

1990

Assessment of Athletic Potential and Augmentation of Performance in Thoroughbred Racehorses.

John Daniel Harkins

Louisiana State University and Agricultural & Mechanical College

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**Assessment of athletic potential and augmentation of
performance in thoroughbred racehorses**

Harkins, John Daniel, Ph.D.

The Louisiana State University and Agricultural and Mechanical Col., 1990

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Ann Arbor, MI 48106



Assessment of Athletic Potential and Augmentation
of Performance in Thoroughbred Racehorses

A dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agriculture and Mechanical College
in partial fulfillment of the requirements for the degree of
Doctor of Philosophy

in

The Interdepartmental Program in Veterinary Medical Sciences

by J. Daniel Harkins
D.V.M., Purdue University, 1975
December 1990

Acknowledgements

I praise God for allowing me to complete this dissertation. I thank Him for guidance and perseverance during the planning, testing, and writing stages of this research.

Secondly, I want to express my appreciation for my wife, Mary, and three children, Johnny, Amanda, and Hannah, for their understanding when I was late or absent because of the time required to complete this study. Mary has been a source of encouragement and assurance, especially during the writing of this dissertation.

I want to thank Dr. Steven G. Kamerling for the use of his horses and facilities, but most of all for his positive encouragement, open mindedness, and willingness to allow me the freedom to pursue my own research interests. His comments and criticisms have always been affirmative and constructive.

I am also grateful to the other members of my advisory committee, Dr. R. E. Beadle, Dr. S. L. Dodd, Dr. C. R. Short, Dr. G. M. Strain, and Dr. C. S. Venugopalan, for their constructive criticism and suggestions in the planning and writing of this dissertation.

Training and testing of the large number of horses used in this project would not have been possible without the able assistance of the many workers associated with Heck Farm including: Mr. Jerry Duncan and Mr. Tim Towne, the jockeys for this study; Dr. Jane Cauthen and Mr. Gary Whatley, assistance passing naso-gastric tube; Mr. Richard Daigle, farm manager; Mr. Micheal Keowen and Ms. Cleo Bagwell, assistance with training and timing of horses, and collection and processing of samples; Mr. Kurt Deshotels, farrier; Mr. Troy Alexander, horse handler.

Special thanks is given to Mr. Raymond Rawls whose assistance with the horses, sense of humor, and friendship are greatly appreciated.

Forward

This dissertation is not written in the conventional style with separate chapters for Methods, Results, Discussion, etc. Rather, each section describes an individual investigation and includes objectives, methods, results and discussion. A global introduction section is included as a prelude to all of the investigations performed. This project included diverse issues from assessment of running ability to factors affecting performance like competition, training, and diet. Therefore, it was hoped that this format would present the data more clearly and enable the reader to more easily follow the rationale used.

The phrase 'this project' is used in this document to refer to the studies performed for this dissertation. It is hoped that this will enable the reader to distinguish between work completed for this project and other studies cited.

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Abstract

The purposes of this dissertation were to 1) determine running abilities of horses on the racetrack, 2) design treadmill standard exercise tests (SETs) to measure exercise variables that correlate with racetrack performance, and 3) assess the influence of training, competition, and ergogenic aids on performance.

Run times of 25 Thoroughbreds were determined during solo and competitive track runs of 1200, 1600, and 2000 meters. Cardiac, respiratory, and hematologic variables were later measured during treadmill SETs. Variables were correlated with average run times. Heart rate scores, V_{200} , plasma lactate values, and maximal oxygen uptake correlated best with performance.

Interval and conventional methods of training were compared to examine the effect of training method on performance. After 7.5 months of training, there was no difference in performance between groups. Interval training (IT) was more labor intensive, but it improved the durability of the horses.

Interval training was used in a study of the treadmill as a training tool. Treadmill IT was the only conditioning received during a 20-week training period. There was a significant decrease in maximal heart rate during a sub-

maximal SET. Cardiovascular fitness was attained solely with treadmill exercise.

The effect of competition was examined by comparing race times for solo and competitive runs. No group ran faster during competition than alone. Run times for males were not affected by competition, while females were consistently slower during competitive runs. There were no differences in run times due to age. The slower competitive run times may have been due to the low quality of horses.

The putative ergogenic effects of sodium bicarbonate and 12% fat supplementation were tested separately by comparing 1600-m run times following administration of the 2 ergogenic aids to a control run. There was no difference between control and bicarbonate run times even though blood pH and lactate were significantly increased following NaHCO_3 administration. Mean run time was significantly faster following fat supplementation when compared to the control diet. The improved run time may have been due to increased availability of blood glucose, better utilization of ketone bodies for fuel, and decreased ketosis in the fat-fed group which delayed the onset of fatigue.

Introduction

The purposes of the first part of this dissertation were 1) to determine the running abilities of 25 Thoroughbred horses at 1200, 1600, and 2000 meters, 2) to determine the measurable variables of performance in racehorses on the racetrack, and 3) to design standard exercise tests (SETs) on the high-speed equine treadmill to assess those variables. The measure of performance used for this project was run time measured during solo and competitive track runs. One of the unique aspects of this project was the correlation of racetrack performance with exercise variables measured on the treadmill during standard exercise tests. The purposes of the second part of this dissertation were to assess the influences of 1) training, 2) competition, and 3) putative ergogenic aids on performance.

One objective of the Thoroughbred racing industry is to produce faster racehorses. Athletic success in racehorses has been correlated with several measurable factors including genetic composition, metabolic nature, conformation, and conditioning. Gaffney and Cunningham (1988) used a statistical analysis of Timeform¹ ratings, which is a handicap system based on performance of the horse, distance raced, and level of competition. They determined that 36% of racing performance was due to heritable factors and

¹ Timeform Publications Ltd, Timeform House, Halifax, Yorkshire

the remaining 64% to other influences including training, nutrition, management, and track conditions.

In a performance study of Thoroughbreds in Great Britain, it was shown that only 49% of the 9675 foals born in 1975 ever entered training between the ages of 2-4 years, only 40% of the total ever started a race, and less than 15% were still racing in their 4th year of age (Jeffcott *et al.* 1982). The estimated cost for maintaining foals in Great Britain that did not have the athletic potential to race was estimated to be \$11 million for one year. The cost of horses that raced but did not win enough money to pay for feed and training was not included in the estimate. The two main reasons for the low percentage of horses starting a race were lack of ability (38%) and unsoundness (28%).

If performance potential of young, unraced horses could be accurately assessed, non-athletic horses could be culled earlier, resulting in substantial savings of training time and money (Gillespie, 1988). It was hoped that techniques to measure potential in unraced horses could be developed, thereby enabling horsemen to concentrate their resources on the better athletes.

This study was designed to run a group of 25 horses over 3 distances (1200, 1600, and 2000 m) typical of competitive Thoroughbred races. Heart rates

(HR) were measured using an on-board heart rate monitor.² Heart rates before exercise and during warm-up were used to assess the degree of excitability of the horse. Maximal HR during the run indicated at what work intensity the horses were performing. Heart rate recovery curves during warm-down were used to evaluate fitness and fatigue. Blood lactate following the runs was obtained as a measure of anaerobic metabolism. Heart rate, peak and total plasma lactate were compared with the time elapsed for each run to determine the correlation of these measurements with performance. It was hoped that by identifying strong correlations between any of the variables and performance times, one or more treadmill standard exercise tests could be designed to measure that variable in unraced horses as an indicator of racing potential.

The equine high-speed treadmill is of significant benefit to equine research because it allows for standardization of work intensity, exercise duration, running surface, and environmental conditions. It enables the investigator to constantly monitor HR, blood lactate, and oxygen uptake during the exercise event. In this study, variables measured on the treadmill³ included: the onset of blood lactate accumulation (the work rate corresponding to a blood lactate level of 4 mmol/l); heart rate (a pre- and post-training heart rate score); and

² VMAX, Equine Racing Systems, Vevay, Ind.

³ Mustang 2200, Kagra International Inc., Fahrwangen, Switz.

the calculated slope, y-intercept, and the velocity attained at a steady-state heart rate of 200 beats/min (V_{200}); maximal oxygen uptake (VO_{2max}); and blood-gas values. Subsequent race track run times were correlated with each of the above values to determine how well each treadmill test reflected performance.

Several investigators have studied the effects of different training methods in horses (Bayly, 1985; Webb *et al.* 1988). In this dissertation, the benefits of different training methods were investigated by comparing interval training with conventional training methods to test the hypothesis that interval training delays the onset of fatigue, speeds recovery from a race, and produces stronger support structures which reduces the incidence of injury (Ivers, 1983).

Competition is intuitively considered to be a stimulus for performance by most horsemen, but human studies have shown that many factors influence how the stress of competition is perceived by the competitor (Triplett, 1898; Michaels *et al.* 1982; Wankel, 1972). The effect of competition as a modifier of equine performance was examined by comparing times from the solo and competitive runs. The effects of age and sex on competition were also examined by comparing the times of solo and competitive runs for those variables.

Many different ergogenic aids (performance enhancing agents) have been used to improve performance in racehorses. Sodium bicarbonate ($NaHCO_3$) is

purported to delay the onset of fatigue by increasing the buffering capacity of the HCO_3^- system. The effect of sodium bicarbonate has not been scientifically examined in maximal-effort exercise in the horse. Therefore, NaHCO_3 was administered to a group of horses and their racing performance and hematologic variables following a 1600-m run, which is a representative distance for most Thoroughbred races, were compared to the same variables following a control run.

Recent investigators (Oldham *et al.* 1989; Webb *et al.* 1987) have proposed that adding 10-15% fat to the diet of horses improves performance in short, high-intensity exercise events. These studies have also shown that muscle glycogen storage is increased while on a high-fat diet, supporting the idea that increased glycogen storage results in improved performance. To test this hypothesis, we administered a diet with increased fat content and assessed its ergogenic and metabolic effects. This was accomplished by comparing run performance and hematologic variables following a 1600-m run to the same variables following a control run.

Literature Review

Introduction

The measurement of athletic performance varies with different athletic events. The measure of performance used for this project was run time measured during solo and competitive track runs. The variables measured and the methods of assessment used were based on human and equine studies of performance potential. Performance potential in horses is defined as the inherent ability of a horse to run swiftly in competitive and solo runs.

Since most performance studies have been completed in humans (Evans & Rose, 1988a), much of the literature cited in this dissertation was from human studies. Since human middle-distance events (400-800 m) are of equal duration (about 50-120 sec) to most Thoroughbred races (1000-2000 m), the human studies cited were generally confined to studies of middle-distance athletes. Several variables used in human exercise studies were adapted for assessment of athletic potential in horses. These variables are discussed below.

There is little variation between training methods used by horse trainers, and training methods have changed only slightly during the past 100 years. However, training methods for human athletes have changed dramatically during this century. The frequent improvements in world records attest to the benefits of alternative training programs (Fox & Mathews, 1974). An alternative training method for horses was examined in this project. The alternative method included long slow distance training followed by repeated bouts of short, intense (interval) training.

Competition is intuitively considered to be an incentive to performance. Since no equine studies have investigated the effect of competition in horses, the literature cited in the literature review was from human competitive studies.

The final 2 sections of this dissertation studied the possible ergogenic effects of sodium bicarbonate (NaHCO_3) and a fat-supplemented diet during a 1600-m run. These putative ergogenic aids have recently received much attention in the lay press and among horsemen but have been scientifically studied by only a few investigators. The findings from those studies will be discussed below.

A. Assessment of athletic potential

Assessment of athletic potential in humans has been accomplished by using varied techniques and measurements such as maximal oxygen uptake (VO_{2max}) (Costill & Winrow, 1970a; Shaver, 1975); lactate indices, including lactate threshold (Yoshida, 1986), onset of blood lactate accumulation (OBLA) (Jacobs *et al.* 1985; Morgan *et al.* 1989; Yoshida *et al.* 1987), and peak lactate (Komi *et al.* 1977a; Svedenhag & Sjodin, 1984; Taunton *et al.* 1981); muscle fiber composition and enzyme activity (Galun *et al.* 1988; Gollnick *et al.* 1972); ventilatory threshold (Powers *et al.* 1983; Reybrouck *et al.* 1986); and morphologic characteristics of runners (Hirata, 1966; Pollock *et al.* 1977). Assessment of equine athletic potential has been attempted by using many of the same measurements, including lactate production during exercise (Bayly *et al.* 1987a; Saibene *et al.* 1985), muscle fiber typing and enzyme activity (Straub *et al.* 1983; Wood *et al.* 1988), heart score based on the QRS complex (Physick-Sheard & Hendren, 1983; Steel, 1963), morphological characteristics of racehorses (Gunn, 1983), and hematologic variables (Blackmore, 1983; Revington, 1983). Measurements that have either been used or could be used to correlate with performance in horses are discussed below with comments on why they were or were not used in this study.

1. Maximal oxygen uptake

a. Human studies

The most commonly used procedure for prediction of athletic performance in man is the test for VO_{2max} (Noakes, 1988). Maximal oxygen uptake is the highest amount of oxygen that can be utilized during maximal-effort exercise. The first English language publication on VO_{2max} was written in 1923 by Hill and Lupton (1923). In that study, it was determined that the requirement of oxygen increased steadily as work load increased, but there was an upper limit which could not be surpassed by further increases in work load. Thus, the curve formed by the oxygen uptake at differing work loads was linear up to VO_{2max} where a plateau was reached. In a later study, Taylor *et al.* (1955) concluded that maximal oxygen utilization was limited by the constraints of the circulatory and respiratory systems.

The earliest studies of VO_{2max} showed that the best distance runners had the highest VO_{2max} (Astrand, 1952; Robinson *et al.* 1937). Subsequent studies have used this principle to predict potential in human athletes (Costill & Winrow, 1970a; Costill *et al.* 1973; Foster *et al.* 1978). However, the predictive value of VO_{2max} is of benefit only in heterogenous groups of athletes with varied athletic abilities. In homogenous groups of equal athletic ability, its predictive

value was greatly reduced (Costill & Winrow, 1970b; Daniels, 1974). The study by Costill and Winrow (1970b) showed that two runners of equal VO_{2max} had different running abilities. The difference was due to a superior running economy of the faster runner. In the study of Daniels (1974), two runners of unequal VO_{2max} had equal running abilities because of a superior running economy in the runner with the lower VO_{2max} . In a review by Shephard (1984), correlations between VO_{2max} and run performance of different distances varied from 0.04 to 0.90. The higher correlations were seen with the longer distances, demonstrating the increasing importance of aerobic capacity with increasing exercise duration. Other elements like technique, state of training, and psychological factors have an influence on performance but are not necessarily reflected in VO_{2max} measurements.

A study correlating run times for 30 untrained college men running 7 different distances (100-yd to 3 miles) with VO_{2max} showed that as distance was increased the correlation coefficients (r-value) increased (Shaver, 1975). The correlation between VO_{2max} and the 100-yd run was -0.08 but was significantly ($P < 0.05$) correlated with the 880-yd run ($r = -0.35$), 1-mile run ($r = -0.43$), 2-mile run ($r = -0.76$), and 3-mile run ($r = -0.82$). As distance of the athletic event increased, VO_{2max} became increasingly important as a predictor of performance (Shaver, 1975). This would be expected since a greater proportion of the required energy for longer distance events are derived from aerobic sources.

b. Equine studies

Maximal oxygen uptake has been measured in horses but has not been correlated with race performance. Evans and Rose (1987) measured VO_{2max} in 6 Thoroughbred horses before and after a 12 week treadmill training period to show that VO_{2max} increased with training. They used a gas collection mask with unidirectional flow valves to collect the expired air. In another study, Evans and Rose (1988b) measured VO_{2max} and determined the repeatability of that measurement in 6 Standardbred horses. The coefficient of variation of VO_{2max} was less than 10% in the six horses (range 1.42 to 9.05 percent).

The use of gas-collection masks with unidirectional flow valves has been questioned because of the possible exacerbation of hypoxemia and hypercapnia (Bayly *et al.* 1987b) that normally occurs in horses during strenuous exercise (Bayly *et al.* 1983b; Bayly & Grant, 1986). Complications related to such a gas-collection system include re-breathing expired air and impedance of flow (Bayly *et al.* 1987b). Bayly *et al.* (1983b) tested 4 gas-collection systems used in horses. Two of the systems incorporated unidirectional flow valves and two were flow-through devices (Seeherman *et al.* 1981). One flow-through system tested used a funnel shaped respiratory mask and pulled air around the head and over the nares of the horse. They concluded that the flow-through system which pulled air over the nares at a flow rate in excess of 6,000 l/min was

superior to the other systems. With this system, there was no difference in arterial partial pressure of oxygen (P_aO_2) and arterial partial pressure of carbon dioxide (P_aCO_2) during exercise with or without the face mask. The maximal measured oxygen uptake was also increased by using the funnel shaped mask flow-through system.

Fedak *et al.* (1981) developed a one-step dilution technique for calibrating a flow-through oxygen uptake measuring system. Use of a measured nitrogen flow eliminated the requirement to calibrate the oxygen analyzer or measure the air flow past the animal. Because of the high correlations between performance and VO_{2max} in the cited human studies, the measurement was used in this project to correlate with run times attained on the track. Because of the limitations of the unidirectional flow valves, the flow-through system described by Seherman *et al.* (1981) was the method of choice for oxygen collection. The one-step nitrogen dilution technique described by Fedak *et al.* (1981) was used to measure oxygen uptake.

2. Plasma lactate

Human and equine exercise studies have used different lactate indices to evaluate plasma lactate levels during exercise including lactate threshold (LT)

(Yoshida *et al.* 1987), onset of blood lactate accumulation (OBLA) (Heck *et al.* 1985; Svedenhag & Sjodin, 1984), and peak lactate concentration, which is the maximal lactate measurement attained during an exercise event (Saibene *et al.* 1985; Schnabel & Kindermann, 1983; Taunton *et al.* 1981).

Lactate threshold (LT) is the work load or oxygen uptake at which the blood lactate begins to rise above resting level during an incremental exercise test (Yoshida *et al.* 1987). Onset of blood lactate accumulation (OBLA) is the velocity (V_{La4}) (Yoshida *et al.* 1987), work load (W_{La4}) (Evans & Rose, 1987; Jacobs *et al.* 1985), or oxygen uptake (Yoshida *et al.* 1987) at which blood lactate reaches a concentration of 4 mmol/l.

a. Human studies

Lactate threshold and OBLA are highly correlated with aerobic capacity (Ivy *et al.* 1980; Jacobs, 1981; Yoshida *et al.* 1987), and endurance capacity (Farrell *et al.* 1979; Jacobs, 1981; Svedenhag & Sjodin, 1984) in humans. The positive correlations illustrate that the highest VO_{2max} values were attained when anaerobic metabolism was limited, thereby preventing a significant rise in blood lactate concentration as exercise intensity increased. Since OBLA and VO_{2max} measure aerobic capacity and since OBLA is determined during sub-

maximal exercise, OBLA measurements may be superior to VO_{2max} for assessment of aerobic capacity.

Peak plasma lactate is a measure of anaerobic capacity (Snow *et al.* 1983a). Saltin and Gollnick (1983) demonstrated that the mechanism for the rapid production of lactate was via anaerobic glycolysis, which has been associated with fast-twitch muscle metabolism. Studies of human athletes have shown that peak lactate following exercise of similar intensity and duration is higher in elite shorter-distance runners than in elite longer-distance runners. Taunton *et al.* (1981) showed that middle-distance runners attained a significantly higher plasma lactate concentration than long-distance runners following an exhaustive treadmill test for VO_{2max} . In a study using elite Swedish runners who competed at distances of 400 m to a marathon (41,950 m), the peak lactate production (range 5.7-18.4 mmol/l) of the shorter-distance runners was significantly greater than for the longer-distance runners (Svedenhag & Sjodin, 1984). It was assumed that elite runners competed in events in which they were successful, so faster middle-distance runners were able to generate lactate more quickly than runners that were less successful at middle-distance events. Komi *et al.* (1977a) showed that sprinters and 800-m runners had higher peak lactate concentrations than longer distance runners after an exhaustive treadmill run. Another study showed that sprinters and middle-distance runners accumulated plasma lactate

significantly faster than longer distance runners during maximal treadmill exercise (Costill *et al.* 1973). Schnabel and Kindermann (1983) showed that elite 400-m runners were able to attain higher maximal arterial lactate concentrations than longer-distance runners or a control group of physical education students not involved in a training program. These studies show that faster short- and middle-distance runners attain a higher concentration of blood lactate at a faster rate of lactate production than less successful runners at those distances.

b. Equine studies

Plasma lactate results from equine studies have been consistent with findings from human studies. Plasma lactate concentration has been proposed to be of significant potential for assessing the athletic ability and state of fitness of a horse (Bayly *et al.* 1987a). In a study of Thoroughbred horses, Bayly *et al.* (1987a) showed that during a run of 1000 m there was a trend toward higher lactates in the faster horses. However, they were unable to show consistent correlations between speed and plasma lactate in individual horses because of environmental and procedural complications. In a study of 16 horses (Thoroughbreds, Standardbreds, and polo ponies), Saibene *et al.* (1985) showed that higher plasma lactate levels (measured 5 min after end of run) were seen

in the faster horses raced over distances of 200, 300, and 400 meters. However, in a study of 12 conditioned Standardbreds, Wilson *et al.* (1983) concluded that the faster horses attained significantly lower blood lactate levels during an incremental submaximal SET with the speed of the final increment causing a heart rate in excess of 220 beats/min. Blood lactate levels were not correlated with actual performance times in this study. The authors also grouped the same horses according to the trainer's perception of the ability of the horse. The group considered to be the 'better horses' attained lower lactate levels during the SET. In a study by Persson and Ullberg (1974) it was also shown that maximum speed of Standardbreds was inversely correlated with blood lactate levels at a speed of 10.0 m/s.

The 2 studies using Standardbreds seem to contradict the findings of the Thoroughbred studies. Standardbred racing probably is more of an aerobic activity than Thoroughbred racing. Standardbreds do have a higher percentage of slow twitch (Type I) and a lower percentage of fast glycolytic (Type IIb) fibers than Thoroughbreds (Valberg & Essen-Gustavsson, 1987) which may indicate why Standardbreds have a higher aerobic potential than Thoroughbreds. The metabolism of Thoroughbreds has been compared to that of human middle-distance runners. However, it may be that the metabolism of Standardbreds resembles that of human marathoners more than middle-distance runners. If this hypothesis is true, then it would be expected that the

faster Standardbreds would perform more work at lower levels of lactate production.

Because of the high correlation of plasma lactate with performance in human and equine studies, various plasma lactate variables (peak lactate, total lactate, V_{La4} , and W_{La4}) were correlated with run times in this dissertation to assess which lactate variables best reflect race track performance.

3. Muscle fiber type

The distribution of fiber types in a skeletal muscle is primarily determined by genetic factors (Komi *et al.* 1977b). Muscle fibers have been grouped into 2 types, slow-twitch and fast-twitch fibers (Gollnick & Matoba, 1984). The contractile speed of muscle fibers is thought to be a function of its myosin, which is part of the myofibrillar complex (Barany, 1967). The different types of myosin have been identified histochemically by their staining property following preincubation in high pH media (pH=10.4). Fast-twitch (type II) fibers stained black and slow-twitch (type I) fibers stained pale. A further subclassification of the fast-twitch fibers has been possible with preincubation in low pH media (pH=4.6 and 4.35). Some of the type II fibers retained the dark stain while others turned pale in the acidic media. This pattern of

staining intensity has resulted in a nomenclature of type I, type IIa (fast twitch high oxidative), and type IIb (fast twitch glycolytic) fibers (Brooke & Kaiser, 1972).

a. Human studies

The first attempt to characterize the fiber composition of human athletes with different athletic ability was by Gollnick *et al.* (1972). The findings of that study indicated that elite endurance athletes had a higher percentage of slow-twitch fibers, while elite power athletes (sprinters, throwers, jumpers) had a higher percentage of fast-twitch fibers. Subsequent studies have found this same distribution of fiber types among endurance and power athletes (Campbell *et al.* 1979; Costill *et al.* 1976; Green *et al.* 1979). However, there has been no conclusive evidence that performance ability can be predicted on the basis of fiber distribution in the muscles (Bell *et al.* 1980; Gollnick *et al.* 1980).

b. Equine studies

While the percentage of type I fibers in the vastus lateralis of man ranged from 0-100%, the range for Thoroughbreds is considerably less (Snow, 1983b). In a study of 50 elite broodmares and 22 elite stallions, Snow and Guy (1981) found $11.0 \pm 0.7\%$ (mean \pm SEM) type I fibers for mares and $7.3 \pm 0.9\%$ for stallions. The small SEM illustrated the limited variation in equine muscle fiber type. Furthermore, Kline *et al.* (1987) demonstrated that the percent of fiber types changed with the depth of the biopsy sample. Lindholm *et al.* (1983) studied the relationship of track performance to fiber type. They biopsied 14 horses before and following 1 year of racetrack training and found no relationship when place of finish or money won was correlated with percentage of the 3 fiber types. Oxidative (citrate synthase and hydroxy-acyl-CoA dehydrogenase) and glycolytic (triose-phosphate dehydrogenase and lactate dehydrogenase) enzymes were also measured. Similarly, there was no correlation between track performance and percentage of these enzymes. In fact, there was more variation attributable to state of fitness and age than to money won or place of finish. In a study of muscle fiber composition of Quarter horses (Wood *et al.* 1988), it was concluded that muscle fiber analysis alone could not be used to predict success in racehorses.

Because of the relatively large variability in muscle fiber type due to age, sex, and state of fitness, fiber typing did not appear to be reliable as a performance predictor. No other equine studies have had favorable results using only the information obtained through muscle biopsies; therefore it was not used in this study.

4. Heart score

Heart score is determined by measuring the duration of the QRS complex to the nearest 10 msec on an electrocardiogram. The electrocardiogram is recorded at 25 mm/sec paper speed on standard bipolar leads I, II, and III, and the mean of the 3 leads is then calculated. The study by Wilson and Herrman (1930) was the first investigation of ventricular depolarization in man, as measured by QRS duration and ventricular weight. Steel (1963) extended this concept to horses and demonstrated a correlation coefficient of 0.89 ($P < 0.01$) for the relationship between heart weight and mean QRS duration, which was the mean of the 3 standard limb leads. He also showed a correlation coefficient of 0.86 ($P < 0.01$) between heart weight and the longest QRS duration. When heart weight was compared with stake race winnings he showed a correlation coefficient of 0.44 ($P < 0.01$). It was believed that a larger heart was able to pump more blood, thereby providing the greater aerobic

power needed in racing. Subsequent studies have presented mixed results on the validity of heart score as a means of predicting performance. Nielsen and Vibe-Petersen (1980) determined the heart score on 230 horses and separated the horses by age. They demonstrated correlations between heart scores and race earnings of $r=-0.28$ ($P<0.05$) for 3-yr olds to $r=-0.57$ ($P<0.001$) for horses 5 yr and older. The correlation was stronger in stallions than in mares. Gross *et al.* (1974) failed to show any significant relationship between heart score and race earnings. They compared the heart scores of 12 yearlings to race earnings after the first year of racing and determined a correlation coefficient of 0.06. Leadon *et al.* (1982) compared the heart scores of 89 racehorses to Timeform ratings and showed correlation coefficients for 2 yr-olds of 0.32 and for 3 yr-olds of 0.36 ($P<0.05$). Nielsen and Vibe-Petersen (1980) and Leadon *et al.* (1982) were able to show statistical significance even with low correlation coefficients because of the large sample size in those studies. Because of a lack of a strong, consistent correlation of heart score and race performance in other equine investigations, heart score was not measured for performance prediction in this dissertation.

5. Morphometric characteristics

a. Human studies

In studies of human athletes, morphometric measurements have been used to show that physical characteristics of elite athletes vary according to ability and the athletic event in which they compete. Bale *et al.* (1985) studied female marathon runners and found that elite runners had a significantly smaller amount of body fat than less successful runners. There was no difference in height, weight, or bone width. They described the elite and good runners in this study as primarily ectomorphic, with a moderately high mesomorphic component. Christensen and Ruhling (1983) also found elite women marathon runners to be of average height but with lower than normal per cent body fat. Katch and McArdle (1973) reported a smaller calf width in superior women marathon runners. Daniels (1974) found that the amount of fat carried by athletes depended on the sport in which they participated. Football linemen and field-event throwers were the heaviest and had the highest per cent of body fat (214 lbs., 14.2% and 218 lbs., 15.5%, respectively). Cross-country runners and distance track men were the lightest and averaged the least per cent of body fat (139 lbs., 7.7% and 147 lbs., 7.8%, respectively). Roskamm (1967) studied the ratio of heart size to total body weight and discovered that competitive cyclists and cross-country skiers had the highest values. Weight

lifters had a lower heart size/body weight ratio than non-athletes. Berg and Bell (1980) measured height, weight, hip width and lower extremity length as expressed as a percentage of standing height, and percent body fat in competitive runners to assess the effect of morphometric characteristics on run times for a 60-yd dash, 440-yd run, and mile run. Heavier body weights were associated with slower run times for the 3 distances. There was a poor correlation between lower extremity length and mile run times but a significant correlation with run times for the shorter runs indicating that long lower extremities may be a disadvantage in sprinting. Hip width/height percentage was also significantly correlated to run times indicating there was an anatomical disadvantage of wide hips because of the increased medial slant of the femoral shaft. Boardman (1933) observed that good endurance runners had longer legs, shorter trunks, and narrower hips in proportion to total height than did less successful endurance runners.

b. Equine studies

Few scientific morphometric studies have been performed in racehorses. Gunn (1983) dissected preserved skeletons of elite Thoroughbreds and compared the morphometric findings with other breeds of horses. He discovered that Thoroughbreds had a greater mass of the hind limbs nearer the hip joint than

other breeds. He concluded that this anatomical configuration allowed an increased frequency of hind leg movement and greater speed when running. In a later study (Gunn, 1987) it was determined that total muscle weight and total bone weight contributed a larger percentage of live body weight in Thoroughbreds than in other breeds. These findings were consistent with another study that demonstrated Greyhound dogs used for racing had a greater muscle growth rate and greater percentage of muscle mass than other breeds of dogs not used for racing (Gunn, 1975). Adult Thoroughbreds were shown to have a higher muscle-to-bone ratio than other breeds of horses. The rate of increase in muscle and bone weight was greater and smaller, respectively, than those rates of increase in other breeds of horses. When growth rates of different muscle groups were compared, the femoral and longissimus groups grew at a greater rate for Thoroughbreds than for other breeds. Since a galloping horse is propelled predominantly by the hind limbs (Hildebrand, 1959), increased growth rate of the femoral muscle group seen in Thoroughbreds indicated a greater propulsive capacity in that breed. Thoroughbreds also had a smaller percentage of fat than other breeds (Gunn, 1987).

Cook (1988) has developed a simple non-invasive palpation technique to assess the degree of recurrent laryngeal neuropathy (RLN), which is a restrictive airway disease of the laryngeal area in racehorses. The author claimed that

performance was directly related to intermandibular width in a group of 48 Thoroughbred broodmares. There was an inverse relationship between the improvement in racing performance from the 2 to 3 yr-old year and the degree of RLN. The less severe the disease, the greater was the improvement in racing performance. Intermandibular width showed no correlation to body size, so it was speculated that a small horse with a wide mandible would have a more efficient oxygen delivery than a large horse.

The methodology used by Gunn (1983), while informative, was not practical for this project. The theory of Cook (1988), while much publicized in the lay literature, has not been well received in the scientific community. Further investigation of the unique anatomical characteristics of elite Thoroughbreds may be useful in performance prediction, but there is insufficient data to correlate performance with morphometric measurements at the present time.

6. Hematologic variables

Investigators have measured different hematologic variables of race horses in an attempt to discover a variable which correlated with race performance with varying results depending on the variable measured. Revington (1983) measured 816 blood samples collected 1-3 hours before racing at an Australian

race track. Race performance was correlated to resting packed cell volume (PCV), red blood cell (RBC) count, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, white blood cell count, neutrophil/lymphocyte ratio, and total plasma protein. No relationship was found between any resting variable measured and race performance. Persson (1967;1969) took blood samples after exercise or after an injection of adrenalin to preclude the wide range of values associated with partial splenic contraction. This collection procedure gave less variation in hemoglobin concentration than samples taken at rest and showed a good correlation with racing performance in Swedish trotters. Stewart (1971) claimed that 82 percent of winners in his study had higher PCV, hemoglobin, and RBC values than the general population mean. He concluded that a horse with a resting PCV more than 1 standard deviation under the population mean for healthy horses in training was apt to perform below the level of capability for that horse (Stewart & Steel, 1974). Performance in such horses was probably limited by the decreased oxygen carrying capacity of the blood.

Total and differential leukocyte counts were not beneficial in performance prediction (Carlson, 1987). Blackmore (1983) analyzed blood samples from over 600 Thoroughbred racehorses for 28 components in a study of the hematological properties of racehorses. When the variables were correlated with Timeform ratings, significant positive correlations were found for sodium

concentration and RBC volume and a significant negative correlation was found for blood urea concentration. Sommer and Felbinger (1983) measured aspartate aminotransferase, creatine kinase, lactate dehydrogenase, alkaline phosphatase, gamma glutamyl transferase, total protein, cholesterol, bilirubin, calcium, phosphorous, magnesium, sodium, and potassium in a group of 26 racehorses. Samples were collected on the day before and on days 1, 2, 3, 4, and 5 after racing. No significant correlation was found between these variables and race performance.

Investigators have performed exhaustive studies on hematologic variables in race horses and found that maximal PCV was more strongly correlated with race performance than any of the other variables measured (Persson, 1967; 1974). Since the oxygen carrying capacity of the blood is dependent on PCV, aerobic capacity will be partially determined by that variable. Packed cell volume can increase 50% from rest to maximal exercise in horses due to splenic contraction. Splenic contraction is not an 'all or none' phenomenon, but rather it is related to the degree of sympathetic stimulation which is determined by mental processes and physical activity (Snow, 1983c). Since maximal PCV is more relevant to maximal oxygen carrying capacity of the blood, and since maximal PCV is more reproducible than resting PCV (because of psychogenic factors), PCV obtained during a maximal effort SET was used in this project.

B. Factors influencing performance

1. Effect of interval training on performance

a. History

The majority of Thoroughbred racehorses are trained with conventional training methods. Conventional training includes jogging, swimming, and walking with intermittent breeze work (exercise at 75% maximal effort) at 7-10 day intervals.

Interval training (IT) has been defined as a method of athletic conditioning with work accomplished in multiple bouts separated by short rest periods (Fox & Mathews, 1981). For example, work bouts were performed at near-maximal effort and lasted 1-2 minutes, with rest periods of 5-10 minutes between bouts (Daniels & Scardina, 1984). A long slow distance (LSD) phase, which consists of numerous conditioning miles each day, precedes the IT phase. The early works of Astrand *et al.* (1960) and Christensen *et al.* (1960) were the foundation upon which subsequent training studies have been based. In those earlier works, the researchers used the term 'intermittent' rather than 'interval'. Since both terms describe a training process of intense exercise

interrupted by short recovery periods, the terms have since become synonymous (Daniels & Scardina, 1984).

b. Metabolic effects

Several studies have described improvements in work capacity with interval training. Westra *et al.* (1985) demonstrated that anaerobic muscle metabolism in rats was enhanced by maximal work efforts of 1 minute followed by a rest period of 4 minutes. This training regimen was sustained for 4-5 work periods until exhaustion occurred. Studies have shown that this type of training improves maximal anaerobic performance by 10-15% (Costill *et al.* 1979; Thorstensson *et al.* 1975). Davies *et al.* (1982) found an increase of 25% in sprinting speed of rats after 4 weeks of sprint (interval) training. After 6 weeks of intense interval training in rats, Westra *et al.* (1985) found a 24% increase in sprinting speed. They demonstrated an enhanced aerobic capacity by a 48% increase in succinate dehydrogenase, a key enzyme in aerobic metabolism. The increased maximal power output was attributed to increased motor recruitment, a greater contribution of power output from aerobic metabolism, and improved muscle economy.

c. Biochemical changes

Studies have shown that the biochemical changes in slow-twitch and fast-twitch muscle fibers are not the same for continuous and interval training regimens (Dudley *et al.* 1982; Henriksson & Reitman, 1976). Interval training increased the oxidative capacities of fast- and slow-twitch fibers in rats (Dudley *et al.* 1982). However, in an interval training study in humans (Henriksson & Reitman, 1976) the oxidative capacity was significantly increased in fast-twitch fibers but was unchanged in slow-twitch fibers. In rats (Dudley *et al.* 1982; Harms & Hickson, 1983) and humans (Henriksson & Reitman, 1976) continuous training at a constant intensity (50-80% VO_{2max}) was most effective in increasing the oxidative capacity of type I (slow-twitch) muscle fibers. Interval training at intensities near or above VO_{2max} was most effective in increasing the oxidative capacity of type II (fast-twitch) muscle fibers (Henriksson & Reitman, 1976). This is because fast twitch fibers appear to be the last fibers recruited during a maximal effort event (Baldwin *et al.* 1977; Terjung, 1976). Dudley *et al.* (1982) found similar results when they studied the effects of intensity and duration of exercise on cytochrome C concentration in type I, IIa, and IIb fibers. They discovered an apparent exercise intensity threshold below which no adaptive change in the muscle occurred. Adaptive changes occurred in type I fibers at low exercise intensities, while type IIb fibers did not show any adaptive response until the exercise intensity reached

a level of 80% VO_{2max} . This was in agreement with the findings of Sullivan and Armstrong (1978) who found that the ratio of type IIb fibers recruited for work increased disproportionately as treadmill speeds increased to supra-maximal levels. There was also a reciprocal relationship between exercise intensity and duration. The time required to bring about an equal change in cytochrome C response was reduced as the exercise intensity was increased.

d. Ventilatory effects

Poole and Gaesser (1985) compared the changes in ventilatory and lactate thresholds in untrained men following continuous and interval training. Maximal ventilation during exercise increased significantly greater for the interval training group than for the continuous training group. Likewise, the increase in ventilatory threshold was significantly greater for the interval training group than for the continuous training group. However, lactate threshold significantly improved for both types of training, and there was no significant difference in the degree of improvement between groups.

e. Interval training in horses

Interval training in race horses has received increased attention since being discussed in a paper by Asheim *et al.* (1970). Interval training was developed for Standardbred racehorses over a hundred years ago (Marvin, 1890). That method of training allowed horses to go rapidly for distances of 200 m then jog slowly for a recovery period. As fitness was attained, the horses were allowed to go rapidly for longer distances up to half (800 m) the race distance of 1600 m. This strategy was similar to the training method of Edington and Edgerton (1976) who suggested that the duration of a single exercise bout should not exceed half the race distance. Lindholm and Saltin (1974b) agreed that the practical distance for a work bout in Standardbreds was 800 m. Gabel *et al.* (1983) were unable to show any differences in plasma lactate, heart rate, cardiac output, and race times between conventional and interval training of 8 Standardbred horses. Despite the lack of significant findings, the authors avowed that IT could improve the race times of horses by 'a few seconds' at a distance of 1 mile. Rodiek *et al.* (1987) compared the effects of interval and conventional training on heart rate, cardiac output, and blood lactate concentration. They maintained equal total physical work for both training regimens, but were unable to show any difference in these variables during a standard exercise test. Comparison of the training schedules of humans and horses has shown that horses were not able to perform as much intense work

as humans without risk of injury (Gabel *et al.* 1983). Daniels and Scardina (1984) listed some typical interval training routines for track athletes, for example 40 x 400-m bouts with a different bout started every 3 min. Interval training for swimmers has involved some of the most intensive conditioning because of the lower risk of injury in water. Some individual swim workouts include: 1) 100-m sprints repeated 100 times with 15-sec rest periods, and 2) 365-m repeated 40 times with 20 second recovery periods (total time of 200 min).

The premise of IT is that it replicates, during the training process, conditions even more extreme than those encountered during competition, thereby causing adaptation to these physiological extremes through increased buffering capacity. This premise has been extrapolated to the race horse. There is a large accumulation of hydrogen ions (H^+) and lactate during intense exercise resulting in reduced skeletal muscle contractility (Donaldson, 1983). Accumulation of H^+ in the muscle cells is believed to be at least partially responsible for fatigue in Thoroughbred racehorses (Terjung *et al.* 1985). Investigators have shown that maximal effort in horses resulted in increased muscle and blood lactate levels (Bayly *et al.* 1987a; Lindholm & Saltin, 1974b; Nimmo & Snow, 1983; Snow *et al.* 1985). Plasma lactate levels rose as the intensity of the exercise increased due to the recruitment of glycolytic, fast twitch fibers. Subjects with a greater anaerobic potential generally had a

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higher capacity for lactate and H⁺ production during intense exercise (Edwards, 1983). Cellular adaptation to increased H⁺ concentration in exercising muscle has been achieved through IT. Fox *et al.* (1987) demonstrated an increased buffering capacity of muscle cells in Standardbreds following an 18-week period of interval training when compared to a control, untrained group. This is consistent with human studies that have shown increased buffering capacity following interval training (Kallings & Persson, 1983; Sharp *et al.* 1986).

Several investigators have shown that IT improved endurance and work efficiency. Thornton *et al.* (1983) demonstrated a reduced plasma lactate level during a submaximal SET following 5 weeks of interval training when compared with a similar pre-training SET. Webb *et al.* (1988) improved the work capacity in Quarter horses following a 6-week period of interval training. The increased work capacity was evident by reduced heart rate and plasma lactate concentrations during an exhaustive cutting performance test when pre-training results were compared to post-training values. Sexton *et al.* (1987) combined interval training and long slow distance work in a 25 week training period for Quarter horses. They demonstrated an increase in stroke volume, arterial partial pressure of carbon dioxide, and running speed at a HR of 140 beats/min and a decrease in body temperature and blood lactate accumulation during exercise. Essen-Gustavsson *et al.* (1989) used an interval

training regimen to show the changes in skeletal muscle following training. They concluded that an intensive training program rapidly increased the oxidative capacity and capillary density in an actively working muscle. Wilson *et al.* (1987) examined the changes in muscle composition and metabolism during an 8 week interval training period. After interval training, the speed producing a plasma lactate of 4 mmol/l was significantly increased, and the number of type IIa fibers was also significantly increased. Gottlieb *et al.* (1989) used a draft-loaded interval training program to demonstrate an increase in cross-sectional area and percentage of type IIa fibers, a decrease in percentage of type IIb fibers, and an increase in citrate synthase activity in the gluteus medius muscle. Less glycogen was utilized in the gluteus muscle and less blood lactate accumulated during a submaximal SET following exercise when compared with a pre-training test.

One of the objectives of this project was to assess any benefits of interval training over conventional training methods. There have been only a few other studies in horses comparing interval training with conventional training methods. Lindholm and Saltin (1974b) concluded that interval training was superior to continuous training in Standardbreds because fast-twitch fibers with low oxidative capacity, which constitute 43% of the cross-sectional area of horse muscle, were only utilized when horses were trained at near-maximal speed. Without near-maximal intensity training, this fiber type remains

inactive except during racing. Gabel *et al.* (1983) were unable to show any differences in race times between horses trained by conventional and interval training methods but stated (without proof) that IT could improve the race times of horses. Rodiek *et al.* (1987) compared the effects of interval and conventional training on heart rate, cardiac output, and blood lactate concentration, but were unable to show any difference in these variables during a standard exercise test. The study of Rodiek *et al.* (1987) maintained equal work loads for the interval and conventional trained horses. Since IT enables the athlete to perform more intense exercise during a training session, that study did not maximize the advantages of IT because the capacity for work is increased by the intermittent rest periods.

Interval training has been at least partially responsible for the improvements in human world records for running and swimming events during the past 40 years (Fox & Mathews, 1974). Record times for Thoroughbred racing have not decreased as rapidly and reluctance to change conventional training methods has been credited as a partial reason (Leerhsen, 1979).

The benefits of IT in racehorses have not been scientifically documented, however the purported advantages have been stated anecdotally (Ivers, 1983). It has been claimed that IT develops more durable animals and delays the onset of fatigue during racing (Ivers, 1983). Daniels and Scardina (1984)

stated that the main reason IT is extensively used in humans is because the athlete is able to maintain near-maximal effort over a longer distance.

Interval training is more labor intensive than conventional methods of training. Some horsemen believe that the musculoskeletal system of horses cannot endure the intense work of interval training. These reasons along with the lack of scientific proof of the benefits of IT in race horses and man's natural tendency to resist change are why IT has not been more accepted on the racetrack. It was hoped that this project would increase the scientific knowledge about interval training and provide horsemen more information about the putative benefits of interval training.

2. Effect of treadmill training on fitness

a. Introduction

The high-speed treadmill has greatly advanced the study of equine exercise physiology. It has been used to study muscle adaptation to training. Hodgson *et al.* (1985) conducted a 7 week treadmill training program in Standardbreds and demonstrated an increase in oxidative capacity of type IIb fibers as indicated by a rise in citrate synthase and 3-hydroxy-acyl CoA dehydrogenase.

Glycogen utilization was 36% lower during a post-training SET than in a pre-training SET. Essen-Gustavsson *et al.* (1989) showed an increase in muscle oxidative capacity and capillary density following 5 weeks of intensive treadmill training in Standardbreds.



b. Muscular and hematological responses

The treadmill has been used to study muscle response to exercise performed at different speeds and resulting changes in hematologic values. Weber *et al.* (1987) used the treadmill to show that lactate turnover rate was directly proportional to cardiac output and plasma lactate concentration. That study also concluded that oxidation of plasma lactate was only a minor source of fuel in horses. From muscle biopsy samples obtained during standardized treadmill exercise, Valberg *et al.* (1989) showed a lowered glycogen and creatine phosphate content, unaltered adenosine triphosphate concentration, and increased intramuscular lactate and glucose-6-phosphate content during exercise. The post-exercise blood chemistry values showed rapid and significant changes from measurements taken while running to illustrate the importance of obtaining blood samples during exercise. Essen-Gustavsson and Valberg (1987) examined the blood ammonia and lactate concentrations during treadmill exercise at different intensities. They determined that increased

blood ammonia and lactate were only seen following higher work intensities. A similar study by Miller and Lawrence (1987) resulted in similar conclusions. Harris *et al.* (1987) investigated the changes in water content and blood density from rest, during exercise, and through recovery. They measured a significant increase in PCV and total protein, and a significant decrease in volume water content following maximal exercise. Because the metabolic changes in muscle and blood occur rapidly during and following exercise, it has been difficult to measure those changes in horses during track exercise. Treadmill testing has allowed investigators to obtain blood and muscle samples during high-intensity exercise, thereby increasing the knowledge of the metabolism in these tissues of horses during exercise.

c. Cardiorespiratory responses

There have been a number of treadmill training studies of cardiovascular and respiratory responses during exercise. Varying results have been obtained. Thornton *et al.* (1983) demonstrated no difference in arterial or mixed venous blood gas tensions, pH, blood lactate, cardiac output, stroke volume, or oxygen uptake during a pre- and post-training SET performed on the treadmill. The lack of a training effect may have been due to the short duration (5 weeks) of the training program. Rodiek *et al.* (1987) were unable to show a difference in

the effect of 10 weeks of treadmill interval and conventional training on heart rate, cardiac output, or blood lactate production. Again, the lack of a training effect may have been because of the brief training period. Bayly *et al.* (1983a) also compared the effects of different treadmill training methods on heart rate, cardiac output, stroke volume, arteriovenous oxygen difference, systemic blood pressure, and venous lactic acid concentration during a SET. They demonstrated no differences in these variables due to training schedules. However, a training effect was shown by a decrease in heart rate and cardiac output when compared to pre-training values. Evans and Rose (1987) measured the difference between VO_{2max} during a treadmill SET before and following a 12-week treadmill training program in Thoroughbred horses. They demonstrated a significant increase in VO_{2max} due to training and concluded that VO_{2max} cannot be accurately predicted from cardiorespiratory indices measured during sub-maximal exercise. In a later study, Evans and Rose (1988a) measured oxygen uptake, heart rate, and arteriovenous oxygen content difference before and after 7 weeks of treadmill conditioning. Since there was no change in maximal heart rate, they concluded that VO_{2max} increased following training due to an increase in maximal cardiac output and stroke volume, and a decrease in arteriovenous oxygen difference. Rose *et al.* (1988) used an inclined treadmill to measure VO_{2max} , maximal oxygen deficit, and oxygen debt in Thoroughbred racehorses. They concluded that 1) restoration of creatine phosphate concentration in muscle was a minor component of the

oxygen debt, 2) not all lactate was cleared by terminal oxidation, 3) some lactate may be cleared via gluconeogenesis and glycogen synthesis, and 4) a large amount of lactate was still present when oxygen uptake approached resting levels. The treadmill has allowed investigators to directly measure oxygen uptake and heart rate of horses during exercise, which has decreased the need to extrapolate data from human exercise studies. This has also revealed differences between human and equine responses to exercise.

d. Gait analysis

The treadmill has been used to study gait abnormalities. Fredricson *et al.* (1983) compared the gait of a Standardbred trotter on the track to the gait of the same horse on the treadmill. They determined that the stride on the treadmill was shorter, but this did not invalidate the treadmill as a research tool. Drevemo *et al.* (1987) utilized the treadmill for a study of early development of gait asymmetries in trotting Standardbreds. They observed a difference between left and right diagonal lengths in trained 18-month old horses. Hjerten and Drevemo (1987) used the treadmill to analyze forces and moments in the extremities of horses during the stance phase of the trot.

e. Training vehicle

The treadmill has been used for conventional training in Thoroughbreds (Evans & Rose, 1988a; Weber *et al.* 1987) and interval training in Standardbreds and Quarter horses (Essen-Gustavsson *et al.* 1989; Sexton *et al.* 1987). Weber *et al.* (1987) used 2 conventional training protocols on the treadmill in a lactate kinetics study in Thoroughbreds. Evans and Rose (1988a) used conventional treadmill training in a study of cardiovascular and respiratory responses to sub-maximal exercise training in Thoroughbreds. Essen-Gustavsson *et al.* (1989) used interval training on the treadmill in a study of muscular adaptation of Standardbreds during intensive training and detraining. Sexton *et al.* (1987) used interval training techniques on the treadmill in a study of cardiopulmonary and metabolic responses to exercise in Quarter horses. Investigators in these studies were able to show a training effect on the measured variables indicating that the treadmill can be used as a training tool for horses.

The treadmill has been used to assess the degree of fitness in horses. Most of the methods used to assess fitness have been based on the relationship between HR or blood lactate concentration and work load (Persson, 1983). Persson and Ullberg (1974) used the relationship of HR to treadmill speed to calculate the velocity at which a horse runs at a steady-state HR of 200

beats/min (V_{200}). The V_{200} is an interpolated or extrapolated velocity calculated from a heart rate vs. speed regression obtained during a standard exercise test. They declared that the V_{200} was a highly reproducible value when strict standardization of test procedure was observed. Fitness has also been assessed by determining the interpolated or extrapolated speed at which a horse attains a steady-state blood lactate concentration of 4 mmol/l (V_{La4}). The V_{La4} has been used as an indicator of endurance performance (Persson, 1983). The V_{La4} is considered to be the approximate anaerobic threshold and is also referred to as the onset of blood lactate accumulation (OBLA). These indexes have also been used in human exercise studies to assess state of fitness. Heck *et al.* (1985) showed that 800-m runners attained a 4 mmol/l blood lactate concentration at a slower speed than 1500-m runners while the OBLA for marathon runners occurred at a higher speed than any group. Svendenhag and Sjodin (1984) demonstrated that elite middle-distance runners (400-800 m) reached OBLA at a slower running speed than elite distance runners (1500-10,000 m). The treadmill allows investigators to determine these values for fitness assessment.

f. Pharmacology

Performance-altering effects of drugs have been investigated using the equine treadmill. Rose and Evans (1987) studied the sympathomimetic effects of clenbuterol on exercise-induced bronchoconstriction. During a SET, they found no differences in cardiovascular or respiratory measurements and concluded that clenbuterol had no major effect on cardiorespiratory functions in healthy racehorses. Manohar (1987) studied the effects of furosemide on systemic circulation in ponies. During severe exercise, furosemide-treated animals demonstrated a decrease in mean aortic pressure but no change in HR, oxygen uptake, cardiac output, or muscle blood flow. Kallings and Persson (1983) studied the effects of theophylline and non-steroidal anti-inflammatory drugs (NSAIDs) on HR and blood lactate concentration following a SET. Theophylline and flunixin caused a significant decrease in the treadmill velocity at which a HR of 200 beats/min was attained when compared to control values. The accumulation of plasma lactate was significantly lower following administration of each NSAID (flunixin, meclofenamic acid, nefopam) when compared with control values.

3. Effect of competition on performance

The effect of competition has been extensively studied in humans, but has not been examined in racehorses. Due to the paucity of information on the effects of competition in horses, most of the research cited in this section was taken from human studies. The degree to which human competitive behavior can be applied to the horse is uncertain, but horses do display some competitive behavioral patterns common to humans (e.g. territoriality, social hierarchy, aggression). Competition has been described as a stress-producing experience since it results in increased heart, metabolic, and respiratory rates, and sweating (Cratty, 1984). In humans, competition has improved and worsened performance depending on the attitude and competitive experience of the participant (Michaels *et al.* 1982).

The first sports psychology research in humans studied the effects of competition on performance and appeared in 1898 (Triplett, 1898). In that initial study, Triplett reviewed bicycle race records and found that racers were faster when riding with or against other cyclists than when riding alone. He suggested that the presence of other riders acted as a stimulant for performance and hence proposed the theory of dynamogenesis. He later tested the theory in the laboratory by having forty children wind fishing reels against the clock either alone or in competition with another child. On average, the

children in competition performed better than the children alone. However, on examination of individual results, he found that only half of the children performed better in competition, one-fourth performed more poorly in competition, and one-fourth showed no difference in performance.

Cratty (1967) proposed that motivation necessary for competition depended on 4 factors: 1) inherent competitiveness, 2) specific competitive situation, 3) nature of the activity, and 4) traits of the competitor.

1) Inherent competitiveness. Buhler (1927) found that if an object was placed between two infants, they would both try to grab it. It was proposed that competition was an inborn trait. Greenberg (1932) used block stacking in a study of children to investigate the age of onset of competition. He concluded that competitive behavior was absent before the age of 3. However, by the age of 5 competitive behavior was present in 70% of the children examined. In the study by Triplett cited earlier (Triplett, 1898), further study revealed that, on average, younger children performed better alone, while older children performed better in competition. The age at which the effect of competition changed from a negative to a positive stimulus was between 10-12 years.

2) Specific competitive situation. Atkinson (1964) concluded that motivation in any competitive situation partially depended on the individual's assessment

of his/her chance of being successful. Kohler (1927) determined that the abilities of competitors should be similar, otherwise the less able competitor lost the urge to try and the better competitor assumed the role of instructor for the less able and also ceased to try. In the work by Buhler (1927) cited earlier, infants that varied by more than 2.5 months of age would not compete for the same object. Von Bracken (1934) found that identical twins, whose abilities were equal, were more competitive than fraternal twins, whose aptitudes were more varied.

3) Nature of the activity. The motivation for competition varied with the type of activity being performed (Coakley, 1978). In a study by Wankel (1972), participants performed an unfamiliar balancing task. Noncompetitive groups performed better in the early stages of learning the task, while competitive groups performed better in the later stages of learning. He concluded that high-ability groups performed best under competitive conditions, while low-ability groups performed poorly when subjected to competitive stress. In a study by Hrycaiko (1978), young boys were required to roll a ball up an incline with a degree of accuracy. The boys were unfamiliar with the task and performed better in the absence of competition than with competition. Lowe (1973) showed that the achievement of Little League baseball players was diminished under highly competitive circumstances. Zajonc (1965) demonstrated that unfamiliar tasks were accomplished more proficiently

without competition than during competition. These studies suggest that age and athletic ability help determine an athlete's capacity to handle competitive situations. Competition was likely to enhance activities that required simple skills or endurance, while performance of complicated skills was inhibited by competition (Coakley, 1978). This idea was supported by a treadmill running study by Higgs (1972). He showed that treadmill running time was longer when the participants ran against competition than when they ran alone.

4) Traits of the competitor. Individuals who consider themselves highly competitive were motivated by competition. However, non-competitive people lost their motivation when confronted with a competitive situation (Coakley, 1978). Cratty (1967) concluded that highly anxious individuals with a high need for achievement performed motor tasks poorly during competitive situations. Conversely, low anxiety individuals with a high need for achievement performed motor tasks well during competition. Similarly, Ogilvie and Tutko (1971) examined personality profiles of 15,000 athletes and concluded that proficiency in sport competition was correlated with a personality characterized by a high need for achievement and low levels of anxiety. Prior to and during competition, an athlete undergoes many physiological changes due to the anxiety of competition (Passer, 1984). An excessively high level of anxiety can result in first a rise and then a lessening of muscle tension resulting in a feeling of weakness (Cratty, 1984).

Other studies have simulated the mental effects of competition with the presence of an audience during the physical event. Worringham and Messick (1983) showed that solitary joggers increased their running speed when passing by a woman seated on a park bench for the purpose of the experiment. This was true for male and female joggers. Michaels *et al.* (1982) simulated the mental stress encountered in athletic situations by having an audience observe the performance. They showed that good pool players were 9% more accurate in their shots when watched by four observers while below-average players dropped 9% in accuracy under the same conditions. Martens (1975) determined that motivation depended on the level of the competitor's tension arousal. When the level of tension arousal was too low or too high, motivation was low. Motivation was highest when the tension arousal of the participants was within a certain limit.

As stated earlier, it is not known to what extent horse behavior in response to competitive situations mimics human behavior. No equine studies have examined this phenomenon. It is hoped that this project will increase the knowledge of the effects of competition in horses and stimulate an interest for more research in this area.

4. Effect of added dietary fat on performance

The traditional diet for horses has been forages and grain. Since grain is a more concentrated source of digestible energy, the percentage of grain in the diet has been increased with work intensity to supply the required extra energy (Duren *et al.* 1987). However, adding excessive amounts of grain to the diet can cause possible complications including colic, azoturia, and laminitis (Hambelton *et al.* 1980).

a. Digestibility of dietary fat by horses

The absence of a gall bladder does not prevent the digestion and utilization of fat by horses (Hintz *et al.* 1978). Investigators have shown that horses can efficiently digest diets that have varied amounts of digestible energy supplied by fat, thus reducing the bulk and the amount of grain in the feed. Webb *et al.* (1987) showed no difference in energy digestibilities between a control (64.9%) and 10% fat-added diet (60.4%). Bowman *et al.* (1977) fed horses a diet containing 20% of the digestible energy in the form of fat and determined that it was 90% digestible. These studies showed that horses are able to digest and utilize fat as an energy source, thereby reducing the likelihood of complications from excessive amounts of grain in the diets of exercising horses. These studies also indicated that palatability of fat was no problem in horses.

b. Effects of dietary fat on muscle glycogen

Maughan and Poole (1981) showed that anaerobic work capacity in man is limited by the quantity of stored glycogen in the muscle cells. Studies have shown that muscle glycogen storage in horses was increased by adding fat to the diet. Meyers *et al.* (1989) fed diets containing 0, 5, and 10% animal rendered fat for a 3-week period. Mean muscle glycogen increased as the percent of dietary fat increased (16.97, 19.60, and 25.77 mg/g wet tissue, respectively) and was significantly greater for the 10% fat-added diet when compared with the 0% fat diet. In the study by Hambelton *et al.* (1980), muscle glycogen storage significantly increased by 46% as the percentage of fat in the diet increased from 0 to 12%. However, muscle glycogen content following the feeding of a diet containing 16% digestible energy in the form of fat decreased (when compared with the 10% fat diet) and was not significantly different from the muscle glycogen content following a 4% fat-added diet. Oldham *et al.* (1989) determined that the muscle glycogen content following a 10% fat-added diet was significantly greater than the muscle glycogen content following a control diet (22.89 and 15.77 mg/g wet tissue, respectively). They also showed an increased utilization of muscle glycogen during a SET following the 10% fat-added diet when compared to the control diet (13.08 and 6.99 mg/g wet tissue, respectively). These studies indicate there is an optimal level of dietary fat (between 10-16%) that results in increased glycogen storage.

Muscle glycogen content decreases when dietary fat percentage is outside of this range.

It has been hypothesized that fat utilization was increased during rest and sub-maximal activity during the 3-week feeding period prior to the race resulting in spared glycogen utilization and increased muscle glycogen storage (Meyers *et al.* 1989). The other studies cited seem to support that conclusion.

c. Effects of dietary fat on performance

Oldham *et al.* (1989) showed a reduction in average sprint time for 6 horses during a near-maximal SET (at a heart rate of 210 beats/min) from 59 sec following a control diet to 56.7 sec following a three-week period on a 10% fat diet. This difference was not statistically significant due to considerable variation in sprint times. Webb *et al.* (1987) showed that the percentage of hindquarter turns executed during a cutting performance test in Quarter horses were significantly higher ($P < 0.01$) when the horses were fed a 10% fat diet than when the horses were fed a control diet. They concluded that the horses worked harder during the cutting performance test when they were fed the 10% fat-added diet. These studies are not conclusive on the ergogenic effect of added dietary fat. This dissertation will test the ergogenic effects of

dietary fat in a competitive running situation. It is hoped that a better understanding of fat metabolism in exercising horses will be achieved.

d. Hematologic effects of dietary fat

Equine nutrition and exercise studies have shown differences in hematologic variables following fat supplementation of the diet.

Glucose. Hambelton *et al.* (1980) demonstrated that plasma glucose decreased during exercise in untrained horses, but increased in trained animals. They also showed that horses on a 16% fat-added diet maintained a higher blood glucose following exercise than horses fed a 4% fat-added diet. Webb *et al.* (1987) demonstrated that blood glucose was maintained at a higher level during a 10-min gallop at 172 m/min on an inclined treadmill in horses fed a 10% fat diet when compared to horses fed a control diet. Hintz *et al.* (1978) showed that the decrease in blood glucose from resting levels during a 37 mile ride at 6 miles/hour was significantly less in horses fed an 8% fat diet compared to the same horses fed a 0% fat-added diet. Pagan *et al.* (1987) confirmed an increase in blood glucose following exercise in horses fed 5, 10, and 20% fat compared to horses fed 0% dietary fat. Duren *et al.* (1987) also showed that blood glucose remained elevated during and following exercise in

horses fed 5-20% fat when compared with the same horses receiving 0% dietary fat.

The reason for the glucose sparing effects is that energy for exercising muscles can be derived from intramuscular stores of glycogen and triglycerides and extra-muscular sources of free fatty acids as well as blood glucose (Miller *et al.* 1985). Mole *et al.* (1971) confirmed that increased utilization of fat as an energy source spared glycogen and blood glucose. These studies consistently showed that horses on fat-supplemented diets maintained higher blood glucose levels during exercise than horses on a control diet. This indicated that fat was utilized for energy to a greater degree in horses fed a fat-supplemented diet when compared to a control diet.

Lipids.

1) Glycerol. Several investigators have shown that an increase in plasma lipids has been observed after exercise. Hickson *et al.* (1977) demonstrated that plasma glycerol increased with exercise in rats for control and fat supplemented groups, but increased at a significantly faster rate for the fat-supplemented group. In an equine nutrition study, Duren *et al.* (1987) also demonstrated the rise in plasma glycerol following exercise. They showed no difference in plasma glycerol between the control and fat-supplemented diets for the pre-exercise and immediate post-exercise samples, but did find a

significant increase in plasma glycerol at 15 min post-exercise for horses fed a diet containing 10% digestible energy in the form of fat when compared to the control diet.

2) Triglycerides. In a human study of volleyball players, Bonetti *et al.* (1988) illustrated that exercise caused a significant rise in plasma triglyceride levels. Duren *et al.* (1987) found lowered plasma triglycerides in horses on a fat-supplemented diet before, during, and following a SET than horses on a control diet.

3) Non-esterified fatty acids. Meyers *et al.* (1989) showed a significant decrease in non-esterified fatty acids in horses fed a 10% fat-added diet during and following a 20-min sub-maximal SET when compared to horses on a 0% fat diet performing the same SET. Hintz *et al.* (1978) showed no difference in free fatty acids in horses during a 37 mile endurance event. McMiken (1983) determined that free fatty acids were the main fuel in equine muscle during rest and light exercise.

These studies show that glycerol is consistently elevated during and following exercise. Glycerol is not metabolized to a large extent during exercise (Newsholme & Leech, 1983), so the rising plasma glycerol following exercise may have indicated a larger degree of lipolysis in the fat fed groups. There is

insufficient data on the metabolism of triglycerides in horses during exercise. The results of the one equine study cited were opposite the results seen in human studies. More investigation of the metabolism of triglycerides by horses is needed. The data on free fatty acid metabolism is also inconsistent and sparse, so more investigation of free fatty acid metabolism in horses is also needed.

Lactate. Duren *et al.* (1987) showed no difference between plasma lactate levels of horses fed a control and 10% fat-added diet before, during, and following a standard exercise test. Pagan *et al.* (1987) showed no difference between the control and fat-added diets in plasma lactate following a high-speed or a long-slow exercise test. Hintz *et al.* (1978) and Meyers *et al.* (1989) also showed no differences in plasma lactate for horses following exercise due to diet. However, Oldham *et al.* (1989) concluded that lactate tended to be higher when the horses were fed the high-fat diet but only with a significance level of $P=0.13$. The study by Oldham *et al.* (1989) had a considerable amount of variation in other results, as well as the lactate measurements, and was considered less credible than the other studies that consistently showed no effect of diet on plasma lactate concentration. This would be expected since lactate does not result from fat metabolism.

5. Effect of induced alkalosis on performance

Investigators have shown that the accumulation of hydrogen ions (H^+) during intense, short duration events reduces glycolysis through the inhibition of the enzyme phosphofructokinase (Hood *et al.* 1988; Wilkie, 1986). Since acidosis results in the disruption of muscle contraction, induced alkalosis could delay the onset of fatigue.

a. Effect on peak buffering capacity

Sodium bicarbonate has been used to induce alkalosis before exercise. Time of pre-exercise administration of $NaHCO_3$ seems to be critical if an ergogenic effect is to be seen. In exercise studies of human athletes Costill *et al.* (1984), Gao *et al.* (1988), and Katz *et al.* (1984) gave $NaHCO_3$ 45-60 min before exercise. Brien and McKenzie (1989), and Wilkes *et al.* (1983) administered treatment over a 2 hr period and exercise commenced after the last capsule was ingested. In equine studies, Hank *et al.* (1985) exercised horses 1.5-2 hr following $NaHCO_3$ administration, Kelso *et al.* (1987) administered treatment 1 hr before exercise, and Lawrence *et al.* (1987) treated horses 1.5-2.5 hr before exercise. These studies made no comment concerning time of maximal buffering effects of $NaHCO_3$. To realize maximal effect from induced alkalosis,

NaHCO₃ should be administered at the proper time before exercise to insure maximal buffering during the exercise event.

b. Effect of H⁺ accumulation

Several human studies have reported that lactic acid, which is the major source of H⁺ in working muscles (Beaver *et al.* 1986), accumulates in body fluids following high-intensity exercise (Hermansen, 1969; Sahlin, 1978). Beaver *et al.* (1986) showed that at the pH of normal muscle tissue, lactic acid is almost completely dissociated into lactate (La⁻) and hydrogen ions (H⁺). Hermansen (1981) proved that the accumulation of H⁺, which is associated with a lowered pH in the working muscle, resulted in a reduced activity of the glycolytic enzymes phosphorylase and phosphofructokinase. Donaldson *et al.* (1978) concluded that the accumulation of H⁺ in muscle tissue resulted in a reduced contraction process. Beaver *et al.* (1986) demonstrated that hydrogen ions are buffered almost entirely by the bicarbonate (HCO₃⁻) buffer system forming H₂CO₃ which breaks down to CO₂ and H₂O. These studies agreed that H⁺ accumulation was a major cause of fatigue during short-duration exercise.

c. Effect on fatigue

Studies in human athletes have attempted to delay the onset of fatigue using sodium bicarbonate (NaHCO_3) as a buffering agent for the accumulating H^+ with differing results depending on the duration and intensity of the exercise. Iwaoka *et al.* (1989) showed that time to fatigue on a cycle ergometer at 95% maximal O_2 uptake was significantly increased from 2.00 ± 0.44 to 2.98 ± 0.64 minutes following pre-exercise treatment with 0.2 g/kg body weight (BW) of NaHCO_3 . Wilkes *et al.* (1983) showed that performance significantly improved in an 800-m run by decreasing mean run times from 2:05.8 seconds for the control run to 2:02.9 seconds when runners were pre-treated with 0.3 g/kg BW NaHCO_3 . Jones *et al.* (1977), Pate *et al.* (1985), and Rupp *et al.* (1983) have also shown beneficial effects from pre-exercise treatment with NaHCO_3 in fatiguing exercises lasting from 2-9 minutes. However, in single exercise events lasting less than 2 minutes no benefits of pre-treatment with NaHCO_3 have been consistently demonstrated. Katz *et al.* (1984) showed that exercise performance in events lasting 45-100 seconds was not influenced by the intake of 0.2 g/kg BW NaHCO_3 in human athletes. Studies by Kindermann *et al.* (1977), McCartney *et al.* (1983), Parry-Billings and MacLaren (1986), and Sjöholm (1986) also failed to show any benefit from pre-treatment with NaHCO_3 in events lasting less than 2 minutes.

In investigations using horses, the fatigue delaying effects of pre-exercise treatment with NaHCO_3 have been equivocal. Kelso *et al.* (1987) claimed a significant improvement in 1600-m run times in Thoroughbreds from 114.0 ± 2.2 sec following placebo treatment to 111.3 ± 1.2 sec following treatment with 0.4 g/kg NaHCO_3 , but only with a significance of $P < 0.10$. Lawrence *et al.* (1987b) failed to show any improvement in endurance from pre-exercise treatment with 0.3 g/kg BW NaHCO_3 in Quarter horses exercised to fatigue on a treadmill for a 20 minute period. These studies were different in intensity and duration, and neither study was able to demonstrate a definite ergogenic effect due to NaHCO_3 .

Human studies have shown that exercise events consisting of repeated bouts responded to pre-exercise treatment with NaHCO_3 even though the individual bouts lasted less than 2 minutes. Improvement in performance was not seen at the beginning of the exercise but in later bouts. Costill *et al.* (1984) showed that time to fatigue in humans during repeated bouts of cycling was increased from 113.5 ± 12.4 seconds for the fifth bout following the control treatment to 160.8 ± 19.1 seconds for the fifth bout following pre-exercise treatment with 0.2 g/kg BW NaHCO_3 . Gao *et al.* (1988) showed a significant improvement of swim times in humans during the fourth and fifth heats of a five-heat (91.4 m/heat) trial following pre-trial treatment with 2.9 mmol/kg NaHCO_3 . Lawrence *et al.* (1987a) showed a significant ($P < 0.05$) reduction of race time in Standardbreds

competing in a mile run from 2:15.4 following a placebo treatment to 2:14.3 following 0.3 g/kg NaHCO₃ treatment. The horses in that study were warmed-up "according to trainer preference" which included at least 1 warm-up heat (frequently more than 1 heat) of 1600 meters in 2:30 performed 45-60 min before race time (A.B. Lawrence, personal communication, 1990).

c. Effect on plasma lactate

In a study in humans, Wilkes *et al.* (1983) showed a significant increase in plasma lactate following an 800-m run in subjects pre-treated with NaHCO₃ when compared to a control run. Gao *et al.* (1988) demonstrated a significant increase in plasma lactate for swimmers pre-treated with NaHCO₃ following a swim SET when compared to a control swim SET. In an equine study, Lawrence *et al.* (1987b) determined a significant elevation in peak lactate from 10.9 ± 1.0 mmol/l for the control group to 15.2 ± 1.5 mmol/l for the NaHCO₃ pre-treatment group when horses were worked to fatigue at a speed of 4.5 m/sec on an 11% grade. The human and equine studies consistently showed an increased lactate accumulation following pre-treatment with NaHCO₃. This would be expected since the amount of lactate produced is dependent on the continuation of glycolysis in the exercising muscles. Since glycolysis stops when H⁺ concentration increases sufficiently to inhibit the glycolytic enzymes,

the increased exogenous HCO_3^- allows glycolysis (and lactate production) to proceed for a longer period.

d. Effect on pH

Lawrence *et al.* (1987b) showed that the effects of NaHCO_3 on pH were significantly elevated prior to exercise, diminished during exercise, and were again evident during recovery ($P < 0.05$). Kelso *et al.* (1987) showed a significantly higher blood pH in the pre-run sample following NaHCO_3 treatment, but the post-run samples, while higher, were not significantly elevated. Mainwood and Worsley-Brown (1975) proposed that the higher muscle-blood pH gradient due to the increased extracellular HCO_3^- facilitated the efflux of H^+ and lactate from the muscle cell. The increased pH would be expected because of the increased buffering of the extracellular H^+ . It would also be expected that the decreased concentration of H^+ before exercise would slow the onset of fatigue. However, the time delay for the efflux of intracellular H^+ and equilibration of intra- and extra-cellular H^+ may diminish the ergogenic effect of NaHCO_3 in short duration events.

Part I. Physiologic Correlates of Performance

Section A
Physiologic Measurements Obtained during
Racetrack Performances

Objectives:

1. To correlate maximal heart rate and peak and total plasma lactate to actual racetrack performance (i.e. run times) of 25 Thoroughbred racehorses during 1200-, 1600-, and 2000-m runs.
2. To determine the effect of run distance on maximal heart rate, peak and total plasma lactate.
3. To determine if the relative performance of horses was consistent over a range of distances.

Materials and Methods:

1. Horses

Twenty-five healthy Thoroughbred racehorses (11 geldings and 14 mares), ranging in age from 3 to 8 years and weighing between 385 and 486 kilograms, were trained for 5 months before completing the runs described in this section.

Training was accomplished on a high-speed equine treadmill⁴ using interval training techniques described in Section D. After the 5 month conditioning period, the horses were stabled at a training track for assessment of running ability.

2. Runs

The protocol for this section was for each horse to complete 10 runs in the following order: 2 solo runs at 1200 m, 2 solo runs at 1600 m, 2 solo runs at 2000 m, 2 runs in competition at 1200 m, and 2 runs in competition at 1600 m. An average time for each pair of runs was determined and correlated with the measured variables (maximal heart rate, peak and total plasma lactate). The same jockey rode all horses during the solo runs. A second jockey was used for the competitive runs which consisted of two equally paired horses. The solo runs were completed first and those run times were used to pair equal ability horses for the competitive runs. Since each horse ran each distance in competition two times, the jockeys alternated horses to minimize any effect on performance due to the rider. Both jockeys were licensed professional riders with racing experience. The jockeys were instructed to ride the horses in the fastest possible time.

⁴ Mustang 2200, Kagra International Inc., Fahrwangen, Switz.

The runs were performed on an 800-m training track with poles dividing it into 200-m segments. Times for each 200-m segment and total run times were recorded to the nearest 0.01 second.

The 10 runs were performed over a 10 week period. The experimental design was for 1 run each week, but some of the runs were rescheduled because of rain and muddy track conditions. The average period between runs was 7 days with a range of 5-14 days.

3. Plasma Lactate

Blood samples for plasma lactate analysis were collected before the runs and at 2, 4, 8, and 16 min post-run by serial sticks into vacutainer tubes⁵ containing potassium oxalate as an anticoagulant and sodium fluoride to prevent the further production of lactate. The tubes were immediately centrifuged and the plasma separated for storage. The plasma samples were frozen at -20 degrees centigrade and later analyzed using a lactate analyzer.⁶ Peak lactate (PLac) was the highest single plasma lactate value recorded, and

⁵ Becton Dickinson, No. 6445, Rutherford, NJ

⁶ Model 23L, YSI Inc., Yellow Springs, Ohio

total lactate (TLac) was the sum of the 5 plasma lactate values obtained before and following the run.

4. Heart rate

Heart rate was recorded at 5 second intervals during each run by an on-board heart rate computer.⁷ The device consisted of a receiver, which was worn on the wrist of the jockey, and a transmitter which was attached to the saddle pad. The transmitter was connected to two electrodes placed on shaved areas of the anterior part of the sternum and left side of the neck anterior to the supraspinatus muscle.

5. Statistics

The average times for each pair of runs were correlated with peak lactate (PLac), total lactate (TLac), and maximal heart rate (MaxHR) using Pearson's correlation. Duncan's multiple range test was used to compare PLac, TLac, and MaxHR from the 1200-, 1600-, and 2000-m runs to assess any differences

⁷ VMAX, Equine Racing Systems, Vevay, Ind.

in these variables due to run distance. All calculations were performed using SAS GLM procedure (SAS Institute Inc, 1985).

Consistency of relative running ability at the different distances was determined by correlating the mean run times for each pair of runs (two 1200-m solo runs, etc.) with all other mean run times. This was done to determine if horses excelled at only one distance or if the relative order of run times was consistent over a range of distances.

6. Diet

The diet consisted of 3.2 kg of a complete, pelleted ration⁸ (14% crude protein) comprised mainly of corn and soy beans with minerals and vitamins added and 3.2 kg Bermuda hay. Feed and hay were fed twice a day. Water was provided ad libitum.

⁸ Purina Pride #200, Purina Mills Inc., St. Louis, MO.

Results:

1. Correlation

Solo, competitive (comp), and average (solo + competitive) mean run times for the 3 distances are listed in Table 1. All horses did not complete all 10 runs because of injuries and a delayed training start for a few horses. Pearson's correlation coefficients, probabilities, and number of observations for the correlations between average run times and PLac, TLac, and MaxHR for each pair of runs are listed in Table 2. There was a significant negative correlation of 1200- and 1600-m run times with PLac and TLac. The correlation between 2000-m run times and PLac ($r=-0.415$, $P<0.0614$) and TLac ($r=-0.399$, $P<0.0732$) approached significance. The strongest correlation was between 1200-m solo run times and PLac ($r=-0.711$, $P<0.0001$) and TLac (-0.726 , $P<0.0001$). There was no significant correlation between MaxHR and run time at any distance.

A comparison among means within a given variable for each run is shown in Table 3. Peak and total plasma lactate values changed similarly across all runs. Peak and total lactate concentrations were lower following the 1200-m solo run than any other run. Competition produced significantly greater lactate concentrations for the 1200-m but not for the 1600-m runs. Lactate

Table 1--Solo, competitive, and average (solo + competitive) times (mean \pm SEM) for 1200-1600- and 2000-m runs.

No	1200 m Runs			1600 m runs			2000 m
	Solo	Comp.	Avg.	Solo	Comp.	Avg.	Solo
1	87.94	88.89	88.42	119.01	126.13	122.57	155.22
2	82.62	83.61	83.11	114.53	114.52	114.53	147.43
3	85.72	DNR	85.72	118.75	DNR	118.75	154.63
4	84.63	DNR	84.63	119.48	DNR	119.48	155.90
5	87.76	88.30	88.03	123.15	124.26	123.70	160.00
6	94.98	DNR	94.98	129.79	DNR	129.79	DNR
7	87.04	DNR	87.04	122.08	DNR	122.08	160.67
8	112.9	DNR	112.9	DNR	DNR	DNR	DNR
9	86.66	88.97	87.81	121.63	121.68	121.65	158.24
10	86.86	87.83	87.34	121.41	125.31	123.36	156.81
11	82.91	83.67	83.29	116.10	117.74	116.92	149.47
12	84.91	85.25	85.08	117.02	117.28	117.15	150.16
13	88.58	87.17	87.87	122.84	124.30	123.57	159.42
14	87.95	89.75	88.85	124.33	124.89	124.61	162.31
15	84.82	83.92	84.37	114.89	117.64	116.26	148.84
16	89.82	92.35	91.08	136.85	131.88	133.54	171.90
17	82.57	83.44	83.00	116.42	116.27	116.34	148.24
18	90.11	87.81	88.96	122.10	123.94	123.02	155.15
19	86.49	DNR	86.49	DNR	DNR	DNR	DNR
20	84.56	84.98	84.77	115.65	118.14	116.89	150.91
21	86.84	89.59	88.21	120.68	123.72	122.20	155.63
22	82.70	84.86	83.78	114.79	116.59	115.69	147.84
23	85.14	86.05	85.59	117.12	DNR	117.12	152.91
24	89.64	89.91	89.78	126.14	122.44	124.29	161.61
25	81.53	DNR	81.53	113.64	DNR	113.64	DNR
X	87.67	87.02	87.25	120.67	121.57	120.74	155.39
SE	1.19	0.61	1.18	1.11	1.08	1.00	1.29

No = Horse number

DNR = Did not run at that distance

Table 2--Pearson's correlation coefficients, probabilities and number (n) of runs used for the correlations between run times and PLac, TLac, and MaxHR.

Run times for:	1200-m Solo Runs (Run 1)	1200-m Comp Runs (Run 2)	1600-m Solo Runs (Run 3)	1600-m Comp Runs (Run 4)	2000-m Solo Runs (Run 5)
PLac R1 (n=25)	-0.711 0.0001				
Plac R2 (n=18)		-0.638 0.0044			
PLac R3 (n=23)			-0.696 0.0002		
PLac R4 (n=17)				-0.606 0.0099	
PLac R5 (n=21)					-0.415 0.0614
TLac R1 (n=25)	-0.726 0.0001				
TLac R2 (n=18)		-0.595 0.0092			
TLac R3 (n=23)			-0.715 0.0001		
TLac R4 (n=17)				-0.599 0.0111	
TLac R5 (n=21)					-0.399 0.0732
MaxHR R1 (n=25)	0.1905 0.3618				
MaxHR R2 (n=18)		0.3016 0.2240			
MaxHR R3 (n=23)			0.1535 0.4843		
MaxHR R4 (n=17)				-0.1655 0.5255	
MaxHR R5 (n=21)					-0.1391 0.5476
PLac = Peak Lactate			TLac = Total Lactate		
MaxHR = Maximal Heart Rate			R1-5 = Runs 1-5		

Table 3--Duncan's multiple range test for PLac, TLac, and MaxHR for each pair of runs.

Run	Variable		
	Mean PLac mmol/l	Mean TLac mmol/l	Mean MaxHR b/min
1200-m Solo	25.7 ^c	97.5 ^c	205 ^b
1200-m Comp	30.7 ^a	117.7 ^a	202 ^b
1600-m Solo	27.3 ^{bc}	103.5 ^{bc}	205 ^b
1600-m Comp	28.4 ^b	109.1 ^b	198 ^c
2000-m Solo	27.0 ^{bc}	104.1 ^{bc}	210 ^a

Within a column means with the same letter are not significantly different (P<0.05)

PLac = peak plasma lactate
 TLac = total plasma lactate
 MaxHR = maximal heart rate
 b/min = beats per minute
 mmol/l = millimoles per liter

concentrations for the solo runs did not differ significantly as a function of distance. The plasma lactate curves following the runs are illustrated in Figure 1. The mean PLac occurred at 8 min post-exercise for the 10 runs. There was a strong correlation ($r=0.98$, $P<0.0001$) between PLac and TLac for all the runs.

The maximal heart rate observed following the 2000-m solo run was significantly greater than that observed after the other runs. Competition was associated with a lower HR at the 1600-m distance but not for the 1200-m run when compared with corresponding solo runs.

Pearson's correlation coefficients, probabilities, and number of observations for the correlations between mean run time of one pair of runs with mean run times of the other pairs of runs are listed in Table 4. Times for all runs were highly correlated with a probability of $P<0.0001$. The lowest correlation was between mean run times for the 1200- and 2000-m solo runs ($r=0.8248$). The highest correlation was between mean run times for the 1600- and 2000-m solo runs ($r=0.9608$). The correlation between mean run times for the 2 competitive runs (R2 and R4) was higher ($r=0.9112$) than the correlations of mean run times for those competitive runs with the solo runs at the same distance (R1 vs R2, $r=0.8700$; R3 vs R4, $r=0.8762$).

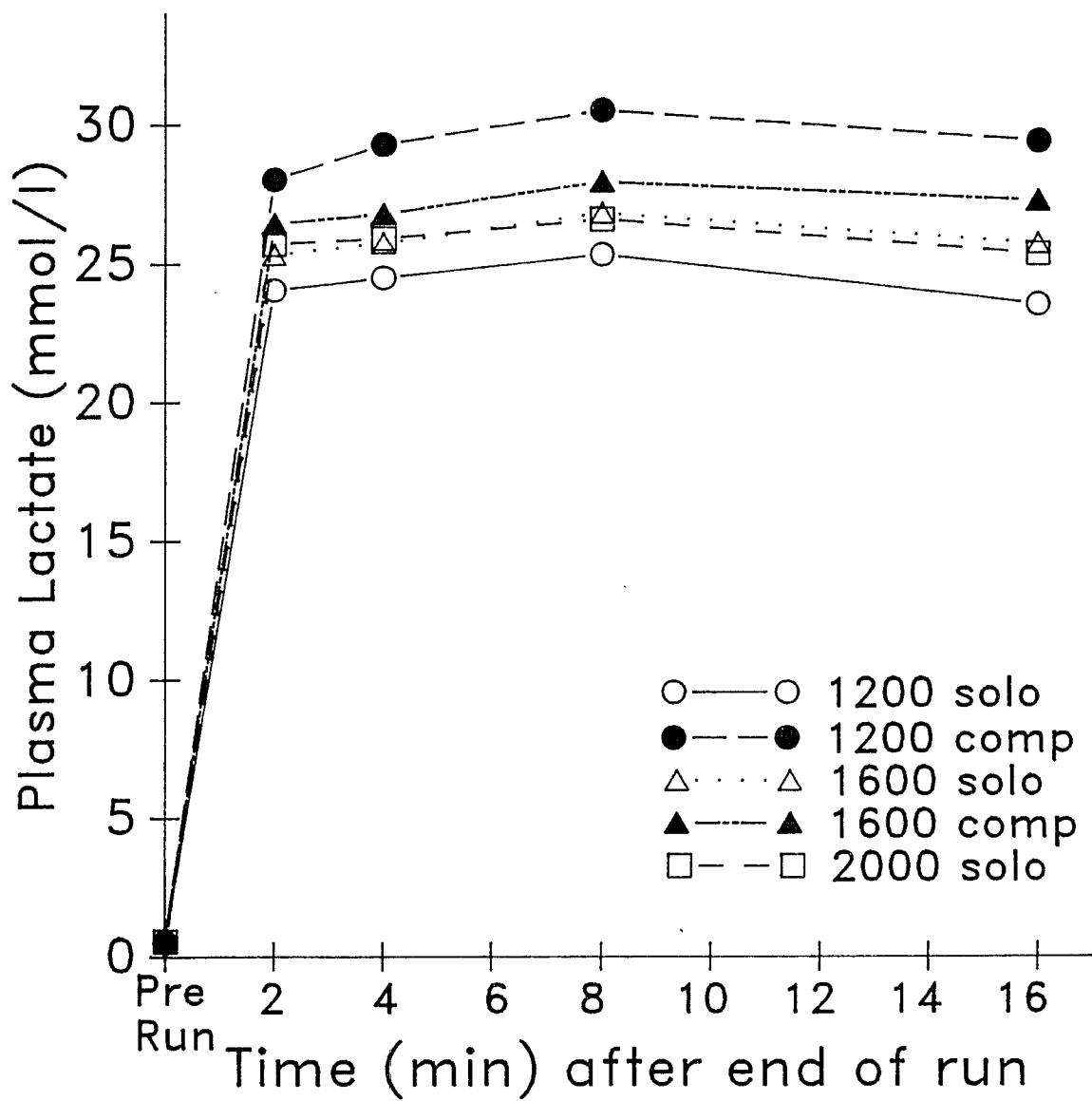


Figure 1--Plasma lactate measured before and at 2, 4, 8, and 16 minutes following each pair of runs.

Table 4--Pearson's correlation coefficients (r), probabilities (P), and number (n) of observations for the correlations of running ability (as measured by run times) between all runs.

Run times for:	1200-m Solo Runs (R1)	1200-m Comp Runs (R2)	1600-m Solo Runs (R3)	1600-m Comp Runs (R4)	2000-m Solo Runs (R5)
R1	r= 1.0000 P= 0.0 n= 25				
R2	r= 0.8700 P= 0.0001 n= 18	1.0000 0.0 18			
R3	r= 0.8341 P= 0.0001 n= 23	0.8808 0.0001 18	1.0000 0.0 23		
R4	r= 0.8706 P= 0.0001 n= 17	0.9112 0.0001 17	0.8762 0.0001 17	1.0000 0.0 17	
R5	r= 0.8248 P= 0.0001 n= 21	0.9225 0.0001 18	0.9608 0.0001 21	0.9082 0.0001 17	1.0000 0.0 21

2. Horses

Only 17 of the original 25 horses completed all 10 runs. Five horses received injuries during the 5-month testing phase and required a recovery period preventing those horses from completing all the runs. Three horses were 2 months late starting the racing schedule; 4 of the runs for those horses were canceled to allow for a timely completion of the study.

Discussion:

1. Plasma Lactate

A negative correlation between plasma lactate and run performance (Table 2) has been demonstrated in exercise studies in both humans and horses. Studies of human athletes have shown that elite shorter-distance runners attain higher peak lactate concentrations following high-intensity exercise than less successful runners. Taunton *et al.* (1981) showed that middle-distance runners attained a significantly higher plasma lactate concentration than long-distance runners following an exhaustive treadmill test for VO_{2max} . Other studies showed that sprinters and middle-distance runners accumulated plasma lactate significantly faster than longer-distance runners during maximal

treadmill exercise (Costill *et al.* 1973; Komi *et al.* 1977). In a study using elite Swedish runners who competed at distances of 400-m to a marathon, the peak lactate production (range 5.7-18.4 mmol/l) of the shorter-distance runners was significantly greater than for the longer-distance runners (Svedenhag & Sjodin, 1984). Because elite runners compete in events in which they are successful, these studies show that faster middle-distance runners are able to produce more lactate at lower work rates than runners that are less successful at middle-distance events. Since human middle-distance events (400-800 m) are of similar duration (about 50-120 sec) to most Thoroughbred races (1000-2000 m), it is assumed that the metabolism of successful human middle-distance runners is similar to the metabolism of successful Thoroughbred racehorses. Therefore, it is concluded that high, rapid lactate production is advantageous for a Thoroughbred racehorse to compete successfully.

Bayly *et al.* (1987a) showed that during a run of 1000 m, there was a trend toward higher lactates in the faster horses. In a study of 16 horses, Saibene *et al.* (1985) showed that higher plasma lactate levels (measured 5 min after end of run) were seen in the faster horses raced over distances of 200, 300, and 400 meters. They also showed a significant correlation between lactate production and speed ($r=0.86$). Since speed and race time are inversely proportional, this positive correlation is consistent with the negative correlation of run time with plasma lactate concentration found in this project.

Peak plasma lactate is a measure of anaerobic capacity (Snow *et al.* 1983a). Saltin and Gollnick (1983) demonstrated that the mechanism for the rapid production of lactate was via anaerobic glycolysis which has been associated with fast-twitch muscle metabolism. The lower correlation of PLac and TLac to race time for the 2000-m runs and higher correlation for the shorter runs is consistent with the belief that the contribution of aerobic metabolism to total energy turnover increases with duration of the exercise event (Cerretelli *et al.* 1980). In the 1200- and 1600-m runs, anaerobic metabolism contributed a higher percentage of the total energy output than in the 2000-m runs. Therefore, the correlation between plasma lactate concentration and run time was higher for the shorter runs than for the 2000-m runs.

The occurrence of peak plasma lactate at 8 min post-run (Figure 1) is consistent with other equine exercise studies. Bayly *et al.* (1987a) showed a peak lactate at 5-10 min following maximal-effort runs of 1000 m. In the training study described in Section C, blood lactate concentration was measured at 0, 5, 10, and 20 min after exercise and showed a peak lactate at 5 min post-run.

Investigators have shown that stressful situations, due to unfamiliar circumstances or disturbing conditions, increase the amount of lactate production during an exercise event. The higher Plac and TLac for the 1200-m

competitive runs (Table 3) were consistent with the findings of Svedenhag and Sjodin (1984) who showed that plasma lactate was significantly higher ($P<0.001$) in elite middle- and long-distance runners following competition than in a solo exhaustive treadmill test. Bayly *et al.* (1987a) showed that there was a tendency toward higher lactate concentrations when horses were run on a soft or muddy track, trained from a starting-gate, or trained with another horse. All of these situations could increase the degree of stress the horse perceives, resulting in increased lactate production.

It is not apparent why PLac and TLac were significantly less than the competitive runs for the 1200-m solo runs but not for the 1600- and 2000-m solo runs (Table 3). Bayly *et al.* (1987a) showed that, although plasma lactate concentration increased with the distance run up to 1000 m, there was no difference in plasma lactate concentration for distances between 1000-1700 m.

The moderate correlation of plasma lactate with run times (Table 2) provides a possible method to estimate the running ability of unraced horses. The fair to moderate correlation between PLac and run time ($r=-0.415$ to $r=-0.711$) demonstrates that relative running abilities of a large group of horses could be determined with a fair degree of accuracy. Persson (1983) demonstrated a high correlation ($r=0.82$) in Standardbreds between speed and blood lactate concentration in a 1000 m run at 10 m/s. Bayly *et al.* (1987a) stated "the

measurement of plasma lactate may have considerable potential with respect to assessing a horse's athletic condition and/or ability."

2. Heart Rate

Heart rate has been measured in many sub-maximal exercise studies but rarely in maximal-intensity exercise events which are necessary for determining MaxHR. Von Englehardt (1977) showed that speed was linearly related to HR in horses up to a HR of about 210 when a departure from linearity occurs. The lack of any significant correlation of maximum HR to run times in this investigation (Table 2) is consistent with other equine exercise studies. Kubo *et al.* (1984) found no significant correlation between maximal HR and run times during runs of 200, 400, and 1000 m and an exhaustive run.

The significantly higher MaxHR for the 2000-m runs (Table 3) was supportive of the findings of Kubo *et al.* (1983). In that study, horses ran 200, 1000, 1600 m and an exhaustive run lasting about 5 minutes. The maximal HR attained during the exhaustive run was about 5% (about 10 beats/min) higher than the maximal HR attained during the other 4 runs. They concluded that maximal HR may not be reached in maximal-intensity events lasting less than 2 min.

It is not apparent why the MaxHR for the 1600-m competitive runs were significantly lower than the other runs.

3. Racing abilities at different distances

The high correlation coefficients ($r=0.8248$ to $r=0.9608$) between the run times for the different distances and conditions (Table 4) demonstrated that horses performed similarly at different distances when compared with the other horses in the project ($P<0.0001$). Racehorses have been generally classified as "stayers", horses that can compete at 1-1.5 miles, or "sprinters", horses that excel at shorter distances. Since most of the "classic races" are one mile or longer, horses are bred and trained to be stayers. Horses that are not successful at the longer distances are raced at shorter distances. Horses that are successful at the shorter distances, become sprinters by default.

This finding could be useful in performance prediction of young horses. The faster horses at shorter distances are probably the faster horses at longer distances as well. There is less chance of injury in shorter distance runs, therefore running speed could be more safely assessed at shorter distances.

It was concluded that plasma lactate correlated well with running performance, however maximal HR did not correlate with performance. It was also concluded that relative performance was consistent at shorter and longer distances.

Section B

Physiologic Measurements Obtained During Treadmill Standard

Exercise Tests: Comparison with Racetrack Performance

Objectives:

1. To correlate actual running performance of 25 Thoroughbred racehorses over 1200-, 1600-, and 2000-m distances to physiologic variables obtained in standard exercise tests on the high-speed treadmill.
2. To determine treadmill tests best correlating with performance.

Materials and Methods:

1. Horses

Prior to the treadmill standard exercise tests, 25 healthy Thoroughbred racehorses (11 geldings and 14 mares ranging in age from 3 to 9 years and weighing between 385 and 486 kilograms) were trained for 5 months on a high-speed equine treadmill⁹ using interval training techniques described in Section

⁹ Mustang 2200, Kagra International Inc., Fahrwangen, Switz.

D. The protocol for measuring the running abilities of the horses on the racetrack was described in Section A. Briefly, average times for solo and competitive runs for each horse were determined at distances of 1200, 1600, and 2000 m (Table 1).

2. Heart rate score standard exercise test (HR-SET)

Since this section describes different treadmill standard exercise tests (SETs), a prefix has been added to distinguish the various tests. Two standard exercise tests for the evaluation of heart rate score vs. work load were performed. Heart rate score is the total number of heart beats recorded during a standard exercise test (HR-SET). The first HR-SET was run in unfit horses after only 4 weeks of minimal treadmill training (Pre HR-SET). The second test was performed after 20 weeks of intense interval training on the treadmill at which time the horses were considered fit enough to endure the stress of racing (Post HR-SET). Both HR-SETs were considered sub-maximal in effort since heart rates did not reach maximal frequency. The tests consisted of a 1-min walk at 1.5 m/s, a 1-min trot at 3.5 m/s, a 5-min gallop at 9.0 m/s, and a 2-min warm-down at 3.5 m/s. The treadmill was not inclined during the HR-SETs.

Heart rates were measured by an on-board heart rate (HR) monitor¹⁰ supplied with the treadmill. The two electrodes for the HR monitor were attached to a girth strap placed over the withers dorsally and just caudal to the axillae ventrally. The electrodes were positioned on the sternum and left side of the thorax midway between the sternum and withers. The hair at these two areas was clipped and a generous amount of electrode gel was applied to the electrode pads to insure proper transmission of the cardiac signal. The HR was transmitted via telemetry from a transmitter in the girth strap to two receivers mounted on the treadmill panel.

The HR was automatically relayed every 5 sec to the display panel above the treadmill and to a computer for storage. During each HR-SET, 12 HR recordings were obtained during the walk (1 min), 12 during the trot (1 min), 60 during the gallop (5 min), and 24 during the warm-down (2 min). The mean number of heart beats for both HR-SETs was measured by computing the area under the HR curve (AUC), which was the mean number of beats during that phase of the exercise. Areas under the curve for the total HR-SETs (Pre 0-9 min, Post 0-9 min), the gallop and warm-down phases (Pre 2-9 min, Post 2-9 min), and the gallop-only phase (Pre 2-7 min, Post 2-7 min) were calculated by integrating the HR recordings for the respective periods. The AUC for each stage was correlated with average run times for each distance to determine the

¹⁰ Hippocard, Bioengineering, Zurich, Switz.

accuracy of AUC as an indicator of performance. Paired t-tests were used to compare the heart rate scores for the Pre and Post HR-SETs for each stage of the test.

3. V_{200} standard exercise test (V_{200} -SET)

The velocity at which a horse can run at a steady-state HR of 200 beats/min is defined as the V_{200} . This variable is calculated from a heart rate vs. speed regression line obtained during a standard exercise test (V_{200} -SET). The HR vs. speed regression is illustrated in Figure 2 which is a regression obtained from a V_{200} -SET performed by one horse. The V_{200} is the speed (on the X-axis) that corresponds to the intersection of the regression line and 200 beats/min (on the Y-axis). The regression line has a slope that is a measure of the change in heart rate per unit change in velocity. The regression line also has a Y-intercept where the line crosses the Y axis. The Y-intercept is the calculated HR at zero speed. The Y-intercept and V_{200} represent the two extremes (however, V_{200} is less than the maximum) of the HR vs. speed regression line while the slope represents the mean change in HR per change in speed for the total curve.

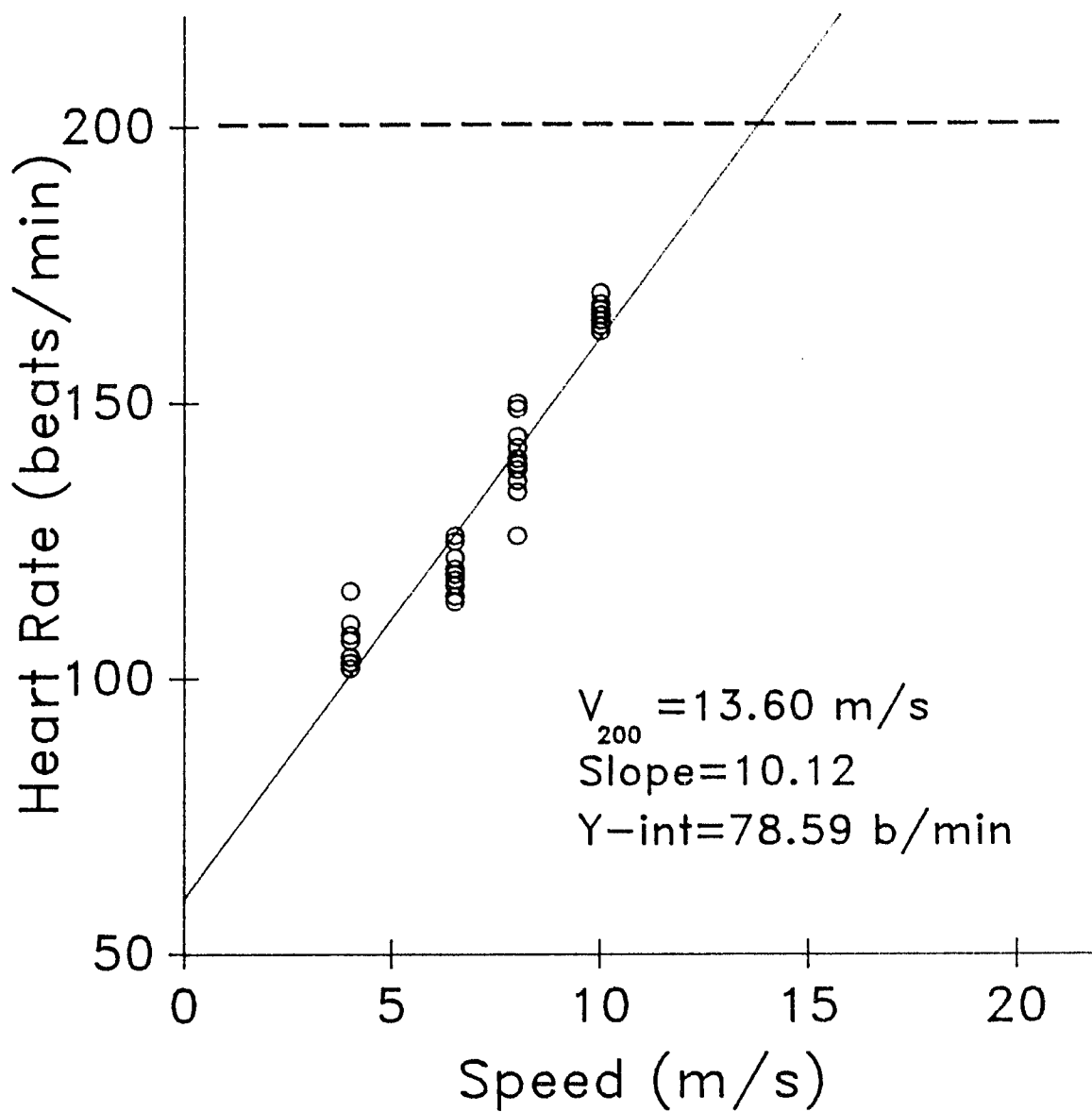


Figure 2--The regression of heart rate plotted against speed showing V_{200} , Y-intercept, and slope of that regression.

The V_{200} -SET was preceded by a 5-min warm-up consisting of a 2 min trot at 4.0 m/s, a 1 min gallop at 6.5 m/s, and a 2 min trot at 4.0 m/s. The V_{200} -SET was an incremental step test performed at 0% treadmill inclination and consisted of a trot at 4.0 m/s for 1 min, a gallop at 6.0 m/s for 1 min, 8.0 m/s for 1 min, and 10 m/s for 1 min. Heart rates were measured at the four different speeds using the HR monitor supplied with the treadmill. The regression line and correlation coefficient (r-value) for HR vs. velocity were determined for each test with slope, y-intercept, and V_{200} calculated from that regression equation. Each horse was tested 6-12 times, and tests with an r-value less than 0.90 were discarded. All horses completed at least four V_{200} -SETs with an r-value greater than 0.90. Coefficients of variation were determined for V_{200} , slope, and Y-intercept to evaluate the reproducibility of each variable.

4. Blood-gas standard exercise test (BG-SET)

A standard exercise test was performed for the analysis of blood gas values including minimum arterial blood pH (Min pH), and minimum arterial oxygen partial pressure (Min PO_2). Minimum arterial oxygen saturation (Min O_2 Sat) was calculated from the measured blood-gas values by the blood-gas

analyzer.¹¹ Since reduced pH during exercise is a cause of fatigue (Wilkie, 1986), Min pH was measured to see if faster horses were able to continue running at lower pH levels than slower horses. Min PO₂ and Min O₂ Sat were measured to determine if reduced blood oxygen concentration or hemoglobin desaturation correlates with performance.

The blood-gas standard exercise test (BG-SET) was a near-maximal effort test in which HRs exceeded 200 beats/min. The BG-SET was preceded by a 5 min warm-up at 0% treadmill inclination (trot at 3.5 m/s for 2 min, gallop at 6.5 m/s for 1 min, and trot at 3.5 m/s for 2 min). The BG-SET consisted of a 1 min trot at 4.0 m/s, a gallop at 6.5 m/s for 1 min, 8.0 m/s for 1 min, 10.0 m/s for 1 min, and 12.0 m/s for 1 min, followed by a 3 min warm-down trot at 4.0 m/s. The BG-SET was performed at a 7% treadmill inclination. Blood samples were drawn at rest, after the warm-up, then at 1 min intervals for 8 minutes through the end of the BG-SET for a total of 10 samples. Samples were obtained from an 18 gauge over-the-needle catheter¹² in the transverse facial artery. The blood gas samples were collected anaerobically in heparinized syringes for blood gas analysis.

¹¹ Model 158, Corning Medical, Medfield, MA

¹² Travenol Laboratories, Inc., Deerfield, IL

The blood gas samples were corrected for temperature effects by continually measuring increasing arterial blood temperature with a 0.029 inch diameter copper/constantan thermocouple.¹³ The probe was inserted into the arterial catheter through a 3-way Y-connector.¹⁴

Blood samples were also collected in vacutainer tubes¹⁵ containing potassium oxalate and sodium fluoride for plasma lactate analysis. Plasma lactate variables measured included peak and total plasma lactate, plasma lactate at the end (8th min) of the BG-SET (Lac 8), and the interpolated or extrapolated time at which the horse attained a 4 mmol/l plasma lactate concentration (T_{La4}). Different plasma lactate variables were measured to determine which variables best correlated with running performance. Peak lactate was the highest single plasma lactate value recorded, and total lactate was the sum of the 9 plasma lactate values obtained during the BG-SET. The Lac 8 measurement was obtained at the end of the highest treadmill speed (12 m/s), and T_{La4} was a calculated measurement of the time of onset of blood lactate accumulation.

¹³ Type PT, Sentsortek Inc., Clifton, NJ

¹⁴ Sorenson Research, Salt Lake City, UT

¹⁵ Becton Dickinson, No. 6445, Rutherford, NJ

Similarly, blood was collected in heparinized microhematocrit tubes¹⁶ for determination of maximum packed cell volume (MaxPCV). The Max PCV was measured because it is an indication of the maximal oxygen carrying capacity of the blood.

The vacutainer tubes for lactate analyses were immediately centrifuged and the plasma separated for storage. The plasma samples were frozen at -20 degrees centigrade and later analyzed using a lactate analyzer.¹⁷

5. Onset of blood lactate standard exercise test (OBLA-SET)

The onset of blood lactate accumulation (OBLA) is the exercise intensity beyond which the blood lactate cannot attain steady-state but rather continues to increase because lactate production exceeds its clearance. The OBLA is considered to be the anaerobic threshold. The OBLA can be described in reference to the velocity (V_{La4}) (Yoshida *et al.* 1987) or work rate (W_{La4}) (Evans & Rose, 1987; Jacobs *et al.* 1985) at which blood lactate reaches a steady-state concentration of 4 mmol/l, which is the numerical value of the approximate anaerobic threshold. In this experiment, plasma lactate concentration was

¹⁶ Chase Instruments Corp., No. 501, Glen Falls, NY

¹⁷ Model 23L, YSI Inc., Yellow Springs, OH

plotted against treadmill speed. The treadmill velocity eliciting a plasma lactate concentration of 4 mmol/l (V_{La4}) was interpolated or extrapolated as described by Jacobs *et al.* (1985). Lactate concentration was also plotted against work rate. The work rate producing a plasma lactate concentration of 4 mmol/l (W_{La4}) was also interpolated or extrapolated in the same manner.

Work rate (watts) was calculated by the following formula (Evans & Rose, 1987):

$$WorkRate = \frac{V * BW * Sin\alpha}{6.12}$$

where V=velocity of the treadmill, BW=Body weight of the horse, Sin alpha=Sine of the angle of treadmill inclination, and 6.12=conversion factor.

The V_{La4} and W_{La4} were measured during a sub-maximal standard exercise test (OBLA-SET). The OBLA-SET was preceded by a 5 min warm-up at 0% treadmill inclination consisting of a trot at 3.5 m/s for 2 min, gallop at 6.5 m/s for 1 min, and trot at 3.5 m/s for 2 min. The OBLA-SET consisted of a 1 min trot at 4.0 m/s, a gallop at 6.5 m/s for 2 min, 8.0 m/s for 2 min, 9.0 m/s for 2 min, and 10.0 m/s for 2 min followed by a 3 min warm-down at 4.0 m/s. The OBLA-SET was performed at a 4% treadmill inclination.

Blood samples were taken from an indwelling 16 gauge jugular catheter¹⁸ before the start of the OBLA-SET, at 1 min intervals through the end of the OBLA-SET and at the end of the warm-down for a total of 11 samples into vacutainer tubes¹⁹ containing potassium oxalate and sodium fluoride for analysis of peak and total plasma lactate. Miller-Graber *et al.* (1989) showed that plasma lactate values obtained from the jugular vein did not differ significantly from mixed venous blood obtained from the pulmonary artery. The vacutainer tubes were immediately centrifuged and the plasma separated for storage. The plasma samples were frozen at -20 degrees centigrade and later analyzed using a lactate analyzer.²⁰ Coefficients of variation were determined for V_{La4} and W_{La4} to evaluate the reproducibility of each variable.

6. Maximal oxygen uptake standard exercise test (VO_{2max} -SET)

Maximal oxygen uptake (VO_{2max}) is the highest amount of oxygen that can be utilized during maximal-effort exercise. This variable was determined during a maximal-effort standard exercise test (VO_{2max} -SET). Prior to the VO_{2max} -SET, the horses were warmed-up with a 3 min trot at 3.5 m/s. The VO_{2max} -SET was

¹⁸ Travenol Laboratories, Inc., Deerfield, IL

¹⁹ Becton Dickinson, No. 6445, Rutherford, NJ

²⁰ Model 23L, YSI Inc., Yellow Springs, OH

an incremental step test in which exercise intensity was increased every 1.5 min by increasing treadmill speed or degree of inclination. The VO_{2max} -SET was started at 0% inclination and consisted of a trot at 4.0 m/s for 1.5 min, a gallop at 6.5 m/s for 1.5 min, and a gallop at 9.0 m/s for 1.5 min. During the next 3 increments, the speed was maintained at 9.0 m/s and the treadmill inclination was increased every 1.5 min to 3.67%, 7.33%, and 11%. The final increment was performed at 12.0 m/s and an 11% inclination. Oxygen uptake was recorded during the last 10 sec of each increment or at the time of fatigue during the final work load. Fatigue was defined as the point at which the horse could no longer maintain treadmill speed even with encouragement from the handlers. The horses required 2-5 sessions on the treadmill wearing a gas collection mask to properly adapt to the gas collection system before VO_{2max} measurements were obtained.

Maximal oxygen uptake was determined with a flow-through breathing system similar to that described by Seeherman *et al.* (1981). The system consisted of a face mask that covered approximately one third of the head (rostral portion). It was attached to a 7 meter section of 12.7 cm diameter flexible hose coated with latex to prevent leakage of air. The hose connected to a 2 meter section of iron pipe in which an annubar flow meter²¹ was mounted. A nylon mesh screen was inserted between the hose and iron pipe to prevent any debris from

²¹ Model DCR-15, Dieterich Standard, Boulder, CO

the horse entering the iron pipe. Another 1.5 meter section of the 12.7 cm hose connected the iron pipe to the blower²² and motor²³ capable of generating a air flow through the system in excess of 100 l/sec.

Air samples were obtained from a port at the connection of the hose and blower before the flow-through air reached the blower. Air samples were analyzed with an oxygen analyzer.²⁴ Precision was improved with a voltmeter²⁵ that read voltage output from the oxygen analyzer to 3 decimal places. The linearity of the oxygen analyzer was determined by measuring the voltage associated with oxygen concentrations varying from 20.9% (room air) to 19.9%. The linearity is illustrated in Figure 3 where the fractional concentration of oxygen was plotted on the x-axis and the corresponding voltage output was plotted on the y-axis. A nitrogen flowmeter²⁶ and purified grade (UHP) nitrogen were used to calibrate the system and calculate the oxygen uptake (liters/min) at the end of each VO_{2max} -SET. Calibration and calculations were done according to the method described by Fedak *et al.* (1981), who developed a one-step dilution technique for calibrating a flow-through oxygen uptake measuring system. Use of a measured nitrogen flow

²² Model 4C329, Dayton Mfg. Co., Chicago, IL

²³ Model 3N668A, Dayton Electric Mfg. Co., Chicago, IL

²⁴ Model OM-11, Beckman Instruments, Fullerton, CA

²⁵ Escort EDM-1341, Cole Palmer, Chicago, IL

²⁶ Model FL-1501A, Omega Engineering Co., Stamford, CT

eliminated the requirement to calibrate the oxygen analyzer or measure the air flow past the animal. The average nitrogen flow for a 100% flow reading on the N₂ flowmeter was determined with a spirometer.²⁷ The N₂ flowmeter was set on 100% and time to fill a known volume of the spirometer was measured. The average flow rate corresponding to the 100% flow setting was calculated.

Temperature of the nitrogen gas exiting the connecting hose from the storage tank was measured. Since gas volume is dependent on temperature of the gas, it was necessary to correct for the difference between temperature of the nitrogen gas and expired air from the exercising horses. The following conversion was used for that calculation:

$$\dot{V}_{N_2} \frac{273 + T_{gas}}{273 + T_{horse}} = \dot{V}_{N_2 corrected}$$

where V_{N_2} =uncorrected volume of nitrogen corresponding to a 100% flow setting, T_{gas} =temperature (degrees C) of nitrogen exiting storage tank, and T_{horse} =temperature (degrees C) of exhaled air from horse during maximal exercise.

Maximal oxygen uptake is expressed at standard temperature and pressure, dry (STPD). Since oxygen uptake was measured at body temperature and

²⁷ Warren E. Collins Inc., P-1700, Braintree, MA

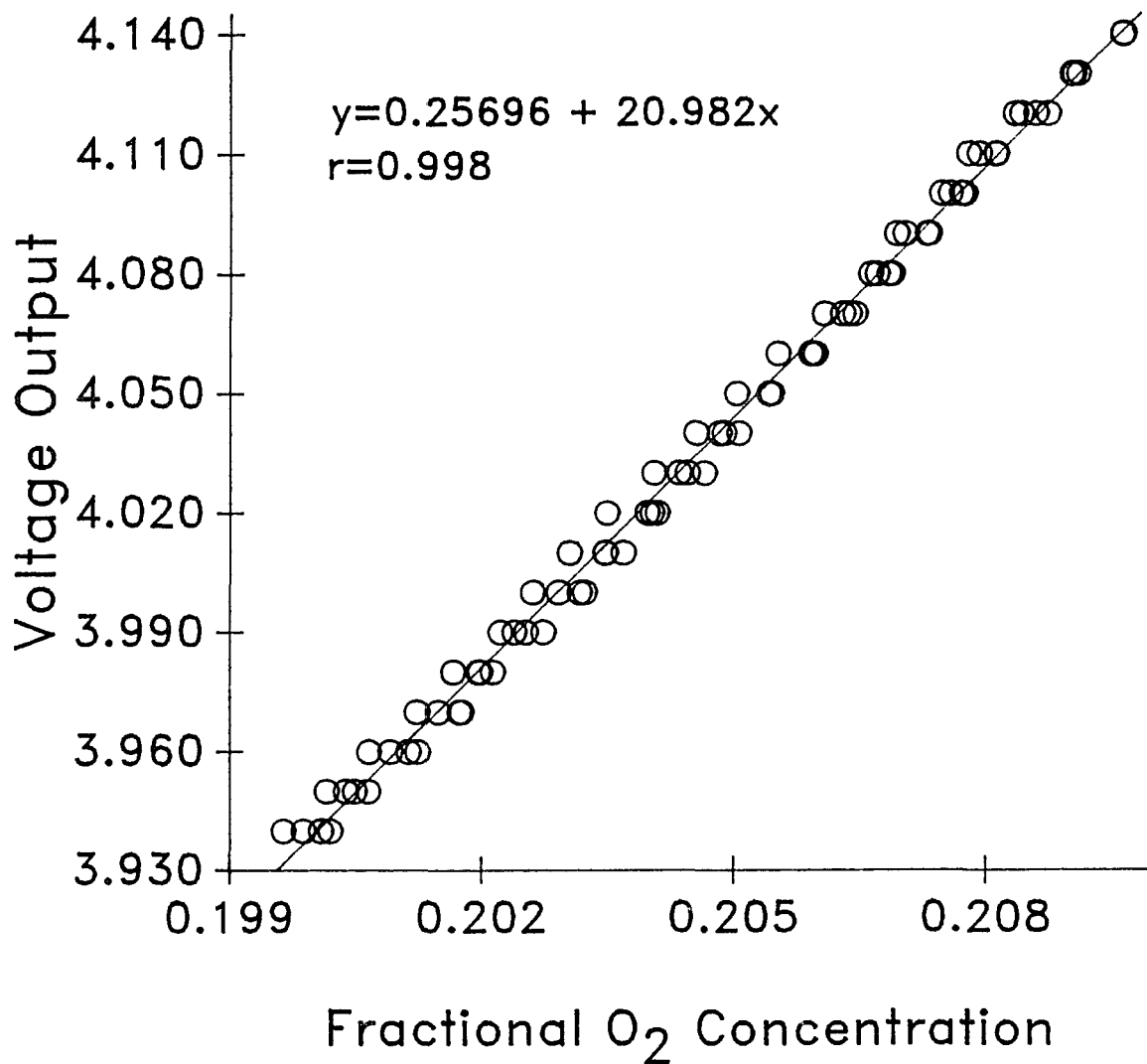


Figure 3--Linearity of oxygen analyzer is illustrated with the fractional concentration of oxygen plotted on the x-axis and the corresponding voltage output plotted on the y-axis.

pressure, dry (BTPD), it was necessary to convert the oxygen uptake from BTPD to STPD. The following formula was used for the calculation:

$$\dot{V}_{O_2(\text{at BTPD})} \frac{273}{273 + T_{\text{horse}}} = \dot{V}_{O_2(\text{at STPD})}$$

where $\dot{V}_{O_2(\text{at BTPD})}$ =oxygen uptake measured at BTPD according to the method of Fedak *et al.* (1981) and T_{horse} =temperature (degrees C) of exhaled air from horse during maximal exercise.

This formula expressed oxygen uptake (at STPD) in liters per minute (l/min). To express oxygen uptake relative to body weight, this number was divided by the weight of the horse (in kilograms) and multiplied by 1000. This final product expressed oxygen uptake in milliliters per kilogram-minute (ml/kg*min). Coefficients of variation (CV) were determined for $\dot{V}_{O_{2\text{max}}}$ to evaluate the reproducibility of this variable.

The level of significance for the correlations between variables and run times was considered to be $P < 0.05$. The terms 'tended to be significant' or 'approached significance' were used to refer to correlations with a P value of 0.05-0.10. All P values were listed in the correlation tables to show any correlation trend with different run distances.

Results:**1. Heart rate score standard exercise test**

The heart rate scores for the stages of each HR-SET are listed in Table 5. Because 3 horses were late starting training, only 22 horses participated in the Pre HR-SET. Because of lamenesses that occurred during training, only 19 of the 22 original horses completed the Post HR-SET. There was a significant decrease in area under the curve (AUC) between the Pre and Post HR-SET for each time period. Figure 10 in Section D illustrates the effect of training on HR.

The correlation coefficients, probabilities, and number of observations for the correlations of AUC of each phase of the HR-SETs with mean run times for each distance are listed in Table 6. The correlations of AUC with average run times were not significant ($P>0.05$) for the 1200-m runs ($r=-0.0535$ to 0.1118). However, the correlations between AUC of the Pre HR-SET and mean run times tended to be significant for the 1600- and 2000-m runs (Pre 2-9 vs. 1600-m, $P=0.0614$). The correlations between run times and Pre HR-SET AUC were stronger than those correlations between run times and Post HR-SET AUC for each time phase and distance except for the 2000-m run (where the correlations for both SETs vs. distance were significant). The strongest

Table 5--Pre- and Post-training heart rate scores measured for the total HR-SET (Pre 0-9, Post 0-9), gallop and warm-down phase (Pre 2-9, Post 2-9), and gallop phase (Pre 2-7, Post 2-7).

Horse	Pre 0-9	Post 0-9	Pre 2-9	Post 2-9	Pre 2-7	Post 2-7
1	1185	1115	927	854	747	680
2	1273	1209	1040	997	818	784
3	1222	1189	1040	1014	841	807
4	1206	1118	992	901	790	719
5	1403	1234	1151	971	911	754
6	1334	946	1121	903	902	711
7	1227	1113	1016	898	829	701
8	1207	1125	1001	914	798	730
9	1250	1113	1059	923	838	707
10	1165	1021	940	815	749	649
11	1220	1113	1033	942	819	758
12	1347	1158	1075	913	878	741
13	1151	1026	937	837	736	657
14	1193	1126	1003	914	816	730
15	1092	1114	925	862	738	681
16	1442	1431	1178	1148	955	937
17	1164	1048	939	844	749	664
18	1068	1045	887	829	720	656
19	1398	1252	1156	1005	915	776
20	1160	No SET	977	No SET	772	No SET
21	1244	No SET	1022	No SET	821	No SET
22	1203	No SET	973	No SET	780	No SET
Mean	1234	1131 *	1020	923 *	816	731 *
\pm SEM	\pm 20.3	\pm 23.5	\pm 17.5	\pm 17.4	\pm 14.2	\pm 14.7

* Significant difference between Pre- and Post-training heart rate scores (P<0.05)

Table 6--Correlation coefficients (r), probabilities (P), and number (n) of heart rate scores for each phase of the HR-SETs correlated with run performance (measured as mean run time) for each distance.

	1200-m Runs	1600-m Runs	2000-m Runs
Pre 0-9	r= 0.0912 P= 0.6864 n= 22	0.4503 0.0464 * 20	0.4427 0.0577 * 19
Post 0-9	r= -0.0535 P= 0.8280 n= 19	0.1790 0.4917 17	0.4825 0.0584 * 16
Pre 2-9	r= 0.0974 P= 0.6744 n= 21	0.4370 0.0614 ^a 18	0.4524 0.0594 * 18
Post 2-9	r= 0.0044 P= 0.9853 n= 20	0.2391 0.3394 18	0.3567 0.1599 17
Pre 2-7	r= 0.1118 P= 0.6296 n= 21	0.4790 0.0380 * 19	0.4970 0.0359 * 18
Post 2-7	r= 0.0195 P= 0.9349 n= 20	0.2430 0.3312 18	0.3449 0.1752 17

Pre 0-9--Heart rate score measured during 0-9 minutes of HR-SET before training

Post 0-9--Heart rate score measured during 0-9 minutes after training

Pre 2-9--Heart rate score measured during 2-9 minutes before training

Post 2-9--Heart rate score measured during 2-9 minutes after training

Pre 2-7--Heart rate score measured during 2-7 minutes before training

Post 2-7--Heart rate score measured during 2-7 minutes after training

^a 0.10>P>0.05

* P<0.05

correlations between AUC and mean run times was for the Pre 2-7 period (5 min gallop) for each distance. However, the strongest correlation (Pre 2-7 min vs. 2000 m, $r=0.4970$) was only considered to be a fair correlation even though highly significant ($P=0.0359$).

2. V_{200} standard exercise test

The average slope, y-intercept, and V_{200} calculated from the HR vs. speed regression line are listed in Table 7. Also listed are the coefficients of variation (CV) within horses for those variables, mean correlation coefficients (r) for the V_{200} -SETs of each horse that describe how well the regression line fit the data points, and number of tests (n) used to calculate the regression line. The number of tests varied for each horse was because only tests with a correlation coefficient greater than 0.90 were used to calculate the mean regression line for each horse. The CV was relatively high for the 3 variables and varied from 4.9 to 27.4% indicating only fair reproducibility. The mean CV was smaller for the V_{200} (11.8 ± 1.17) than for the slope (17.6 ± 1.93) and Y-intercept (13.8 ± 1.88) indicating better reproducibility for that variable.

Correlation coefficients, probabilities, and number of horses used for the correlations of slope, Y-intercept, and V_{200} with average racetrack run times for

Table 7--Slope (Slp), Y-intercept, V_{200} , coefficients of variation (CV), mean correlation coefficients for the V_{200} -SETs of each horse (r), and number of tests (n) performed for these V_{200} -SET measurements.

No	Slp	CV%	Y-Int.	CV%	V_{200} (m/s)	CV%	r	n
1	6.90	14.7	70.13	7.2	19.10	9.2	0.92	6
2	8.30	27.4	74.92	22.7	15.67	17.5	0.94	11
5	6.64	17.8	96.13	16.4	15.87	12.2	0.92	8
9	7.98	12.2	70.65	10.1	16.45	11.9	0.95	8
11	8.02	14.3	77.48	10.0	15.46	16.2	0.94	10
12	12.8	8.4	57.81	6.9	11.13	4.9	0.93	7
13	6.54	6.7	75.08	10.1	19.37	8.9	0.94	8
14	7.40	25.9	77.79	17.2	16.63	6.4	0.95	8
15	9.65	16.4	78.06	9.5	13.13	18.1	0.96	7
17	7.20	14.0	77.20	10.9	17.21	12.9	0.94	8
18	8.78	35.1	62.14	36.5	16.56	18.4	0.90	6
20	8.79	15.1	70.72	10.0	15.28	16.7	0.95	5
22	8.65	10.9	123.0	16.0	8.85	9.5	0.91	4
24	7.74	22.8	80.85	11.2	15.58	8.3	0.95	8
25	9.15	22.9	66.17	11.9	14.69	5.8	0.95	6
X	8.31	17.6	77.21	13.8	15.40	11.8	0.94	7.3
SE	0.39	1.93	3.853	1.88	0.679	1.17	0.01	0.4

No= Horse number

each distance are listed in Table 8. The correlation of slope to run time was not significant for the 1200-m runs, but approached significance ($P=0.0600$ and $P=0.0888$, respectively) for the 1600- ($r=-0.4961$) and 2000-m ($r=-0.4714$) runs. The correlations for V_{200} with average run times tended to be significant ($P=0.0957$ and $P=0.0650$, respectively) for the 1200- ($r=0.4459$) and 2000-m runs ($r=0.5058$) and attained significance ($P=0.0363$) for the 1600- runs ($r=0.5434$). There was no significant correlation of Y-intercept with average run times for any of the run distances.

3. Blood gas standard exercise test

The Min pH, Min PO_2 , Min HCO_3^- , and Min O_2 Sat are listed in Table 9. The resting values for each variable (not listed) were within normal limits. There was a wide variation among horses for each variable with a range for Min pH of 7.125 to 7.384, Min PO_2 of 45.7 to 87.1 mm Hg, Min HCO_3^- of 8.2 to 23.4 mmol/l, and Min O_2 Sat of 78.2 to 95.2%.

Packed cell volume (resting, maximal, and percent change), Lac8, Max Lac, TLac, and T_{Lac4} are listed in Table 10. The resting values for each measurement (PCV and plasma lactate) were within normal limits. There was a wide variation among horses for each measurement with a range for resting

Table 8--Correlation coefficients (r), probabilities (P), and number of horses (n) used in the correlation of Slope, Y-intercept, and V_{200} with mean run times for 1200, 1600, and 2000 m.

	1200-m Runs	1600-m Runs	2000-m Runs
Slope	r= -0.3624	-0.4961	-0.4714
	P= 0.1843	0.0600 ^a	0.0888 ^a
	n= 15	15	14
Y-int	r= -0.0781	-0.0427	-0.0851
	P= 0.7820	0.8800	0.7723
	n= 15	15	14
V_{200}	r= 0.4459	0.5434	0.5058
	P= 0.0957 ^a	0.0363 [*]	0.0650 ^a
	n= 15	15	14

Slope = Slope of the regression line

Y-int = Y intercept of regression line

V_{200} = Speed at which the horse attained a steady-state heart rate of 200 beats/min

^a $0.10 > P > 0.05$

^{*} $P < 0.05$

Table 9--Min pH, Min PO₂, Min HCO₃⁻, and Min O₂ Sat obtained during the BG-SET.

Horse	Min pH	Min PO ₂ (mmHg)	Min HCO ₃ ⁻ (mmol/l)	Min O ₂ Sat (%)
1	7.211	63.1	14.7	82.7
2	7.181	69.7	12.3	89.0
5	7.275	82.9	16.5	93.5
9	7.357	75.2	22.4	94.3
11	7.312	78.1	18.9	93.7
12	7.125	67.1	8.2	82.4
13	7.360	87.1	19.3	95.5
14	7.330	64.3	23.4	87.8
15	7.278	70.8	15.3	89.3
17	7.284	56.0	16.4	82.0
18	7.309	68.8	17.8	90.4
20	7.290	67.4	18.6	90.5
24	7.285	45.7	19.4	78.2
25	7.384	81.5	22.1	95.2
Mean	7.278	69.8	17.5	88.9
± SEM	0.018	2.83	1.06	1.44

Table 10--Packed cell volume at rest, maximal level, and per cent change, final plasma lactate measurement (Lac 8), maximal plasma lactate attained, sum of plasma lactate samples (TLac), and calculated time at which horse reached a 4 mmol/l plasma lactate concentration ($T_{L_{0.4}}$) for the BG-SET.

No.	PCV			Lac 8 mmol/l	Max Lac mmol/l	TLac mmol/l	$T_{L_{0.4}}$ min
	Rest	Max	% Increase				
1	0.54	0.67	22.9	16.9	18.9	95.8	2.28
2	0.40	0.67	68.4	17.5	18.4	106.3	1.76
5	0.46	0.61	32.6	10.3	12.1	61.0	3.18
9	0.40	0.61	52.5	6.9	9.3	42.8	3.79
11	0.43	0.61	41.9	11.8	13.2	64.3	2.29
12	0.45	0.65	44.4	22.8	22.8	128.0	1.10
13	0.43	0.57	33.7	5.3	8.2	34.3	4.13
14	0.40	0.59	49.4	7.2	9.3	43.4	3.72
15	0.46	0.62	34.5	12.7	15.0	77.4	2.35
17	0.40	0.64	58.8	15.0	17.4	95.1	2.03
18	0.50	0.67	34.0	13.1	14.5	67.4	3.15
20	0.43	0.60	39.5	11.2	13.1	68.6	2.53
24	0.34	0.59	73.5	9.6	11.1	45.0	4.22
25	0.46	0.59	28.3	7.7	10.7	48.6	3.58
Mean	0.44	0.62	43.9	12.0	13.85	69.86	2.91
\pm SEM	0.01	0.01	3.86	1.25	1.10	7.13	0.24

No.= Horse number

PCV of 0.34 to 0.54, Max PCV of 0.57 to 0.67, Lac 8 of 5.3 to 22.8 mmol/l, Max Lac of 8.2 to 22.8 mmol/l, TLac of 34.3 to 128.0 mmol/l, and T_{La4} of 1.10 to 4.22 min. The mean resting PCV was 0.44 ± 0.01 and the mean Max PCV was 0.62 ± 0.01 , which represented an average increase in PCV of $43.9 \pm 3.86\%$ during maximal exercise.

Table 11 illustrates the rapid and substantial changes in PCV as exercise intensity changed. Note that maximal PCV (62%) was reached within one minute after maximal work intensity (12.0 m/s measured at 10 min of the BG-SET). Note also the gradual decline in PCV during the warm-down phase (4.0 m/s trot).

Correlation coefficients, probabilities, and number of horses used to correlate Min pH, Min PO_2 , Min HCO_3^- , Min O_2 Sat, Max PCV, Lac 8, Max Lac, TLac, and T_{La4} with run times are listed in Table 12. There were no significant ($P < 0.05$) correlations for Min PO_2 and Min O_2 Sat with run time for any distance. The correlation between Min HCO_3^- and run time was not significant for 1200- and 1600-m runs, but was significant ($P < 0.05$) for the 2000-m runs ($r = 0.6224$). The correlations for Max PCV and Min pH with run times were not significant for the 1200- and 1600-m runs, but approached significance ($P = 0.0701$ and $P = 0.0854$) for the 2000-m runs ($r = -0.5175$ and $r = 0.4951$, respectively). The correlation for Lac 8 with run time was not significant for the 1200- and 1600-m runs, but was significant for the 2000-m runs ($r = -$

Table 11--The change in PCV% from rest to maximal intensity (at 7% incline) during the BG-SET measured at 1 min intervals.

Time min	Rest	WarmUp	BG-SET					Warm-down		
	0	5	6	7	8	9	10	11	12	13
No.	Speed (m/s)		4.0	6.5	8.0	10	12	4.0	4.0	4.0
1	54	56	56	58	62	65	67	67	66	66
2	40	53	57	59	64	64	66	67	65	63
5	46	50	51	52	54	56	60	61	59	58
9	40	50	51	52	53	56	59	60	61	57
11	43	48	50	52	54	57	61	60	58	57
12	45	58	58	60	62	64	65	65	64	64
13	43	43	47	49	52	57	57	57	57	56
14	40	47	50	53	54	56	59	58	58	55
15	46	53	55	56	58	60	62	61	59	60
17	40	51	52	54	58	63	64	64	63	61
18	50	54	56	57	59	61	64	65	67	64
20	43	44	43	45	52	58	59	60	59	57
24	34	44	44	46	49	51	55	58	58	59
25	46	48	49	51	55	56	58	59	57	56
X	44	50	51	53	56	59	61	62	61	60
SEM	1.3	1.2	1.2	1.2	1.1	1	0.9	0.8	0.9	0.9

Table 12--Correlation coefficients (r), probabilities (P), and number (n) of horses used to correlate parameters with average run times for each distance.

	1200 M Runs	1600 M Runs	2000 M Runs
Min pH	r= 0.0834 P= 0.7767 n= 14	0.2061 0.4796 14	0.4951 0.0854 ^a 13
Min PO ₂	r= -0.2203 P= 0.4492 n= 14	-0.1168 0.6910 14	0.0238 0.9384 13
Min HCO ₃ ⁻	r= 0.2280 P= 0.4330 n= 14	0.3314 0.2471 14	0.6224 0.0231 [*] 13
Min O ₂ Sat	r= -0.2009 P= 0.4910 n= 14	-0.1065 0.7170 14	0.0772 0.8021 13
Max PCV	r= -0.0583 P= 0.8431 n= 14	-0.1774 0.5441 14	-0.5175 0.0701 ^a 13
Lac 8	r= -0.2796 P= 0.3330 n= 14	-0.3950 0.1621 14	-0.6765 0.0111 [*] 13
Max Lac	r= -0.3231 P= 0.2599 n= 14	-0.4322 0.1227 14	-0.7011 0.0076 [*] 13
TLac	r= -0.3883 P= 0.1701 n= 14	-0.4891 0.0759 ^a 14	-0.7411 0.0037 [*] 13
T _{La4}	r= 0.5050 P= 0.0655 ^a n= 14	0.5939 0.0251 [*] 14	0.8304 0.0004 [*] 13

Min pH = Minimum arterial pH

Min PO₂ = Minimum arterial partial pressure of oxygen

Min HCO₃⁻ = Minimum arterial HCO₃⁻

Min O₂ Sat = Minimum arterial oxygen saturation

Max PCV = Maximum packed cell volume attained

Lac 8 = Plasma lactate value at end of BG-SET

Max Lac = Maximum plasma lactate attained

TLac = Sum of plasma lactate values during BG-SET

T_{La4} = Time at which horse attained a steady-state plasma lactate concentration of 4 mmol/l

^a 0.10>P>0.05

^{*} P<0.05

0.6765). The correlation for Max Lac with run time was not significant for the 1200- and 1600-m runs, but again was significant for the 2000-m runs ($r=-0.7011$). The correlation for TLac with run time was not significant for the 1200-m run, approached significance ($P=0.0759$) for the 1600-m run ($r=-0.4891$), and became significant ($P=0.0037$) for the 2000-m runs ($r=-0.7411$). The correlation for T_{La4} with run time approached significance ($P=0.0655$) for the 1200-m runs ($r=0.5050$) and attained significance for the 1600- ($r=0.5939$) and 2000-m runs ($r=0.8304$). Note that the correlation between each variable and run distance became stronger with increased distance, except for Min PO_2 and Min O_2 Sat (which showed no significant correlation with run times).

4. Onset of blood lactate standard exercise test

The velocity (V_{La4}) and work rate (W_{La4}) at which blood lactate reached a steady-state concentration of 4 mmol/l obtained during the OBLA-SET and the coefficients of variation (CV) for those values are listed in Table 13. The V_{La4} varied from 1.0 to 10.23 m/s (mean= 8.32 ± 0.48 m/s). The W_{La4} varied from 265.7 to 3050.5 watts (mean= 2359.1 ± 144.2 watts). The CV for the V_{La4} and W_{La4} were the same since those values were obtained from the same OBLA-SETs, and varied from 0.11 to 22.00% (mean= $9.43 \pm 1.72\%$).

Table 13-- V_{La4} , W_{La4} , and VO_{2max} values with coefficients of variation (CV) for each parameter.

No.	V_{La4} (m/sec)	CV%	W_{La4} (Watts)	CV%	VO_{2max} (ml/kg* min)	CV%
1	9.63	5.00	2467.9	5.00	138.4	5.10
2	5.78	16.00	1569.4	16.00	174.6	2.56
3	8.55	22.00	2284.9	22.00	170.0	3.70
4	9.96	0.11	3050.5	0.11	163.5	4.68
5	8.94	4.00	2507.6	4.00	163.1	5.47
6	10.23	9.00	3045.7	9.00	174.3	1.77
9	9.29	3.00	2593.8	3.00	178.3	2.31
11	8.00	21.19	2438.8	21.19	179.8	2.31
12	6.92	21.00	1843.4	21.00	191.1	0.51
13	9.36	2.00	2620.1	2.00	165.1	0.69
14	10.00	0.80	3082.7	0.80	170.4	1.36
15	7.37	20.04	2239.4	20.04	166.8	3.23
17	5.95	17.00	1827.4	17.00	172.0	2.59
18	10.15	7.00	2958.3	7.00	156.3	2.20
19	10.21	4.00	2955.4	4.00	167.4	0.88
20	7.55	13.41	1957.4	13.41	189.4	5.81
22	1.0	4.00	265.7	4.00	175.4	5.09
23	9.67	0.12	2496.1	0.12	171.1	3.23
24	8.71	15.02	2784.3	15.02	158.2	2.32
25	9.15	4.00	2193.8	4.00	177.3	4.74
Mean	8.32	9.43	2359.1	9.43	170.1	3.03
+SEM	0.48	1.72	144.2	1.72	2.53	0.36

No.= Horse number

Correlation coefficients, probabilities, and number of horses used for the correlations of V_{La4} and W_{La4} are listed in Table 14. There was a significant correlation between V_{La4} and run times for the 1200-, 1600-, and 2000-m runs ($r=0.5124$, 0.5768 , and 0.6854 , respectively). There were also significant correlations between W_{La4} and run times for the 3 distances ($r=0.5783$, 0.6670 , and 0.7162 , respectively). Note the stronger correlations as distance increased.

5. Maximal oxygen uptake standard exercise test

The VO_{2max} values obtained during the VO_{2max} -SET and coefficients of variation (CV) for those measurements are listed in Table 13. Values for VO_{2max} varied from 138.4 to 191.1 ml/kg*min (mean= 170.1 ± 2.53 ml/kg*min). The CV varied from 0.51 to 5.81% (mean= $3.03 \pm 0.36\%$).

Correlation coefficients, probabilities, and number of horses used for the correlations of VO_{2max} with run times are listed in Table 14. The correlation between VO_{2max} and run time was not significant for the 1200-m runs, approached significance ($P=0.0691$) for the 1600-m runs, and attained significance ($P=0.0435$) for the 2000-m runs.

Table 14----Correlation coefficients (r), probabilities (P), and number (n) of horses used for the correlations of V_{La4} , W_{La4} , and VO_{2max} with times for the 1200-, 1600-, and 2000-m runs.

	1200 M Runs	1600 M Runs	2000 M Runs
V_{La4}	r= 0.5124 P= 0.0209 * n= 20	0.5768 0.0097 * 19	0.6854 0.0024 * 17
W_{La4}	r= 0.5783 P= 0.0076 * n= 20	0.6670 0.0018 * 19	0.7162 0.0012 * 17
VO_{2max}	r= -0.3492 P= 0.1428 n= 19	-0.4379 0.0691 ^a 18	-0.5102 0.0435 * 16

V_{La4} = Velocity at which horse maintained a steady-state plasma lactate concentration of 4 mmol/l

W_{La4} = Work rate at which horse maintained a steady-state plasma lactate concentration of 4 mmol/l

VO_{2max} = Maximal oxygen uptake

^a 0.10>P>0.05

* P<0.05

Discussion:**1. Heart rate score standard exercise test**

Decreased heart rate following training has been well documented in human studies (Ekblom *et al.* 1968). The significant decrease in heart rate scores following training in this study (Table 5) was consistent with other equine exercise studies (Bayly *et al.* 1983a; Sexton *et al.* 1987; von Englehardt, 1977).

The HR-SETs were performed to determine if the number of heart beats during a standard exercise test in an unfit or fit condition correlated with running ability (measured by mean run times). Since HR is related to the oxygen carrying ability of the blood (Astrand & Rodahl, 1986), this SET was a measure of aerobic capacity. Since the duration of the 1200-m run was shorter and the horses ran at maximal effort the entire distance, aerobic metabolism contributed a smaller portion of the total energy expenditure when compared to the longer runs. During the 1600- and 2000-m runs, the horses were restrained by the jockeys during the initial portion of the runs to delay the onset of fatigue presumably by preventing the early accumulation of lactate, inorganic phosphate, and H^+ associated with fatigue (Wilkie, 1986). Aerobic metabolism is of increasing importance in longer distance events (Skinner & McLellan, 1980). This probably explains the stronger correlations

between area under the curve (AUC) and mean run times for the 1600- and 2000-m runs compared to the 1200-m runs.

Correlation between AUC and run time was consistently stronger for the Pre HR-SET than for the Post HR-SET (Table 6). The positive correlations of low heart rate scores with quicker run times in the untrained horses indicated that the faster horses were able to perform the sub-maximal HR-SET at a lower heart rate (HR) than the slower horses. With training, this inherent superiority of the faster horses to perform the same work load at a lower HR was not as evident as indicated by the weaker correlations between run times and AUC in the Post HR-SET. Athletically superior horses appear to perform the same work load at lower HRs than slower horses. This phenomenon was more evident in unconditioned horses. Conditioning may improve cardiac efficiency in slower horses more than faster horses. Likewise, more athletic horses may not detrain during periods of limited activity to the extent of inferior horses. It appears that conditioning narrows the HR range between faster and slower horses during a similar exercise. The narrower HR range weakened the correlation between sub-maximal HR and run times.

The AUC for the gallop phase (2-7 minute period) of the HR-SET correlated better with mean run times than AUC for any other phase. Since the gallop phase was included in the other periods measured, it was concluded that HR

during the gallop period had a stronger influence on correlation of AUC with run times than HR during any other period of the HR-SET. The higher work rate of the gallop phase increased the range of individual steady-state heart rates. This is illustrated by the much higher SEM during the gallop phase (Table 21--Section D). In Section A, no correlation was found between maximal HR and run times. However, sub-maximal HR appears to show a fair correlation with run times for 1600- and 2000-m runs ($r=0.4790$ and 0.4970 , respectively).

2. V_{200} standard exercise test

Other investigators have used V_{200} and slope of the heart rate vs. speed curve to evaluate exercise tolerance and fitness in Standardbred horses (Persson & Ullberg, 1974; Persson *et al.* 1983). The Y-intercept has not been used to correlate with fitness or race performance. The Y-intercept is a calculated value of the HR at rest on the treadmill. Table 7 shows that the Y-intercept values are higher than would be expected for resting HR values. However, a horse standing on the treadmill would have a HR above resting values because of the anticipation of exercise. Therefore, the Y-intercept values appear to be within normal range for standing HR. Even though the Y-intercept values were representative of actual HRs at zero speed on the treadmill, the lack of

any significant correlation with run times indicates that resting HR values are of no benefit in predicting maximal-effort performance.

The relatively high CVs for the 3 variables from the V_{200} -SETs indicated that these measurements were not highly reproducible. This was in contrast to Persson (1983) who stated that the slope and V_{200} of a HR vs. speed regression were highly reproducible if strict standardization of the test procedure was observed. (However, the degree of reproducibility was not indicated in that study.) Strict standardization was followed during data collection for the V_{200} -SET in the present investigation. All tests were run following a 5 min warm-up. Constant environmental conditions were maintained. Since HR during exercise has been shown to be influenced by exogenous factors (Persson, 1983), external distractions were eliminated by restricting entry to the exercise area during testing and minimizing movement and noise in the treadmill room. Horses were frequently excited, and possibly apprehensive, at the beginning of exercise. After a few minutes of galloping, excitement seemed to decrease. To reduce the effect of excitement and anxiety on HR, horses were put through multiple (2-4) V_{200} -SETs on the same day in an attempt to improve reproducibility. However, there was no improvement in reproducibility when sequential V_{200} -SETs were compared with similar V_{200} -SETs performed on other days. The decreased reproducibility of V_{200} in this study when compared with the studies by Persson may have been because the horses used for this

investigation were Thoroughbreds while Persson used Standardbreds. Thoroughbreds are generally considered a more excitable breed than Standardbreds and may have been fearful or nervous to a greater degree than the horses used by Persson. Furthermore, Standardbreds trotted during the entire SET of Persson while Thoroughbreds changed gaits from a trot to a gallop as treadmill speed increased. The transition speed at which horses changed gaits varied, but usually occurred between 4.0 and 6.5 m/s. It may have been discomfoting for the horses to maneuver if they could not trot or gallop comfortably at one of these speeds. This would result in an altered HR and would decrease reproducibility. For future tests, the treadmill speed protocol should probably be changed to speeds that could be performed at the same gait. Likewise, an increased time for each work rate might allow the HR to reach a more reproducible steady-state.

As mentioned in the protocol for this study, tests with a correlation coefficient (r-value) less than 0.90 were not used to determine the mean regression for each horse. The average correlation coefficient for the tests used to determine the slope, Y-intercept, and V_{200} of the mean regression equation for each horse was 0.94. This illustrates that the points used to calculate the regression line for each V_{200} -SET were not scattered but were in close proximity to the regression line. However, because of the wide variation between separate V_{200} -

SETs for the same horse, it was concluded that a regression line determined by HR vs. speed did not exhibit high reproducibility within horses.

The slope of the linear regression line is a measure of the change in HR per unit change of treadmill speed. The negative correlation between slope and run time demonstrated that the HR of faster horses increased at a faster rate per change in speed than did the HR of slower horses during a near-maximal SET. The positive correlation between V_{200} in a near-maximal intensity SET and run time demonstrated that faster horses attained a steady-state HR of 200 beats/min at a lower velocity than did the slower horses. These two findings indicated that the HRs of faster horses increased more rapidly during near-maximal intensity exercise than the HRs of slower horses running at a similar speed. Slope and V_{200} showed a fair correlation ($0.45 < P < 0.55$) with run times, however Y-intercept demonstrated no relation to performance (Table 8).

3. Blood gas standard exercise test

a. Minimum pH

The lack of any significant correlation of Min pH with run times (Table 12) at 1200- and 1600-m and the tendency for a significant effect for the 2000-m runs

may be explained by the effect of pH on muscle tissue and the difference in buffering capacities between horses. In vitro research has shown a positive correlation between muscle fatigue and the free H^+ concentration in muscle (Hill, 1955). A build up of H^+ in muscle tissue has an inhibitory effect on the glycolytic enzyme phosphofructokinase (Trivedi & Danforth, 1966). Blood pH is not equal to, but fluctuates with the intracellular pH of working muscles (Hood *et al.* 1988). Therefore, blood pH is related to muscle pH. The tendency for a positive correlation between Min pH and run times for the 2000-m runs indicated that faster horses were capable of faster running at a lower pH than slower horses. The important factor in determining the onset of fatigue is the buffering capacity of the muscle to neutralize the inhibitory effect of the H^+ generated by glycolysis (Brien & McKenzie, 1989). Since the bicarbonate system is the main buffer for neutralizing H^+ (White *et al.* 1964), the correlation of Min pH with run performance should be similar to the correlation of Min HCO_3^- to run performance. These correlations were consistent in our study (see discussion of Minimum HCO_3^-).

b. Minimum HCO_3^-

The bicarbonate (HCO_3^-) buffer system is the main buffering mechanism in the muscle cell and extracellular fluid (White *et al.* 1964). Therefore, it is

responsible for buffering the majority of the H^+ produced during intense exercise. The concentration of HCO_3^- in the blood decreases significantly during a maximal effort exercise test (Snow *et al.* 1983a). Snow *et al.* (1983a) demonstrated a decrease in blood HCO_3^- from 26.6 ± 1.1 mmol/l before a 1.6 km run to 19.5 ± 2.9 mmol/l after 0.8 km, 15.4 ± 0.9 mmol/l after 1.6 km, 10.6 ± 0.9 on stopping, and 6.6 ± 0.8 mmol/l 5 minutes after exercise. Since H^+ accumulation in the muscle has been shown to be at least partially responsible for the onset of fatigue (Hill, 1955), the ability to buffer the H^+ should correlate with performance. There was no significant correlation between blood HCO_3^- and times for the 1200- and 1600-m runs (although the correlation for the 1600-m run was higher). However, the correlation between HCO_3^- and 2000-m run times was significant (Table 12). It would be expected for the longer distance runs to show a stronger correlation than shorter runs because as more H^+ are produced with increasing distance, the bicarbonate buffering system becomes more depleted and therefore, a limiting factor for muscle contraction (Beaver *et al.* 1986). The positive correlation between Min HCO_3^- and run times indicated that the faster horses were capable of greater speeds at lower concentrations of bicarbonate. This was consistent with the lower Min pH seen in faster horses.

c. Minimum PO₂

The lack of any significant correlation of minimum PO₂ and minimum O₂ saturation with run times (Table 12) can be explained by considering the amount of oxygen delivered to the muscle during intense exercise. Several studies have shown that hypoxemia does occur in horses during heavy exercise (Bayly *et al.* 1983b; Persson *et al.* 1987; Thornton *et al.* 1983). Wagner *et al.* 1989) determined that hypoxemia was due to elevated PCO₂, mild ventilation/perfusion mismatching, and increased alveoli-end capillary diffusion disequilibrium. However, splenic contraction can increase hemoglobin content by 50% over resting values during severe exercise. The increased hemoglobin content prevents any significant decrease in the quantity of oxygen carried in arterial blood (Jones *et al.* 1989). Therefore, differences in partial pressure of arterial oxygen (PO₂) would not be expected to significantly affect performance. This hypothesis is supported by the increased PCV seen during maximal-intensity exercise (Table 11).

d. Minimum O₂ Saturation

Hemoglobin in elite human distance runners desaturates with near-maximal intensity exercise (Dempsey *et al.* 1984). Studies have shown that the

hemoglobin of horses also desaturates during maximal intensity exercise (Bayly *et al.* 1983b). However, a recent equine study on blood-gas measurements during exercise from rest to VO_{2max} showed that correcting blood temperature to resting or rectal temperature overestimates the degree of desaturation. It revealed that if blood temperature was corrected to working muscle temperature, which is higher than resting or working rectal temperature, the degree of desaturation was less than 1% (Jones *et al.* 1989). Therefore, if hemoglobin does not desaturate there should be no correlation of O_2 desaturation with run times, which was consistent with the findings of this project (Table 12).

e. Maximum packed cell volume

Table 10 shows the resting and maximal PCV reached during the BG-SET and the per cent change. These values were consistent with the findings of other studies that measured PCV during near-maximal intensity exercise (Harris & Snow, 1988; Persson, 1967). Maximal PCV is of importance during aerobic exercise since the oxygen carrying ability of the blood is a function of PCV and has been correlated with performance in other equine studies (Persson, 1967; Persson, 1983). Persson (1983) showed that the velocity attained at a HR of 200 beats/min had a strong correlation with total red cell volume ($r=0.95$). The

tendency ($P=0.0701$) toward a negative correlation between Max PCV with run time ($r=-0.5175$) for the 2000-m runs demonstrated that faster horses had higher Max PCV than slower horses. A stronger correlation between Max PCV and run times would be expected for longer runs because total energy needs during a run of 2000 meters is more dependent on aerobic metabolism than shorter runs.

Animals react differently to hypodermic needles and the restraint required for blood sample collection. External stimulation results in splenic contraction and increased circulation of red blood cells in more excitable horses. This must be considered when interpreting the resting PCV and per cent change seen in Table 10. Some of the resting PCV values appear elevated (e.g. Horses 1 and 18), which may have been due to fear. Elevated resting PCV would also reduce the percent change from rest to maximal value. There was no significant correlation of resting PCV with run times. This is consistent with studies that have shown resting PCV to be an unreliable indicator of the oxygen carrying capacity of the blood (Persson, 1967).

f. Plasma lactate (Lac 8, Max Lac, TLac, T_{La4})

The negative correlations of Lac 8, Max Lac, and TLac and the positive correlation of T_{La4} with run time illustrated that higher plasma lactate levels were measured in the faster horses (Table 12). This was consistent with human and equine exercise studies. Heck *et al.* (1985) showed that 800-m runners attained a 4 mmol/l blood lactate concentration at a slower speed than 1500-m runners. They also showed that OBLA was higher for marathon runners than any other group. Svendenhag and Sjodin (1984) demonstrated that elite middle-distance runners (400-800 m) reached OBLA at a slower running speed than elite distance runners (1500-10,000 m). In the same study, they demonstrated that elite middle-distance runners also reached significantly higher peak lactate concentrations than longer distance runners following an exhaustive treadmill run. Taunton *et al.* (1981) revealed that middle distance runners attained a significantly higher blood lactate concentration following a 5 min exhaustive treadmill run when compared with long distance runners. In an equine exercise study, Bayly *et al.* (1987a) showed that during a run of 1000 m, there was a trend toward higher lactates in the faster horses. In a study of 16 horses, Saibene *et al.* (1985) demonstrated that higher plasma lactate levels (measured 5 min after the end of the run) were seen in the faster horses raced over distances of 200, 300, and 400 meters. They also showed a correlation coefficient between lactate production and speed of $r=0.86$. Since

speed and race time are inversely proportional, this finding is consistent with the negative correlation between run time and plasma lactate concentration seen in this investigation.

The correlation of Lac 8, Max Lac, TLac, and T_{La4} with run times became consistently stronger with increased distance. Since ATP and creatine phosphate accounts for a greater portion of the anaerobic energy expended during shorter distances (Astrand & Rodahl, 1986), plasma lactate concentration would be expected to better correlate with run times for distances that rely less on these sources of energy. In addition, anaerobic capacity may have been more completely expended in the longer runs, thereby maximizing any correlation between anaerobic capacity and run times.

The different plasma lactate measurements were used to find the variable having the strongest correlation with run time. Since the correlation between T_{La4} and run time was strongest, an onset of blood lactate standard exercise test (OBLA-SET) was designed to more accurately determine the point at which plasma lactate reached a concentration of 4 mmol/l. The OBLA-SET is discussed in the following section.

4. Onset of blood lactate standard exercise test

The strong correlations of W_{La4} and V_{La4} with run times (Table 14) are consistent with human and other equine exercise studies. This was discussed in the preceding segment. The greater correlation between W_{La4} and run times than between V_{La4} and run times was consistent with other investigators (Jacobs *et al.* 1985; Yoshida, 1986) who have shown stronger correlations between work load and performance when the work load was expressed relative to body weight.

The correlations of W_{La4} and V_{La4} with run times were significant for each distance, and were more strongly correlated with run times than T_{La4} (from the BG-SET) for the 1200- and 1600-m runs. However, the correlation between T_{La4} and run times for the 2000-m runs was greater than the correlation of W_{La4} and V_{La4} with run times for that distance. This may have been due to the inherent differences in these measurements. In shorter distance runs, the energy needed to overcome the inertia of a body at rest is proportionately greater than for runs of longer distances. The W_{La4} and V_{La4} are dependent on speed and/or body weight which are factors involved in overcoming inertia. The unique variable in T_{La4} is time. The 2000-m runs lasted longer than the shorter runs, and would be less influenced by inertia and the forces necessary to overcome it.

Investigators (Davis *et al.* 1976; Jacobs, 1981; Jacobs *et al.* 1985) have shown that the onset of blood lactate correlated well with endurance performance. Furthermore, variables measured during sub-maximal exercise like W_{La4} and V_{La4} correlated as well with performance as maximal-effort variables like VO_{2max} (Daniels *et al.* 1978; Katch *et al.* 1978). The results from this project agreed with those findings. Since sub-maximal measurements are more safely obtained in racehorses, W_{La4} and V_{La4} may be more valuable than VO_{2max} for assessing performance potential.

5. Maximal oxygen uptake standard exercise test

Maximal oxygen uptake (VO_{2max}) has been shown to be the most important measurement for success in long-distance events for humans (Astrand, 1952; Robinson *et al.* 1937). Astrand (1952) and Robinson *et al.* (1937) were among the first investigators to show that maximal oxygen uptake was highest in the best distance runners. The increasing correlation between VO_{2max} and run times for longer distances in this project was consistent with a human study (Shaver, 1975) correlating run times with VO_{2max} for 30 untrained college men running 7 different distances (100-yd to 3 miles). That study showed that as distance was increased the correlation coefficients (r-value) increased (Shaver, 1975). The correlation coefficient between VO_{2max} and the 100-yd run was only

-0.08 but was significantly ($P < 0.05$) correlated with the 880-yd run ($r = -0.35$), 1-mile run ($r = -0.43$), 2-mile run ($r = -0.76$), and 3-mile run ($r = -0.82$). As distance of an athletic event increased, VO_{2max} became increasingly important as a predictor of performance (Shaver, 1975). This would be expected since longer distance events derive a greater proportion of the required energy from aerobic sources.

The type of gas collection system can influence VO_{2max} measurements. The system used in this investigation was similar to the flow-through system used by Seeherman *et al.* (1981). With that system, there were no differences in arterial partial pressure of oxygen (PO_2) and arterial partial pressure of carbon dioxide (PCO_2) during exercise with or without the face mask. Measured oxygen uptake was also increased by using the mask flow-through system. The system used for this investigation consisted of a respiratory mask over the rostral portion of the head and pulled air around the head and over the nares of the horse. The system had a flow rate in excess of 6,000 l/min. The high flow rate prevented the loss of any expired air.

Maximal oxygen uptake has been measured in horses but has not been correlated with race performance in the same group of horses. Hoppeler *et al.* (1987) used a flow-through gas collection device to measure VO_{2max} in Standardbreds. Evans and Rose (1987) measured VO_{2max} in 6 Thoroughbred

horses before and after a 12 week treadmill training period to show that VO_{2max} was increased with training. They used a gas collection mask with unidirectional flow valves to collect the expired air. In another study (Evans & Rose, 1988b), they measured VO_{2max} and determined the repeatability of that measurement in 6 Standardbred horses. The coefficient of variation (CV) of VO_{2max} was less than 10% in the six horses (range 1.42 to 9.05 percent). This was consistent with the CV (mean= $3.03 \pm 0.36\%$, range 0.51 to 5.81%) found in this investigation.

It was concluded that no resting variables (resting PCV, standing HR [Y-intercept]) correlated with run times. The variables showing the strongest correlation with run times were V_{200} , onset of blood lactate measurements (T_{La4} , V_{La4} , and W_{La4}), blood lactate concentrations (peak lactate and total lactate) and VO_{2max} . The strongest correlations of these variables with run times were consistently seen with the 2000-m runs. The variables showing the strongest correlation with 1200-m run times was the onset of blood lactate variables (T_{La4} , V_{La4} , and W_{La4}). Since the OBLA variables correlated best with the 1200-m runs (compared to the other variables), and since those measurements are obtained during sub-maximal exercise, T_{La4} , V_{La4} , and W_{La4} were determined to be the best correlates of running performance.

Part II. Factors Influencing Performance

Section C

Influences of Training Method on Racing Performance

Objectives:

1. To describe alternative and conventional training protocols used to athletically condition horses.
2. To demonstrate differences in heart rate recovery curves, lactate production, rate of lactate disappearance from plasma, and onset of fatigue in horses trained by alternative and conventional methods.
3. To compare running performance of each training group over a distance of 1000 meters.

Materials and Methods:

1. Horses

Eight 4-5 yr old Thoroughbreds (seven geldings and one mare) with previous race track experience were randomly assigned to two different training groups, a conventional training (CT) group and an alternative training (AT) group.

The AT regimen consisted of long slow distance (LSD), which consisted of slow galloping each day, and interval training (IT). Horses had been on pasture and had not received any conditioning for at least six months prior to the beginning of this investigation.

2. Training methods

Since an objective of this study was to compare running abilities of each training group over a distance of 1000 meters, the training programs were designed to prepare the horses for maximal-effort 1000 m runs. The text that follows describes the training programs employed for each group. There were differences in the exact protocols for individuals within the groups. Therefore, the training regimens presented are for a prototypic alternatively and conventionally trained horse. The AT schedule was adapted from the interval training methods described by Ivers (1983).

a. Alternative training

The alternative training program was divided into three phases: Phase 1--Long Slow Distance work (LSD), Phase 2--Interval training for cardiovascular fitness work, and Phase 3--Interval training for speed work. The end points

Table 15--Schedule for alternative training

Phase	Time to complete	Heat parameter	Beginning regimen	Ending regimen
1	14-20 weeks	Dist/day Speed Frequency	1600m 1600m/5:00 6/week	9600m 1600m/3:30 6/week
2a	5 weeks	Dist/day Time/Heat No. Heats Frequency	3200m 7:30, 7:30 2 2/week	3200m 6:15, 6:10 2 2/week
2b	6 weeks	Dist/day Time/Heat No. Heats Frequency	2400m 4:35,4:30, 4:25 3 2/week	2400m 4:20,4:15, 4:10 3 2/week
2c	6 weeks	Dist/day Time/Heat No. Heats Frequency	1600m 2:50,2:45, 2:40,2:35 4 2/week	1600m 2:10,2:03, 2:00,1:55 4 2/week
3	4 weeks	Dist/day Time/Heat No. Heats Frequency	1200m 1:40,1:35, 1:32,1:30 4 2/week	1200m 1:28,1:25, 1:21 3 2/week
Final Taper	2 weeks	Dist/day Time/Heat No. Heats Frequency	800m 1:00,0:56, 0:54 3 2/week	800m 0:54,0:50 2 2/week

of each phase are summarized in Table 15.

Phase 1: During the first week of training, LSD began with trotting for 800 meters, followed by 1600 meters of slow galloping over a 5 min period (Table 15). The distance galloped was increased 800 m each week providing the work load was tolerated. Speed was gradually increased over an 8 week period to 1600 m/4:00, and distance galloped was gradually increased to 6400 m/day. By the end of Phase 1, the distance galloped was 9600 m at a speed of 1600 m/3:30. Long slow distance was performed 6 days with one rest day per week. The time required to complete Phase 1 varied among the group from 14-20 weeks, depending on how quickly each horse was able to tolerate more strenuous work loads.

Phase 2: Phase 2 started after LSD had been increased to 9600 m a day. During this period, IT was performed twice a week. During the IT sessions, speed was gradually increased and the total distance travelled was divided into multiple heats separated by short rest periods. The rest periods lasted 10 min at the beginning of Phase 2 and were gradually shortened to 5 minutes by the end of Phase 3. At the finish of each heat, horses were warmed-down by cantering 400 meters, trotting 400 meters, then walking.

Phase 2a: The first training session consisted of two 3200-m heats run at 7:30/heat (speed=1600m/3:45), separated by a 10 minute rest period (Table 15). This was followed by a 1600-m warm-down that consisted of slow galloping and trotting. The second training session was performed 4 days later and consisted of the same regimen, except the heats were run in 7:13 and 7:04 respectively.

The same protocol as outlined above was followed for the next eight training sessions (4 weeks), but each heat was run progressively faster by 5 to 10 seconds/heat. The first heat was designed to always be the slower heat. If the horse ran a subsequent heat slower than a previous heat, it was interpreted as an indication of fatigue and training was stopped for the day. This protocol was adopted to preclude injury, which may more readily occur during fatigue. The final two 3200-m heats were run in 6:15 and 6:10 respectively (Table 15).

Phase 2b: After the above level of performance was achieved, the distance of each heat was reduced to 2400 m, the number of heats was increased to three, rest periods between heats were shortened to 7 minutes, and the speed of each heat was progressively increased (Table 15). During the first week of this phase, the training session consisted of three 2400-m heats run in 4:35, 4:30, and 4:25. This corresponded to a speed of 1600m/3:03, 1600m/3:00, and 1600m/2:57, respectively. Three days later this regimen was repeated with the

three heats run in 4:20, 4:15, and 4:10, respectively. This regimen continued for 5 more weeks.

Phase 2c: For the next level of performance, the distance for each heat was reduced to 1600 m, the number of heats was increased to four, the rest periods were maintained at 7 minutes, and the speed was continually increased (Table 15). The first 4-heat training session consisted of 1600-m heats run in 2:50, 2:45, 2:40, and 2:35. This routine was repeated 3 days later. The next week, two 4-heat work-outs were performed in 2:45, 2:40, 2:35, and 2:30. For the following five weeks, training occurred twice a week, and the heat times were reduced by 5-10 seconds each week. The final 4-heat workout consisted of 1600-m heats run in 2:10, 2:03, 2:00, and 1:55 (Table 15). On non-training days, the horse was galloped at 1600 m/3:00 for 3200-6400 m depending on how the horse recovered from the previous training session. During Phases Two and Three, there were 2 interval training days, 3 long slow distance days, and 2 rest days each week.

Phase 3: This phase was a continuation of Phase 2 except distances were typically shorter and speeds were faster. The number of heats was maintained at four, the distance of each heat was reduced to 1200 m, and the rest periods between heats were reduced to 5 minutes. The first workout of Phase 3 consisted of four 1200-m heats in 1:40, 1:35, 1:32, and 1:30 (Table 15).

At this speed, the times for each workout were only reduced by 1-3 seconds per heat. For example, during the next work out the heats were run in 1:37, 1:34, 1:30, and 1:27. There were six more training sessions (3 more weeks) at this distance, then the number of heats was reduced to three. The reason for the reduction in number of heats was because heart rate had frequently remained elevated above a predetermined threshold level of 110 beats/min following the third heat (after two minutes rest). This phase of training occurred during the late summer when high ambient temperature and humidity prolonged the time required for heat dissipation. Therefore, the reduced number of heats precluded injury due to hyperthermia or excessive fatigue. The last workout consisted of three 1200-m heats run in 1:28, 1:25, and 1:21.

Final taper: The first workout of the final taper consisted of three 800-m heats run in 1:00, 0:56, and 0:54 with rest periods of 5 min between heats. This was repeated three days later. Four days after this workout, the horse ran three more heats in 0:58, 0:55, and 0:53. This was also repeated three days later. The final workout of the training program consisted of two 800-m heats run in 0:54, and 0:50.

b. Conventional training

The second group of horses underwent a conventional training (CT) program of the same duration as the AT program. The training schedule for the first four weeks of the CT horses was similar to the AT schedule in distance galloped, but swimming was also used for conditioning of the CT group. During week 5, the horses on the AT schedule increased the distance galloped (Table 15), but the CT horses never galloped farther than 4000-m on a single day (Table 16). The following is a description of the CT schedule.

As mentioned in the alternative training section, during the first week the prototypic CT horse galloped 1600 m/day at a rate of 1600m/5 min (Table 16). The distance galloped was increased by 800 m/day each week until the maximum distance of 4000 m per day was reached (9600m/day for AT group). Speed was gradually increased over the next 10 weeks until 1600m/3 min was achieved. This speed and distance were maintained for the duration of the training period. Twice a week the horse also swam in an 11-meter diameter equine swimming pool. During the first pool workout, 6 laps were swum which required 5 minutes of strenuous exercise to complete (Table 16). Distance was increased by 1 lap/day each week until the swimming workouts consisted of 24 laps which required about 20 minutes to perform. After 6 weeks of galloping and swimming, horses were breezed (run at 75% maximal effort) over a

Table 16--Schedule for conventional training

Training Period	Training Variable	Beginning of Period	End of Period
Weeks 1-6	Gallop Distance	1600m	4000m
	Speed	1600m/5:00	1600m/4:00
	Frequency	4/week	4/week
	Laps Swam	6	11
	Frequency	2/week	2/week
Weeks 7-12	Gallop Distance	4000m	4000m
	Speed	1600m/4:00	1600m/3:15
	Frequency	3/week	3/week
	Laps Swam	12	17
	Frequency	2/wk	2/wk
	Breeze Distance Frequency	400m 1/week	400m 1/week
Weeks 13-18	Gallop Distance	4000m	4000m
	Speed	1600m/3:15	1600m/3:00
	Frequency	3/week	3/week
	Laps Swam	18	23
	Frequency	2/wk	2/wk
	Breeze Distance Frequency	600m 1/week	600m 1/week
Weeks 19-24	Gallop Distance	4000m	4000m
	Speed	1600m/3:00	1600m/3:00
	Frequency	3/week	3/week
	Laps Swam	24	24
	Frequency	2/wk	2/wk
	Breeze Distance Frequency	800m 1/week	800m 1/week
Weeks 25-32	Gallop Distance	4000m	4000m
	Speed	1600m/3:00	1600m/3:00
	Frequency	3/week	3/week
	Laps Swam	24	24
	Frequency	2/wk	2/wk
	Breeze Distance Frequency	1000m 1/week	1000m 1/week

distance of 400 m once a week for 6 weeks (Table 16). Breeze distance was increased to 600 m for the next 6 weeks, then to 800 m for 6 weeks, and finally to 1000 m for the last 6 weeks of the training period. The breezes were not designed to be run in a specific time. Rather, the jockey was instructed to allow the horse to run at 75% maximal speed. Consequently, running time for each breeze was dependent on the jockey's assessment of the running ability of the horse. During a typical week, CT horses were galloped on the track for 3 days, breezed for 1 day, swum for 2 days, and rested for 1 day.

3. Standard exercise tests

Four maximal-effort standard exercise tests (SETs) were performed during the training period: 1) a pre-conditioning 400-m run after 2 weeks of slow galloping (unfit 400-m SET), 2) a 400-m run after 5 months of training (fit 400-m SET), 3) a 1000-m run after 7 months of training (1st 1000-m SET), and 4) a second 1000-m run (2nd 1000-m SET) one week after the first 1000-m SET.

The warm-up protocol for each SET was the same for all horses. The horses were trotted and cantered in a clockwise direction around the track for 1600-m. The horses were then stopped, turned in a counterclockwise direction, and began each SET from a running start 200 m before the starting line. Horses

were run at the same time in their training, and under similar environmental and track conditions. The same jockey was used for every horse during every SET. The horses ran at maximal speed for the entire distance during each SET. Run times were recorded for all SETs to the nearest 0.01 second. In addition, for the 1000-m SETs, the average time for each 200-m segment was recorded. Immediately after completing the SET, each horse was stopped within 400 m of the finish line, returned to the training barn and walked for the next 30 minutes. During the 30 minute recovery period, heart rates were continually recorded at 15 second intervals, and plasma lactate samples were collected at 0, 2, 5, 10, 15, 20, 30, 60, and 120 minutes after the runs.

4. Lactate

Blood samples were drawn from a pre-placed jugular catheter and collected in vacutainers²⁸ containing oxalate as an anticoagulant, and fluoride to stop glucose metabolism and further lactate production. The tubes were immediately centrifuged and the plasma removed for storage. Plasma samples were frozen at -20 degrees centigrade and later analyzed using a lactic acid kit²⁹ and a wide-bandwidth spectrophotometer.³⁰

²⁸ Becton Dickinson, No. 6445, Rutherford, NJ

²⁹ Sigma Chemical Co., 826-B, St. Louis, MO

5. Heart rate monitor

Two types of heart rate monitors^{31,32} were used in this project. The electrodes for the Hippocard monitor had been sewn into the saddle pad and were placed on the horse's sternum and left side of chest midway down the chest wall. The VMAX monitor used 2 paste-on electrodes³³ placed on shaved areas of the sternum and left side of neck just anterior to the scapula. The receivers for both systems were worn on the wrist of the jockey and were set to record and store the heart rates every 15 seconds. The stored information was then down-loaded with the system interface to a computer equipped with an RS232 serial input. Heart rates were measured during SETs for both groups and all IT sessions in Phases Two and Three.

A training HR threshold limit was established for horses in the AT group. Within 2 minutes after each heat, the heart rate had to return below 110 beats/min or all subsequent heats scheduled for that day were canceled. This threshold limit assured adequate recovery of the horse between heats which reduced the chances for injury (Erickson *et al.* 1987).

³⁰ Milton Roy Co., Bausch & Lomb Spectronic 2000, Rochester, NY

³¹ Hippocard PEH2000, Bioengineering, Zurich, Switz.

³² VMAX, Equine Racing Systems, Vevay, IN

³³ Vermont Medical Inc., A-10007, Bellows Falls, VT

6. Diet

The diet was a complete pelleted ration (14% crude protein) comprised mainly of corn and soy beans with added minerals and vitamins. Each horse consumed 5.4-6.8 kg of pelleted feed per day. The hay provided was sun cured coastal bermuda hay with a crude protein content of 10-12%. Each horse was fed 5.4-6.8 kg of hay per day. Water was provided ad libitum.

7. Statistical analyses

Statistical analyses (SAS Institute Inc, 1985) were performed on the following: peak heart rates and recovery curves, total plasma lactate levels and lactate clearance curves, speeds for each SET, and mean speeds for each 200-m in the two 1000-m SETs. The mean speed was determined by dividing the distances (200-, 400-, and 1000-m) by the time required to cover that distance.

Blood lactate concentration plotted against time produced an exponential curve. Analysis of variance was performed on the fast phase of the lactate clearance curves, which was the 5th (peak lactate) to the 60th minutes, to determine the best curve fit. The lactate clearance curves for each group in

each SET were compared to determine differences in rates of lactate disappearance. Significance was established at $P < 0.05$.

The heart rate data plotted against time also produced an exponential curve. The fast-phase of the heart rate recovery curve leveled off at about 2.5 minutes after the end of the run. The fast-phase portion of each curve was linearized and an analysis of variance was performed on the regression equation for this line to determine how accurately the equation described the regression ($P < 0.05$). The slopes of the regression lines for each group in each SET were compared to determine differences in heart frequency recovery rates ($P < 0.05$).

Student's t-test was used to compare the speeds during each SET and each 200-m segment of the 1000-m SETs.

Results:

1. Lactate

There were no differences between the AT and CT groups in total plasma lactate, peak lactate, plasma lactate at each sampling time post-run, or lactate clearance rate for the unfit 400-m SET or fit 400-m SET (Table 17). Therefore,

Table 17--Mean plasma lactate concentrations and clearances (Clr) (for 400-m runs) at various distances and stages of athletic conditioning

	400-m Unfit	400-m Fit	1st 1000-m	2nd 1000-m
AT				
Total	92.75	86.23	155.30 ^a	116.48 [*]
Lac	± 2.61	± 4.19	± 6.13	± 3.14
Peak	16.08	16.30	25.60 ^{* a}	19.85 [*]
Lac	± 0.98	± 0.42	± 1.18	± 0.17
Clr	-0.2296	-0.2802		
CT				
Total	96.68	90.68	140.45	146.78
Lac	± 7.83	± 5.20	± 5.53	± 9.70
Peak	16.18	16.00	22.48	23.50
Lac	± 1.09	± 1.04	± 0.48	± 1.14
Clr	-0.2153	-0.2412		
Pooled (AT+CT)				
Total	94.71	88.45		
Lac	± 4.18	± 3.43		
Peak	16.13	16.15		
Lac	± 0.68	± 0.52		
Clr (slope)	-0.2225	-0.2607 ^b		

^{*} Significant difference (P<0.05) between AT and CT horses within the same SET

^a Significant difference (P<0.05) between 1st and 2nd 1000-m SET within a group

^b Significant difference (P<0.05) between pooled fit vs. unfit horses

AT and CT values for the unfit 400-m SET were pooled and compared with pooled AT and CT values for the fit 400-m SET to determine the effect of conditioning on these variables. There was no difference in total plasma lactate, peak lactate, or plasma lactate at each sampling time post-run when pooled unfit 400-m SET values were compared with pooled fit 400-m SET values. However, there was a significant increase in plasma lactate clearance rate (slope) for the pooled fit 400-m SET compared to the pooled unfit 400-m SET (Table 17). A linear regression best described the pooled fit and pooled unfit lactate clearance rates. This is illustrated in Figure 4 by the greater slope for the fit group compared to the unfit group.

Following the first 1000 m run, there was no significant difference between the AT and CT groups for total plasma lactate. However, plasma lactate concentrations at 2 and 5 minutes post-run were significantly higher for the AT group compared to the CT group (Figure 5). Peak lactate occurred at 5 minutes post-run for both the AT and CT groups. There was also a significant increase in the lactate clearance rate (measured from the peak lactate at 5 min to 60 min after the run) for the AT group compared to the CT group following the first 1000-m run (Table 17). Quadratic equations best described the rate of lactate clearance for both groups. This is illustrated in Figure 6.

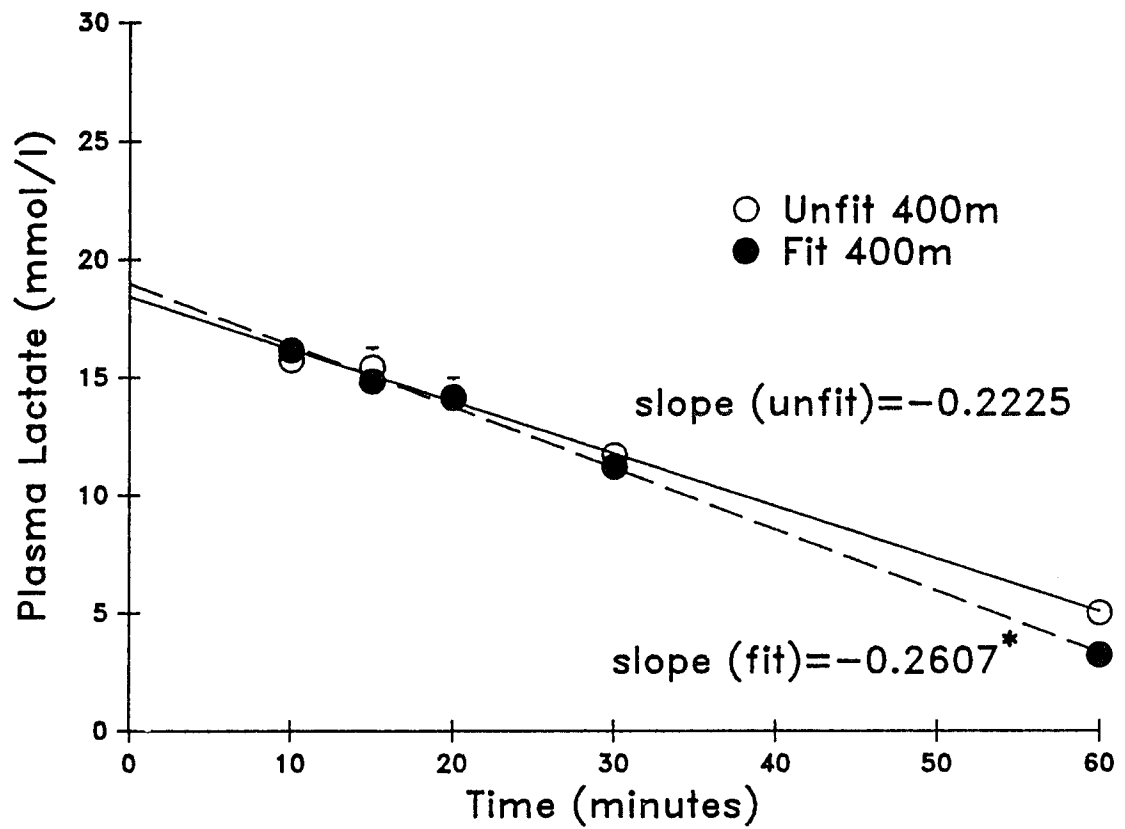


Figure 4--Lactate clearance for the pooled unfit and pooled fit horses (after 5 mo. training) during the 400-m SETs. Each point represents an average of 8 horses \pm SEM.

* Significant difference ($P < 0.05$) between slopes of unfit vs. fit horses

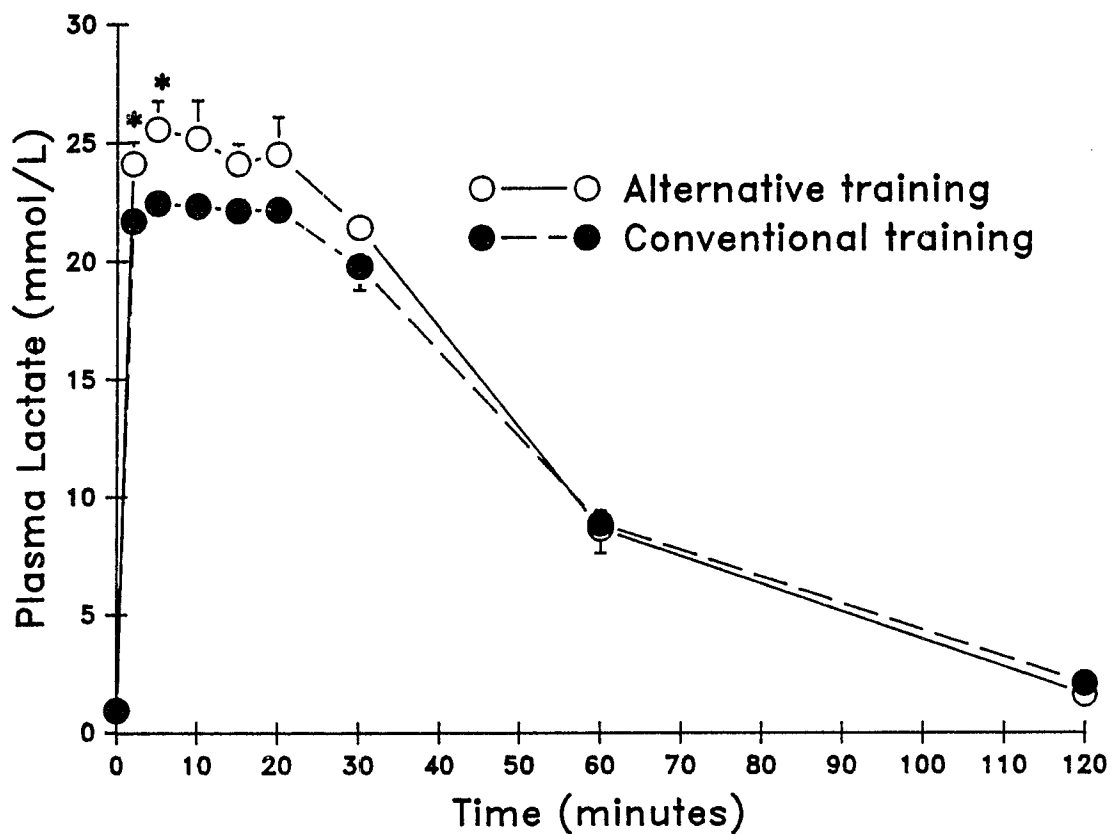


Figure 5--Plasma lactate levels (mean \pm SEM) over time following the first 1000-m SET. Each point represents the average of 4 horses \pm SEM.

* Significant difference ($P < 0.05$) between AT and CT groups

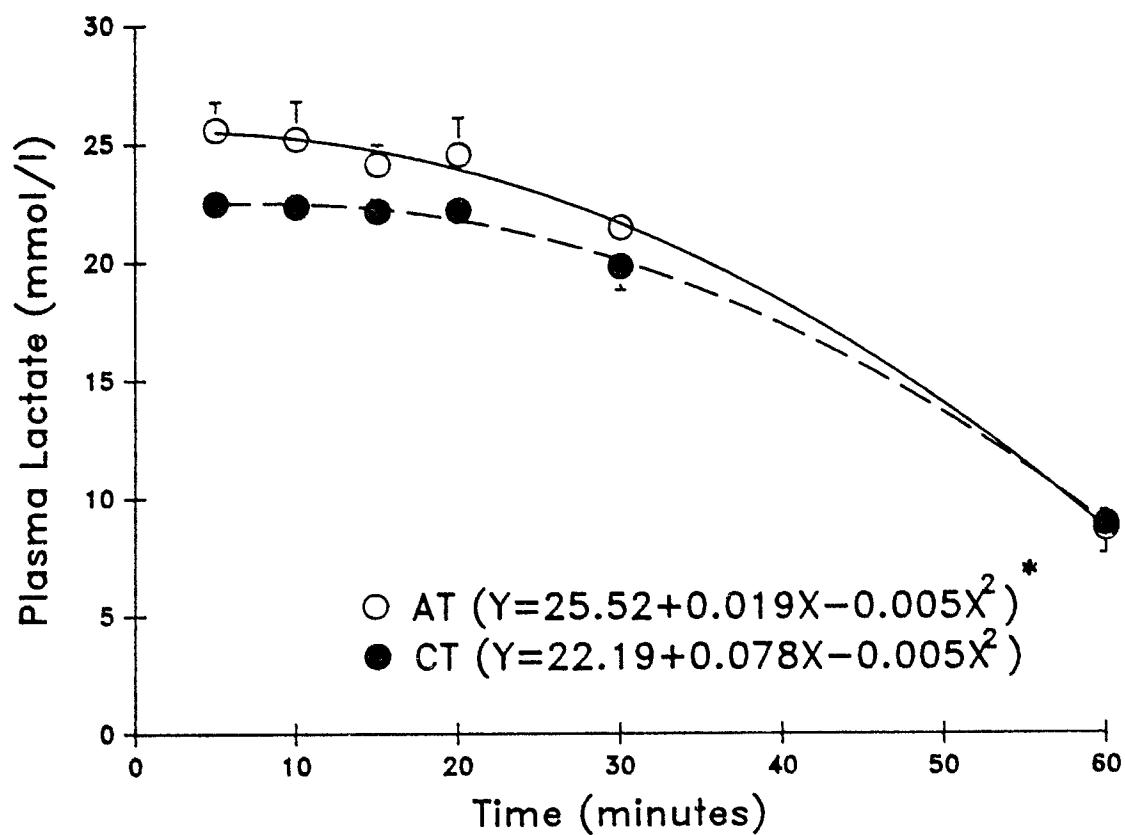


Figure 6--Calculation of lactate clearance rates from 5-60 minutes following the 1st 1000-m SET for the AT and CT horses. Each point represents the average of 4 horses \pm SEM.

* Significant difference ($P<0.05$) between clearance rates for AT and CT groups

Following the second 1000-m run, there was a significant decrease in total and peak plasma lactate for the AT group compared to the CT group (Table 17), and a decrease in plasma lactate at 2, 5, 10, 15, 20, 30, and 60 minutes after the run (Figure 7). Quadratic equations best described the rate of lactate clearance for both groups. There was no difference between groups in the lactate clearance rates following the second 1000-m run (Figure 8, Table 17).

When total plasma lactate and plasma lactate at each sampling time for the first 1000-m run were compared to those values obtained for the second 1000-m run, there was no difference between runs for the CT group. However, for the AT group, there was a significant decrease in peak and total plasma lactate, and plasma lactate measured at 2, 5, 10, 15, 20, and 30 minutes after the second 1000-m run (Figure 9, Table 17) compared to the first 1000-m run.

2. Heart rate

Heart rate data were statistically compared among the various test runs. Mean peak heart rates and heart rate recovery slopes (regression coefficients) are reported in Table 18. There were no significant differences in peak heart rate between unfit and fit horses or between AT and CT groups at the different periods of training or test run distances (Table 18).

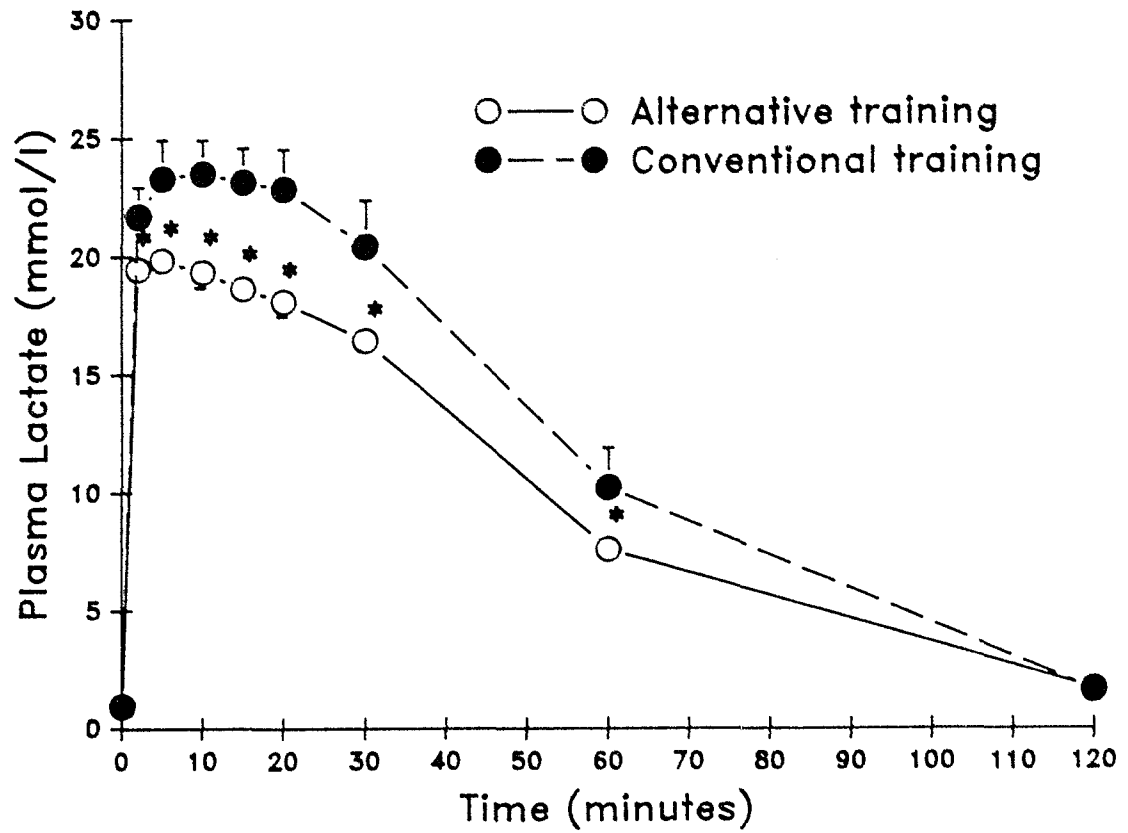


Figure 7--Plasma lactate levels (mean \pm SEM) following the 2nd 1000-m SET. Each point represents the average of 4 horses \pm SEM.

* Significant difference ($P < 0.05$) between AT and CT groups

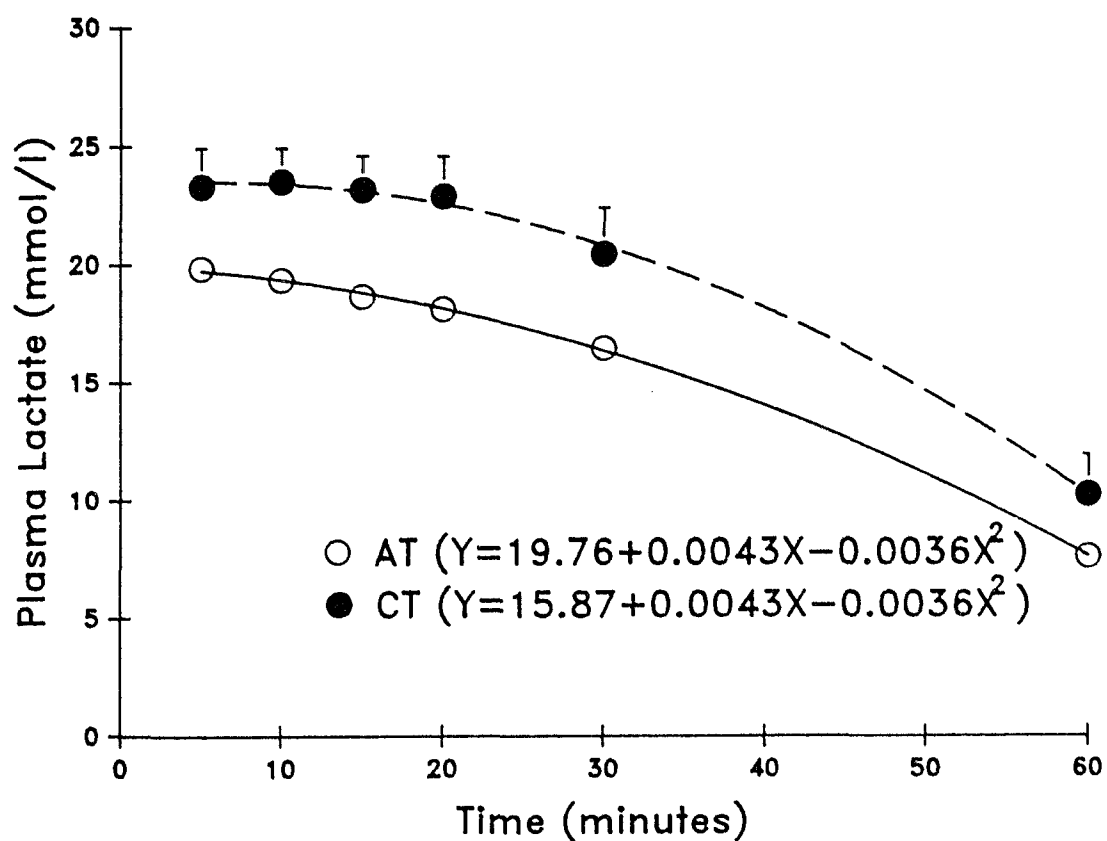


Figure 8--Calculation of lactate clearance rate from 5-60 minutes following the 2nd 1000 m run for the AT and CT groups. Each point represents the average of 4 horses \pm SEM.

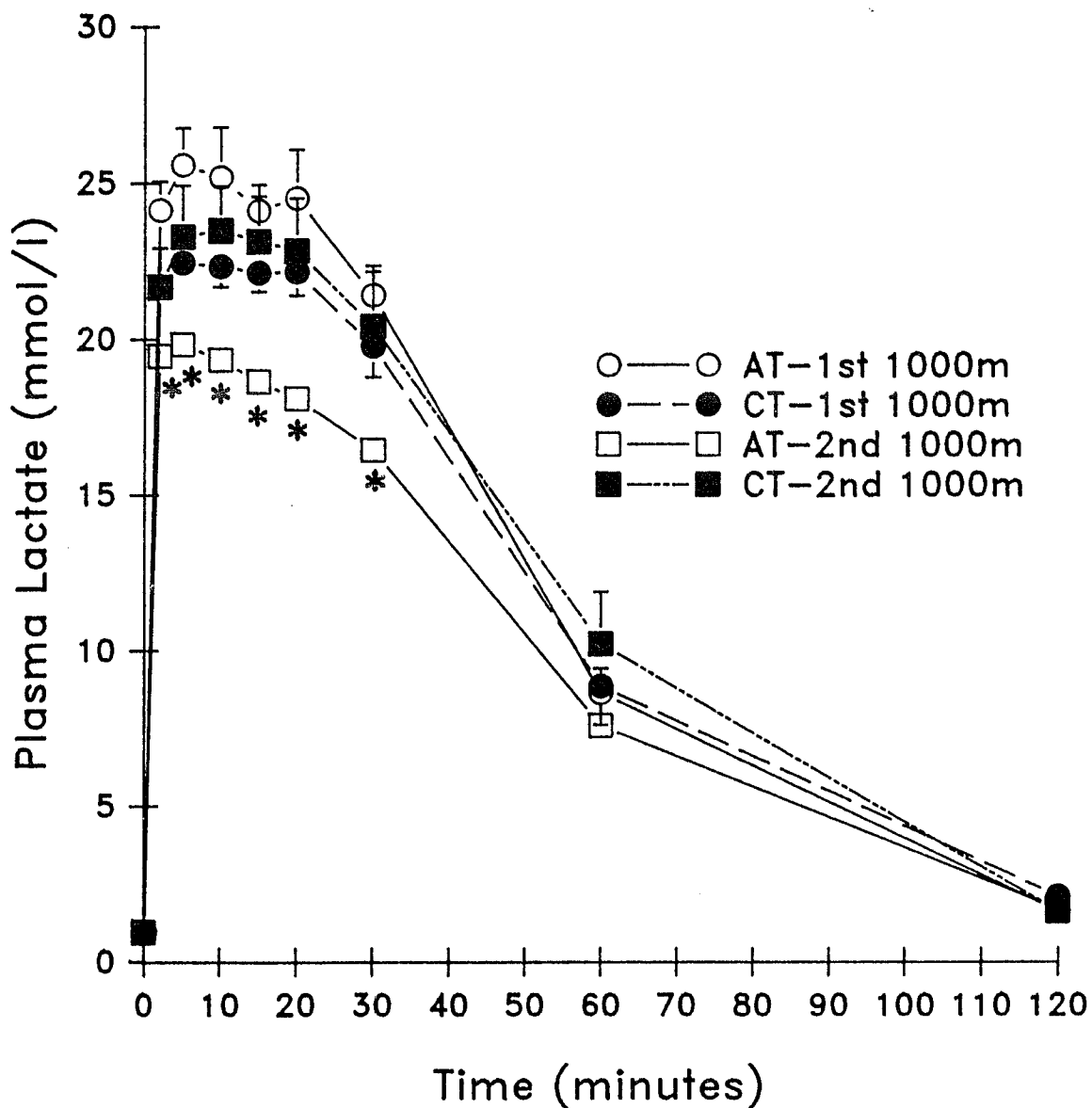


Figure 9--Plasma lactate levels for AT and CT groups following the 1st and 2nd 1000-m SETs (Figs. 5 and 7 combined).

* Significant difference ($P < 0.05$) between 1st and 2nd 1000-m SET for AT group

3. Standard exercise test speed

There was no significant difference in the running speed for the two groups during any SET and no significant differences in the speeds for each 200-m segment of the 1000-m SETs (Figure 10). There was no significant difference when the pooled race times for the unfit 400-m SET was compared with the pooled race times for the fit 400-m SET after 5 months of training. The recorded times for each SET and each 200-m segment of the 1000-m SETs are reported in Table 19.

There was no difference in the rates of deceleration when the decrease in speed for the AT group was compared with the decrease in speed for the CT horses. Figure 10 shows the acceleration and deceleration in speed over the 1000-m SETs divided into 200-m segments. The speed at the end of each 200-m segment represents the average speed for each group of horses during that segment. However, it was interesting to note that during a maximal effort peak speed was typically achieved at a distance of 400 m and deceleration occurred in a linear fashion for the remaining 600 meters.

Table 18--Peak heart rates and regression coefficients (Slope) of the HR recovery curves in AT and CT horses at various distances and stages of athletic conditioning.

SET	400-m Unfit	400-m Fit	1st 1000-m	2nd 1000-m
AT				
Peak HR (b/min)	197 ± 3.35	199 ± 3.39	203 ± 3.57	212 ± 5.40
Slope	-0.6883	-0.6676	-0.5265	-0.6300
CT				
Peak HR (b/min)	199 ± 2.50	199 ± 6.21	206 ± 1.25	201 ± 9.11
Slope	-0.7021	-0.6259	-0.5908	-0.5630

Table 19

a. Mean time \pm SEM (seconds) for each group during each SET.

SET	AT	CT
400-m Unfit	24.19 \pm 0.86	23.70 \pm 0.50
400-m Fit	23.61 \pm 0.29	23.24 \pm 0.30
1st 1000-m	66.86 \pm 0.49	67.36 \pm 0.63
2nd 1000-m	66.09 \pm 0.48	66.73 \pm 0.47

b. Average speed \pm SEM (meters/min) for each 200-m segment.

SET	200-m segment	AT	CT
1st 1000-m	0-200	921.0 \pm 7.24	918.5 \pm 15.37
	200-400	978.3 \pm 6.83	971.3 \pm 11.70
	400-600	944.4 \pm 8.70	936.8 \pm 18.93
	600-800	860.1 \pm 9.17	863.9 \pm 10.42
	800-1000	806.9 \pm 12.75	789.9 \pm 10.56
2nd 1000-m	0-200	931.2 \pm 7.39	918.7 \pm 21.07
	200-400	998.6 \pm 9.01	962.1 \pm 24.16
	400-600	942.6 \pm 8.48	931.8 \pm 13.07
	600-800	880.7 \pm 8.86	869.6 \pm 15.03
	800-1000	809.3 \pm 18.59	804.8 \pm 13.00

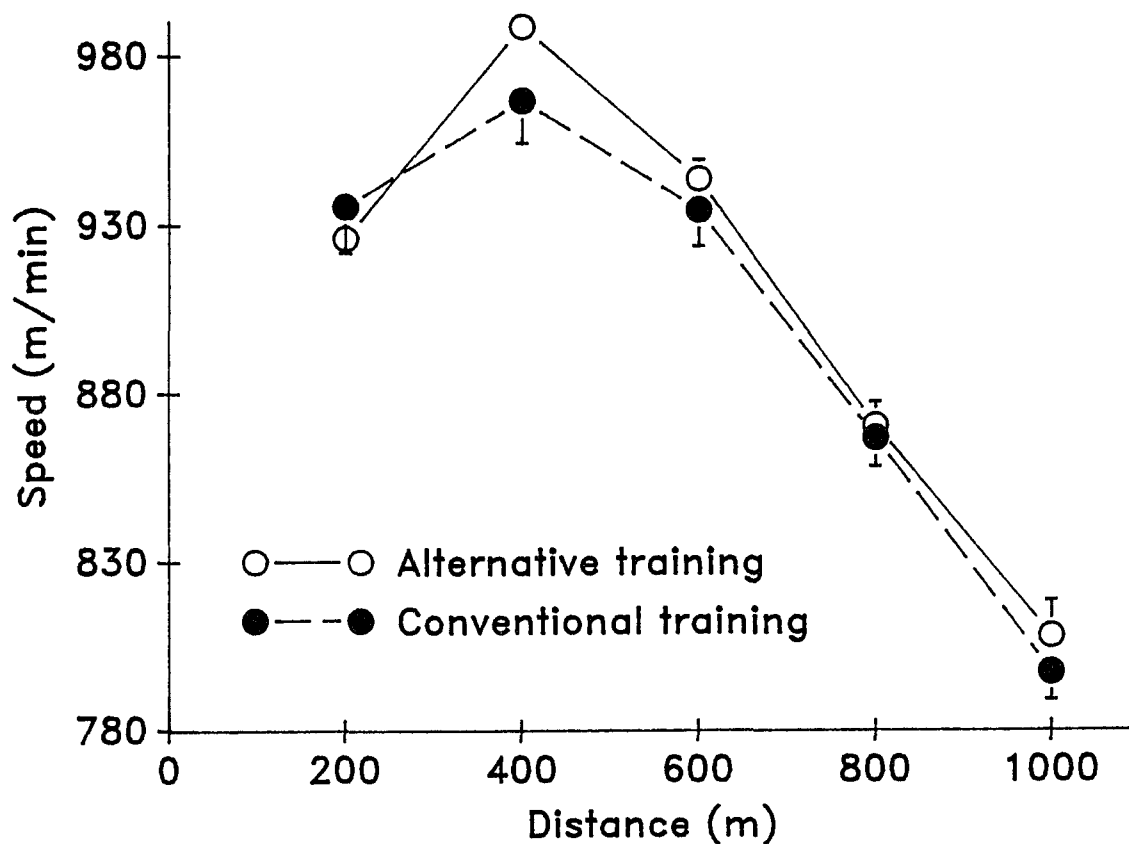


Figure 10--Acceleration and deceleration curves for AT and CT groups during the 1000-m SETs. The curves are a compilation of both 1000-m SETs. There was no difference in run times between 1st and 2nd SETs within or between groups.

Discussion:1. Lactate

Many investigators have studied the rate of lactate production and clearance from blood in horses and man (Bayly *et al.* 1987a; Gollnick *et al.* 1986; Judson *et al.* 1983; Snow *et al.* 1985; Thornton *et al.* 1983). In severe exercise, there is a dramatic increase in the blood lactate level from its resting value of about 1 mmol/l (Gollnick *et al.* 1986; Gottlieb *et al.* 1988). Hermansen and Vaage (1977) reported plasma lactate levels in man of 30 mmol/l following exercise. Snow *et al.* (1985) observed plasma lactate levels in horses of 35 mmol/l following maximal exercise.

The increased rate of lactate clearance for the pooled trained horses (following the fit 400-m SET) when compared with the pooled untrained group (following the unfit 400-m SET--Figure 4) was consistent with the findings of other investigators (Bayly *et al.* 1987a; Miller & Lawrence, 1987). Donovan and Brooks (1983) showed that training dramatically increased the clearance rate of lactate from the blood. Furthermore, adequate training of horses reduced the plasma lactate levels following a sub-maximal standard exercise test (Miller & Lawrence, 1987). The lowered plasma lactate concentration was probably not the result of a reduction in the amount of lactate produced but

was due to an increase in the rate of lactate removal (Donovan & Brooks, 1983).

The increased rate of lactate clearance seen in the AT group following the first 1000-m SET (Figure 6) indicated a more efficient clearance of lactate by that group. Donovan and Brooks (1983) contended that there was no difference in the amount of lactate produced during a sub-maximal SET before training when compared to lactate production during a SET after training. They maintained that the difference in plasma lactate levels was explained entirely by the increased efficiency in the rate of lactate clearance that occurs as a result of training, specifically, the maintenance of splanchnic blood flow during higher levels of activity in better trained subjects. In untrained animals, blood flow to the splanchnic area is shunted away to the working muscles at a lower intensity of exercise, thereby reducing the lactate removing capability of the liver and kidneys.

The elevated peak lactate levels seen in the AT group after the first 1000-m SET (Figure 5, Table 17) may have been evidence of a higher level of conditioning in that group. In human studies, well conditioned subjects produced higher peak lactates than less trained individuals during a maximal-effort SET (Edwards, 1983). The potential for higher peak lactate concentration was a result of training at or above the "onset of blood lactate"

(OBLA) level. Fast twitch fibers, especially type IIb fibers, have the highest potential for lactate production, and are the last fibers recruited during a maximal effort event (Baldwin *et al.* 1977). To improve the anaerobic power potential of muscle cells, it is necessary to expose the cells to overload stresses that are greater than the work levels at which the cells are currently being trained. Therefore, the work loads have to be progressively increased for continued performance improvement. While both groups of horses in this project probably performed the majority of their training above OBLA level, the intense training regimen for the AT group allowed for a greater amount of work above this threshold level.

The reasons for the wide variation in plasma lactate for the AT group between the first and second 1000-m runs (Figure 9), but not in the CT horses in this project, were not readily apparent. There were no systematic errors performed in the lactate analysis which could have explained this difference. The lowered plasma lactate levels in the AT group could have been due to 1) decreased anaerobic glycolysis, 2) alteration in the rate of lactate efflux from the muscles, 3) increased rate of lactate clearance by the liver, 4) increased rate of lactate utilization by the muscles, or 5) a combination of any of the above. It would have been interesting to have performed a third 1000-m SET 1 week later to assess any changes in plasma lactate following that run.

The rate of plasma lactate clearance is determined by the lactate concentration in the muscle cells, the rate of blood flow to muscles, the amount of lactate utilized for fuel by the heart and skeletal muscles, and blood flow to the liver and kidneys (Jorfeldt *et al.* 1978). Raising the concentration of lactate in the blood increases the rate of blood lactate removal in trained and untrained subjects (Donovan & Brooks, 1983; Dodd *et al.* 1984). Trained subjects are able to maintain a higher rate of lactate clearance at a lower plasma lactate concentration. Less fit subjects must attain higher blood lactate levels to realize a similar higher rate of lactate clearance (Donovan & Brooks, 1983).

Since lactate clearance rate is partially determined by plasma lactate concentration (Donovan & Brooks, 1983), the lower plasma lactate levels for the AT group following the second 1000-m SET (Table 17) may have been expected to result in a lower rate of lactate clearance. However, there was no difference in the lactate clearance rates for the two groups following the second 1000-m SET (Figure 8). The expected lower lactate clearance rate may have been offset by the fact that the AT group was more intensely trained and thereby maintained a higher degree of splanchnic circulation during the SETs.

2. Heart rate

Exercise physiology studies in human athletes have shown that the heart rates of better conditioned individuals have a faster return to normal following exercise (Gould & Dye, 1935). In a review by von Engelhardt (1977), it was stated that the heart rates of better trained horses returned to resting levels faster than the rates of less trained horses following sub-maximal exercise. Miller and Lawrence (1987) also showed that the heart rates of conditioned horses returned toward normal levels faster than the same horses in an unconditioned state following sub-maximal exercise.

The lack of any significant differences in peak heart rates or heart rate recovery curves between AT and CT horses may have been due to equivalent degrees of cardiovascular fitness produced by both training methods. The lack of a significant difference in heart rate recovery curves between the pooled unfit and pooled fit horses (after 5 mo. of training) may have been because 400-m was not a long enough distance to reveal any differences due to training. A longer distance run at maximal effort would probably create a greater oxygen debt in both fit and less fit horses resulting in a prolonged heart rate recovery. However, the fit group would be expected to have a faster heart rate recovery than the less fit group because of greater oxygen delivery to muscles

and increased clearance of lactate from exercising muscles (Donovan & Brooks, 1983).

3. Run speeds

The speeds of the AT horses did not vary significantly from the speeds of the CT horses for the 400- and 1000-m distances (Figure 10, Table 19a). The small sample size, relatively short run distances, and wide range of running abilities within each group may have precluded the detection any significant differences in running speeds due to training methods alone.

Since a delay in the onset of fatigue has been one of the alleged advantages of IT, fatigue was assessed by comparing the decrease in speed for each 200-m segment. Figure 10 is a composite of both 1000-m SETs and shows the initial increase in speed up to 400-m, followed by a gradual decrease in speed for each 200-m segment thereafter. Although there was not a significant difference in speed for each 200-m segment between groups, the speed for the AT group during the last 200-m segment appeared to decrease at a slightly slower rate than the speed for the CT group. At a longer distance, the speed for the CT group might have continued to decrease at a greater rate than the AT group resulting in a statistical difference in run times. The jockey that rode the

horses during the SETs commented that the AT horses "finished stronger" than the CT horses during the 1000-m SETs indicating that he felt the AT horses tired to a lesser degree than the CT horses.

4. Horses

The horses used in this study had been donated to the School of Veterinary Medicine and were of varying abilities. Before the study began, it was uncertain that the horses could endure the intense work load required for interval training without becoming lame. Although the horses were sound when donated, some had been donated because of an inability to remain sound during normal training. Other horses had been donated because they lacked speed. Therefore, the horses used were not top racetrack performers.

Training was done on an 800-m training track with tight turns and minimal banking in the curves. All of these factors prevented training times from being faster. The training schedule and heat times were meant to serve as a guide for designing an alternative training program and not as a definitive timetable. Horses with greater racing potential could be worked at faster speeds with more demanding schedules.

5. Complications

The most commonly seen complication during LSD was occasional swelling of the forelimb fetlock joint capsules and tendon sheaths that was probably due to the longer distances being jogged during the LSD phase. The affected legs were massaged with a camphor liniment and wrapped with support bandages to reduce the fluid accumulation. Two of the four AT horses developed edema in the front legs but showed no lameness so the training was continued. After six weeks of training, these swelling problems did not recur even though the speed and the total distance galloped each day were steadily increased. The swelling probably subsided because the supporting tissue structures of the legs were strengthened, the production of inflammatory mediators due to tissue damage was minimized, and venous and lymphatic circulation from the leg improved after the first several weeks of LSD.

One AT horse exhibited exercise-induced pulmonary hemorrhage (EIPH) routinely during the LSD phase and the early part of Phase 2. That horse was not expected to be able to finish the program, but improved significantly after 4 months of training. The horse rarely exhibited any hemorrhage during the final half of training, which occurred during the cooler fall months. The milder weather may have contributed to the absence of EIPH. There were no other complications that were unique to the AT group of horses.

Two of the AT horses and one of the CT horses experienced mild respiratory infections, and there were a few leg lacerations common to horses in training. All horses that began the AT and CT programs finished without any serious injury or disability.

6. Jockey

One of the most difficult aspects of interval training is finding a rider willing to stay on the horses for the full time required to gallop the prescribed distance. At the end of Phase 1 when the horse was galloping 9600-m a day, the rider spent about 30 minutes aboard each horse, which was tiring work even for a young, experienced rider.

7. Trainer

Interval training was more labor-intensive than conventional training for the trainer as well as the jockey. The trainer must attend all the work sessions because each horse has to be evaluated for excessive fatigue or hyperthermia following each heat. The work schedule for each horse was planned and

modified on a daily and individual basis. This was necessary to prevent serious injury from overwork.

8. Heart rate monitor

Portable heart rate monitors have been used to assess stress in horses in training (Evans & Rose, 1986; Foreman & Rabin, 1984). A heart rate monitor was essential for evaluating the cardiovascular stress encountered during the intense training sessions of Phase 3. During that phase, the heart rate reached near-maximal level of 210-220 beats per minute during each heat. The heart rate monitor provided a means for evaluating how quickly the horse recovered following each heat. The established heart rate recovery threshold of 110 beats/min (within two minutes of the end of the heat) was exceeded a few times in late summer when temperature and humidity were elevated, and the subsequently scheduled heats were canceled. With one horse, an exception to this rule was made. It was difficult training this particular horse because of its desire to run at maximal effort during each training session. The other horses began to relax after one heat with heart rates consistently returning to below 110 following each heat. However, the aberrant horse typically became more excited with subsequent heats. Because of this emotional artifact, a

higher heart rate recovery threshold of 125 beats per minute was established for that horse.

More consistent results were obtained with the VMAX monitor than the Hippocard. The Hippocard frequently recorded erroneous readings during high speeds. Part of the problem was due to excessive movement of the Hippocard electrodes during galloping. The self-adhering electrodes of the VMAX system had minimal movement and provided more reliable results.

9. Alternative training

Light to moderate work following an intense exercise event increases the rate of lactate removal by increasing blood flow to the fatigued muscles and mobilizing the accumulated lactate for fuel (Weber *et al.* 1987). The rest periods enabled the horse to undergo more high-intensity training than would have been possible during conventional training by allowing a partial clearance of lactate from the muscles during the light-intensity exercise (rest) periods (Bayly, 1985).

The reason the AT horses were able to safely endure the work intensity of that program may have been because the work loads were increased gradually and

individually. The major precaution for AT is that the training regimen of each horse must be continually revised.

Because of the time and labor involved in the AT protocol, it does not appear to be a practical method of training for all horses. Horses racing in claiming races, which are the most common races in the U.S., may not be good candidates for AT because the trainer could lose the 6-9 months invested in training the first time the horse started a race. Alternative training regimens requiring a shorter period of time have been used (Sexton *et al.* 1987; Thornton *et al.* 1983), but a reduced amount of time for LSD and IT fails to properly prepare the support structures of the horse for the stresses encountered during racing.

Section D

Assessment of Treadmill Interval Training on Fitness

Objectives:

1. To develop interval training methods for the high-speed equine treadmill.
2. To show the effects of interval training on heart rate with sub-maximal standard exercise tests prior to and following 20-weeks of treadmill training.

Materials and Methods:

1. Horses

Nineteen healthy, Thoroughbred horses (10 geldings and 9 mares) ranging in age from 3 to 9 years and weighing between 406 and 540 kilograms were used in this exercise study. All horses had previous racing and/or training experience, but were not high quality racehorses. The horses were trained on a high-speed treadmill³⁴ using interval training techniques adapted from the interval training study on the training track described in Section C. None of

³⁴ Mustang 2200, Kagra International Inc., Fahrwangen, Switz.

the horses had been involved in a training program for at least six months prior to the onset of this study.

2. Training schedule

The twenty week training period is detailed in Tables 20a and 20b. During the first week, each horse was harnessed and introduced to the treadmill for the first time. During this week, each horse was walked on the treadmill at a speed of 1.5 m/s for 2 min, trotted at 3.5 m/s for 10 min, then walked at 1.5 m/s for a 2-min warm-down for a total distance of 2370 meters/day for 5 consecutive days then rested 2 days (Table 20a). This routine was repeated during the next 2 weeks except that speed was gradually increased so that at the end of week 3 each horse was trotted at 4.5 m/s and covered a total distance of 3070 m for 5 days/week. During the entire training period each horse received the same schedule of 5 days of training and 2 days rest per week.

Each horse was galloped for the first time on the treadmill during week 4. The distance and speed of the daily gallop were gradually increased from an initial speed of 7.6 m/s for 5 min (2280 m) during week 4 to 9.0 m/s for 10 min (5200 m) during week 11. This was performed for 5 consecutive days each week. A

Table 20a--Pre-interval training schedule (weeks 1-12)

Wk	<u>Walk</u> (1.5 m/s)	<u>Trot</u> (4.0 m/s)	<u>Walk</u> (1.5 m/s)		Total Time	Total Dist
1	180m	2100m	90m		13 min	2370m
2	180m	2400m	90m		13 min	2670m
3	180m	2800m	90m		13 min	3070m
	<u>Walk</u> (1.5 m/s)	<u>Trot</u> (3.5 m/s)	<u>Gallop</u> Dist-Speed	<u>Trot</u> (3.5 m/s)	Total Time	Total Dist
4	90m	420m	2280m-7.6m/s	630m	11 min	3420m
5	90m	420m	3200m-7.6m/s	630m	11 min	4340m
6	90m	420m	3200m-8.0m/s	630m	11 min	4340m
7	90m	420m	4000m-8.0m/s	630m	11 min	5140m
8	90m	420m	4800m-8.0m/s	630m	11 min	5940m
9	90m	420m	4800m-8.5m/s	630m	11 min	5940m
10	90m	420m	4800m-9.0m/s	630m	11 min	5940m
11	90m	420m	5200m-9.0m/s	630m	11 min	6340m
12	90m	420m	*3360m-8.0m/s	630m	11 min	4500m

Wk=week

* Gallop was performed at 7% treadmill inclination

3-min warm-up period before and a 3-min warm-down period following the gallop were always included in the daily regimen. During week 12, the treadmill was inclined for the first time to 7% and the gallop speed was reduced to 8.0 m/s for 7 min for a total distance of 4500 m 5 days/week.

Interval training (IT) was started during week 13 and continued through week 20 (Table 20b). Interval training was performed twice a week, at a 7% incline, with progressively faster heats. Heats were separated by warm-down periods lasting 2-4 min depending on the time required for the heart rate (HR) to return below a threshold level of 110 beats/min. During the warm-down periods, each horse was trotted at 3.5 m/s at a 0% incline. If the HR did not return to the threshold level after 5 min, the speed was further reduced to 1.5 m/s. If the HR did not return below 110 beats/min within 1 minute at 1.5 m/s, further training was canceled for the day.

The first IT session during week 13 consisted of two 1600-m heats run at 9.0 m/s and 9.5 m/s, respectively, separated by a 4-min warm-down period. The total distance for this training session was 5210 m. The second training session was performed 3 days later and consisted of the same routine.

The number of heats was maintained at 2 for weeks 13-14, then increased to 3 during weeks 14-18, and finally increased to 4 during weeks 19-20. As the

Table 20b--Treadmill interval training schedule

On interval training days (weeks 13-20):

Wk	Ht	Walk (1.5 m/s)	Trot (3.5 m/s)	Gallop Speed-Dist	Trot (3.5 m/s)	Total Time	Total Dist
13	1	90m	420m	9.0m/s-1600m	700m		
	2			9.5m/s-1600m	800m	16 min	5210m
14	1	90m	420m	10.0m/s-1600m	700m		
	2			10.5m/s-1600m	800m	15 min	5210m
15	1	90m	420m	11.0m/s-1200m	600m		
	2			11.3m/s-1200m	600m		
	3			11.8m/s-1200m	600m	17 min	5910m
16	1	90m	420m	11.5m/s--800m	600m		
	2			11.8m/s--800m	600m		
	3			12.0m/s--800m	600m	15 min	4710m
17	1	90m	420m	12.0m/s--800m	600m		
	2			12.3m/s--800m	600m		
	3			12.5m/s--800m	600m	15 min	4710m
18	1	90m	420m	12.5m/s--800m	600m		
	2			12.8m/s--800m	600m		
	3			13.0m/s--800m	600m	15 min	4710m
19	1	90m	420m	13.0m/s--600m	600m		
	2			13.3m/s--600m	600m		
	3			13.6m/s--600m	600m		
	4			13.9m/s--600m	600m	18 min	5310m
20	1	90m	420m	13.7m/s--600m	600m		
	2			14.0m/s--600m	600m		
	3			14.3m/s--600m	600m		
	4			14.6m/s--600m	600m	18 min	5310m

Wk=week, Ht=heat

Interval training was completed 2 days/wk

Gallops were performed at 7% treadmill inclination

HR < 110 beats/min during warm-down period

On non-interval training days (weeks 13-20)

Walk (1.5 m/s)	Trot (3.5 m/s)	Gallop (9.0 m/s)	Trot (3.5 m/s)	Total Dist/ Time
90m	420m	3200m	630m	4340m
1 min	2 min	6 min	3 min	12 min

number of heats increased, the distance was shortened and the speed was progressively increased. By week 20, speed for each of the four heats had increased to 13.7 m/s, 14.0 m/s, 14.3 m/s, and 14.6 m/s, respectively, compared to the initial 9.0 m/s for the first heat of the first training session. Distance had also been reduced to 600 m/heat from the initial 1600 m/heat. Total distance worked during all training sessions did not change substantially. During this 8 week period (week 13-20) each horse was trained twice a week at a 7% inclination with 3-4 days between training sessions.

On days when the horses did not undergo IT during weeks 13-20, a more conventional exercise routine was performed. This consisted of a walk at 1.5 m/s for 1 min (90 m), a trot at 3.5 m/s for 2 min (420 m), a gallop at 9.0 m/s for 6 min (3200 m), and a warm-down at 3.5 m/s for 3 min (630 m) for a total distance of 4340 m. This was performed 3 days/week, and the horses were rested 2 days/wk. All treadmill exercise on non-interval training days was done at 0% treadmill inclination.

3. Standard exercise test

To evaluate cardiac fitness a standard exercise test (SET) was performed. The SET was sub-maximal, since the HR did not reach maximal frequency, and

consisted of a 1-min walk at 1.5 m/s, a 1-min trot at 3.5 m/s, a 5-min gallop at 9.0 m/s, and a 2-min warm-down at 3.5 m/s. The treadmill was not inclined during the SET. The first SET was run during week 5, and the second SET was run during week 20.

4. Heart rate

Heart rates were measured by an on-board heart rate (HR) monitor³⁵ supplied with the treadmill. Two electrodes for the HR monitor were attached to a girth strap and placed on the sternum and left side of the horse's chest midway between the sternum and withers. The hair at these two areas was clipped and generous amounts of electrode gel were applied to the electrode pads to insure adequate transmission of the cardiac signal. The HR was transmitted via telemetry from a transmitter in the girth strap to two receivers mounted on the treadmill panel.

The HR was automatically relayed every 5 sec to the display panel above the treadmill and to the computer for storage. During the SET 12 HR recordings were obtained during the walk (1 min), 12 during the trot (1 min), 60 during the gallop (5 min), and 24 during the warm-down (2 min). Differences in heart

³⁵ Hippocard, Bioengineering of Zurich, Switz.

rates before and after training were assessed by comparing the mean number of beats for the first (pre-training) SET to the mean number of beats for the second (post-training) SET by computing the area under the HR curve (AUC) for both SETs. The AUC was calculated by integrating the HR recordings for the entire SET and for each stage of the SET (walk, trot, gallop, and warm-down). Student's t-test was used to compare the AUC for the HR of the entire SET and at each stage ($P < 0.05$).

5. Diet

The diet was a complete, pelleted ration (14% crude protein, 2585 Kcal/kg digestible energy) comprised mainly of corn and soy beans with minerals and vitamins added. Each horse consumed 6.4 kg of the pelleted feed twice a day. No hay was fed because of barn restrictions. Water was provided ad libitum.

Results:**1. Heart rate**

The HR responses during the first (pre-training) and second (post-training) SETs are illustrated in Figure 11. Note the rapid rise in HR immediately following an increase in treadmill speed. Peak heart rates were achieved within 1 minute of the start of the gallop phase. Note also the steady decrease in HR following the initial rise. This is most obvious for the gallop phase since it was longer and provided more time for a steady-state HR to be reached. The decrease in the gallop phase HR began at about the 3 min mark in the post-training SET whereas it did not appear to decrease until 4-4.5 min in the pre-training SET. Note the more rapid decrease in HR during the warm-down trot following the gallop in the second SET.

There was a significant decrease ($P < 0.01$) in AUC for the total SET (min 0-9), gallop portion (min 2-7), and warm down portion (min 7-9) during the post-training SET when compared to the pre-training SET (Table 21). There was no significant difference in AUC during the walk (0-1 min) or warm-up trot (1-2 min) portions of the pre- and post-training SETs. Mean heart rates are also listed in Table 21. There was a strong correlation ($r = 0.99$) between mean HR and AUC.

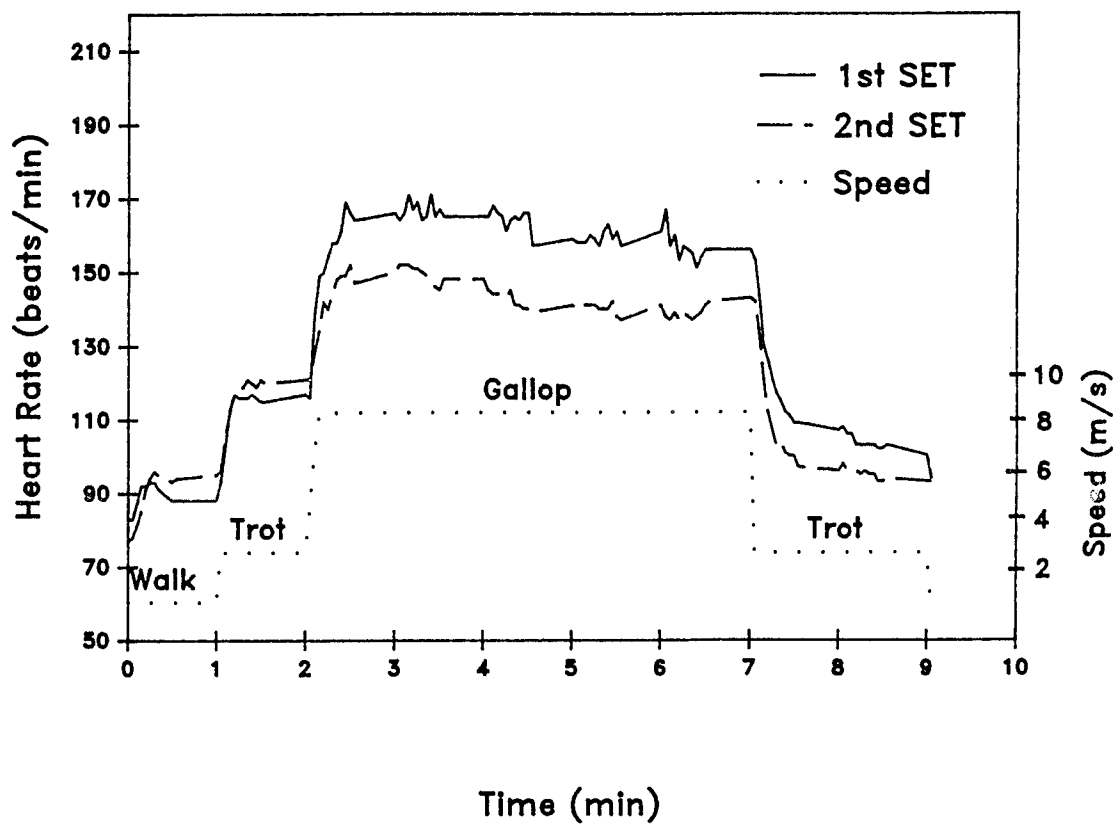


Figure 11--Mean heart rates for horses during the Pre HR-SET (performed during week 5 of training period) and Post HR-SET (performed during week 20 of training period).

1st SET, N=22

2nd SET, N=19

Table 21--Mean \pm SEM number of heart beats and mean HR (beats/min) for the different phases of the standard exercise tests (SETs).

Portion of SET Measured	No. of Beats (Mean \pm SEM)	Heart Rate (Mean \pm SEM)
1st SET--total (0-9 min)	1242 \pm 23	136 \pm 1
2nd SET--total (0-9 min)	1138 \pm 23 *	125 \pm 1
1st SET--walk (0-1 min)	91 \pm 3	91 \pm 1
2nd SET--walk (0-1 min)	89 \pm 4	89 \pm 1
1st SET--trot (1-2 min)	115 \pm 3	116 \pm 1
2nd SET--trot (1-2 min)	113 \pm 4	112 \pm 1
1st SET--gallop (2-7 min)	801 \pm 15	160 \pm 1
2nd SET--gallop (2-7 min)	713 \pm 18 *	143 \pm 1
1st SET--warm down (7-9 min)	225 \pm 7	112 \pm 1
2nd SET--warm down (7-9 min)	205 \pm 4 *	102 \pm 1

Numerical values are expressed to the nearest whole number
N=19

* Significant difference (P<0.01) between 1st and 2nd SET

Discussion:

1. Heart rate

The first SET was run during the fifth week of treadmill training while the horses were still relatively unconditioned. This SET was delayed until the fifth week to allow the horses time to adapt to the treadmill room surroundings and the motion of the treadmill. This adaptation period helped to prevent lameness and precluded false elevation of HR due to excitement. The steady-state HRs during the gallop varied from 142-187 beats/min (Figure 11) between horses. A maximal speed of 9.0 m/s at 0% inclination during the first SET was considered to be moderately strenuous in unconditioned horses even though the HRs were not near-maximal. A main objective when designing the SET was to prevent musculoskeletal injury during the 5-min gallop.

The second SET was performed during week 20. Three of the horses showed less than a 10 beats/min improvement in mean HR, while 16 demonstrated a 10-27 beats/min improvement in mean HR during the second SET. The significant decrease in HR during the second SET was in agreement with the findings of other investigators who used HR as an indicator of fitness. Sexton *et al.* (1987) showed a significant reduction in mean HR during a standard exercise test at 2.8 m/s (12% grade) from 180 ± 7 beats/min to 150 ± 3

beats/min following 12 weeks of long slow distance and interval training. Bayly *et al.* (1983a) showed a reduction in mean HR in 7 Standardbred horses following 11 weeks of treadmill training during a SET at 0% inclination and a speed of 2.6 m/s from 179 ± 29 beats/min before training to 139 ± 11 beats/min following training. Rodiek *et al.* (1987) showed a decreased mean HR in 9 Quarter horses after 10 weeks of treadmill training during a SET at 3 % inclination and a speed of 3.3 m/s from 133 beats/min before training to 124 beats/min following training. These studies measured HR during a trot to assess fitness, although work load was increased with an inclined treadmill. Thornton *et al.* (1983) showed that the V_{200} (the calculated speed at which a steady-state HR of 200 is maintained) of 5 Standardbred trotters was increased from 7.35 ± 0.45 to 7.92 ± 0.56 m/s following 5 weeks of interval training on the treadmill.

The mean HR during the gallop phase of the SET was reduced from 160 ± 1 beats/min in the first SET to 143 ± 1 beats/min during the second SET. The amount of work performed was identical for the two SETs, so the lowered HR during the second SET illustrated the increased cardiac efficiency in conditioned horses.

2. Interval training

A few scientific studies have shown the benefits of IT in Standardbred racehorses (Lindholm & Saltin, 1974b; Thornton *et al.* 1983), Thoroughbred racehorses (described in Section C), and cutting horses (Webb *et al.* 1988). The study described in Section C concluded that IT on the track is more labor intensive than other conventional methods of training, and may be practical only in higher quality racehorses. Interval training is used almost exclusively in human athletes competing in middle-distance (400-800 m) events (Brooks & Fahey, 1985), which require 45-110 sec to complete. Most Thoroughbred races cover a distance of 1000-2000 m (5-10 furlongs) and require 50-120 sec to complete. Despite the similarity between human middle-distance events and Thoroughbred races, and the proven benefit of IT in human middle-distance athletes, IT has been ignored by the majority of Thoroughbred trainers. As the benefits of IT of Thoroughbred racehorses become more evident, trainers will begin to utilize the principles of IT. One of the disadvantages of IT is the increased labor required. With the treadmill that drawback is reduced.

3. Treadmill

There are many advantages to using the treadmill as a training tool. The treadmill belt is always dry with a consistent surface to insure proper footing. Track maintenance is reduced. A rehabilitating horse can be exercised without having to bear the weight of a jockey. Since a jockey is not required, the expertise of a rider is eliminated and the labor needed for applying and removing riding tack is reduced. The treadmill can be inclined to increase the work load without increasing speed. Since a primary cause of lameness in Thoroughbred racehorses is damage to weight bearing structures due to excess forces on those structures (Stashak, 1987), maintaining a lower exercising speed should decrease the chance of injury. Housing the treadmill in a climate regulated building allows for environmental control of the exercising horses.

There are also disadvantages to exercising horses on the treadmill, the most obvious being the initial costs of the treadmill and the building in which it must be housed. The treadmill requires 40-45 seconds to reach maximal speed of 14.5 m/s, but a horse can reach maximal speed (16-18 m/s) on a racetrack in about half that time, so the rapid acceleration necessary for winning sprint races cannot be simulated on the treadmill. Since the horse does not move relative to the ambient air, there is no air resistance to overcome and no cooling effect of wind while the horse is galloping on the treadmill. These 2

situations can be countered by placing fans in front of the treadmill and synchronizing the speed of the treadmill with the number of revolutions of the fans. The absence of a jockey during exercise can be beneficial to a horse recovering from injury, but in a healthy horse a jockey is of benefit to provide the weight required for racing and to perceive how the horse is performing. The horse does not set the pace of the work-out but only maintains the speed set by the treadmill. The effect of this phenomena on treadmill training has not been studied.

4. Horses

The horses adapted very rapidly to the unusual surroundings and movement of the treadmill. None of the horses had previous treadmill experience, and a few were very reluctant to walk onto the belt at first. With patience and gentle encouragement, every horse trotted on the treadmill the first day of introduction.

The horses were not galloped until week 4 to allow for adaptation to the firm treadmill surface. This was the first treadmill study for the investigators and caution was used to preclude injury to the horses. Subsequently, horses have been galloped at 10 m/s the first day on the treadmill after 10-15 minutes of

walking and cantering. It is no longer believed that a 3-week introductory period is necessary before galloping a horse on the treadmill.

5. Heart rate monitor

Heart rate monitors have been used to assess the degree of cardiovascular stress at which a horse in training is working (Evans & Rose, 1986; Foreman & Rabin, 1984). Interval training requires close monitoring of the exercising horse to prevent excessive fatigue during exercise. A heart rate monitor is necessary to apprise the trainer of the horse's level of fatigue by indicating how quickly the HR returns to a threshold level following a training heat.

During the final 4-5 weeks of training in this study, the workouts were designed for the HR to approach maximal limits. The HR monitor indicated at what intensity the horse worked during the heat and how quickly the horse recovered following the heat. A HR recovery threshold limit of 110 beats/min within 2 min of the end of the heat was established in the track IT study (described in Section C) and was implemented in this study as well.

Section E

Effect of Competition, Gender, and Age on Performance

Objectives:

1. To examine the effect of competition on running performance.
2. To examine the effects of age and sex on competitive performance.

Materials and Methods:

1. Horses

Eighteen healthy Thoroughbred racehorses (8 geldings and 10 mares) ranging in age from 3 to 8 years (Mean=5.9 yrs) and weighing between 385 and 476 kilograms (Mean=451.2 kg) were conditioned on a high-speed treadmill³⁶ over a period of 5 months using interval training techniques described in Section D. Following conditioning, the horses were stabled at a training track for completion of the solo and competitive runs. At the track, conditioning was maintained with conventional training methods.

³⁶ Mustang 2200, Kagra International Inc., Fahrwangen, Switz.

2. Runs

Each horse completed a series of 2 runs alone and 2 runs in competition over 1200 and 1600 m distances for a total of eight runs. The 1200-m run, unlike the 1600-m run, was a maximal effort run for the entire distance. The same jockey rode all horses during the solo runs. A second jockey was used for the competitive runs. Since each horse ran each distance in competition twice, the jockeys alternated horses to minimize any effect on performance due to the rider. Both jockeys were licensed professional riders with racing experience. The jockeys were instructed to ride the horses in the fastest possible time.

The solo runs were completed first and those run times were used to pair equal ability horses for the competitive runs. The runs were performed on an 800-m training track with poles dividing it into 200-m segments. Run times were recorded for each 200-m segment and for the total distance for all the runs to the nearest 0.01 second.

The 8 runs were performed over a 10 week period. (The horses also performed two 2000-m runs alone during this time that were not part of the competition study.) The experimental design allowed for 1 run each week, but because of rain and muddy track conditions some of the runs were rescheduled. The

average interval between runs was 7 days with a range of 5-14 days between runs.

3. Blood samples

Blood samples for plasma lactate analysis were collected before the runs and at 2, 4, 8, and 16 min post-run into vacutainers³⁷ containing potassium oxalate as an anticoagulant and sodium fluoride to prevent further production of lactate. The tubes were immediately centrifuged and the plasma separated for storage. The plasma samples were frozen at -20 degrees centigrade and later analyzed using a lactate analyzer.³⁸ Peak lactate was the highest single plasma lactate value recorded, and total lactate was the sum of the 5 plasma lactate values obtained.

³⁷ Becton Dickinson, No. 6445, Rutherford, NJ

³⁸ Model 23L, YSI Inc., Yellow Springs, Ohio

4. Heart rate

Heart rate was recorded at 5 second intervals during each run by an on board heart rate computer.³⁹ The device consisted of a receiver, which was worn on the wrist of the jockey during the runs, and a transmitter which was attached to the saddle pad. The transmitter was connected to two electrodes placed on shaved areas of the sternum and left side of the neck just anterior to the scapula and low enough not to interfere with the bridle reins.

5. Environmental factors

On each test day the runs were completed before 9:30 AM to minimize the effects of temperature and humidity on run times. A hygrometer-thermometer was used to measure temperature and humidity each time a horse ran. A daily track soil sample was obtained from the same 2 sites at the same time each test day for calculation of soil moisture. The soil samples were weighed before and after a 24 hr drying period (at 200 degrees C) to determine moisture content.

³⁹ VMAX, Equine Racing Systems, Vevay, Ind.

6. Diet

The diet consisted of 3.2 kg of a complete, pelleted ration (14% crude protein, 2585 Kcal/kg digestible energy) comprised mainly of corn and soy beans with minerals and vitamins added and 3.2 kg Bermuda hay. The pelleted ration and hay were fed twice a day. Water was provided ad libitum. The horses were fed the previous evenings but not on the mornings of the runs.

7. Statistics

Analysis of covariance was used to evaluate any effects on performance due to humidity, temperature, and soil moisture. Total run times for the solo and competitive runs were compared using a paired t-test after the run times had been mathematically adjusted for any environmental effects. Tests for the simple main effect of competition within each 200-m segment were performed using multivariate analysis of variance with repeated measures.

To assess the effects of sex on competitive performance, the horses were grouped by gender. Times for the solo and competitive runs for each gender group were compared using paired t-tests after run times had been adjusted for any environmental effects. To assess the effects of age on competitive

performance, the mean age of 5.9 years-old was used as the dividing age for younger and older groups. The times for the solo and competitive runs for each age group were compared using paired t-tests after run times had been adjusted for any environmental effects. Times for the younger group were compared with times for the older horses for each run using t-tests to determine any difference in performance due to age. Times for females were compared with times for males for each run using t-tests to determine any difference in performance due to sex. Significance was set at $P < 0.05$.

Duncan's multiple range test was used to compare maximal heart rate (MaxHR), peak lactate, and total lactate from the 1200-m solo, 1600-m solo, 1200-m competitive, and 1600-m competitive runs to assess any differences in those parameters.

Results:

1. Run times

Run times for all horses, and run times according to age, sex, and distance are listed in Table 22. There was no significant ($P > 0.05$) interaction for soil moisture x sequence of run, temperature x sequence of run, or humidity x

Table 22--Mean (\pm SEM) run times (seconds) for all horses, and according to age, sex and distance.

	1200 m Solo	1200 m Competition	1600 m Solo	1600 m Competition
All Horses	86.92 * \pm 0.33 (n=18)	88.87 \pm 0.33 (n=18)	119.94 * \pm 0.85 (n=17)	121.35 \pm 0.78 (n=17)
Younger (<5.9 yr)	86.09 \pm 0.69 (n=8)	86.94 \pm 0.60 (n=8)	119.58 \pm 1.10 (n=7)	120.60 \pm 0.77 (n=7)
Older (>5.9 yr)	86.34 \pm 0.59 (n=10)	87.06 \pm 0.64 (n=10)	120.21 \pm 1.23 (n=10)	121.90 \pm 1.22 (n=10)
Males	84.80 ^a \pm 0.55 (n=8)	85.20 ^a \pm 0.56 (n=8)	117.72 ^a \pm 0.94 (n=8)	118.84 ^a \pm 0.91 (n=8)
Females	87.37 * \pm 0.55 (n=10)	88.45 \pm 0.45 (n=10)	122.04 * \pm 1.20 (n=9)	123.71 \pm 0.95 (n=9)

* Significant difference ($P < 0.05$) between solo and competitive run times within a given distance.

^a Significant difference ($P < 0.05$) in run times between genders within a given distance.

sequence of run for the 1200- or 1600-m runs. No environmental factors significantly affected performance during the 1600-m runs, but temperature did have a significant effect on performance during the 1200-m runs.

The analysis of covariance procedure determined that there was a linear relationship between the dependent variable (run time) and the covariate (temperature). A statistical manipulation (Freund *et al.*, 1986) was performed to correct for the effect of temperature on the run times by adjusting the run times as if all runs had been performed at the same temperature. The correction factor used to adjust the run times was determined from the linear regression of temperature and run times.

After the 1200-m runs were adjusted for the effect of temperature, the mean time for the competitive runs was significantly slower (88.87 ± 0.33 sec) than the mean time for the solo runs (86.92 ± 0.33 sec) (Table 22). However, competitive run times were significantly slower than the solo run times for the 1200-m runs even before corrections for environmental effects were made. The mean time for the 1600-m competitive runs (121.35 ± 0.78 sec) was also significantly slower than the mean time for the solo runs (119.94 ± 0.85 sec) (Table 22).

Figure 12 shows the time required to run each 200-m segment for the 1200- and 1600-m runs. As seen in Figure 12a, the horses ran the final 400 meter segment (800-1200 m) significantly faster alone than in competition. The same phenomenon was seen in the 1600-m runs. Note the significant increase in time required to run the final 600 meter segment during the competitive 1600-m runs (Figure 12b). The initial 400 meter segment for the 1600-m solo runs was significantly slower than the competitive runs, but the final three 200-m segments were significantly slower for the competitive runs.

There was no significant difference in mean times between solo and competitive runs at both distances between age groups (Table 22). There was a tendency ($P < 0.10$) for the 1200- and 1600-m competitive runs to be slower than the solo runs for the younger and older groups.

The mean times for females were significantly slower for the competitive 1200- and 1600-m runs (88.45 ± 0.45 and 123.71 ± 0.95 sec) than for the solo runs (87.37 ± 0.55 and 122.04 ± 1.20 sec). There was no significant difference in mean times for the male 1200- and 1600-m between the competitive and solo runs. The male horses were significantly faster than the female horses during the competitive and solo 1200- and 1600-m runs. There were no differences in the mean run times between the older and younger horses for the solo and competitive 1200- and 1600-m runs (Table 22).

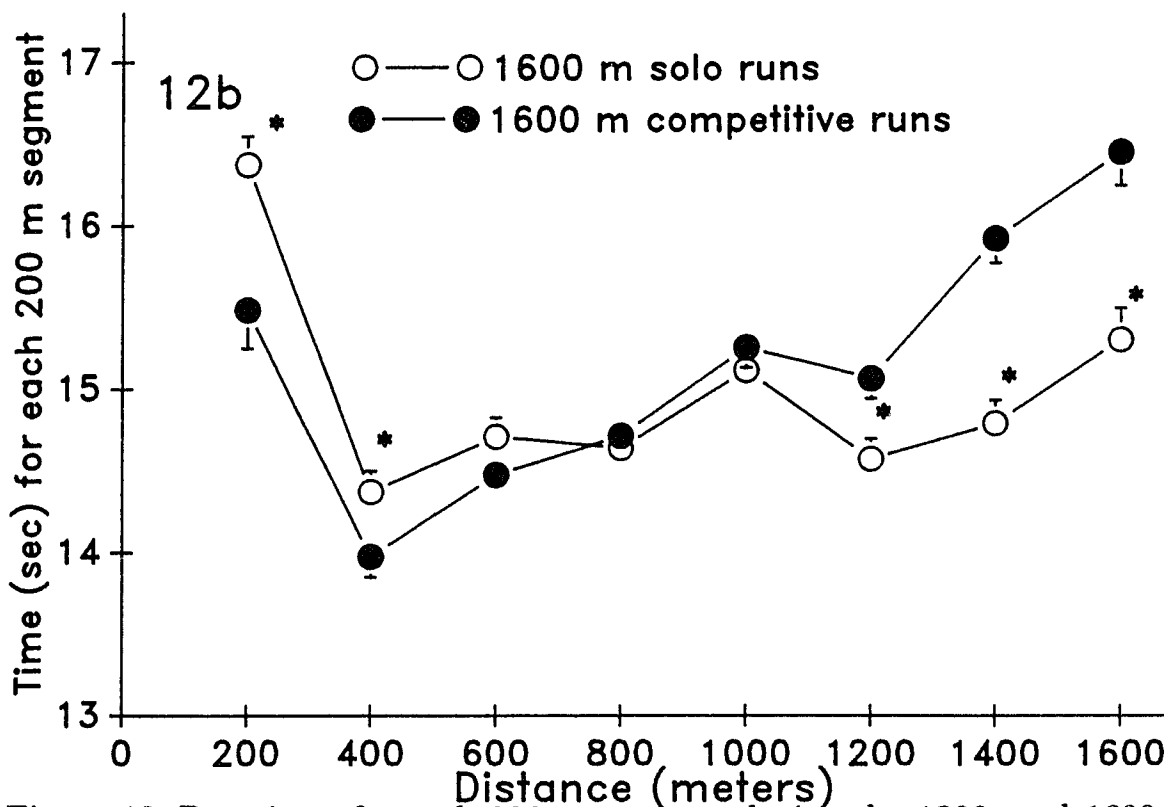
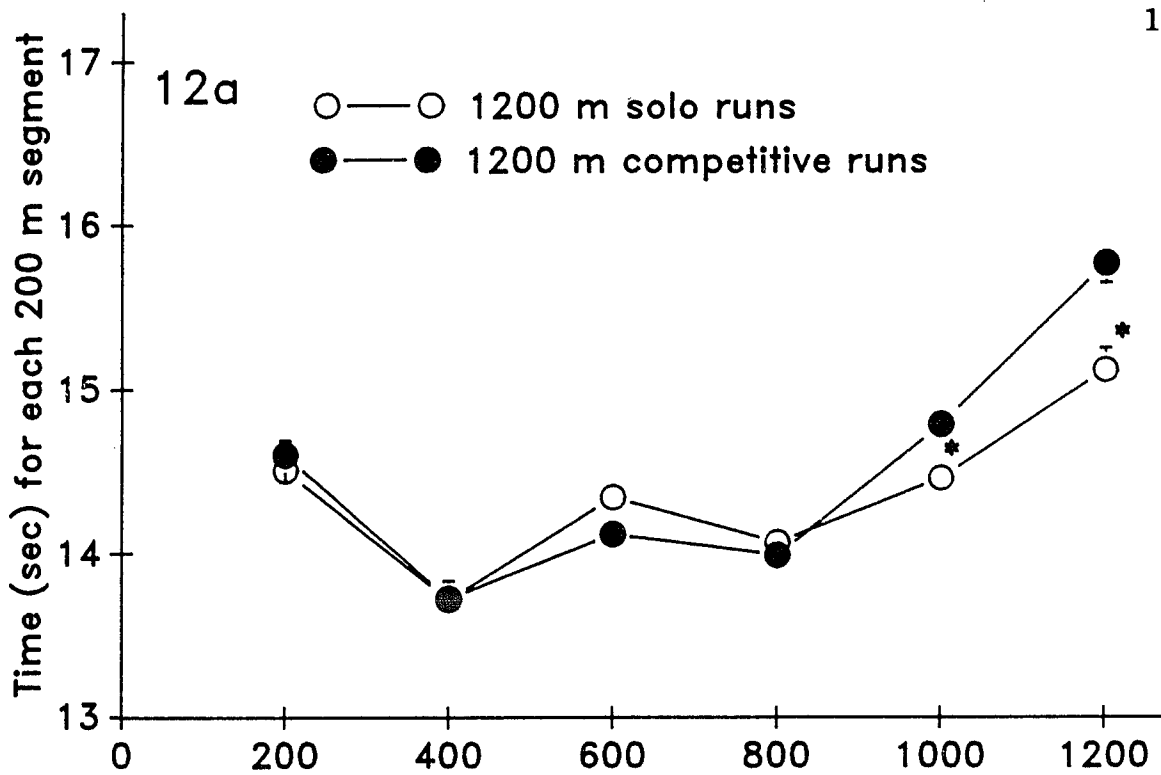


Figure 12--Run times for each 200-m segment during the 1200- and 1600-m solo and competitive runs. (N=18)

* Significant difference ($P < 0.05$) between solo and competitive segment run times.

2. Heart rate

Mean values for MaxHR, peak lactate, and total lactate are listed in Table 23. The mean peak HR for the 1600-m competitive runs was significantly less (198 ± 1.40) than the mean peak HR for the 1200-m competitive runs (201 ± 2.19), solo runs (205 ± 1.42), and 1600-m solo runs (205 ± 1.26). There was no significant difference in the mean MaxHRs for the other runs.

3. Peak and total lactate

Duncan's multiple range test for peak and total lactate showed that the peak (30.7 ± 0.46 mmol/l) and total (117.7 ± 1.74 mmol/l) lactate for the 1200-m competitive runs were significantly higher than those values for the other runs (Table 23). The mean peak lactate occurred at 8 min post-run for the competitive and solo runs at both distances.

Table 23--Mean values (\pm SEM) for peak heart rate, peak lactate, and total plasma lactate for each pair of runs.

Runs	1200-m Solo	1200-m Competition	1600-m Solo	1600-m Competition
Maximal Heart Rate (b/min)	205 \pm 1.42 (n=36)	201 \pm 2.19 (n=36)	205 \pm 1.26 (n=33)	198 [*] \pm 1.40 (n=33)
Peak Lactate (mmol/l)	26.0 \pm 0.82 (n=36)	30.7 ^a \pm 0.46 (n=36)	27.3 \pm 0.63 (n=33)	28.4 \pm 0.75 (n=33)
Total Lactate (mmol/l)	98.1 \pm 3.10 (n=36)	117.7 ^b \pm 1.74 (n=36)	103.5 \pm 2.48 (n=33)	109.1 \pm 2.89 (n=33)

^{*} Significantly different from MaxHR of other runs (P<0.05)

^a Significantly different from Peak Lactates of other runs (P<0.05)

^b Significantly different from Total Lactates of other runs (P<0.05)

Discussion:1. Run times

Since there is a shortage of research information on the effects of competition in horses, most of the research cited in this segment of the discussion was obtained from human studies. The degree to which human competitive behavior can be applied to the horse is uncertain, but horses do display some competitive behavior patterns common to humans (e.g. territoriality, social hierarchy, aggression).

2. Age

Triplett's initial study of the effect of competition on performance (Triplett, 1898) revealed that younger children performed better alone, while older children performed better in competition. Other human studies have shown that young adolescents are more affected by competition than any other age group (Butt, 1987). These studies suggest that age helps determine an athlete's capacity to handle competitive situations.

Both older and younger groups of horses showed a tendency ($P < 0.10$) to run slower during competition than alone. The lack of any significant difference between solo and competitive run times due to age may have been because of the high mean age of the horses. The youngest horse was 3 yr-old, five were 4 yr-olds, and the rest were 5 years old or older. All horses had been involved in training and/or racing before being donated for exercise research. The horses in the younger group had been in training for at least 1.5 years and were probably old enough and had enough racetrack experience to preclude any effects due to age. A study using inexperienced 2 and 3 yr-old horses would be required to evaluate the effect of age on competitive performance. The lack of any difference in run times between the older and younger groups for all runs (Table 1--Section A) illustrated the equality of running abilities for the two groups.

3. Sex

Females seemed to be more affected by competition than males. This was evident by significantly slower competitive than solo run times for females at both distances, and no significant difference in competitive and solo run times for males at either distance. This finding does not necessarily mean the females were less competitive. The females may have been more competitive

and therefore, more stressed by the other horses on the track resulting in slower competitive run times. In human competitive studies, the psychological stress of competition is measured with the Sport Competition Anxiety Test (SCAT) (Martens, 1977), which is generally accepted as the best indicator of anxiety in athletes (Cooley, 1987). In a study of elite young athletes (Ahlgren & Johnson, 1979), female gymnasts scored significantly higher on the SCAT than male gymnasts indicating a higher level of anxiety during competition. In a review of the effect of gender on anxiety of competition, Layman (1974) concluded that females were less likely than males to experience a decline in anxiety once the competition began. The anxiety experienced due to competition may have persisted for a longer period of time in the female horses than the males resulting, at least in part, in slower competitive run times.

The faster mean run times for males compared to females was expected since it is generally believed that males are superior to females (for most species) in any athletic event that depends on aerobic power and muscular strength (Astrand & Rodahl, 1986). For example, fillies have won the Kentucky Derby only three times in over 115 years. Fillies generally do not compete with colts in stake races because of a lack of success racing against larger, stronger males.

4. Quality of horses

Michaels *et al.* (1982) showed that good pool players were 9% more accurate in their shots when encountering stresses similar to the stress of competition while below-average players dropped 9% in accuracy under the same conditions. Wankel (1972) also concluded that high-ability groups performed best under competitive conditions, while low-ability groups performed poorly when subjected to competitive stress. These studies suggest that athletic ability helps determine an athlete's capacity to handle competitive situations.

The horses used for this study were healthy and sound, and a few had won parimutual races. However, overall they were not quality racehorses and could be considered a "low-ability group" of horses. The horses had been donated to Louisiana State University School of Veterinary Medicine because of a lack of success on the racetrack. A few of the horses had won races, but the majority failed to win and were considered unprofitable for the owners. The increase in run times seen during competition for these horses may correspond to the way that "below-average" humans react to competition in the studies cited earlier (Butt, 1987; Cratty, 1981). None of the groups ran the competitive runs faster than the solo runs. Only 3 of the 18 horses in this study showed an improvement in run times for the 1200-m competitive runs when compared

with the solo runs, and 4 showed an improvement in run times for the 1600-m competitive runs.

5. Anxiety of competition

Psychologists identify two types of anxieties in athletes: 1) a person's general level of anxiety which is a personality trait, and 2) situational anxiety which is a short-term condition of stress caused by the athletic event. In general, athletes who are commonly anxious are often most upset and anxious during competitive situations (Cratty, 1984). Horses also demonstrate a range of anxiety from calm to very nervous, and excessive anxiety due to competition may result in poorer run times. Horsemen generally prefer a horse to be calm in the paddock before a race instead of "lathered up" from excessive perspiration due to anxiety.

Prior to and during competition, an athlete undergoes many physiological changes. Activation of the sympathetic nervous system results in increases of respiration, cardiac output, blood flow to working muscles and away from non-functioning organs, muscle tension, and glycolysis. All of these changes can positively affect performance (Cratty, 1984). However, elevated lactate and H^+ accumulation associated with increased glycolysis lowers muscle pH (Sutton

et al. 1981) resulting in an earlier onset of fatigue (Wilkie, 1986). An excessively high level of anxiety can result in first a rise and then a lessening of muscle tension creating a feeling of weakness (Cratty, 1984). In this study, the highest peak and total plasma lactate values were obtained during the 1200- and 1600-m competitive runs. However, the average speed for those runs was less than for the solo runs. The increased cellular metabolism and glycolysis seen during high-anxiety situations like competition (Cratty, 1984) may have been demonstrated during the 1200- and 1600-m competitive runs by the elevated plasma lactate values and slower run times.

Some of the horses were noticeably more anxious when running with another horse. The increased anxiety was demonstrated by shying from the other horse, constantly watching the other horse, increased sweating, and decreased responsiveness to the jockey's instructions. The distraction of the other horse may have decreased the horse's concentration on running and resulted in slower competitive run times.

6. Onset of fatigue

An increased state of anxiety during the competitive runs may be evident in a comparison of the run times for each 200-m segment of the competitive and

solo runs (Figure 12). Figure 12a illustrates an earlier onset of fatigue in the 1200-m competitive runs by the significantly slower run times for the final 400 meters. Since the 1200-m run was a maximal effort run for the entire distance, there was no significant difference between 200-m segment times for solo and competitive runs for the first 800 meters. Therefore, the earlier onset of fatigue was not the result of horses running the early portion of the competitive runs at a faster pace than the solo runs. The earlier onset of fatigue may have been due to an excess of metabolic waste products (H^+ and inorganic phosphate) (Wilkie, 1986) from increased anxiety.

An earlier onset of fatigue was also seen in the 1600-m run, where the first 400 meters of the competitive runs were run significantly faster than the solo runs (Figure 12b). To run the fastest possible time over a distance of 1600-m, it was necessary for the jockeys to restrain the horses at the beginning of the run to (theoretically) prevent early accumulation of lactate, H^+ , and inorganic phosphate that lead to fatigue (Wilkie, 1986). An elevated state of anxiety with an associated increased release of catecholamines (Smith *et al.* 1988) would make it more difficult for the jockey to restrain the horse early in the run. Fatigue appears to have occurred much earlier in the competitive runs as indicated by the increased run time during the final 600 m when compared to times for the solo runs.

7. Plasma lactate

The higher peak and total plasma lactate values for the competitive runs (only plasma lactate values for the 1200-m competitive run were statistically higher) were consistent with the findings of Bayly *et al.* (1987a) who showed that there was a tendency toward higher lactate concentrations when horses were run on a soft or muddy track, trained from a starting-gate, or trained with another horse. All of these situations can add stress by introducing unfamiliar conditions or disturbing circumstances for the exercising horse.

The plasma lactate concentration did not increase with distance in our study, which is also consistent with the findings of Bayly *et al.* (1987a). They showed that, although plasma lactate concentration increased with the distance run up to 1000 m, there was no difference in plasma lactate concentration for distances between 1000-1700 m.

8. Heart rate

It was not apparent why the mean MaxHR for the 1600-m competitive runs was less than the mean MaxHR for the other runs in this study. Heart rate has been shown to be linearly related to exercise intensity, up to the maximal

heart rate (von Englehardt, 1977). Kubo *et al.* (1984) showed that the actual maximal HR of horses was not reached until after 5 min of near-maximal effort galloping, but that there was no difference in the peak HR measured in horses during runs of 200-1600 m.

The horses ran more slowly in competition than alone. Females performed more poorly during both competitive runs, while there was no difference in solo and competitive run times for the males at either distance. There was no difference in run times between competitive and solo runs due to age within the limited age span of these horses. No group showed improvement in run times when running with competition, which may be a reflection of the quality of horses used for this study.

Section F

Effects of Added Dietary Fat on Performance

Objectives:

1. To assess any ergogenic effect of 12% dietary fat on performance of Thoroughbred racehorses in a 1600-m run.
2. To assess the effect of a fat-added diet on metabolism following maximal-effort exercise.

Materials and Methods:

1. Horses

Fifteen healthy Thoroughbred racehorses (8 geldings and 7 mares), ranging in age from 3 to 8 years and weighing between 407 and 499 kilograms, had been in training and racing projects for 10 months prior to this investigation. During the first 5 months, the horses were conditioned on a high-speed treadmill.⁴⁰ The conditioning was described in Section D. During the final

⁴⁰ Mustang 2200, Kagra International Inc., Fahrwangen, Switz.

5 months, the horses were stalled at a training track for completion of the studies described in the earlier sections of this dissertation. Conventional track exercise was used to maintain conditioning during that period. All horses made 11 runs (6 alone and 5 in competition) during the final 5 months of the exercise studies reported in this dissertation at distances from 1200-2000 m. Fitness of the horses was considered to be at a plateau during the exercise studies since times for the previous runs had not shown any progressive improvement. The absence of a progressive improvement in run times was evidence that no training effect due to the runs had occurred.

2. Treatments

The control diet (Table 24) consisted of 3.1 kg of a complete, pelleted ration⁴¹ twice a day (14% protein, 2% fat, 2478 Kcal/kg digestible energy [DE]). The ration was comprised mainly of corn and soy beans with minerals and vitamins added. Bermuda hay (1772 Kcal/kg DE⁴²) was also fed at a rate of 3.0 kg twice a day. This diet provided 26000 Kcal/day.

⁴¹ Purina Pride #200, Purina Mills Inc., St. Louis, MO.

⁴² National Research Council, Washington, DC

Table 24--Contents, digestible energy, amount fed, and total Kcal/day of the control and fat-supplemented diets. Note that both diets supplied 26000 Kcal/day.

	Digestible Energy (Kcal/kg)	Amount Fed (kg/day)	Kcal/day Intake
Control Diet			
Pelleted Feed	2478	6.2	15363
Bermuda Hay	1772	6.0	10637
Total Daily Intake			26000
Fat-supplemented Diet			
Pelleted Feed	2478	9.44	23400
Corn Oil	9009	0.29 (177 ml BID)	2600
Total Daily Intake			26000

The fat-supplemented diet (Table 24) consisted of 4.7 kg of the same complete, pelleted ration twice a day (14% protein, 2% fat, 2478 Kcal/kg DE) with 177 ml corn oil (9009 Kcal/kg) added that accounted for 10% of the DE. The pelleted ration contained 2% fat, so the added corn oil produced a feed with 12% of the DE from fat sources. The experimental diet did not include hay. This allowed more pelleted feed in the ration which diluted the concentration of corn oil and increased palatability. The horses were weighed prior to each run to insure that any change in muscle glycogen content was not due to weight gain from increased DE intake. There was no difference ($P < 0.05$) in mean body weight (Table 25) prior to each run.

3. Runs

The horses were run in pairs to simulate racing conditions. Horses were paired according to performance in previous 1600-m runs. The runs were performed on an 800-m training track on 3 different days. Four pairs of horses competed on run days 1 and 2 (control runs) and 8 pairs of horses competed on run day 3 (fat run). Because of muddy track conditions, twelve days elapsed between the two control run days. Fat supplementation of the diet was begun the day after the control runs were finished and continued for a 21-day period. The fat run was performed at the end of the 21-day fat-

Table 25--Pre-run weights of each horse following the control and fat-added diets. There was no significant difference between the weights for each run.

Horse	Control Diet Wt. (kg)	Fat-added Diet Wt. (kg)	Difference
1	476.7	484.4	7.7
2	458.6	468.1	9.5
3	427.7	426.8	-0.9
4	415.5	422.7	7.3
5	426.4	421.4	-5.0
6	471.7	490.3	18.6
7	417.8	414.6	-3.2
8	492.6	480.4	-12.2
9	427.7	426.8	-0.9
10	452.7	457.2	4.5
11	455.4	460.4	5.0
12	445.4	440.0	-5.4
13	412.3	407.3	-5.0
14	499.4	494.0	-5.4
15	407.3	412.3	5.0
Mean	445.8	447.1	1.3
\pm SEM	7.4	7.7	2.0

supplementation period. Since there was an odd number of horses in the study, one horse ran against a horse of equal running ability whose time was not included in this investigation. The same two jockeys were used for both 1600-m runs. The horse pairings and riding assignments were the same for the control and fat runs. The riders were licensed professional jockeys and were instructed to ride the horses in the fastest possible time.

The horses were fed the evening prior to but not on the morning of the runs. On each run day, the runs were completed between 8-10 AM. A hygrometer-thermometer was used to measure temperature and humidity when each pair of horses arrived at the track. Soil samples for the measurement of soil moisture percent were taken from the same two areas of the track at the same time on each run day. The soil samples were weighed before and after a 24 hr drying period (at 200 degrees C) to determine soil moisture content. These values for the three run days of this investigation are listed in Table 26. Times were recorded for each 200-m segment of the 1600-m run and for the total distance to the nearest 0.01 second.

Rain and muddy track conditions caused postponement of the second control run. Conditioning prior to the control runs and during the fat supplementation period was maintained with conventional track and treadmill exercise. Horses were exercised equally during the 3 week period prior to each run. This

Table 26--Percent ambient humidity, temperature, and % soil moisture for each hour of each run day. Runs following control diet were on days 1 and 2, and run following fat-added diet was on day 3.

Time	Day	% Ambient Humidity	Ambient Temperature (degrees C)	% Soil Moisture
8 AM	1	85	10.0	8.2
	2	97	12.8	5.5
	3	87	4.4	8.4
9 AM	1	77	12.2	
	2	91	16.7	
	3	78	8.9	
10 AM	1	72	16.7	
	2	84	20.0	
	3	65	11.1	

eliminated disparate work schedules as a cause of glycogen storage differences.

4. Blood samples

Blood samples were collected from the jugular vein in vacutainer tubes⁴³ containing potassium oxalate and sodium fluoride just prior to the run, and at 2, 4, 8, and 16 minutes after completion of the run. Plasma was removed by centrifugation and stored at -20 degrees C until future analysis for plasma lactate (LA), glucose (GLU), triglycerides (TRIG), glycerol (GLOL), beta-hydroxybutyric acid (BHBA), and non-esterified fatty acids (NEFA).

Serum levels of GLOL, TRIG, GLU, NEFA, and BHBA were determined on a Beckman Synchron CX5⁴⁴ automated chemistry analyzer. Serum glucose was determined with a Beckman glucose reagent cartridge.⁴⁵ Hexokinase catalyzed the transfer of a phosphate group from adenosine triphosphate (ATP) to glucose to form adenosine diphosphate (ADP) and glucose-6-phosphate. The glucose-6-phosphate was then oxidized to 6-phosphogluconate with the concomitant reduction of nicotinamide adenine dinucleotide (NAD) to NADH

⁴³ Becton Dickinson, No. 6445, Rutherford, NJ

⁴⁴ Beckman Instruments, Brea, CA

⁴⁵ Beckman Instruments, No. 442640, Brea, CA

(reduced form) by the catalytic action of glucose-6-phosphate dehydrogenase. The change in absorbance was monitored at 340 nm.

Serum triglycerides concentration was determined with a Beckman triglycerides reagent cartridge.⁴⁶ The TRIG were hydrolyzed to glycerol and free fatty acids by the action of lipase. A sequence of 3 coupled enzymatic steps using glycerol kinase, glycerol-1-phosphate dehydrogenase, and diaphorase caused the reduction of 2-iodophenyl-3-nitrophenyl tetrazolium chloride to a colored formazan product. The change in absorbance was monitored at 520 nm. The triglyceride method made no correction for endogenous glycerol in the sample, and therefore included free glycerol and triglyceride.

Glycerol, NEFA, and BHBA methods were adapted for the Beckman Synchron CX5 (Tulley & Harkins, 1990). The free glycerol procedure was an adaptation of the Sigma method⁴⁷ for TRIG in which the saponification step was omitted. Glycerol kinase reacted with glycerol and ATP to form glycerol phosphate and ADP. Pyruvate kinase acted on ADP and phosphoenolpyruvate to form pyruvate and ATP. Lactate dehydrogenase caused pyruvate and NADH to

⁴⁶ Beckman Instruments, No. 442770, Brea, CA

⁴⁷ Sigma Diagnostics, No. 320-UV, St. Louis, MO

form lactate and NAD. The amount of NADH oxidized was proportional to the original glycerol concentration.

Serum NEFA were determined with a WAKO diagnostic kit.⁴⁸ This method relied upon the acylation of Coenzyme A (CoA) by fatty acids in the presence of added acyl CoA synthetase. The acyl CoA thus produced was oxidized by added acyl CoA oxidase with the generation of hydrogen peroxide. Hydrogen peroxide, in the presence of peroxidase, permits the oxidative condensation of 3-methyl-N-ethyl-hydroxyethyl-aniline with 4-aminoantipyrine to form a purple colored adduct which was measured colorimetrically at 550 nm.

Analysis of beta hydroxybutyric acid was performed with a Sigma diagnostic kit⁴⁹ and was a modification of a procedure by Koch and Feldbruegge (1987). It was based on the conversion of beta hydroxybutyrate and NAD to acetoacetate and NADH as catalyzed by the enzyme beta hydroxybutyrate dehydrogenase. The change in NADH was measured at 340 nm. Therefore, the change in absorbance was directly proportional to beta hydroxybutyrate in the serum samples.

⁴⁸ WAKO Pure Chemical Industries, Ltd., No. 990-75401, Osaka, Japan

⁴⁹ Sigma Diagnostics, No. 310-UV, St. Louis, MO

Serum lactate was analyzed using a Sigma diagnostic kit.⁵⁰ That technique utilized the enzyme lactate dehydrogenase, which catalyzed the reversible reaction of lactate and NAD to pyruvate and NADH. Excess NAD was used to force the reaction to completion in the direction of lactate formation. The increased absorbance at 340 nm due to NADH formation was proportional to the lactate originally present.

5. Muscle samples

Biopsy sites were clipped and scrubbed using aseptic techniques. A local anesthetic⁵¹ was injected 5 minutes before a 1 cm skin incision was made over the middle gluteal muscle and extended through the fascia layer over the muscle. Muscle samples were taken from a depth of 10 cm. Muscle biopsies were taken from the same biopsy site before and after each run from the middle gluteal muscle according to the technique of Snow (1983b) and immediately frozen in isopentane cooled in liquid nitrogen. The muscle samples were stored at -80 degrees C until analysis. Muscle glycogen content was determined according to the method of Hassid and Abrahams (1957). That method consisted of the digestion of muscle tissue in hot potassium hydroxide.

⁵⁰ Sigma Diagnostics, No. 726-UV, St. Louis, MO

⁵¹ Lidocaine hydrochloride 2%, Pan Vet, Inc., Springfield, MO

Glycogen was precipitated with ethanol and hydrolyzed with 0.6 N hydrochloric acid. Glycogen content was calculated from the amount of glucose in the hydrolysate.

After the pre-run biopsy was obtained, the biopsy site was covered with an adhesive bandage. The horses walked to the training track, warmed-up with a trot and slow gallop for 800 m, then started the 1600 m run. Within 1 minute after the runs, post-run muscle samples were taken from the same biopsy site. Glycogen utilization was determined by subtracting the post-run from the pre-run muscle glycogen content.

6. Statistics

Times for the runs following the control and fat-added diets were compared using a paired t-test. The effects of fat supplementation on plasma LA, GLU, TRIG, GLOL, BHBA, and NEFA were determined by comparing those values to the same variables following the control diet using multivariate analysis of variance with repeated measures. Tests for the simple main effect of diet within each 200-m segment were performed using multivariate analysis of variance with repeated measures. Times for each 200-m segment of the control and fat-added runs were contrasted by determining the confidence interval for

the simple main effect of treatment. Analysis of covariance was used to evaluate any effects on performance due to humidity, temperature, or soil moisture. Significance was set at $P < 0.05$.

Results:

1. Run times

Individual run times for the 1600-m runs following each diet and differences appear in Table 27. The average run time was significantly improved ($P < 0.05$) from 120.3 ± 0.46 sec after the control diet to 118.9 ± 0.46 sec following the 12% fat-added diet. There were no significant variances for soil moisture x run, temperature x run, or humidity x run interactions, or main effect of soil moisture for the control and fat-added treatments. Ambient humidity and temperature did have a significant main effect on run times. Humidity and temperature were strongly correlated with each other, so the factor with the higher significance, which was humidity, was used to correct the run times. A statistical manipulation (Freund *et al.*, 1986) was performed on the run times to adjust the times as if all runs had been performed at the same humidity. After the run times were corrected for the effects of humidity, there was still a significant difference in mean times between the runs following the

Table 27--Run times and differences for each horse during the runs following the control and fat-added diets.

Horse	Control Diet (sec)	Fat-added Diet (sec)	Difference (sec)
1	122.23	120.29	1.94
2	113.68	112.80	0.88
3	124.33	121.59	2.74
4	124.27	120.84	3.43
5	117.56	118.65	-1.09
6	118.58	116.91	1.67
7	124.46	123.86	0.60
8	125.88	120.72	5.16
9	119.22	118.57	0.65
10	116.88	114.31	2.57
11	122.02	121.29	0.73
12	118.14	117.34	0.80
13	119.34	117.70	1.64
14	121.96	124.44	-2.48
15	116.38	113.46	2.92
Mean	120.3	118.9 *	
± SEM	± 0.46	± 0.46	

Humidity adjusted times

Mean	120.7	118.2 *
± SEM	± 0.44	± 0.44

* Significant difference between control and fat-added diets (P<0.05)

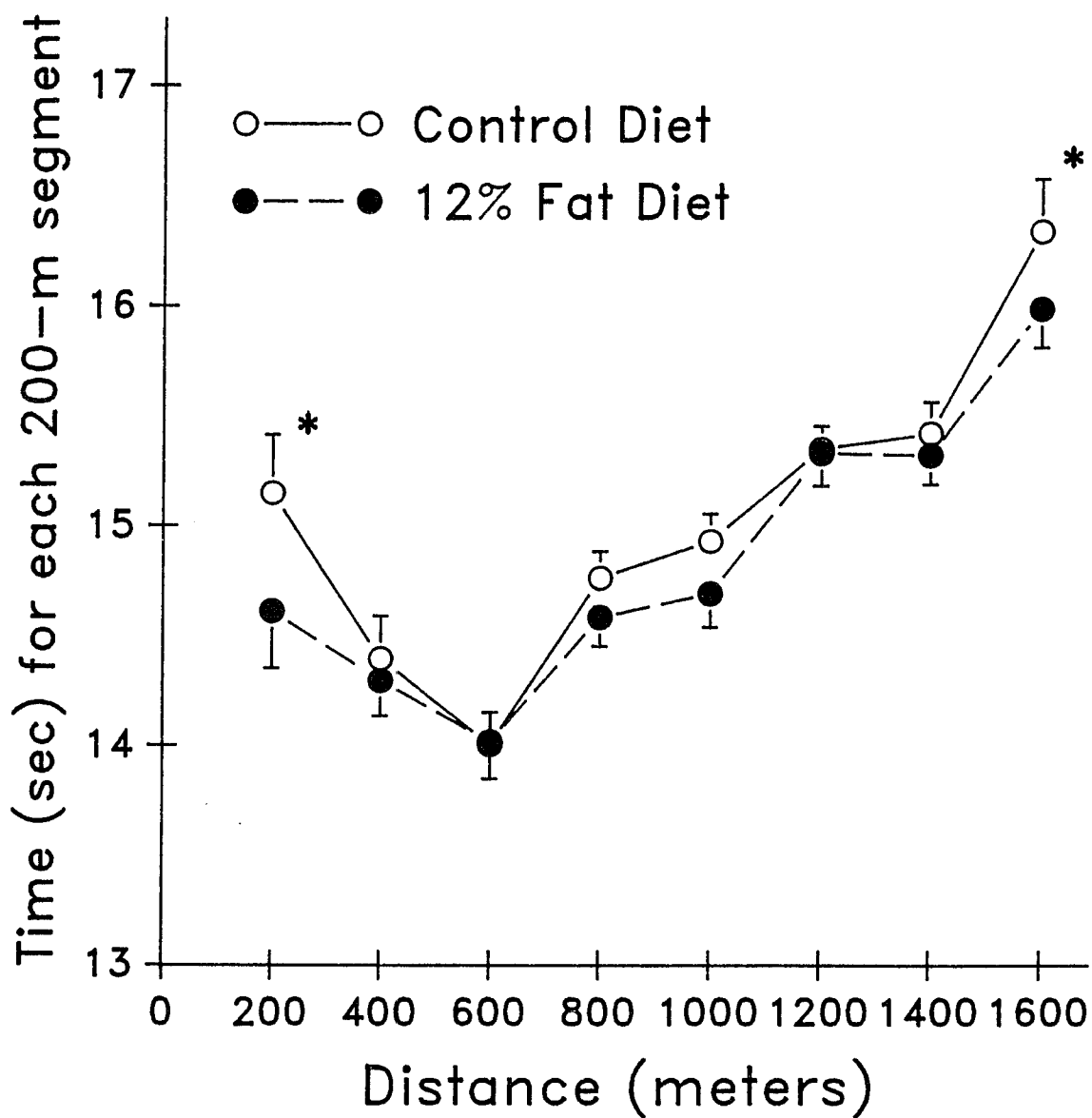


Figure 13--Run times for each 200-m segment of the runs following the control and fat-added diets.

N=15

* Significant difference ($P < 0.05$) in segment run times following the control and fat-added diets

control and fat-added diets (Table 27). In fact, the difference between the corrected mean times for the fat-added and control runs (118.2 ± 0.44 and 120.7 ± 0.44 sec, respectively) was greater than the difference for the uncorrected mean run times. Figure 13 shows the time required to run each 200-m segment of the control and fat-added runs. The first and last 200-m segments (0-200 m and 1400-1600 m) of the run following fat supplementation were significantly faster than the run following the control diet. This indicated a faster acceleration and stronger finish during the fat-added run. Note the similarities in performance times for the intervening segments. Note also the similar time to peak speed (600 m) during both runs.

2. Blood samples

Figure 14 shows the values for plasma GLOL (Figure 14a), TRIG (Figure 14b), NEFA (Figure 14c), BHBA (Figure 14d), GLU (Figure 14e), and LA (Figure 14f) before and after each run. The variables that have normal limits established for horses (NEFA, GLU, and LA) were within those limits. Values that do not have normal ranges established were within the range reported by other investigators.

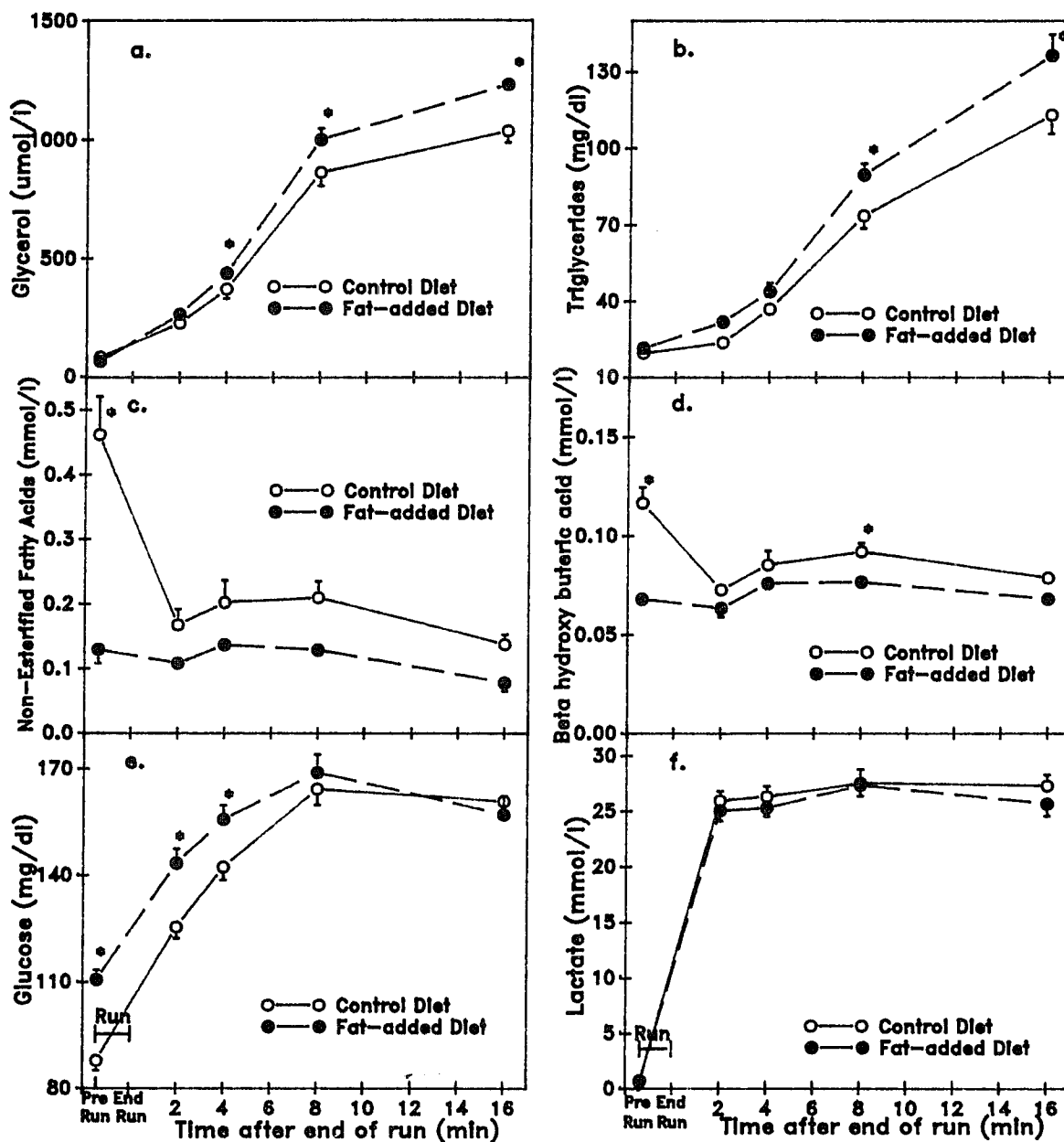


Figure 14--Plasma values during the runs following the control and fat-added diets for a) GLOL, b) TRIG, c) NEFA, d) BHBA, e) GLU, and f) LA.

N=15

* Significant difference ($P < 0.05$) between values for the control and fat-added diets

There was no difference between diets for the pre-run or 2-min post-run values for GLOL, but the 4-, 8-, and 16-min post-run values for the fat-added diet were significantly higher than the control diet values (Figure 14a). There was no difference between diets for the pre-run, 2-, or 4-min post-run values for TRIG, but the 8- and 16-min post-run values for the fat-added diet were significantly higher than those for the control diet (Figure 14b). The only significant difference between diets for NEFA was seen in the pre-run sample where that value for the control diet was markedly elevated (Figure 14c). The pre-run and 8-min post-run samples for BHBA were significantly higher for the control diet than for the fat-added diet (Figure 14d). The pre-run, 2-, and 4-min post-run GLU values for the fat-added diet were significantly higher than those values for the control diet, but there was no difference between diets for the 8 and 16-min post-run GLU values (Figure 14e). There was no difference between diets for any of the values for LA (Figure 14f).

3. Muscle samples

Muscle glycogen content of the pre- and post-run samples for the control and fat-added diets are listed in Table 28. There was a significant difference between pre- and post-run mean glycogen contents for the control (32.3 ± 2.00 and 22.4 ± 2.34 mg/g wet tissue, respectively) and fat-added diets (37.4 ± 1.42

Table 28--Pre- and post-run muscle glycogen, and glycogen utilization for each horse after control and fat-added diets.

No.	Control Diet Muscle Glycogen (mg/g wet tissue)			Fat-added Diet Muscle Glycogen (mg/g wet tissue)		
	Pre- run	Post- run	Glycog Utiliz	Pre- run	Post- run	Glycog Utiliz
1	38.5	23.6	14.9	17.6	11.8	5.8
2	33.2	28.0	5.2	40.4	30.2	10.2
3	25.8	20.0	5.8	29.5	29.1	0.4
4	46.9	35.1	11.8	43.7	21.7	22.0
5	39.6	25.7	13.9	31.9	29.3	2.6
6	39.6	27.4	12.2	34.6	35.6	-1.0
7	31.8	25.3	6.5	33.8	30.7	3.1
8	35.7	36.3	-0.6	28.3	25.8	2.5
9	38.8	28.5	10.3	39.6	29.5	10.1
10	47.8	29.0	18.8	29.8	14.4	15.4
11	31.0	26.7	4.3	43.2	8.6	34.6
12	38.0	26.9	11.1	17.1	7.6	9.5
13	38.6	28.1	10.5	33.2	22.9	10.3
14	35.1	28.2	6.9	35.2	28.8	6.4
15	40.1	31.9	8.2	26.1	10.4	15.7
Mean± SEM	32.3 ±2.00	22.4 ±2.34	9.3 ±1.21	37.4 ±1.42	28.8 ±1.02	9.9 ±2.32

and 28.8 ± 1.02 mg/g wet tissue, respectively). Furthermore, the pre- and post-run mean muscle glycogen content values for the fat-added diet were significantly greater than the corresponding values for the control diet. However, the differences between the mean pre- and post-run muscle glycogen values (glycogen utilization) for the fat-added and control diets (9.3 ± 1.21 and 9.9 ± 2.32 mg/g wet tissue, respectively) were not significantly different. The muscle sample glycogen contents are represented in Figure 15.

Discussion:

1. Run times

Thirteen of the fifteen horses in this study showed an improvement in run times following the fat-added diet when compared with the control diet. The improvement in mean run time following the fat-added diet was consistent with the findings of other equine studies that used fatiguing or near-maximal effort SETs for evaluation. Oldham *et al.* (1989) showed a reduction in average sprint time during a near-maximal SET (at a heart rate of 210 beats/min) from 59 sec following the control diet to 56.7 sec following a three-week period on a 10% fat diet. However, the difference in that study was not significant. No sample mean variances were included in the data. There may have been

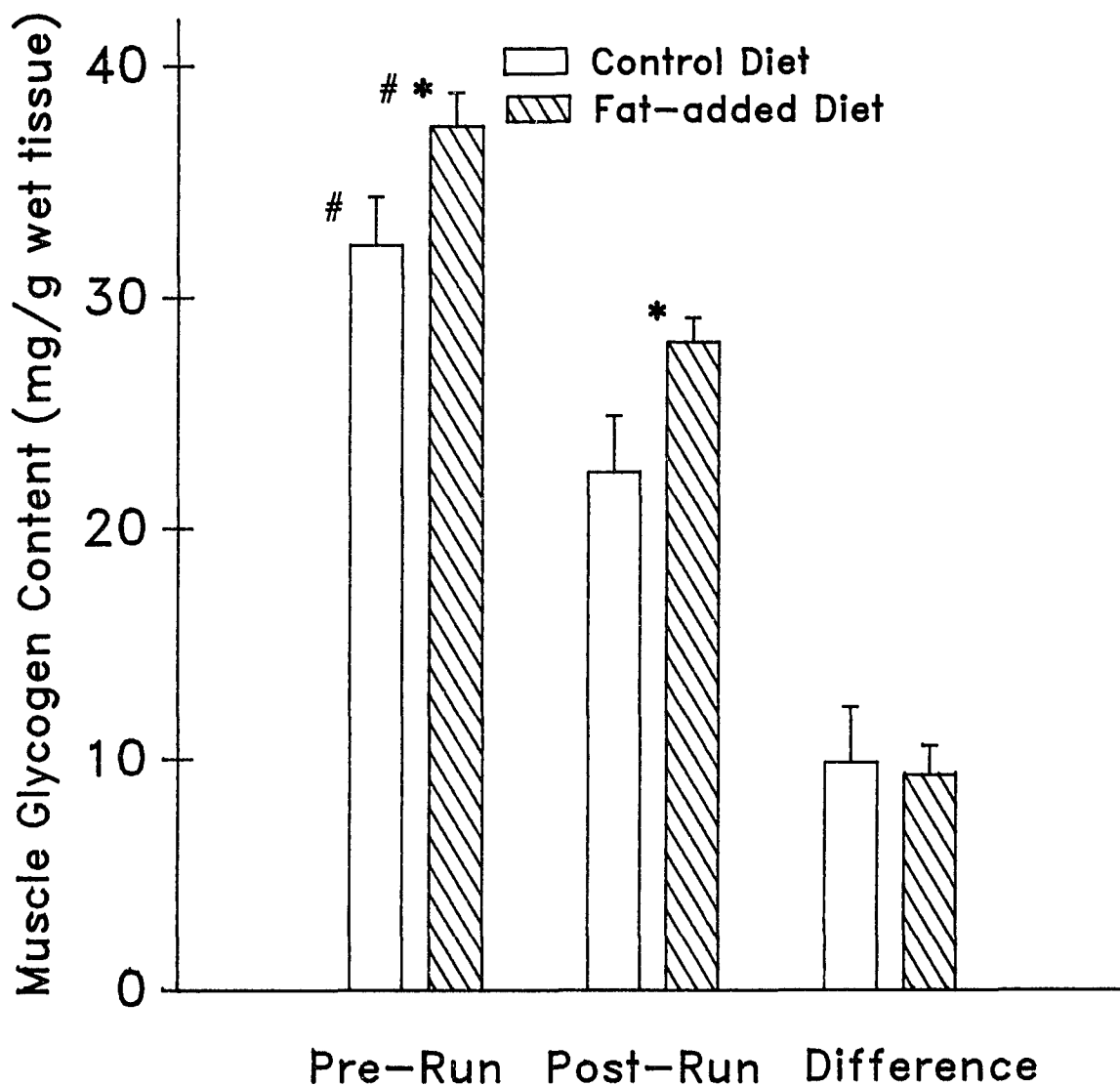


Figure 15--Pre- and post-run muscle glycogen content and glycogen utilization (Difference) following the control and fat-added diets.

N=15

* Significant difference ($P < 0.05$) in muscle glycogen content between control and fat-added diets

Significant difference ($P < 0.05$) between pre- and post-run muscle glycogen content within diets

considerable variation in sprint times between treatments or among horses since 2.3 sec is a substantial time difference in an event lasting less than 1 minute. Webb *et al.* (1987) showed that the percentage of hindquarter turns executed during a cutting performance test in Quarter horses were significantly higher ($P < 0.01$) when the horses were fed a 10% fat diet when compared to a control diet. They concluded that the horses worked harder during the cutting performance test when they were fed the 10% fat-added diet.

Figure 13 illustrates that the first and last 200-m segments were significantly faster during the run following the fat-added diet when compared with the run following the control diet. Results from Section E (Figure 12b) showed that a significantly faster speed at the beginning of the run resulted in an earlier onset of fatigue. The onset of fatigue is attributed to the accumulation of lactate, H^+ , and inorganic phosphate (Wilkie, 1986). The earlier onset of fatigue noted was illustrated by significantly slower 200-m segment times at the end of the run (Figure 12b). That phenomenon was not seen during the run following the fat-added diet. Even though the horses ran faster at the beginning of the run following the fat-added diet, they were still able to complete the final 200-m segment significantly faster than in the run following the control diet. This finding suggests that the fat-added diet delayed the onset of fatigue.

2. Muscle samples

The significant increase in pre-run glycogen storage in our study following a 21-day fat-added diet was in agreement with the findings of other equine nutrition and exercise studies. Hambelton *et al.* (1980) increased pre-exercise muscle glycogen 46% following a 3-week period on a 12% fat-added diet. Meyers *et al.* (1989) increased glycogen content at rest from 16.97 ± 1.90 mg/g wet tissue to 25.77 ± 1.90 mg/g wet tissue by adding 10% fat to the diet for a 3 week period. Oldham *et al.* (1989) increased pre-exercise muscle glycogen content from 15.77 to 22.89 mg/g wet tissue following a 3 week 10% fat-added diet. These studies showed a greater increase in muscle glycogen storage following fat-supplementation of the diet than was found in this project. The difference may have occurred because the horses in this project sustained a greater amount of exercise than the horses in the other studies. It has been hypothesized that fat utilization was increased during rest and sub-maximal activity during the 3-week feeding period prior to the run, thereby sparing glycogen utilization and increasing muscle glycogen storage (Meyers *et al.* 1989). Increased muscle activity in the horses in this project may have reduced the digestible energy available for storage as glycogen.

Glycogen utilization accounted for the difference between pre- and post-run muscle glycogen content. The similar values for glycogen utilization during the

runs following the fat-added (9.9 ± 2.32 mg/g wet tissue) and control (9.3 ± 1.21 mg/g wet tissue) diets was contrary to the study by Oldham *et al.* (1989). They showed a significant increase in glycogen utilization following a 10% fat-added diet (13.09 mg/g wet tissue) compared to glycogen utilization following a control diet (6.99 mg/g wet tissue). The reason for this difference is not readily apparent.

Snow *et al.* (1987) concluded that fatigue in horses subjected to short, intense exercise bouts is probably not related to glycogen depletion. Muscle glycogen content was still relatively high in the post-run samples after the fat-added and control diets (28.8 ± 1.02 and 22.4 ± 2.34 mg/g wet tissue, respectively) (Table 28). This decrease represented only a 23% and 31% depletion of muscle glycogen during the runs following the fat-added and control diets, respectively. This percent glycogen depletion was consistent with the findings of Hodgson *et al.* (1984), who showed that glycogen utilization in the middle gluteal during 800-1200 m competitive runs was only 25-40% of the available stores. Miller *et al.* (1985) also showed only a 25% glycogen depletion in Quarter horses performing a 15-18 minute exhaustive exercise bout. Since differences between the mean pre- and post-run samples (glycogen utilization) for both runs were the same, increased glycogen utilization does not appear to be the reason for the decreased run time following the fat-added diet.

3. Blood samples

a. Glycerol

The similar pre-run and 2 min post-run values of plasma GLOL for both diets, and the significant increase of GLOL at 4-16 min post-run following the fat-added diet (Figure 14a) was consistent with the findings of Duren *et al.* (1987). They showed no difference between the control and fat-added diets for the pre-exercise and immediate post-exercise samples, but did show an increase in plasma GLOL at 15 min post-exercise for horses fed a 10% fat-added diet. Human studies have shown that GLOL increases with exercise for both control and fat supplemented groups, but GLOL increases at a faster rate for fat-added diets (Hickson *et al.* 1977), which is consistent with data from this project. Glycerol is not metabolized to a large extent during exercise (Newsholme & Leech, 1983), so the rising plasma glycerol following exercise may have indicated a larger degree of lipolysis in the fat fed horses resulting in a build-up of the lipolytic metabolites including glycerol.

b. Triglycerides

The elevation of plasma triglycerides following the 1600-m run (Figure 14b) was opposite to the result seen in the equine study of Duren *et al.* (1987). They demonstrated lowered plasma TRIG before, during, and following an exercise test in horses on a fat-supplemented diet. No other equine exercise studies were found that measured the effect of exercise on TRIG. In a human study of volleyball players (Bonetti *et al.* 1988), exercise caused a significant rise in plasma TRIG levels. The elevated plasma TRIG can be generally explained by the increased lipolysis in adipose tissue stimulated by the release of catecholamines. This also increases the release of NEFA from adipose tissue, utilization of NEFA by muscle tissue, and re-synthesis of TRIG in the liver as very low density lipoproteins (Newsholme & Leech, 1983).

c. Non-esterified fatty acids

The lowered pre-run NEFA for the run following the fat-added diet compared with the control diet (Figure 14c) may have been due to increased NEFA utilization. The increased NEFA utilization may have been a result of altered enzyme activity that occurred during the 21-day fat supplementation of the diet. The lowered NEFA in the blood following exercise was consistent with

the findings of Meyers *et al.* (1989) who also showed a significant decrease in NEFA during and following a 20-min sub-maximal exercise test in horses fed a 10% fat-added diet when compared to a control diet. Hintz *et al.* (1978) showed no difference in NEFA in horses during a 37 mile endurance event. Non-esterified fatty acids are the main fuel in equine muscle during rest and light exercise (McMiken, 1983). The NEFA plasma level may not reflect NEFA utilization. Because the half-life of NEFA is relatively short (approx. 2 min) (Newsholme & Leech, 1983), the actual steady state concentration tells little about the rate of utilization. The increase in plasma glycerol concentration is probably a better indicator of NEFA utilization (Newsholme & Leech, 1983). The decrease in plasma NEFA following exercise, along with the increased plasma glycerol and triglycerides following exercise, probably indicated an increased mobilization and utilization of NEFA for fuel due to increased fatty acid enzyme activity.

d. Beta-hydroxybutyric acid

The horses were not fed the morning of the run. Therefore, the horses were in a ketotic condition as indicated by the elevated pre-run BHBA in the control group (Figure 14d). The pre-run BHBA for the fat-added diet may not have been elevated because of altered metabolism due to the increased fat in the

diet. This altered metabolism may have allowed for better fuel utilization of ketone bodies during fasting. The increased state of pre-run ketosis following the control diet when compared with the values following the fat-added diet may have resulted in an earlier onset of fatigue due to increased H^+ concentration associated with the ketotic state. If this was the main reason for the better performance of the fat-fed group, feeding a meal a few hours prior to the run may have eliminated this advantage following the fat-added diet. There was no mention of ketosis in the other cited studies that showed improvements in performance (Oldham *et al.* 1989; Webb *et al.* 1987), so this may not have been the main reason for the improved run times associated with the fat-added diet.

e. Glucose

The elevated pre- and post-run blood glucose for the run following the fat-added diet (Figure 14e) was also consistent with the findings of other equine studies. Hambelton *et al.* (1980) showed that plasma glucose decreased with exercise in untrained horses, but increased in trained animals. They also showed that horses on a 16% fat-added diet maintained a higher blood glucose following exercise than horses fed a 4% fat-added diet. Webb *et al.* (1987) demonstrated that blood glucose was maintained at a higher level during a 10-

min gallop at 172 m/min on an inclined treadmill in horses fed a 10% fat-added diet compared with horses on a control ration. Hintz *et al.* (1978) showed that the decrease in blood glucose from resting levels during a 37 mile ride at a rate of 6 miles/hour was significantly less in horses fed an 8% fat diet compared to the same horses fed a 0% fat-added diet. Pagan *et al.* (1987) showed an increase in blood glucose following exercise in horses fed 5, 10, and 20% fat compared to horses fed 0% dietary fat. Duren *et al.* (1987) also showed that blood glucose remained elevated during and following exercise in horses fed 5-20% fat when compared with the same horses receiving 0% dietary fat.

The reason for the blood glucose sparing effects before and during exercise is that energy for exercising muscles can be derived from intramuscular stores of glycogen and triglycerides and extramuscular sources of non-esterified fatty acids as well as blood glucose (Miller *et al.* 1985). Mole *et al.* (1971) showed that increased fat utilization as an energy source spared glycogen and blood glucose. The exact mechanism for this is not completely understood. In work cited by Gollnick (1985), a high-fat diet resulted in a decreased respiratory exchange ratio (R) during exercise while consumption of a high-carbohydrate diet generated a higher R while performing at the same exercise intensity. They concluded that metabolism was altered by dietary intake. The enzyme systems were modified to utilize different ratios of energy substrates (primarily carbohydrate and fat) (Duren *et al.* 1987). Lindholm *et al.* (1974a) showed that

Standardbreds utilized blood glucose as well as muscle glycogen for carbohydrate sources during intense exercise. The carbohydrate sparing effect of the dietary fat may have elicited a higher resting blood glucose which permitted increased blood glucose utilization during the run following the fat-supplemented diet.

As noted in the discussion for BHBA, the horses were not fed the morning of the run and were in a ketotic state as indicated by the elevated BHBA for the control group. The fat-fed group may have maintained a higher blood glucose through increased fatty acid enzyme activity resulting in increased utilization of NEFA and ketone bodies for fuel thereby sparing blood glucose (Newsholme & Leech, 1983).

f. Lactate

The lack of differences in post-exercise plasma lactates between diets (Figure 14f) is consistent with the findings of other equine studies. Duren *et al.* (1987) showed no difference in plasma lactate levels between the control and 10% fat-added diets before, during, or following an exercise test consisting of a 1600-m run at 13.4 m/s followed by a 200-m sprint at 16 m/s. Pagan *et al.* (1987) showed no difference between the control and fat-added diets in plasma lactate

following a high-speed or a long-slow exercise test. Oldham *et al.* (1989) stated that lactate "tended to be higher when the horses were fed the high-fat diet", but only with a significance level of $P=0.13$. Other equine studies also showed no differences in plasma lactate due to diet (Meyers *et al.* 1989; Hintz *et al.* 1978). Different plasma lactate concentrations between the 2 diets would not be expected since anaerobic metabolism of glycogen results in the formation of lactate and the amount of glycogen utilized during the runs was equal.

In conclusion, 10% fat supplementation to a total of 12% of the diet of horses acted as an ergogenic aid in a short duration, 1600-m event. The fat-added diet increased muscle glycogen storage. However, that does not appear to be the reason for the decreased running time since glycogen utilization was equal in the runs following the control and fat-added diets. The faster mean run time following the fat-added diet seems to be due to increased availability of blood glucose, better utilization of ketone bodies for fuel, and a reduced state of ketosis that may have postponed the onset of fatigue when compared with the run following the control diet.

Section G

Effects of Induced Alkalosis on Performance

Objectives:

1. To determine the time of maximal buffering capacity for NaHCO₃.
2. To assess the effect of NaHCO₃ on blood-gas values, plasma lactate, and run time of Thoroughbred racehorses.

Materials and Methods:

1. Horses

Sixteen healthy Thoroughbred racehorses (8 geldings and 8 mares), ranging in age from 3 to 8 years and weighing between 385 and 476 kilograms had been in training and exercise studies described in earlier sections for 10 months prior to participating in this study. The first 5 months of training were accomplished on a high-speed treadmill⁵² using interval training techniques described in Section D. During the final 5 months the horses were stabled at a training track for completion of the exercise studies. Conditioning was

⁵² Mustang 2200, Kagra International Inc., Fahrwangen, Switz.

maintained with conventional track exercise. All horses completed 10 runs (6 alone and 4 in competition) at 1200-2000 meters prior to this investigation.

2. Treatments

Two different treatments were administered via naso-gastric tube 3 hours prior to the run: experimental treatment-- NaHCO_3 (0.4 g/kg body weight [BW]) dissolved in 1 liter H_2O , and control treatment--1 liter H_2O . The two treatments were administered in a randomized cross-over design, and the jockeys were unaware of which treatments had been administered.

3. Runs

The horses were paired according to performance in previous 1600-m runs. The same two jockeys were used for both 1600-m runs. The pairings and riding assignments were the same for the control and experimental treatment runs. The jockeys were licensed professional jockeys and were instructed to ride the horses in the fastest possible time. The runs were performed on an 800-m training track on 4 different days with 4 pairs of horses competing each day. There was an average of 14 days between the control and experimental

runs. Conditioning was maintained between runs with conventional training to insure equal fitness for both runs.

The horses were fed the evening prior to, but not the morning of the run. Three hours after treatment the horses were walked to the training track, warmed-up with a trot and slow gallop for 800 m, then started the 1600-m run from a moving start. All runs were completed between 8-10 AM. A hygrometer-thermometer was used to measure ambient temperature and humidity when each pair of horses arrived at the track. Soil samples for the measurement of soil moisture percent were taken from the same two areas of the track at the same time each run day. The soil samples were weighed before and after a 24 hr drying period (at 200 degrees C) to determine moisture content. These values for the 4 run days of this study are listed in Table 29. Times were recorded for each 200-m segment and the total distance to the nearest 0.01 second.

4. Blood samples

Blood samples were collected from the jugular vein before treatment was administered, 3 hr later just prior to the run, and at 2, 4, 8, and 16 minutes after the run. The samples were collected anaerobically in heparinized

Table 29--Ambient humidity, temperature, and soil moisture content for each hour of each run day.

Time	Day	% Ambient Humidity	Ambient Temperature (degrees C)	% Soil Moisture
8 AM	1	100	10.0	8.6
	2	100	8.3	8.4
	3	85	10.0	8.2
	4	97	12.8	5.5
9 AM	1	100	12.8	
	2	92	12.2	
	3	77	12.2	
	4	91	16.7	
10 AM	1	80	17.8	
	2	76	15.6	
	3	72	16.7	
	4	84	20	

syringes for blood gas analysis of venous blood pH, partial pressure of carbon dioxide ($P_v\text{CO}_2$), and bicarbonate (HCO_3^-). The heparinized syringes were capped and immediately placed in an ice bath. Blood-gas analysis was performed within 1 hour of the time the sample was drawn with a pH/blood-gas analyzer.⁵³ Blood was also collected in vacutainer⁵⁴ tubes containing potassium oxalate and sodium fluoride for plasma lactate analysis. Sample tubes were immediately centrifuged and the plasma removed and stored at -20 degrees C until analyzed. Plasma lactate analysis was performed with a lactate analyzer.⁵⁵

5. Peak buffering capacity

In a separate experiment with a separate group of horses, six conditioned Thoroughbred racehorses (3 geldings and 3 mares), ranging in age from 3 to 8 years and weighing between 390 and 508 kilograms, were used to determine the time of maximal buffering capacity after administration of sodium bicarbonate.

⁵³ Model 158, Corning Medical, Medfield, MA

⁵⁴ Becton Dickinson, No. 6445, Rutherford, NJ

⁵⁵ Model 23L, YSI Inc, Yellow Springs, OH

Each horse was treated via naso-gastric tube with 0.4 g/kg BW NaHCO₃. Blood samples were drawn anaerobically into heparinized syringes from the jugular vein before treatment and at 30 min intervals thereafter until the buffering effects of NaHCO₃ had peaked and started to decline. (Venous blood samples were collected through 4 hours post-treatment.) Blood-gas samples were immediately placed in an ice bath and analyzed within 1 hr. Peak buffering capacity was determined with a pH/blood-gas analyzer⁵⁶ by measuring pH of venous blood. Time to peak buffering capacity was the elapsed time between NaHCO₃ administration and maximal pH level. To simulate run day conditions, the horses were not fed the morning of treatment until after the maximal pH level had been reached and the test was terminated.

6. Diet

The diet consisted of 3.2 kg of a complete, pelleted ration (14% crude protein, 2585 Kcal/kg digestible energy) comprised mainly of corn and soy beans with minerals and vitamins added and 3.2 kg Bermuda hay. The pelleted ration and hay were fed twice a day. Water was provided ad libitum.

⁵⁶ Model 158, Corning Medical, Medfield, MA

7. Statistics

To assess the change in buffering capacity due to NaHCO_3 , the pH values at each sampling time were compared using multivariate analysis of variance with repeated measures ($P < 0.05$). Run times for the treatment and control runs were compared using a paired t-test. The effects of pre-run treatment with NaHCO_3 on blood lactate, venous PCO_2 , HCO_3^- and pH were assessed by comparing those values before and following the control run to the corresponding values before and following the NaHCO_3 treatment run using multivariate analysis of variance with repeated measures. Values at individual sampling times were compared by contrasting for the simple main effect of the NaHCO_3 treatment. Analysis of covariance was used to evaluate any effects on performance due to humidity, temperature, or soil moisture. Significance was set at $P < 0.05$.

Results:**1. Peak buffering capacity**

Figure 16 shows the change in pH of venous blood following administration of the NaHCO₃ to the six horses in the peak buffering experiment. Blood pH was significantly higher than the pre-treatment sample at 2.5 and 3.0 hrs after treatment. Maximal pH was reached between 2.5 and 3.0 hours following treatment after which time venous pH began to decrease.

2. Run times

Run times for the control and NaHCO₃ 1600-m runs appear in Table 30. There was no significant improvement in mean run times from 120.57 ± 0.46 sec for the control run to 120.51 ± 0.55 for the run following NaHCO₃ treatment. Figure 17 shows the time required to run each 200-m segment of the control and NaHCO₃ runs. There were no differences in run times for any of the 200-m segments when compared by treatments. There was no significant effect on performance from any measured environmental factor.

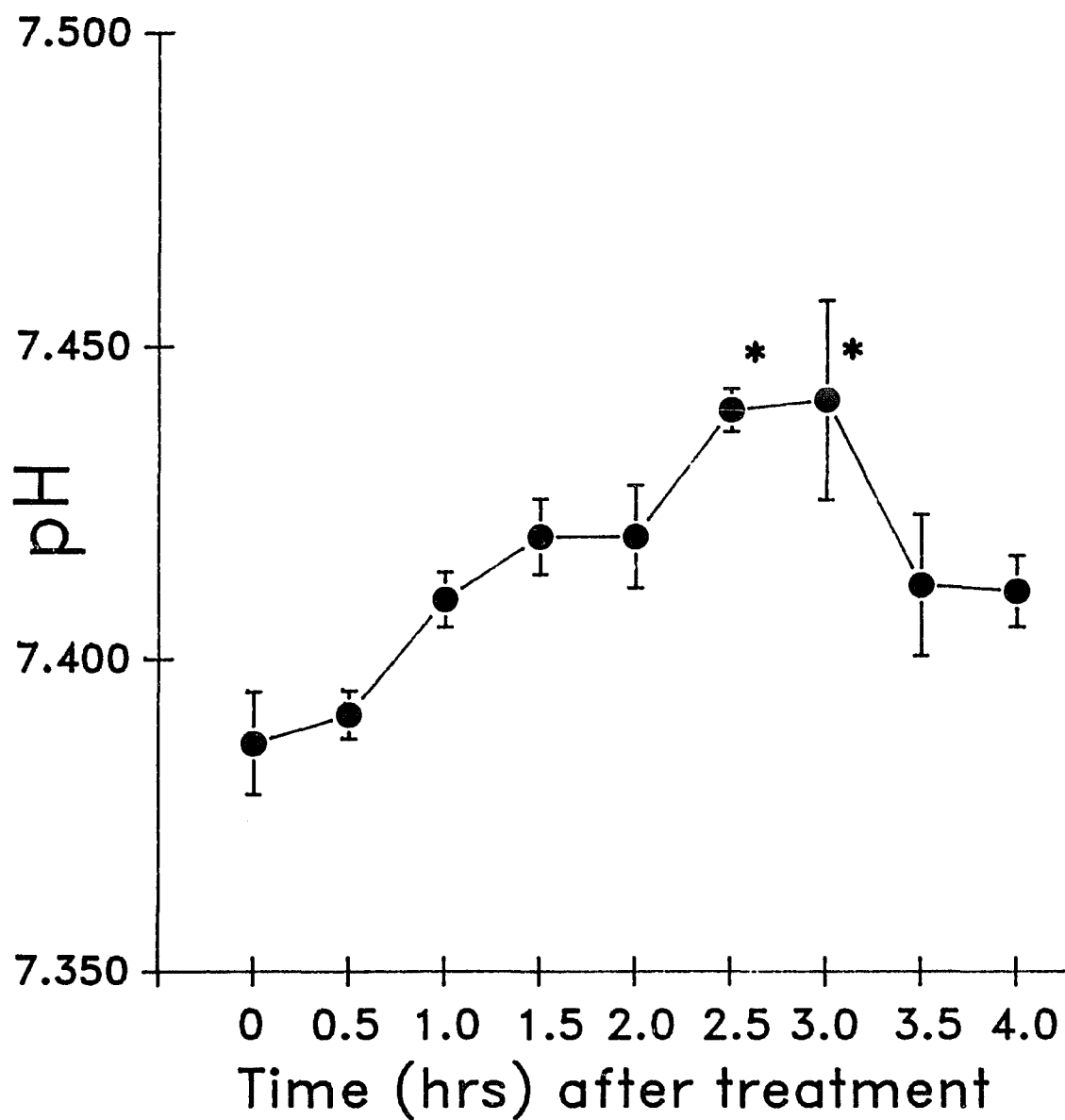


Figure 16--The change in venous blood pH following treatment with Na Bicarbonate. Peak buffering capacity was reached at 2.5-3 hrs following treatment.

N=6

* Significantly different ($P < 0.05$) from pre-treatment (Time 0) value

Table 30--Run times and differences for each horse following control and NaBicarb treatments.

Horse	Control Run (sec)	Treatment Run (sec)	Difference (sec)
1	122.23	122.22	0.01
2	113.68	114.66	-0.98
3	124.33	122.03	2.30
4	124.27	125.47	-1.20
5	117.56	116.18	1.38
6	118.58	115.07	3.51
7	124.46	121.52	2.94
8	125.88	132.17	-6.29
9	119.22	118.50	0.72
10	116.88	113.69	3.19
11	122.02	124.47	-2.45
12	118.14	119.41	-1.27
13	124.12	126.71	-2.59
14	119.34	118.50	0.84
15	121.96	122.12	-0.16
16	116.38	115.47	0.91
Mean ± SE	120.51 ± 0.55	120.57 ± 0.46	

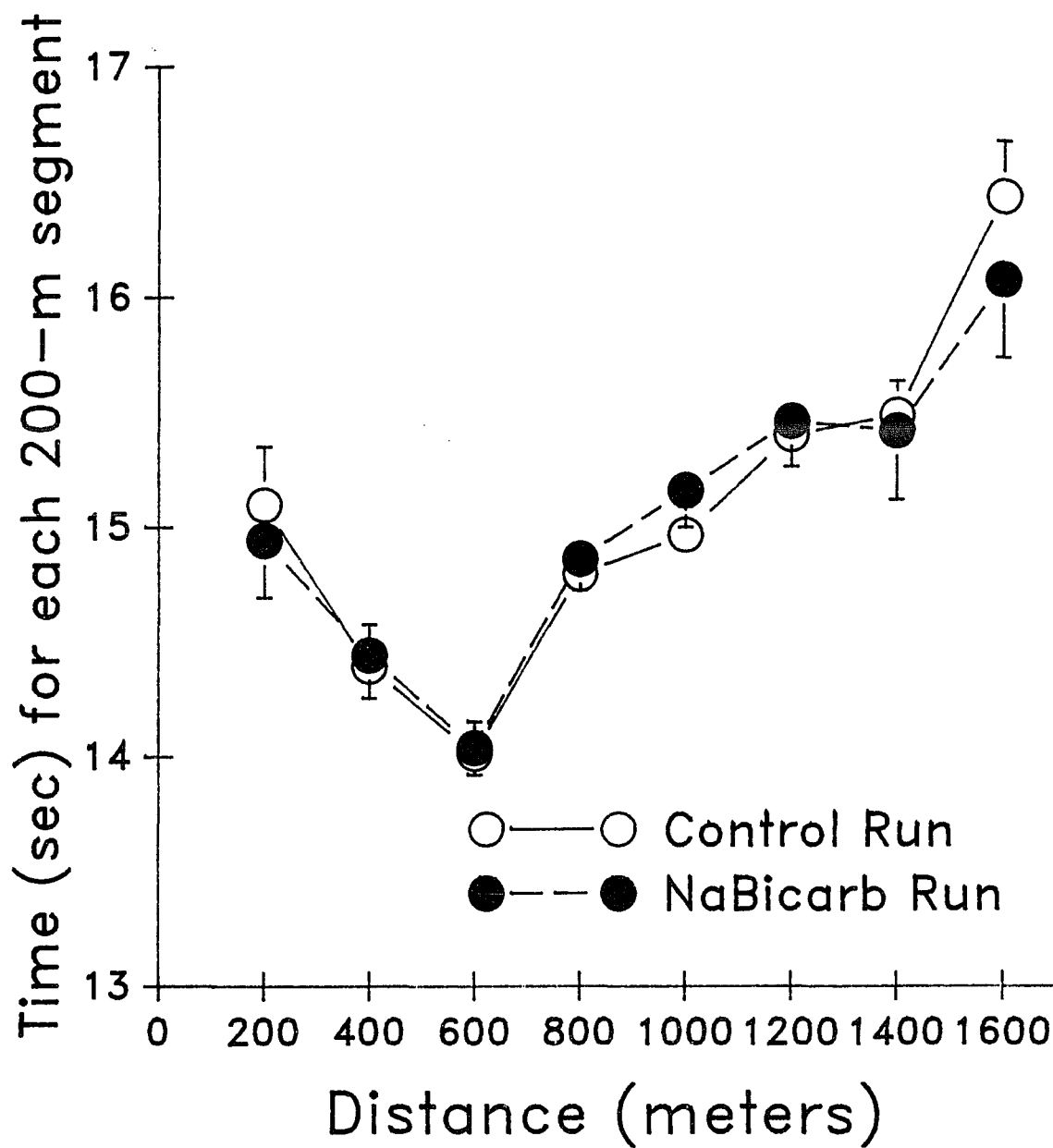


Figure 17--Time (sec) required for each 200-m segment following control and NaBicarb treatments.
N=16

3. Blood samples

Venous plasma lactate, pH, PCO_2 , and HCO_3^- values for the control and NaHCO_3 runs are shown in Figure 18. Pre-treatment values of plasma lactate, venous pH, PCO_2 , and HCO_3^- for both the control and NaHCO_3 treatments were within normal range. Pre-run (post-treatment) values for plasma lactate, venous pH, PCO_2 , and HCO_3^- obtained 3 hours later (Figure 18), were not significantly different from the pre-treatment samples when compared within treatments. There were no significant differences between treatments for pre-run samples for plasma lactate, venous PCO_2 , and HCO_3^- . However, pre-run mean pH was significantly greater following NaHCO_3 treatment (7.458 ± 0.005) when compared to the control treatment (7.421 ± 0.004). There was also a significant elevation in venous pH following NaHCO_3 treatment at each post-run sampling time (Figure 18b). All post-run pH values were significantly decreased from pre-run values within groups.

The post-run mean plasma lactate values (Figure 18a) were significantly increased following NaHCO_3 treatment when compared to control treatment values. Peak mean lactate was reached at 8 minutes after the runs for both treatment conditions, and was significantly increased from 27.84 ± 1.21 for the control treatment to 31.28 ± 1.43 for the NaHCO_3 treatment. There was a significant increase for both the control and NaHCO_3 groups in post-run lactate

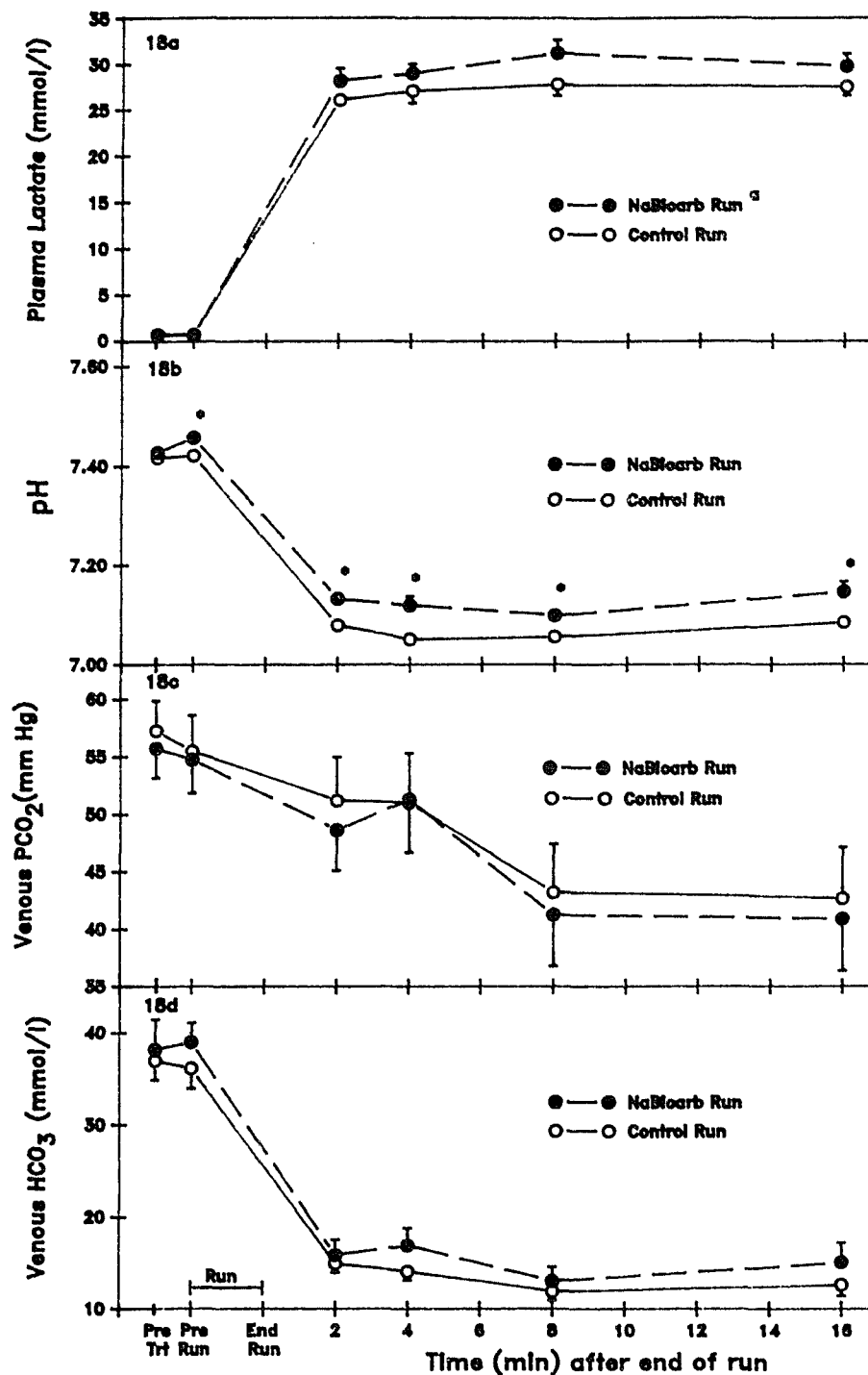


Figure 18--a) plasma lactate, b) pH, c) venous PCO₂, and d) venous HCO₃⁻ for control and NaBicarb treatments before and following 1600-m runs. N=16

^a Significant difference (P<0.05) between control and NaHCO₃ treatment post-run curves

*

Significant difference between control and NaHCO₃ values for that sample time

concentration (Time=2-16 min) above pre-run plasma lactate (0.86 ± 0.09 and 0.76 ± 0.04 mmol/l, respectively).

There were no significant differences in pre- or post-run venous PCO_2 (Figure 18c) and HCO_3^- levels (Figure 18d) between treatments. There was a significant decrease in post-run venous PCO_2 for the control and NaHCO_3 groups for the 8-16 min samples (8 min= 42.7 ± 4.44 and 41.4 ± 3.64 mmol/l, respectively) from pre-run values (55.1 ± 2.88 and 54.3 ± 2.87 mmHg, respectively). There was also a significant decrease in all post-run venous HCO_3^- values for the control and NaHCO_3 groups (minimum HCO_3^- = 11.9 ± 0.89 and 13.1 ± 1.52 mmol/l, respectively) from pre-run values (37.0 ± 2.02 and 38.2 ± 3.13 mmol/l, respectively).

Discussion:

1. Peak buffering capacity

In studies of induced alkalosis in humans, NaHCO_3 has been administered 45-120 min before the start of exercise. In the studies by Costill *et al.* (1984), Gao *et al.* (1988), and Katz *et al.* (1984) NaHCO_3 was ingested 45-60 min before exercise. Brien and McKenzie (1989), and Wilkes *et al.* (1983) administered

treatment over a 2 hr period and exercise commenced after the last capsule was ingested. These studies did not comment on the time of maximal buffering effect of the NaHCO_3 .

In the equine studies cited, Hank *et al.* (1985) exercised horses 1.5-2 hr following NaHCO_3 administration, Kelso *et al.* (1987) administered treatment 1 hr before exercise, and Lawrence *et al.* (1987a) treated horses 1.5-2.5 hr before exercise. These studies made no comment concerning time of maximal buffering effects of NaHCO_3 . The 2.5-3.0 hr post-treatment peak buffering capacity seen in this study indicated that these other equine studies may not have optimized the buffering effect of the administered NaHCO_3 .

Lactic acid is the major source of hydrogen ions (H^+) in the working muscles during intense exercise, with H^+ contributions of other organic acids being relatively small. Bicarbonate, protein, and phosphate buffer systems aid in buffering the increasing H^+ concentration during exercise (Beaver *et al.* 1986). Accumulation of intracellular H^+ has been well documented as a cause of muscle fatigue (Donaldson *et al.* 1978; Hermansen, 1981; Wilkie, 1986). Investigators have shown that intracellular pH is not affected by NaHCO_3 administration (Katz *et al.* 1984), but the efflux of H^+ from the muscle cells may be augmented by an increased extracellular pH (Mainwood & Worsley-Brown, 1975). Since the pH of extracellular fluid affects the

intracellular pH and since intracellular pH has been shown to be a limiting factor during intense exercise (Hultman & Sahlin, 1980), increased efflux of H⁺ from the cell could delay the onset of fatigue. A lack of improvement in run times indicated that onset of fatigue was not delayed by a presumed increase in efflux of H⁺ from the muscle cells in maximal effort events of 2 minutes or less.

2. Run times

The lack of improvement in run times (Table 30) in this study was consistent with the findings of other investigators using exercises of similar intensity and duration. Several investigators have used NaHCO₃ as a pre-exercise treatment to delay the onset of fatigue in humans. Iwaoka *et al.* (1989) showed that time to fatigue on a cycle ergometer at 95% maximal O₂ uptake was increased from 2.00 ± 0.44 to 2.98 ± 0.64 minutes following pre-exercise treatment with 0.2 g/kg BW of NaHCO₃. Wilkes *et al.* (1983) showed that performance significantly improved in an 800-m run when runners were pre-treated with 0.3 g/kg BW NaHCO₃. In that study, mean run times were decreased from 2:05.8 seconds for the control run to 2:02.9 seconds for the NaHCO₃ run. Jones *et al.* (1977), Pate *et al.* (1985), and Rupp *et al.* (1983) have also shown beneficial effects from pre-exercise treatment with NaHCO₃ in fatiguing

exercises lasting from 2-9 minutes. However, in single exercise events lasting less than 2 minutes the benefits of pre-treatment with NaHCO_3 have not been consistently demonstrated. Katz *et al.* (1984) showed that exercise performance in events lasting 45-100 seconds was not influenced by the intake of 0.2 g/kg BW NaHCO_3 in human athletes. Studies by Kindermann *et al.* (1977), McCartney *et al.* (1983), Parry-Billings and MacLaren (1986), and Sjöholm (1986) also failed to show any benefit from pre-treatment with NaHCO_3 in events lasting less than 2 minutes.

In investigations using horses, the fatigue delaying effect of pre-exercise treatment with NaHCO_3 has been equivocal. Kelso *et al.* (1987) claimed a significant improvement in 1600-m run times in Thoroughbreds from 114.0 ± 2.2 sec following placebo treatment to 111.3 ± 1.2 sec following treatment with 0.4 g/kg NaHCO_3 , but only with a significance level of $P < 0.10$. Lawrence *et al.* (1987b) failed to show any improvement in endurance from pre-exercise treatment with 0.3 g/kg BW NaHCO_3 in Quarter horses exercised to fatigue on a treadmill for a 20 minute period.

In this project, the lack of any improvement in performance following NaHCO_3 treatment may have been due to the short duration of the run. The mean run times for both runs was about 2 minutes, the lower limit in human studies showing an ergogenic effect from pre-exercise treatment with NaHCO_3 . Since

most Thoroughbred and all Quarter horse races last less than 2 minutes, NaHCO_3 probably is not an important ergogenic aid in those events.

In Thoroughbred races that last longer than 2 min (> 1.25 miles), pre-treatment with NaHCO_3 may have a beneficial effect. Figure 18 suggests that the horses pre-treated with NaHCO_3 were able to maintain speed better during the final 200-m segment. This may have been due to a delayed onset of fatigue from increased H^+ buffering by the HCO_3^- . However, there was no significant difference in mean run times for the final 200-m segment between the NaHCO_3 and control runs. However, at longer distances the divergence between the two curves may increase, resulting in faster run times during the later portions of a run in horses pre-treated with NaHCO_3 .

Human studies have shown that exercise events consisting of repeated bouts respond to pre-exercise treatment with NaHCO_3 even though the individual bouts may last less than 2 minutes. Improvement in performance is not seen at the beginning of the exercise but in later bouts. Costill *et al.* (1984) showed that time to fatigue in humans during repeated bouts of cycling was increased from 113.5 ± 12.4 seconds for the fifth bout following the control treatment to 160.8 ± 19.1 seconds for the fifth bout following pre-exercise treatment with $0.2 \text{ g/kg BW NaHCO}_3$. Gao *et al.* (1988) showed a significant improvement of swim

times in humans during the fourth and fifth heats of a five-heat (91.4 m/heat) trial following pre-trial treatment with 2.9 mmol/kg NaHCO₃.

The significance of this finding may be of benefit to trainers of Standardbred horses because of the multiple-heat nature of Standardbred training and racing. Lawrence *et al.* (1987a) showed a reduction ($P < 0.10$) of race time in Standardbreds competing in a mile race from 2:15.4 following a placebo treatment to 2:14.3 following 0.3 g/kg NaHCO₃ treatment. The horses in that study were warmed-up "according to trainer preference" which included at least 1 (frequently more than 1) warm-up heat of 1600 meters in 2:30 performed 45-60 min before run time (A.B. Lawrence, personal communication, 1990). Since a majority of Standardbred races last longer than 2 minutes, and because of the multiple pre-race warm-up heats used with that breed, pre-race treatment with NaHCO₃ may supply an ergogenic benefit to Standardbred racehorses.

3. Blood samples

The increased plasma lactate after exercise in the NaHCO₃ treated horses (Figure 18a) was consistent with the findings of studies in humans (Iwaoka *et al.* 1989; Katz *et al.* 1984; Wilkes *et al.* 1983) and horses (Lawrence *et al.*

1987b; Hank *et al.* 1985). Lawrence *et al.* (1987b) showed an elevation in peak lactate from 10.9 ± 1.0 mmol/l for the control group to 15.2 ± 1.5 mmol/l for the NaHCO₃ group when horses were worked to fatigue at 4.5 m/sec on an 11% grade. Hank *et al.* (1985) showed higher plasma lactate values for each sampling time during a 6 min treadmill standard exercise test. Studies have shown that plasma lactate concentration increased with exercise severity (Gollnick *et al.* 1986; Gottlieb *et al.* 1988) so the higher peak lactate values seen in this project were probably due to the more intense nature of the exercise.

The increased blood pH for the pre-run and all post-run samples after NaHCO₃ treatment was consistent with the findings of studies in humans (Costill *et al.* 1984; Katz *et al.* 1984; Wilkes *et al.* 1983) and horses (Lawrence *et al.* 1987a; Lawrence *et al.* 1987b). Lawrence *et al.* (1987b) showed that pre-run treatment with NaHCO₃ caused a significant elevation in pH prior to exercise. They showed a decreased effect during exercise but the effect was again evident during recovery ($P < 0.05$). Kelso *et al.* (1987) showed a significantly higher blood pH in the pre-run sample following NaHCO₃ treatment, but the post-run samples, while higher, were not significantly elevated.

The non-significant difference between control and NaHCO₃ values for venous HCO₃⁻ following exercise were consistent with the findings in other equine

studies (Kelso *et al.* 1987; Lawrence *et al.* 1987b). While the cell membrane is readily permeable to H^+ , it is essentially impermeable to HCO_3^- (Robin, 1961). Hence, only the buffering capacity of the extracellular fluid is increased by the administration of $NaHCO_3$ (Gao *et al.* 1988), and the intracellular pH is not directly affected by a change in the pH of the extracellular milieu (Costill *et al.* 1984). Mainwood and Worsley-Brown (1975) proposed that the higher pH gradient due to the increased extracellular HCO_3^- facilitated the efflux of H^+ and lactate from the muscle cell. Even though the venous HCO_3^- was not significantly greater for the horses treated with $NaHCO_3$, the slight elevation of HCO_3^- following treatment with $NaHCO_3$ may have augmented the efflux of H^+ from the muscle cells to delay the onset of fatigue.

Treatment with $NaHCO_3$ caused a significant elevation in blood pH and allowed for a significantly greater production of lactate. However, the increased buffering capacity and increased lactate production did not result in a significant difference in run times for a 1600-m run. This was probably because of the brevity of the exercise event. It was concluded that $NaHCO_3$ did not effect performance in a 1600-m run.

Summary and Conclusions

Part I.

Objectives:

- 1) To determine the running abilities of 25 Thoroughbred horses at 1200, 1600, and 2000 meters alone and in competition.
- 2) To determine the measurable variables of performance in racehorses on the racetrack.
- 3) To design standard exercise tests (SETs) on the high-speed equine treadmill to assess those variables.

An average time for each pair of runs was determined and used to correlate with the measured variables. The relative order of finish was not affected by run distance. Faster horses at shorter distances were also the faster horses at longer distances. Peak and total plasma lactate concentrations following the runs correlated well with performance times. Faster horses attained higher plasma lactate values. Maximal HR was higher during the 2000-m runs than during the 1200- and 1600-m runs. Maximal HR did not correlate well with run time.

The treadmill SET variables that best correlated with times for the track runs were pre-training heart rate score, V_{200} , onset of blood lactate accumulation

measurements (T_{La4} , V_{La4} , and W_{La4}), blood lactate concentrations (peak and total lactate) and VO_{2max} . It was concluded that athletically superior horses performed a similar sub-maximal work load at a lower heart rate than athletically inferior horses. The lowered HR for the faster horses was more evident while the horses were unfit. The relatively high variance associated with V_{200} caused that variable to be less reliable as a performance correlate. Since higher plasma lactate concentrations (peak lactate, total lactate) and lower OBLA values were seen in the faster horses at distances ranging from 1200-2000 m, it was concluded that high lactate production at relatively low work loads was advantageous for success in racehorses at those distances. High aerobic capacity (VO_{2max}) was also determined to be beneficial in runs of 1200-2000 m.

This project demonstrated that variables measured during sub-maximal exercise like W_{La4} and V_{La4} correlated as well with performance as maximal-effort variables like VO_{2max} . Since sub-maximal measurements are more safely obtained in racehorses than maximal-effort values, it was concluded that W_{La4} and V_{La4} would be of value in performance prediction.

Part II.

Objectives: To assess the influences of:

- 1) training,
- 2) competition, and
- 3) putative ergogenic aids on performance

Interval training was more labor intensive than conventional training and was deemed impractical for all horses. However, it was concluded from this study that higher quality horses could benefit from the more intense exercise encountered during interval training.

Peak heart rates, heart rate recovery curves, and run times were not significantly different between the alternatively and conventionally trained groups. However, higher lactate production and increased plasma lactate clearance by the alternatively (IT and LSD) trained group demonstrated an increased anaerobic capacity. This project did not provide sufficient information to support or refute the purported advantages of interval training due to the small sample size.

Twenty weeks of high-speed treadmill training employing interval training methods significantly lowered the HR in Thoroughbred racehorses performing a SET. The degree of improvement in HR due to treadmill training showed

that the treadmill could be used to develop a high degree of fitness in horses. The treadmill was useful for training in inclement weather and for evaluating fitness and racing ability. However, the treadmill cannot totally replace training on the track. Horses still need track training to learn to carry a jockey, break from the starting gate, run in a straight line, negotiate turns, run on dirt or turf, pass other horses, and adjust to the presence of other horses on the track.

As stated previously, interval training was shown to be more labor intensive and time consuming than conventional forms of training. However, the reduced amount of labor required for treadmill training and the freedom from the constraints of weather makes interval training on the treadmill a more practical option to conventional training methods. Human performance in middle-distance events has steadily improved over the past few decades, and at least part of the improvement in performance has been attributed to improved training methods including interval training. More research showing the benefits of interval training in horses may encourage horsemen to change their established methods of training.

The horses used for this project ran more slowly when running in competition than when running alone. The slower times for the competitive runs probably occurred because of an earlier onset of fatigue in those runs when compared

with the solo runs. Females performed more poorly during both competitive runs, while there was no difference in solo and competitive run times for the males at either distance. There was no difference in run times between competitive and solo runs due to age. No group showed improvement in run times when running with competition, which may be a reflection of the quality of horses used for this study and the susceptibility of the horses to stress induced impairment of performance.

Maximal heart rate did not vary between competitive and solo runs except for an unexplainable decrease in maximal heart rate for the 1600-m competitive run. Peak and total plasma lactate values tended to be higher for the 1600-m competitive runs, and were significantly higher for the 1200-m competitive run.

There was a significant decrease in 1600-m run time following a 12% fat-added diet. Plasma glycerol, triglycerides, non-esterified fatty acids, beta hydroxybutyric acid, and glucose levels following the run were significantly altered by the addition of fat to the diet. The fat-added diet increased muscle glycogen storage. However, that does not appear to be the reason for the decreased run time since glycogen utilization was equal for both runs. The faster mean run time for the fat-added diet may have been due to increased availability of blood glucose, better utilization of ketone bodies for fuel, and a reduced state of ketosis following the fat-added diet. These factors may have

postponed the onset of fatigue when compared with the control diet. The 12% fat diet was acceptable after a few days of adaptation.

Maximal buffering capacity was reached 2.5-3 hrs after administration of NaHCO_3 in horses. Pre-treatment with NaHCO_3 caused a significant elevation in blood pH and allowed for a significantly greater production of lactate. However, the increased buffering capacity and increased lactate production did not result in a significant difference in run times. This was probably due to the brevity of the exercise event. Longer distance events and/or exercise events with multiple heats may benefit from pre-run treatment with NaHCO_3 .

Future studies:

Since some correlates of performance have been identified, the next procedure would be to measure these variables in unraced 2-year olds and predict racing performance from those measurements. The racing careers of the 2-year olds would then be closely followed to determine the accuracy of the predictions.

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Appendix

Glossary of terms and abbreviations

AT	Alternative method of training
AUC	Area under the curve
BG-SET	Blood-gas standard exercise test
BHBA	Beta-hydroxybutyric acid
BTPD	Body temperature and pressure, dry
BW	Body weight
CT	Conventional methods of training
DE	Digestible energy
GLOL	Glycerol
GLU	Glucose
H⁺	Hydrogen ion
HCO₃⁻	Bicarbonate ion
HR	Heart rate
HR-SET	Heart rate score standard exercise test
IT	Interval training
La⁻	Lactate

Lac 8	Plasma lactate at the end (8th min) of the BG-SET
LSD	Long slow distance
LT	Lactate threshold
MaxHR	Maximal heart rate
MaxPCV	Maximum packed cell volume
Min pH	Minimum arterial blood pH
Min P_aO₂	Minimum arterial oxygen partial pressure
Min O₂ Sat	Minimum arterial oxygen saturation
Min HCO₃⁻	Minimum blood bicarbonate
NaHCO₃	Sodium bicarbonate
NEFA	Non-esterified fatty acids
OBLA	Onset of blood lactate accumulation
OBLA-SET	Onset of blood lactate standard exercise test
P_aCO₂	Arterial partial pressure of carbon dioxide
P_aO₂	Arterial partial pressure of oxygen
PCV	Packed cell volume
PLac	Peak lactate

RBC	Red blood cell
RLN	Recurrent laryngeal neuropathy
SET	Standard exercise test
STPD	Standard temperature and pressure, dry
T_{La4}	Calculated time at which a 4 mmol/l plasma lactate concentration is attained
TLac	Total lactate
TRIG	Triglycerides
V₂₀₀	Velocity attained at a steady-state heart rate of 200 beats/min
V₂₀₀-SET	V ₂₀₀ standard exercise test
V_{La4}	Velocity at which blood lactate reaches a concentration of 4 mmol/l
VO_{2max}	Maximal oxygen uptake
VO_{2max}-SET	Maximal oxygen uptake standard exercise test
W_{La4}	Work load at which blood lactate reaches a concentration of 4 mmol/l

Vita

J. Daniel Harkins was born in Greenville, Kentucky on April 11, 1947. He received a Bachelor of Science in Animal Science from the University of Kentucky in December, 1970. In the fall of 1971, he entered the School of Veterinary Medicine at Purdue University. He graduated with a Doctor of Veterinary Medicine in June, 1975. After completing an internship and residency at the Animal Medical Center in New York City, he operated a private practice for 8 years at Lake Almanor, California. In August, 1986 he was awarded a graduate assistantship by the School of Veterinary Medicine, Louisiana State University where he pursued a Ph.D. degree in veterinary physiology. These studies were guided by Dr. Steven G. Kamerling, Associate Professor, Department of Veterinary Physiology, Pharmacology, and Toxicology. At present, Dr Harkins is a post-doctoral fellow at Cornell University, Ithaca, NY.

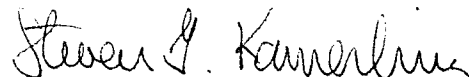
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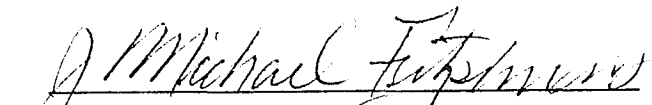
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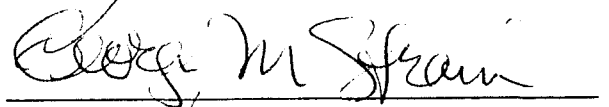

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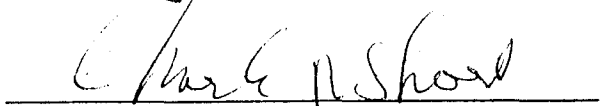

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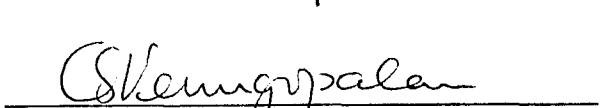
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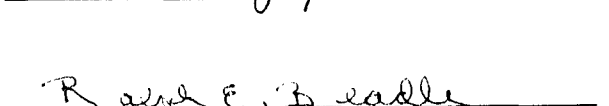












Date of Examination:

August 23, 1990