-2 velocity ($p < 0.05$). There was no significant difference ($p < 0.05$) in the velocities of W1-2 to W6 or of W6 to W10. This would imply that an intense training program (see Table 3) of greater than four to six weeks is necessary for any significant alterations to occur in the plasma lactate/swimming velocity relationship.

Ekblom (1969) has demonstrated that maximal plasma lactate concentrations increase significantly with training. In the present study there was no significant change in the peak lactate concentrations at any of the weeks of testing (Table 4). In fact, as discussed above, the peak lactate values are lower than reported previously for maximal swimming efforts. A possible reason that the peak values failed to increase with training was the initial high level of fitness of the

present subjects. Other possible reasons for the low peak lactate and the failure of these peak plasma lactates to increase following training have been postulated previously, e.g. low subject motivation or failure to draw blood samples at times corresponding to peak lactate concentrations.

The subjects in the present study demonstrated a linear increase in plasma lactate concentration with increases in intensity, i.e. swim velocity (see Figure 2). Others (Karlsson and Saltin, 1970; Hermansen and Stensvold, 1972; and Senay and Kok, 1977) have demonstrated an exponential increase in plasma lactate concentrations for treadmill running and bicycling above 50-60% of a subject's $\dot{V}O_2$ max, i.e. anaerobic threshold. To date there has been little research performed describing the response of plasma lactate to increasing intensity for swimming. The submaximal work loads in the present study were designed to be significantly above anaerobic threshold (Skinner and McLellan, 1980). It was felt that the high submaximal work loads (80, 86, and 92% of maximal effort) in the present study accounted for the linearity of the plasma lactate concentration/swimming intensity relationship.

From an applied standpoint, the question has often arisen concerning the validity of using a single plasma lactate point to predict the changes in the lactate curves following training. It was felt that if the slope of this linear relationship did not change when the lactate curve shifted to the right with training, then a single plasma lactate point could be an accurate predictor to determine the changes in the plasma lactate/swimming velocity relationship. There were no

significant differences ($p < 0.05$) between the slopes of the mean regression analyses for any of the weeks of testing.

In an attempt to determine the accuracy of a single plasma lactate point as a predictor of the changes in the plasma lactate/swimming velocity relationship, the velocity corresponding to 100 mg/100 ml was used for W1-2, W10, and estimated for W10 using the slope of W1-2 and a single lactate value following the 86% swim test at W10. The values (mean and standard deviation) were 1.87 ± 0.20, 2.20 ± 0.34, and 2.10 ± 0.27 yd/sec for W1-2, W10, and W10 determined with a single lactate point, respectively. The two velocities for W10, i.e. actual and predicted from one lactate point were not significantly different ($p < 0.05$) and were highly correlated ($r = 0.91$). However, both actual and predicted velocities for W10 were significantly greater ($p < 0.05$) than the velocity for W1-2. Therefore, it would appear valid to use a single plasma lactate point to determine the subsequent changes in the plasma lactate/swimming intensity relationship. It would be necessary to develop a regression analysis, using at least three lactate points, for each individual at the onset of training. The slope of the initial regression analyses could be used with a single plasma lactate point at a later date to determine the changes in the plasma lactate/swimming velocity relationship. The ability to use a single lactate point to determine the changes in the lactate curve could be of practical use to the swim coach who is concerned with the evaluation of training, detraining, and possible overtraining.

It seems reasonable to assume that if training causes the plasma lactate curve to shift towards the right then detraining would cause the curve to shift back to the left. It would also seem reasonable to postulate that the plasma lactate/swimming intensity curve would provide a physiological index to determine the presence of the overtrained state, i.e. overtraining would also result in a shift in the curve back towards the left (see Figure 1).

It is not possible to use the individual data from the present study to define overtraining. Any abrupt change in the individual regression analyses (see Appendix C) are likely the result of illness or errors in methodology. Subject 10, however, deserves further comment. The individual regression analysis for Subject 10 (Appendix C) illustrates a shift in the plasma lactate/velocity curve towards the left at week six. While it is impossible at this time to determine if the shift towards the left is the result of overtraining, it does seem reasonable to assume that it may have been the result of overtraining and not the result of detraining since intense training procedures were being followed at that time. The submaximal plasma lactate values at W6 are higher than the submaximal plasma lactate values during the first two weeks of training, which is contradictory to the results of others (Ekblom, 1969; Hermansen, 1971; and Saltin and Karlsson, 1971).

CHAPTER VI

SUMMARY

Ten collegiate varsity swimmers were studied in an attempt to determine changes in plasma lactate values for standardized swimming velocities following swim training, and to determine if these changes could be used in the development of a model to identify overtraining. It was not the intention of this study to quantify the overtraining syndrome since there was no attempt to define overtraining either psychologically or physiologically.

The swimmers trained six days/week for a total of ten weeks. All subjects trained 5,000 yards/day for weeks one (W1) and two (W2), then increased training to 12,000, 14,000, 15,000, and 16,000 yards/day for weeks three (W3), four (W4), five (W5), and six (W6), respectively. All subjects trained 16,000 yards/day from W6 until week ten (W10). In addition to the swim workout, all subjects were required to attempt three weight lifting workouts/week except for W1 and W2 when five weight lifting workouts/week were required.

Each subject performed three submaximal and one maximal swim test during W1, W2, W6, and W10 of the training program. The submaximal swim tests consisted of 500-yard front crawl swims at intensities corresponding to 80, 86, and 92% of the subject's best time recorded for that distance within the previous 12 months. The maximal swim test

consisted of an all-out 200-yard front crawl swim. Peak plasma lactate concentrations were determined following each swim test. All plasma lactate values were expressed as the difference between the pre-exercise and the post-exercise values, in an attempt to better estimate the change in lactate which resulted from exercise, controlling for daily fluctuations in resting values. Multiple regression analyses were performed using W1-2, W6, and W10 lactates as the dependent variables and the swim velocity for each of these weeks as the independent variables.

The plasma lactate values for the submaximal swims decreased throughout the ten weeks of training. The plasma lactate values following the 80 and 86% swim tests decreased significantly ($p < 0.05$) by W10 and W6, respectively. The values following the 92% swim test decreased throughout the ten weeks of training, however, the magnitude of decrease was not statistically significant ($p < 0.05$) between any of the test weeks. It was postulated that this was due to the relatively high work load and to the high standard deviation. There were no significant differences for the peak values following the maximal tests between any of the weeks of testing.

The decreases in submaximal plasma lactate concentrations indicate that training caused the plasma lactate/swimming velocity curve to shift to the right. To determine when, during the training program the shift in the lactate curve occurred, the intercepts of the mean regression analyses were tested for significant differences ($p < 0.05$). The only significant differences occurred between W6 and

W10 with the intercept at W10 shifting to the right. Since there were no significant changes in the slopes of the curves for W1-2, W6, and W10, this would indicate that training shifted the entire curve to the right. To further substantiate when the shift in the lactate curve occurred, a velocity corresponding to a delta lactate of 100 mg/100 ml was determined for each individual for each test week. Only the velocity at W10 was found to be significantly greater than the velocity at W1-2. It was therefore concluded that an intense training program of greater than four to six weeks is necessary for any significant alterations to occur in the lactate/swimming velocity curve in elite swimmers.

In an attempt to determine the accuracy of a single plasma lactate point as a predictor of changes in the lactate/swimming velocity relationship, a velocity corresponding to 100 mg/100 ml was estimated for W10 using the slope from W1-2 and the single lactate value following the 86% swim test at W10. The two velocities for W10, i.e. actual and predicted, from one lactate point were not significantly different ($p < 0.05$) yet highly correlated ($r = 0.91$). However, both actual and predicted velocities for W10 were significantly greater ($p < 0.05$) than the velocity for W1-2. It was concluded that it is valid to use a single plasma lactate point to determine the changes in the lactate/intensity relationship subsequent to training.

It seems reasonable to conclude that if training causes the plasma lactate curve to shift to the right then detraining would cause the curve to shift back to the left. It also seems reasonable to

postulate that the lactate/swimming intensity curve could provide a physiological index to determine the presence of the overtrained state, i.e. overtraining would also result in a shift in the curve back to the left.

APPENDIX A

MEDICAL HISTORY QUESTIONNAIRE

MEDICAL HISTORY QUESTIONNAIRE

NAME_____

AGE _____

SEX _____

1. HEIGHT_____

2. WEIGHT_____

3. DO YOU HAVE A HISTORY OF CHEST PAIN? YES_____ NO_____

4. DO YOU HAVE A HISTORY OF HYPERTENSION (blood pressure of 150/100 or greater)? YES_____ NO_____

   DOES (DID) ANYONE IN YOUR IMMEDIATE FAMILY (mother, father sister, brother, or grandparents) HAVE A HISTORY OF HYPERTENSION? YES_____ NO_____

5. DO YOU HAVE A HISTORY OF CORONARY ARTERY DISEASE (angina, myocardial infarction, or coronary artery bypass surgery)? YES_____ NO_____

   DOES (DID) ANYONE IN YOUR IMMEDIATE FAMILY HAVE A HISTORY OF CORONARY ARTERY DISEASE (angina, myocardial infarction, sudden death, or coronary artery bypass surgery)? YES_____ NO_____

6. DO YOU HAVE DIABETES? YES_____ NO_____

   DOES (DID) ANYONE IN YOUR FAMILY HAVE DIABETES? YES_____ NO_____

7. PLEASE LIST ALL MEDICATIONS YOU ARE CURRENTLY USING_____

   _____

   _____

   _____

8. PLEASE LIST ANY OTHER MEDICAL CONDITIONS WHICH MIGHT CONFLICT WITH A VIGOROUS PROGRAM OF PHYSICAL ACTIVITY. ALSO, LIST AND EXPLAIN ANY DISABILITY OR TROUBLE YOU HAVE WITH YOUR BACK, SHOULDERS, ELBOWS, EARS _____

_____

_____

_____

_____

APPENDIX B

INFORMED CONSENT FORM

SUBJECT CONSENT

Study Title: Lactate Values as a Prediction for Swim Performance

I understand that I am being asked to voluntarily participate in a study entitled "Lactate Values as a Prediction for Swim Performance." The purpose of this investigation is to determine if the amounts of lactic acid in my blood following swims of different intensity can be used to predict my fastest swimming times while noting the changes in the amounts of lactic acid as a result of swim training. Lactic acid is present normally in my muscles after I exercise. I understand that my participation in this study is totally voluntary and that I may withdraw from the study at any time without ill will on the part of the investigators. I further understand that I will participate in the following procedures.

1. Evaluation of Body Composition

I will be participating in a determination of my body composition by being weighed under water ten times. I will exhale all of the air out of my lungs while totally submerged underwater for a period of five to ten seconds, seated in a chair suspended from a scale. Prior to this determination, I will perform two tests to determine my residual lung volume, which is the air remaining in my lungs following a maximal expiration. This will involve breathing into and out of a spirometer for a period of five to ten seconds.

I will also have my body composition assessed by skinfold calipers. Skinfold calipers measure the thickness of skin, and fat. Each

method to determine my body composition will be repeated after the end of the training period.

2. Evaluation of the Amount of Lactic Acid

To evaluate the amount of lactic acid in my blood, I realize that a technician will draw a blood sample of 2 ml (approximately a teaspoon), four to five minutes after I finish each swim test. The swim tests will consist of swimming the front crawl stroke for 400 meters a total of three different times and a 200-meter swim one time. These swims will occur on three different days with no more than two 400-meter swims occurring on the same day. The 400-meter swim will be at 60, 75, and 90% of my best 400-meter time that I have done within the last year. The four swim tests and blood draws will be repeated at the end of the first, second, sixth, and tenth weeks. I realize that I will swim a total of 12 400-meter tests and four 200-meter tests and have a total of 16 blood samples drawn.

I know of no conditions in my medical history which would cause me to think that I cannot participate in this study. I also realize that whenever the skin is punctured, there is a risk of infection or blood leakage around the area. These possibilities will be minimized by adhering to sterile procedures. I understand that during the blood draw there is the usual discomfort associated with the initial puncture of the skin with the needle. There is also the possibility of a hemotoma associated with drawing blood (some blood leaking from the vein and collecting under the skin).

3. Training

I realize that the major emphasis of this study is to evaluate my adaption to swim training.

I understand that as a member of this study, I will be required to train six days per week for a total of ten weeks. I will begin training a total of 10,000 yards to 12,000 yards per day for the first three weeks. The total yardage per day will increase to 15,000 yards per day by the fourth week.

4. Conditions of Participation

As a participant in this study, I will gain an understanding of my physiological profile, both prior to and following a period of endurance training. I will also be in much better physical condition and will have a more favorable body composition. I am also aware that these findings may have significant implications for the prediction of peak swimming speeds.

I understand that all information concerning my performance during this study will be kept confidential and all data will be filed according to a subject number identification code system. I realize that all procedures will be under the constant and direct supervision of physicians who are involved with the Exercise and Sport Sciences Laboratory in McKale Center.

I also understand that this consent form will be filed in an area designated by the Human Subjects Committee with access restricted to the principle investigator or authorized representatives of the department.

I am also aware that in the event of injury resulting from any of the above stated procedures I will receive no compensation for wages, time lost, medical expenses, or hospitalization.

I understand that my involvement in this study will not cost me any money. Likewise, I realize that I will receive no monetary compensation in exchange for my participation.

I have read the above "Subject's Consent" form. The nature, demands, risks, and benefits of the project have been explained to me. I understand that I may ask questions and that I am free to withdraw from the project at any time without ill will. A copy of this consent form will be made available to me upon request.

_____ _____

Subject's Signature Date

_____ _____

Witness' Signature Date

I have carefully explained to the subject the nature of the above project. I hereby certify that to the best of my knowledge the subject signing this consent form understands clearly the nature, demands, benefits, and risks involved in participating in this study. A medical problem or language or educational barrier has not precluded a clear understanding of his involvement in this project.

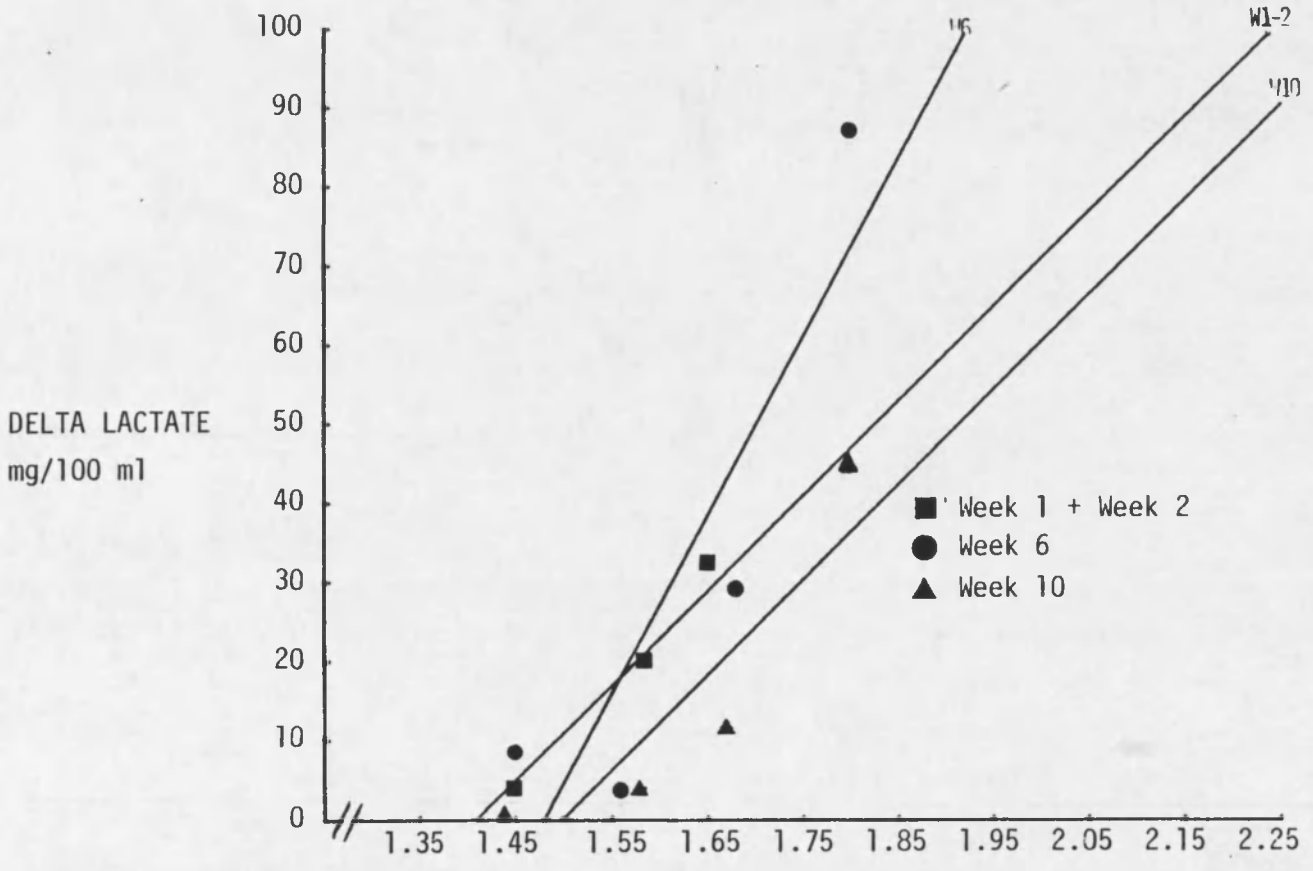
_____ _____

Date

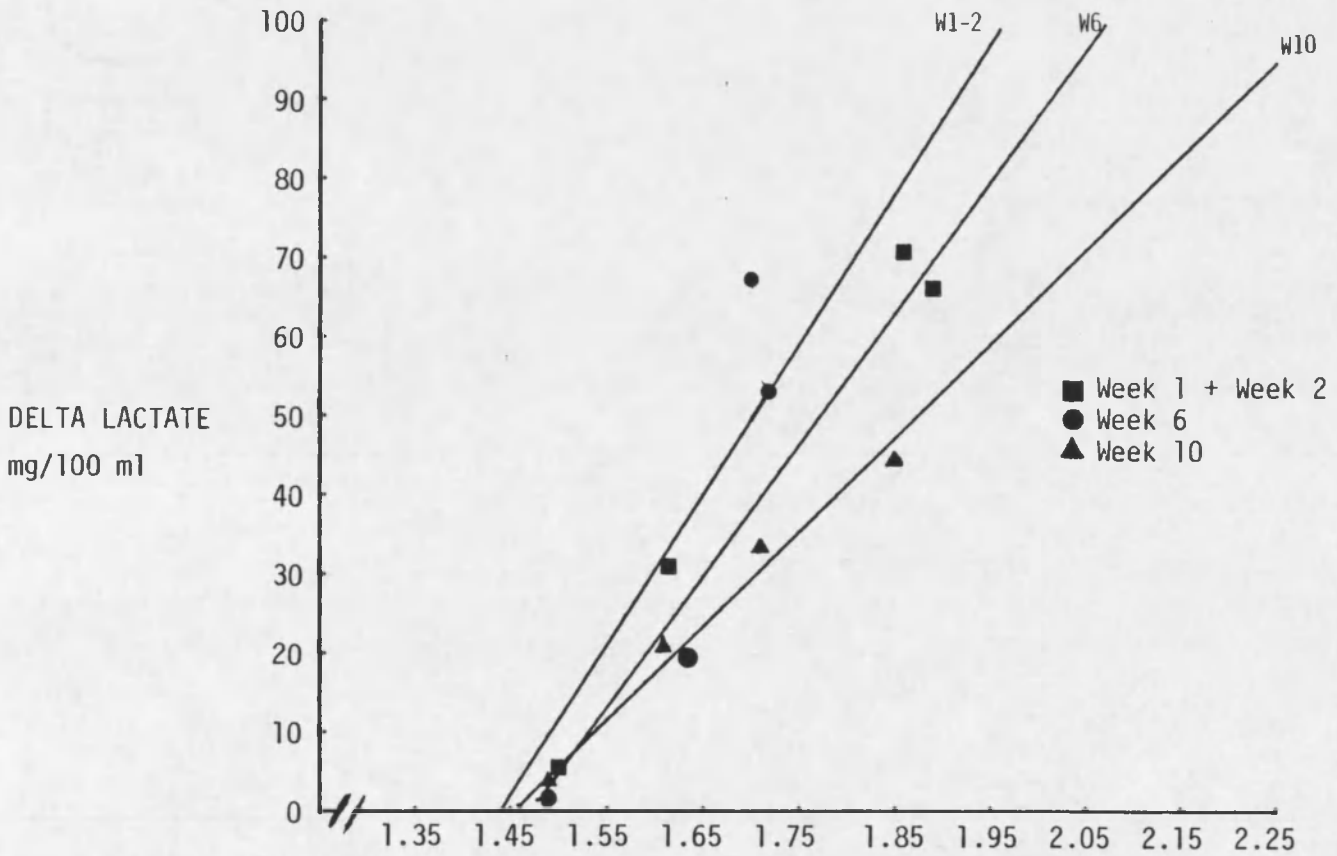
APPENDIX C

INDIVIDUAL REGRESSION ANALYSES

DELTAL LACTATE VERSUS SWIM VELOCITY



Individual Regression Analysis for Subject 01



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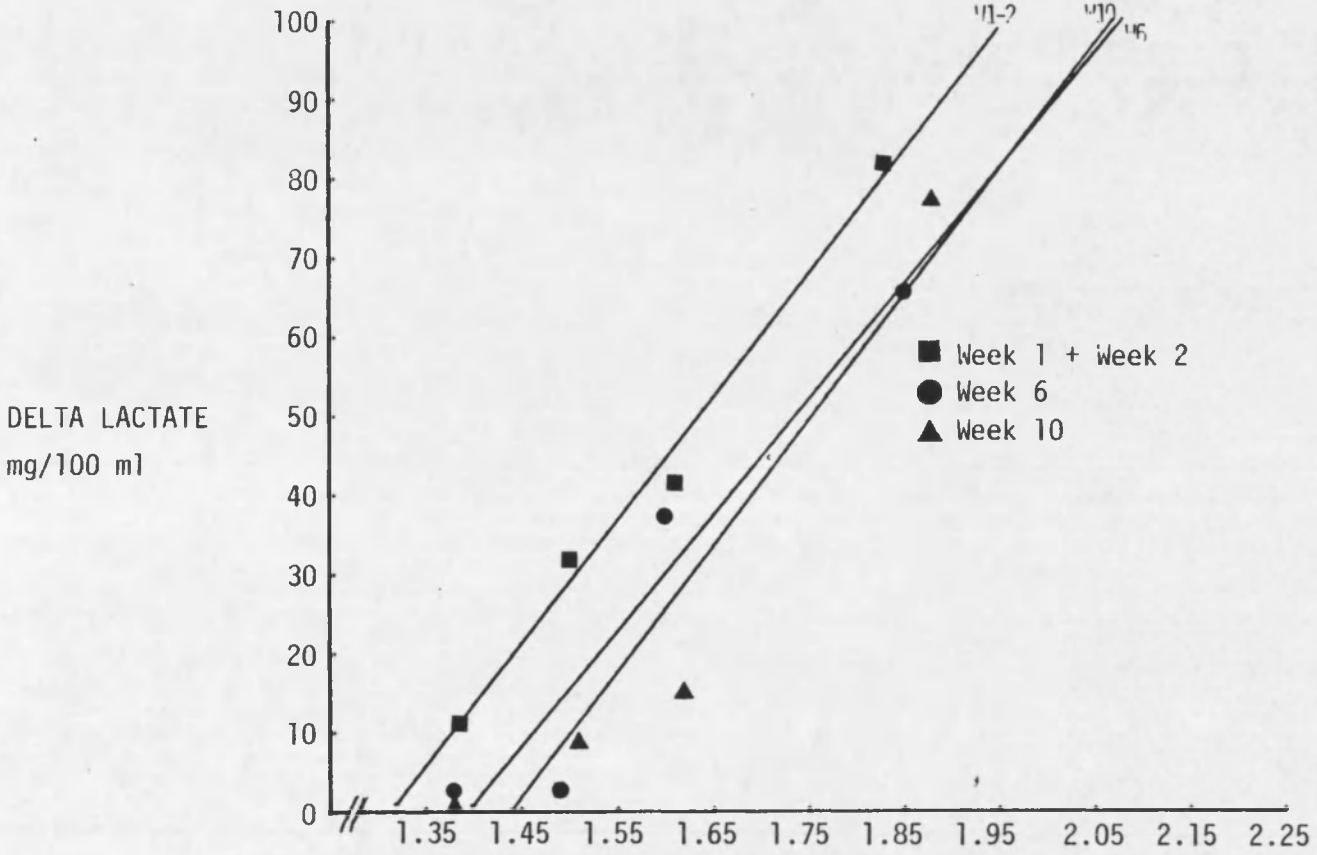
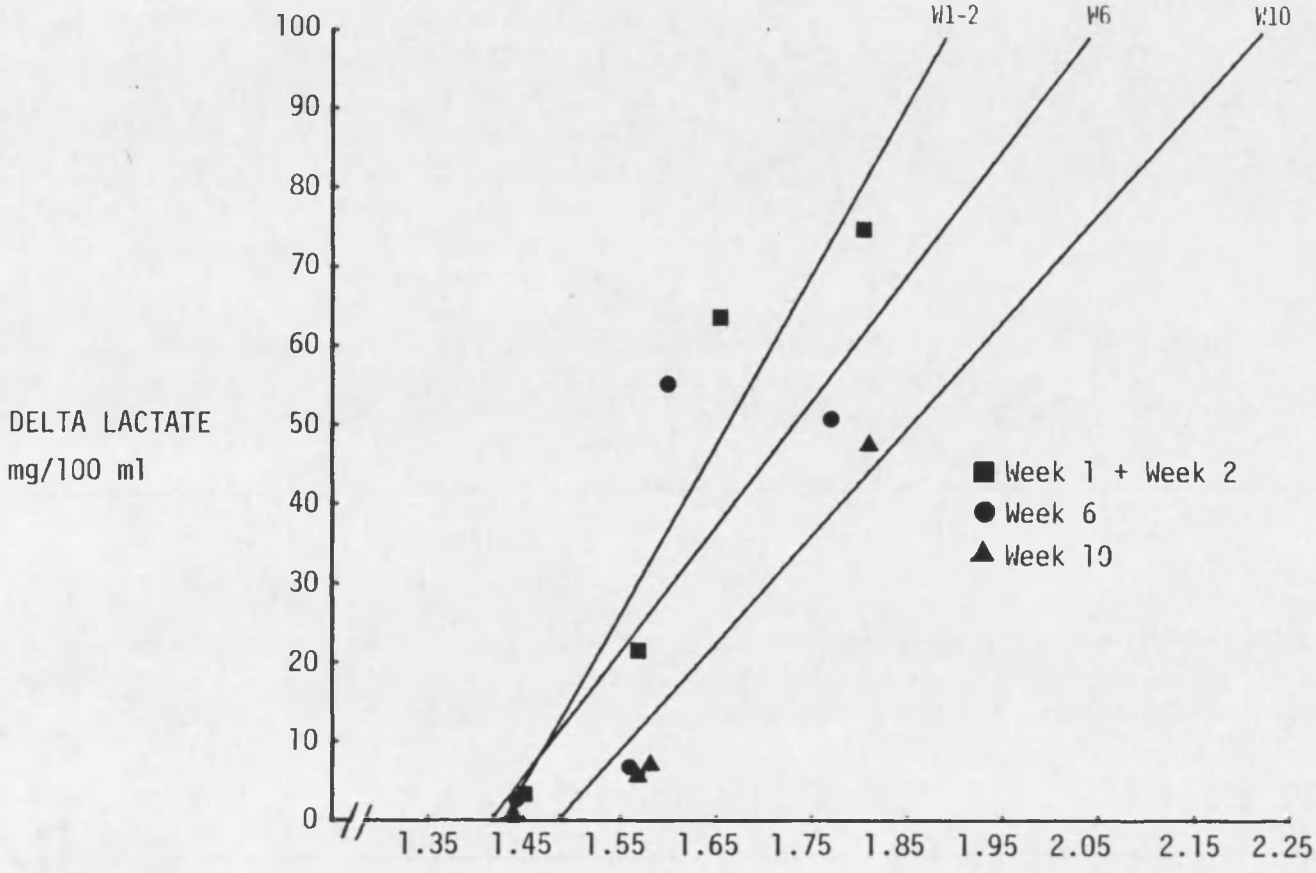


Individual Regression Analysis for Subject 03



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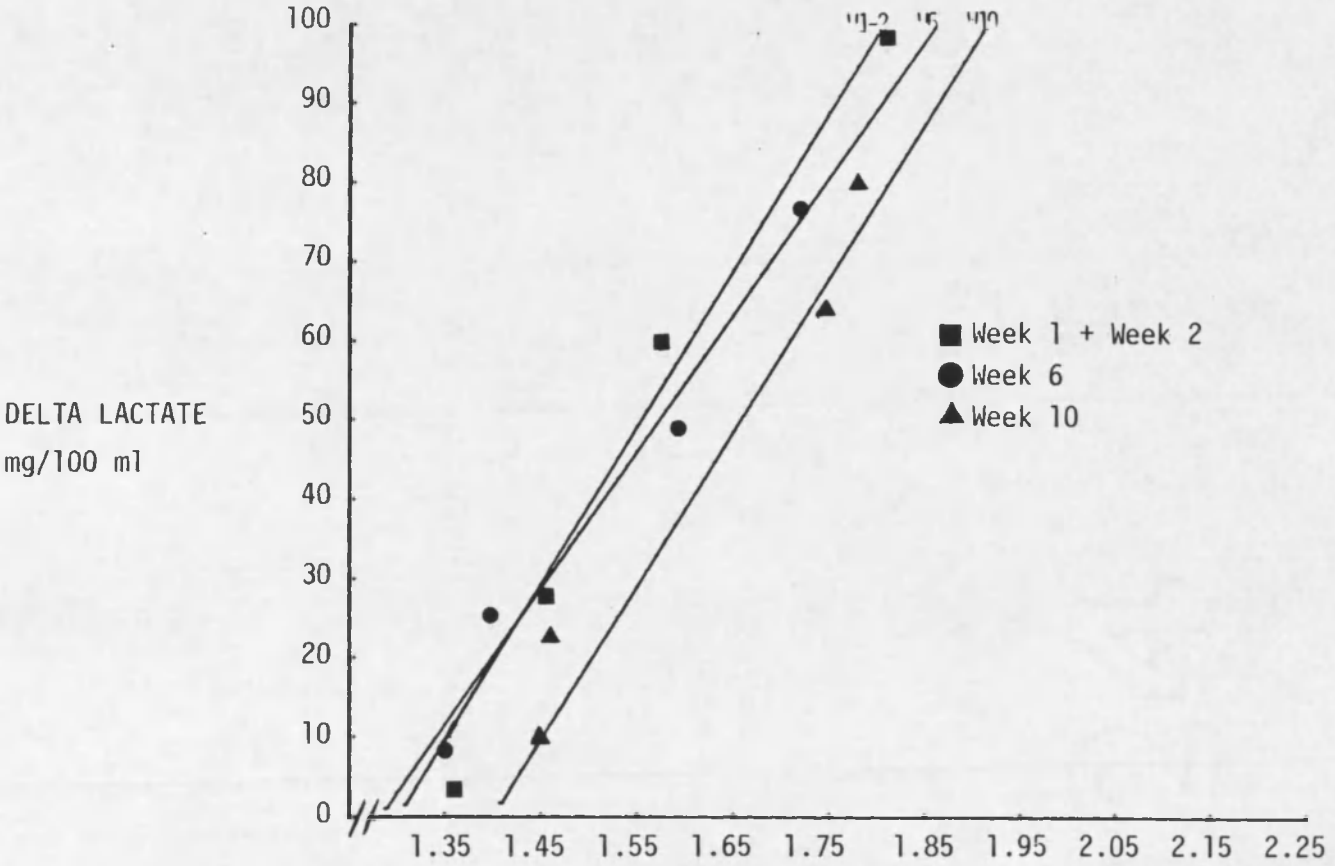

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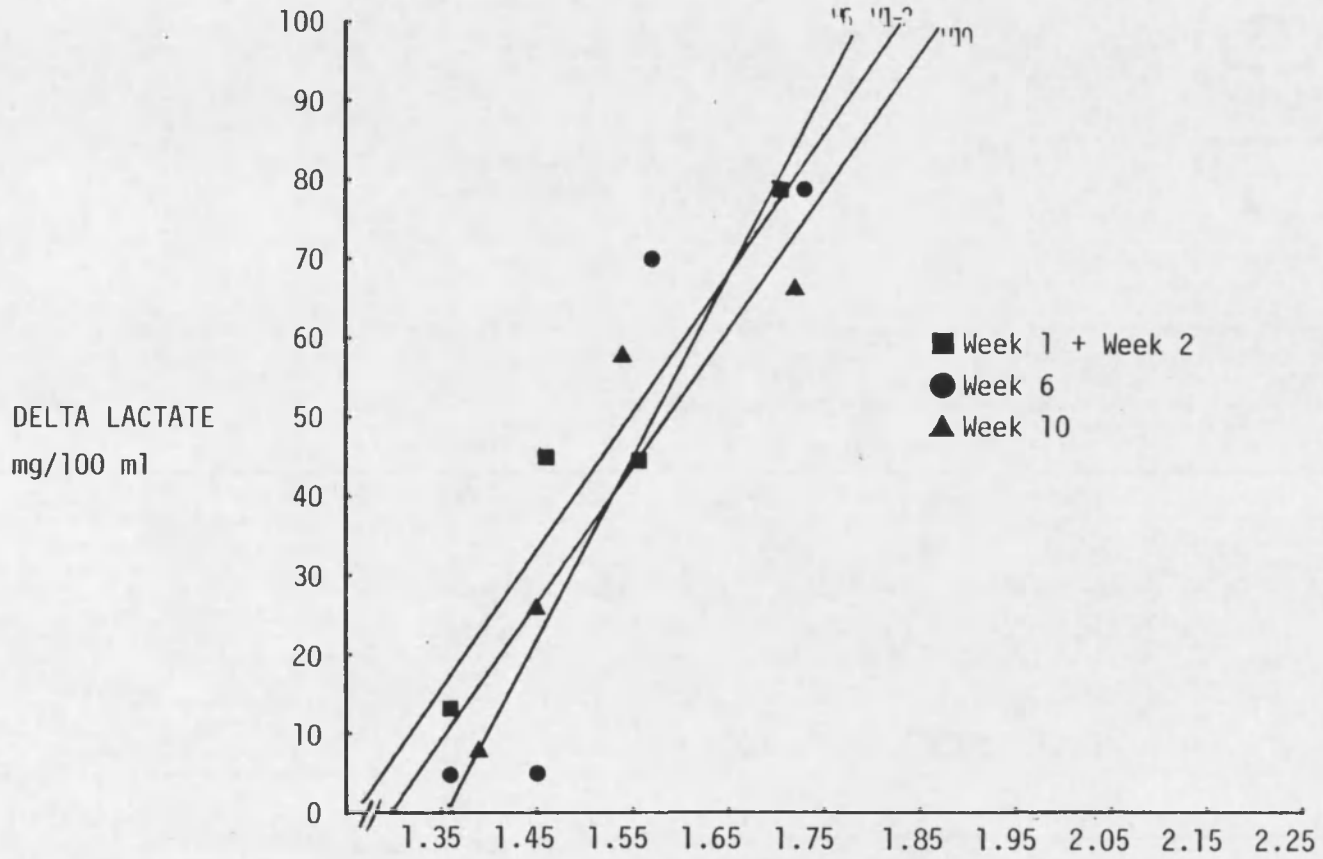

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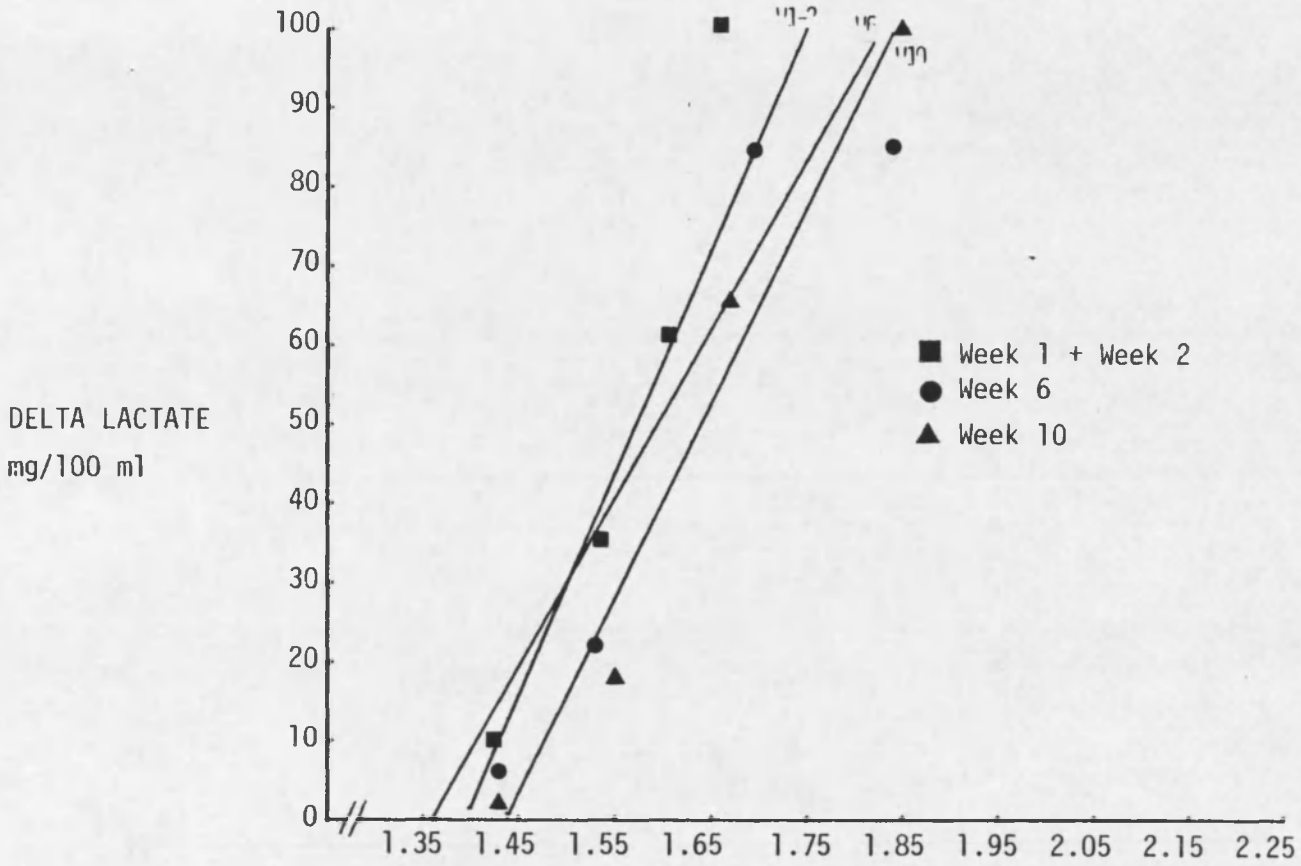

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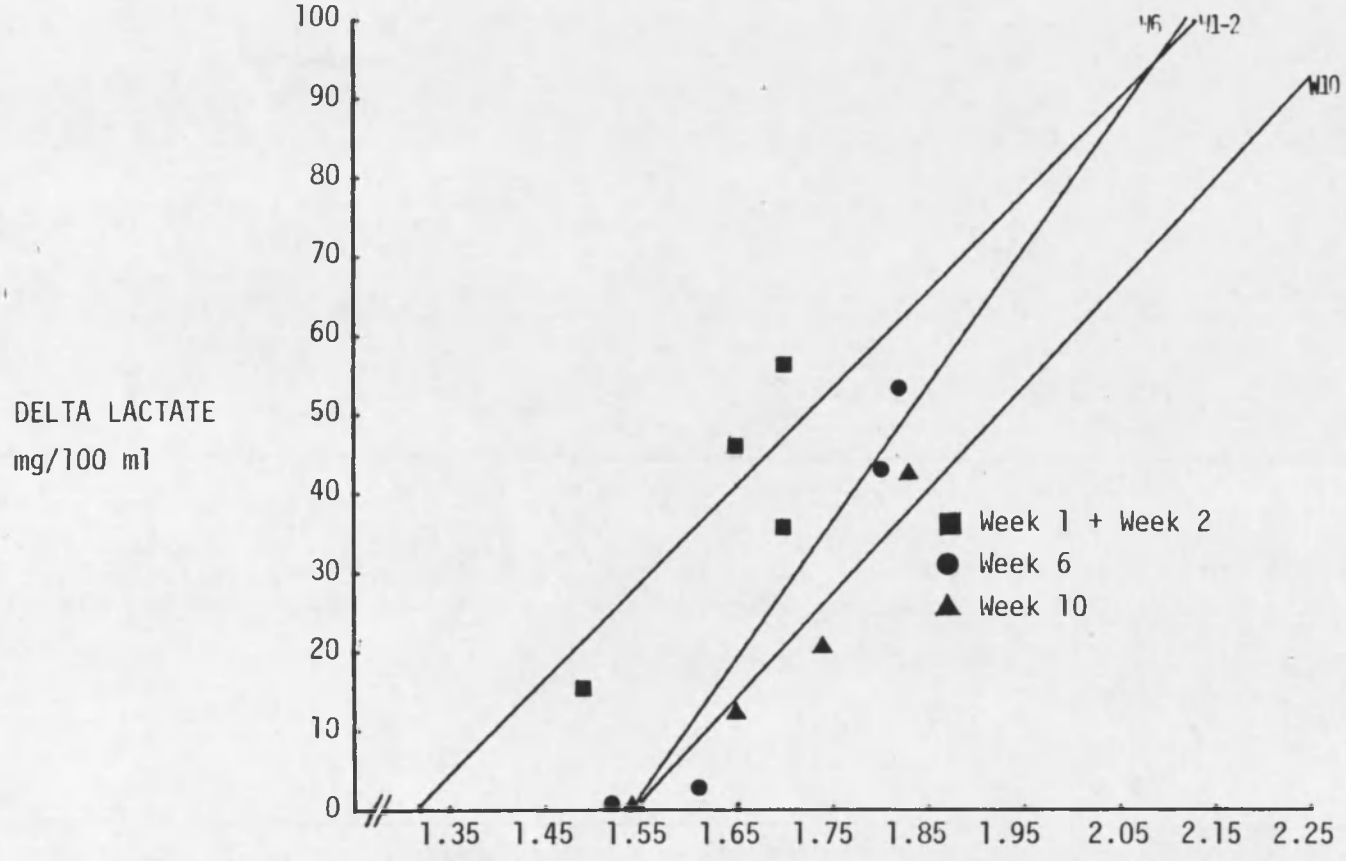
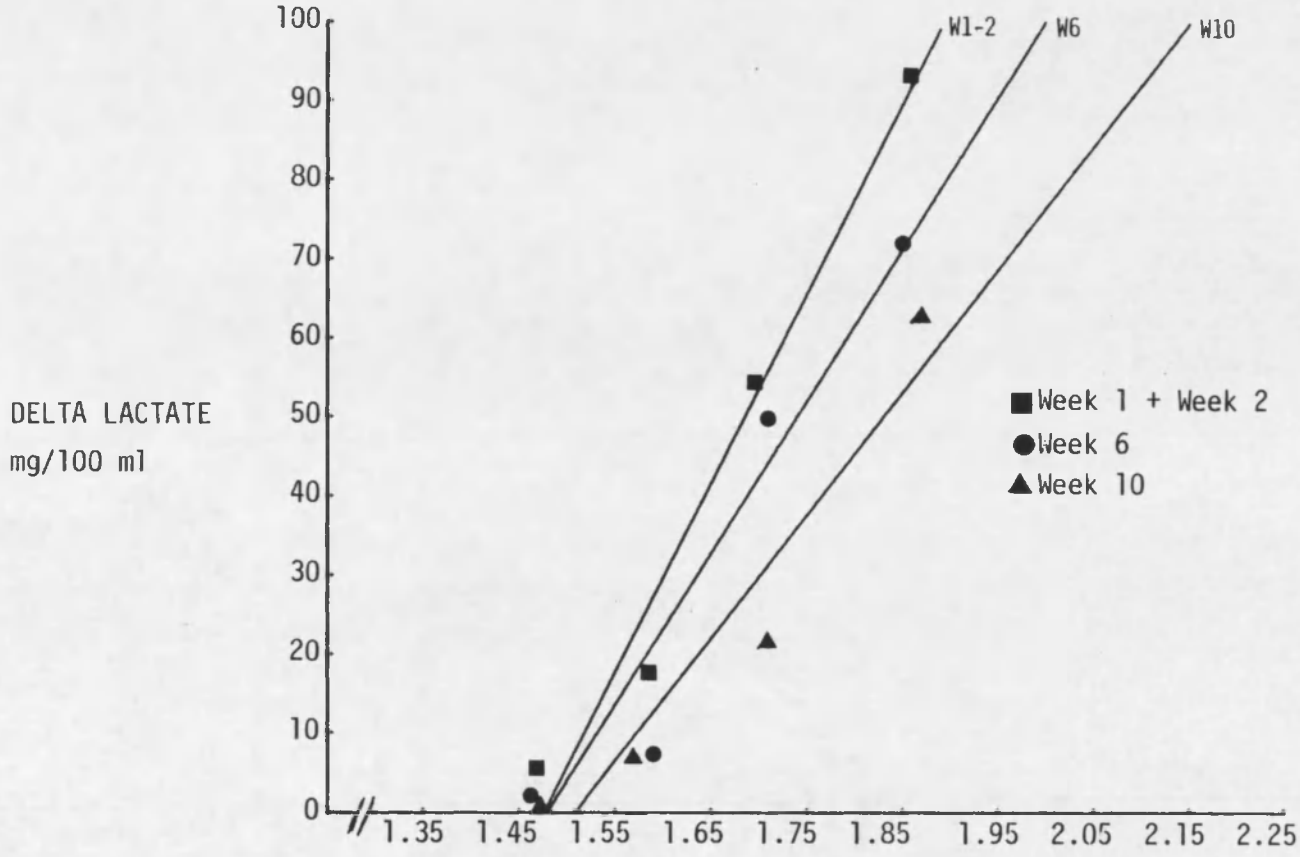
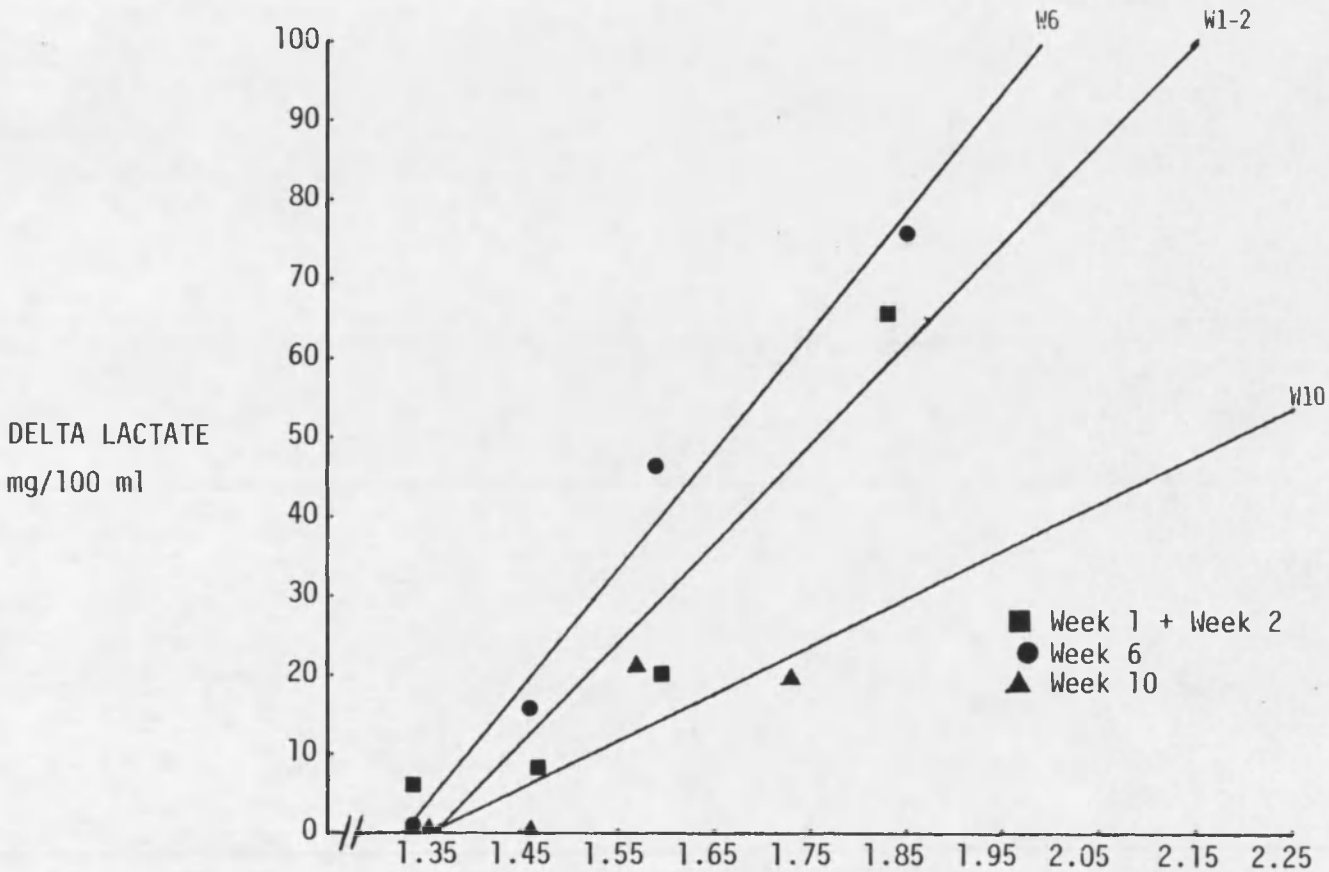


Individual Regression Analysis for Subject 08



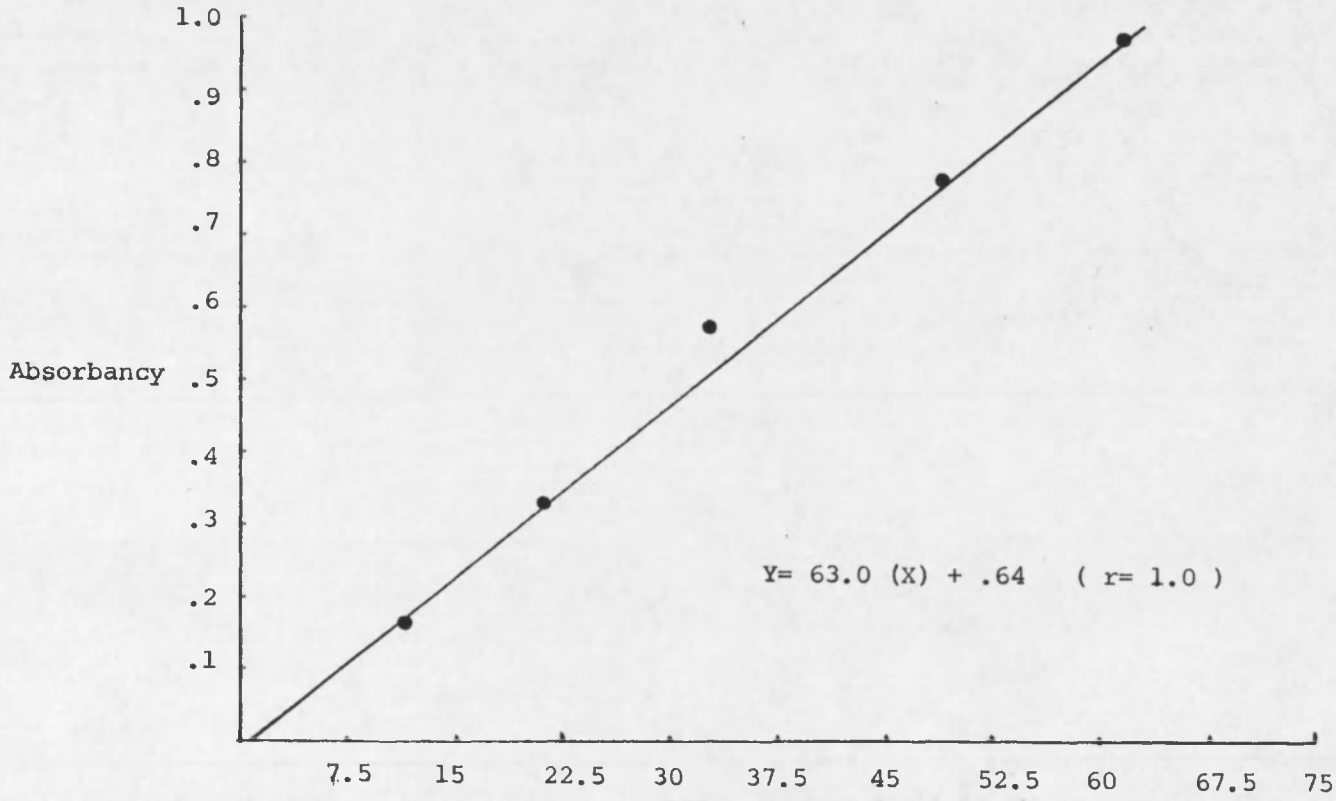
Individual Regression Analysis for Subject 09



Individual Regression Analysis for Subject 10

APPENDIX D

SAMPLE CALIBRATION CURVE



Sample Calibration Curve

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