LACTATE AND GLUCOSE RESPONSES TO EXERCISE IN THE HORSE: INFLUENCE OF INTERVAL TRAINING AND DIETARY FAT

by

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Introduction

The nutritional needs of performance horses are largely determined by the amount of work and training. The energy and nutrient requirements of mature horses at maintenance can usually be met by feeding good quality roughage. The increased energy demands of hard work, however are met by feeding starch in the form of grain.

Addition of fat to the diets of equine athletes in place of carbohydrate has potential benefits, especially for submaximal work. Fat can be used to increase energy density and reduce bulk, hence weight in the large bowel. Use of fat instead of large amounts of soluble carbohydrate results in fewer fermentative disturbances, such as colic and laminitis, and exertional rhabdomyolysis. One nutritional strategy for stamina in humans, carbohydrate loading, may be conducive to exertional rhabdomyolysis in horses (Kronfeld and Downey, 1981).

Fatty acid oxidation results in less carbon dioxide production and respiratory effort, compared to glucose oxidation. It spares muscle glycogen and should result in less lactic acid production during aerobic exercise, hence delay fatigue. These metabolic advantages may be limited to low intensity exercise, perhaps less than 25 % VO_2 max. This limit may be raised, we propose, by a second strategy

for stamina, fat adaptation, achieved by interval training horses fed a high fat diet (Kronfeld and Downey, 1981).

Horses adapted to fat have also performed sprinting. This may be due to reduced bulk (mass in the large bowel). It is not readily explained in terms of metabolic regulation for working muscles.

The objective of this experiment was to examine the influences of fat adaptation on certain metabolic responses in mature Arabian horses to an incremental exercise test. More specifically an attempt was made to determine the lactate and glucose threshold in horses.

Literature Review

Energy Sources

Exercise basically involves conversion of chemical energy into the mechanical energy of muscle contraction. The immediate source of chemical energy is adenine triphosphate (ATP). There are four energy systems that replete ATP (McGilvery, 1975):

1) The phosphagen system of ATP and Phosphocreatine (PCr);

2) Breakdown of glycogen to lactic acid (glycolysis);

3) Oxidation of glucose;

4) Oxidation of fatty acids (FA).

High energy phosphates, ATP and PCr, are stored in muscle fibers. ATP used in contraction is rapidly replenished by transfer of a high energy phosphate from PCr. This system supplies ATP for a brief period of exercise, after which working muscles must be able to rapidly increase their metabolic rate to provide enough ATP for the activity to continue. Training and diet have not been shown to influence the availability of ATP and PCr.

Glycolysis is the breakdown of glycogen resulting in lactic acid. ATP is derived from glycolysis at about 0.25 to 0.5 the rate from PCr (McGilvery, 1975). Glycolysis takes place in the cytosol and is catalyzed by

phosphorylase. Epinephrine and calcium release during exercise increase activity of phosphorylase which in turn increases glycogenolysis.

ATP can also be replenished by oxidation of glucose at a rate of about half that of ATP generation from glycolysis. Glucose is extramuscular in origin and delivered to the muscles by the blood. Glucose entry rate determines its oxidation rate in mitochondria. The entry rate of glucose is regulated by insulin and the counterregulatory hormones, glucagon, growth hormone, cortisone, and catecholamines. Oxidation of glucose can be increased by carbohydrate diets and decreased by high fat diets and by fasting.

The fourth energy system that generates ATP is oxidation of fat. Oxidation of FA has half the power output of glucose oxidation, because the rate of FA transport into cells and mitochondria is relatively slow (McGilvery, 1975). Plasma free FAs and lipoproteins supply FA for use by the muscle. Some FAs are also released from adipose cells in the muscle and become directly available to muscle cells (Hurley, 1980).

During exercise FA are released from adipose tissue and are taken up by the liver. FA are converted into triglycerides and ketones in the liver and released into the blood for utilization as substrates predominantly by muscle.

FA are the main substrate for ATP synthesis during low intensity, endurance type work.

Oxidation of FA can be limited by plasma free FA concentration, carnitine, and mitochondrial enzymes. Cellular uptake of plasma free fatty acid, is by diffusion, hence is increased by higher plasma free FA concentrations. Carnitine plays a part in transportation of fatty acyl CoA across the mitochondrial membrane.

The contribution that each ATP source makes to the energy need during exercise depends on many factors, such as intensity, duration, amount of conditioning, diet and nutritional status, genetic makeup and health (Gollnick, 1988). During exercise it is necessary to use both aerobic and anaerobic mechanisms, and a balance of many ATP sources occurs. Power output is proportional to the rate of ATP generated (Table 1), which roughly increases by a factor of 2 in each step from FA oxidation, glucose oxidation, lactic acid production, to PCr donation (McGilvery, 1975).

At low power, the supply of oxygen and FA to the muscle via the circulation is adequate, and FA is the preferred fuel source. FA oxidation increases levels of citrate which inhibits phosphofructokinase (PFK), a key regulatory enzyme of glycolysis. Thus utilization of glycogen and glucose is inhibited with increasing levels of citrate, thus FA

Table 1. Power output of fuels.

Fuel	Maximum Power umoles/g/sec
ATP, PCr	1.6 to 3.0
Glycogenolysis	1.0
Oxidation of glucose	0.5
Oxidation of fat	0.24

oxidation tends to spare carbohydrates.

PFK is also responsive to levels of ATP and CP. When the ATP:ADP ratio decreases due to consumption of ATP in the cell, the inhibitory effect on PFK by citrate is decreased and carbohydrate utilization is enhanced (Hodgson, 1985). Thus influences act on PFK to determine the balance of utilization of FA and carbohydrate.

Lactate Threshold

The lactate threshold is the work intensity or speed at which plasma lactate concentration begins to rise sharply above a resting baseline level or an initial moderate slope. It is one manifestation of a controversial concept, the lactate threshold (Davis, 1985; Brooks, 1975). Several mechanisms have been proposed (Davis, 1985; Brooks, 1975). One concept emphasizes that exercise induced lactate accumulation is related to oxygen uptake by working muscle. It assumes that the plasma lactate increase reflects the lactate produced in the muscle (Davis, 1985). However, generation of lactate probably occurs during all levels of exercise and lactate can accumulate in muscles during completely aerobic conditions (Connett, 1984).

Another factor is substrate predominance. When glycogen is broken down under the influence of epinephrine, or when glucose enters muscle cells more rapidly during

hyperglycemia, production of lactate is more rapid (Kronfeld and Downey, 1981).

Alternatively, lactic acid can be formed at a rate that exceeds its clearance from the blood. In other words, blood lactate increases because lactate removal mechanisms do not keep up with lactate production. In this view blood lactate does not adequately reflect muscle lactate (Brooks, 1986).

Despite arguments about its physiological interpretation, anaerobic threshold and lactate threshold have been used empirically, to assess the fitness of athletes.

Since distance runners compete at an exercise intensity slightly above lactate threshold, it is a consistent and powerful prediction of performance in aerobic exercise (McArdle, 1986). A high correlation has been found in humans between lactate threshold and performance (Snow and Vogel, 1987).

The lactate threshold has been determined through incremental step tests in rats and humans but not horses (Kinderman et al., 1979). In humans it has been used to design interval training programs, and it has correlated with racing performance (Ivy, et al., 1981). In rats it has been increased by high fat diets.

If the lactate threshold occurs at higher speeds with both training and high fat diets, this delay in fatigue would help to win races.

Attempts have been made to determine the lactate threshold in horses, however only a few number of steps were used (Lindner, et al., 1991). In humans, a lactate concentration of 4 mM/L has been used to indicate the onset of blood lactate accumulation (OBLA) (Kindermann et al., 1979; Weltman et al., 1990; McClellan et al., 1989). OBLA is the concentration of blood lactate at the point where it begins to rise sharply above a resting baseline level. A lactate concentration of 4 mM/L determined in humans has been adopted for use in horses (Persson, et al., 1983; Sloet, et al., 1985; Thornton, 1985; Wilson et al., 1982). In human cyclists, curve analysis for the lactate threshold has been done using least square regression (Ivy et al., 1981). Also, the lactate threshold has been correlated to heart rate. In humans, a correlation between lactate threshold and heart rate was .60 (Dwyer and Bybee, 1983). In humans correlation coefficients have been determined for OBLA and treadmill velocity (r=0.91) and for OBLA and maximal oxygen consumption (r=0.98) (Farrell, et al., 1979). Lactate threshold is 50 to 60% maximal oxygen consumption rate in untrained subjects and can improve to 70 to 75% with training (MacDougall, 1977; Kronfeld and Downey, 1989).

However curve analysis without a sufficient number of points lacks accuracy and precision.

Influence of Training

Training increases the ability of a muscle fiber to utilize oxygen. This is associated with an increase in the number and size of the mitochondria. There is also an increase in capillaries around the muscle fibers.

The relationships between mitochondria, muscle and whole body respiration has been studied by Davie et al. (1981). After a ten week program of endurance treadmill running, the mitochondrial content of the muscle increased 100 % as did absolute tissue oxidative capacity. Maximum oxygen consumption increased only 14 % with training, thus was a poor indicator of endurance. Training increased the mitochondrial enzymes available to break down more free fatty acids for use as a fuel, shifted metabolism towards oxidation of fat, spared muscle glycogen, reduced lactate accumulation, and helped to delay fatigue. The ability to conserve the glycogen and to forestall its depletion are important physiological adaptations resulting from endurance training (Karlsson and Saltin, 1971).

Strategies for Stamina

Endurance athletes often modify their diets to improve specific performances. Two of the primary methods used are carbohydrate loading and fat adaptation (Kronfeld and Downey, 1981). Carbohydrate loading can be used to increase muscle glycogen concentration and thus delay fatigue. This method is used consistently in humans, however the small digestive capacity of the stomach and small intestine limits use of carbohydrate loading in the horse. Use of large amounts of soluble carbohydrate in the equine may be conducive to colic and laminitis, and exertional rhabdomyolysis. Alternatively, fat adaptation is another method that can be used. In the horse, fat can be used to increase energy density and reduce bulk, and thereby reduce fermentative disturbances. Fat adaptation has been used to increase oxidation of fatty acids and consequently spare muscle glycogen during an event. The use of these two methods depends on duration and intensity of exercise (Kronfeld and Downey, 1981).

Fat Use in Horses

The potential of fat supplementation for horses was summarized at the start of this Introduction. Past studies of dietary fat in horses will now be reviewed.

Digestibility of Added Dietary Fat. Horses fall into the classification of nonruminant herbivores or hindgut

fermentors. The hindgut, or the cecum and colon, is specialized for microbial fermentation of plant fiber. Protein, fat, vitamins and minerals are digested and absorbed in the stomach and small intestine. Large meals of grain overwhelm the digestive capacity of the stomach and small intestine. This results in rapid fermentation in the hindgut of the grain carbohydrates which can lead to digestive problems including colic and laminitis (Jackson and Pagan, 1990). One way of avoiding digestive problems is by adding dietary fat to the diets of horses with increased energy needs. This provides a more concentrated energy source without the added bulk that overwhelms the stomach and small intestine.

Addition of corn oil to the diet of standardbred geldings has resulted in a mean apparent digestibility for fat of 88% (Bryant, 1969). These horses were very capable of digesting the fat from a semipurified diet. They showed no digestive disorders, such as diarrhea, previously seen in horses fed high fat levels.

When ponies were fed from 0% to 20% levels of corn oil, there was a linear increase in apparent digestibility of energy and fatty acids as corn oil levels increased (Bowman, 1977). Apparent digestibility of crude protein was not affected. Serum cholesterol demonstrated a quadratic effect

as levels of corn oil increased in the ration. Serum triglycerides were not affected.

Corn oil was added at 15% and 30% of the energy to the diets of pony geldings in 3x3 Latin Square design (Kane et al., 1979). It increased the energy available to the ponies, apparent digestibility of EE, digestible energy and metabolizable energy. These changes increased the energy balance since heat production was not altered. The ponies utilized the added corn oil with no detrimental effects.

The digestibility and heat production of three different fats, corn oil, blended fat and inedible tallow were examined in pony geldings (McCann, 1987). Added fat increased metabolizable energy (ME), apparent fatty acid digestibility and the energy balance. There was no difference in heat production and DM digestibility. Digestible energy tended to be higher for the corn oil diet than for the others. The digestibility coefficient for EE increased the most for corn oil and inedible tallow. NDF, ADF and cell contents were highest for corn oil but did not differ significantly among diets. Fat supplementation increased metabolizable energy with no effect on heat production and therefore increased the energy balance. Serum calcium and magnesium levels stayed within the normal range with the added fat.

The effects of exercise on nutrient utilization were studied in eight light horse mature geldings fed a 14 % added fat diet (Worth, 1988). Before conditioning, added fat decreased apparent digestibility of DM, cell contents, energy, NDF. It increased apparent digestibility of EE. After conditioning, the horses on the fat supplemented diet had a higher apparent digestibility for CP, ADF, energy and EE than the horses on the control diet.

Mature Quarter Horse geldings were fed a 0, 5, or 10 % animal fat diets in a 3X3 Latin Square design (Meyers et al., 1987). Addition of fat to the diet allowed a decreased feed intake to maintain constant body weight and condition.

Yearling Quarter Horses were fed diets containing 0, 5 and 10 % added fat (Scott et al., 1987). Diets were equal in nutrient to calorie ratio. Digestibility studies revealed that horses on the 10 % fat diet had higher apparent digestibilities of protein, EE and NDF. Also, ADG was higher and feed intake was lower for the horses on the 10 % fat diet. Thus, horses on the high fat diet were eating less but were also gaining more weight each day than the horses on the control diet. It was concluded that yearling horses can be fed fat in high energy diets to replace some of the carbohydrates.

Feeding 10 % added fat reduced the DE requirements of six mature horses (Potter et al., 1987). Horses fed 10 %

added fat had a lower feed intake and lower DE intake. The digestible energy required for maintenance during no activity was calculated and subtracted from the total digestible intake. The remaining DE consumed was defined as the DE required for work. The DE for work was adjusted for constant work loads then subtracted from total DE intake to estimate maintenance requirements for DE in other periods. The DE required for maintenance for horses on the 10 % fat diet was met with 75 and 95 % of the DE required by the horses on the control diet. It was concluded that the 10 % fat diet was more efficient for DE used for maintenance than the control diet. This effect was possibly due to a lower heat of fermentation which resulted in a decrease in energy needs for thermal regulation.

Bone growth of weanlings was not affected by dietary fat (Davison et al., 1987). Weanlings fed a 10 % fat diet had lower concentrate intakes, lower feed to gain ratios and higher average daily gains than controls. Bone density changes, closure of epiphyseal plates were similar for both groups of weanlings. No radiographic skeletal abnormalities were found. Digestion of EE and NDF were higher for the fat fed horses. Concentration of the hormones thyronine and triiodothyronine were similar in both groups. It was concluded that fat can be fed to weanlings as an efficient energy source to support growth and development.

Addition of fat to the equine diet can increase apparent digestibility of energy and EE. It can reduce the amount of feed needed for weight gain. Horses can utilize the additional fat with no detrimental effects.

Aerobic Exercise. The addition of dietary fat is beneficial to aerobic exercise. It has been shown to increase endurance by sparing muscle glycogen and protecting against decreases in blood glucose. Rats exposed to a high fat diet were capable of prolonged, intense exercise (Miller et al.,1984). Two groups of rats were fed a low carbohydrate diet (78 % fat, 21 % protein, 1 % carbohydrate) or a normal diet (11 % fat, 20 % protein, 69 % carbohydrate). Rats on the low carbohydrate diet (LCD) had significantly decreased resting blood glucose levels. The LCD animals had lower blood lactate levels at rest and after a run to exhaustion. They also ran significantly longer than the rats on the normal diet (ND). Resting glycogen levels in the soleus and red vastus lateralis muscle were significantly lower in the LCD rats. At exhaustion LCD rats were almost depleted of glycogen whereas those of the ND rats were only partially depleted. The enzyme activity of 3-hydroxylacyl CoA dehydrogenase increased during the trial period. The diet and exhaustion appeared to increase citrate synthase (CS) activity. No significant change was found in 3-hydroxybutyrate dehydrogenase. The untrained

rats exposed to a high fat diet were capable of prolonged intense exercise regardless of limited glycogen stores. It was concluded that the improvement in performance partly resulted from muscular adaptation to the diet that seemed to increase fat oxidation and concomitantly spare glycogen.

The effects of feeding a 10 % fat supplemented diet on muscle glycogen were studied in 12 mature Thoroughbreds (Scott, et al., 1991). The horses were divided into three groups and fed to achieve moderately low, moderate or moderately high body fat content. Horses were trained for 28 d. A standard exercise test (SET) was performed before and after a 28 d training period. The horses on the fat supplemented diet had significantly greater muscle glycogen concentration than horses on the control diet. The horses in the moderate and moderately high body fat condition had more resting muscle glycogen and used greater amounts of muscle glycogen during exercise than horses in the moderately low body fat condition. There was also no significant difference in horses fed the moderately low fat diet and controls. It was concluded that horses maintained in moderate body condition and fed 10 % fat supplemented diet had an optimum combination of maximum muscle glycogen concentration and minimal body fat.

A glycogen sparing effect has been found in horses after 12 weeks on a 10.5 % added fat diet (Greiwe et

al.,1989). Six two year old horses of light horse breeding were conditioned aerobically for 12 weeks. Three horses were fed a 10.5 % fat supplemented diet and three horses were fed a control diet. Each horses was conditioned on a treadmill 5 d/wk at 2.9 m/s until its heart rate reached 160 beats per minute. A standard exercise test (SET) was performed at 0, 6, and 12 weeks of training. During the SET's, the horses were walked at 1.56 m/sec for 3 min then trotted at 2.90 m/sec until their heart rate stabilized at 160 BPM, and then walked at 1.34 m/sec for 5 min to cool out. An initial preexercise blood sample and muscle biopsy were taken, a second blood sample was taken after 3 min of trotting, a third blood sample was taken when the heart rate stabilized at 160 BPM, and a final blood sample and muscle biopsy were taken after 10 min of recovery at a slow walk.

The fat fed horses ate 0.3 kg/d less than the control horses and gained 0.03 kg/d more in BW than the control horses (Greiwe, 1989). There were no significant differences between diets for any of the blood parameters. However, there was a conditioning effect on blood components: lactate and glucose significantly increased, and FFA and cholesterol significantly increased in fat fed horses compared to controls. There were no significant differences between fat fed and control horses in serum protein. Muscle glycogen decreased 20 % in the fat

supplemented horses after exercise in the final SET, whereas it decreased 68 % in the control horses (Greiwe, 1989). Resting levels of glycogen after the twelve week experimental period were 47 % higher in the control horses than in the fat horses. It was concluded that there was a sparing effect of muscle glycogen in the fat supplemented horses, addition of fat had a negative effect on resting muscle glycognen, and that a longer adaptation period may be needed to evaluate all of the effects from added dietary fat.

The addition of an 8 % fat diet has been shown to protect against decreases in blood glucose during trail rides (Hintz et el., 1978). Six horses were divided into two groups, one of which was given a control diet and the other was given an 8% added fat diet. They were ridden fifteen miles a day three times per week. Two trail rides of 37 miles were conducted two weeks apart, then the diets were switched. The horses were given a three week adjustment period and two more trail rides were conducted. Horses on the 8% fat diet required 15% less feed than the control diet. Muscle glycogen decreased during exercise but was not affected by dietary fat. Blood glucose decreased after the ride for both groups, and this decrease was greater for the control horses. Plasma FFA increased significantly during exercise in both diet groups. Blood

lactate levels increased after exercise but did not significantly differ between diets.

The effect of feeding three diets supplemented with starch, protein or fat on endurance type performance was examined by Slade et al. (1975). Three horses were assigned to one of the three diets in a Latin Square design. The horses were exercised for one to two hours daily and at the end of a five week period they were taken on an endurance ride. The ride was completed in an 8 to 10 hour period and included 35 to 40 miles of mountainous terrain. Food was witheld from the horses immediately before the ride and during the ride. They were given water after the third and sixth hour of the ride. Blood samples, respiration rate, and pulse rate were taken ten minutes after exercise on the day preceding the endurance ride and following the ride. Blood samples were also taken following the ride.

Blood samples indicated that horses on the fat supplemented diet performed the best and horses on the protein supplemented diet performed the worst (Slade et al., 1975). Blood glucose levels increased on the fat supplemented diet and decreased on the other two diets indicating that there was less dependence on glucose as an energy source in horses fed fat. Blood gases and pH indicated that horses on the fat supplemented diet had the least metabolic acidosis and the horses on the protein

supplemented diet showed the most metabolic acidosis. Hematocrit, hemoglobin and total protein levels indicated less dehydration for the fat supplemented diet. Horses on the protein supplemented diet had higher pulse and respiration rates after exercise, suggesting that it was least effective in developing stamina. Horses on the fat supplemented diet did not sweat as much as horses on the other two diets. It was concluded that feeding fairly high levels of fat may be beneficial for endurance work.

The influence of dietary energy source on substrate utilization during different exercise intensities was examined by Pagan et al., (1987). Three Standardbred horses were used in a 3X3 Latin Square design. The horses were fed a diet high in protein (20% CP), carbohydrate (12% CP) or fat (15% soybean oil) during each one month period. The first two weeks of each period consisted of light work on a treadmill two to three times per week. The horses performed a high speed exercise test during the third week. During the high speed test, the horses performed at the fastest speed that could be maintained at their natural gaits for a prolonged period of time. Two horses performed at 10 m/s and one horse performed at 9 m/s. Blood samples were taken at rest, every two minutes during exercise, immediately after exercise and two and five minutes after exercise. The fourth week consisted of a long slow exercise test on a

treadmill. During the long slow exercise test, the horses were trotted at 5 m/sec for 105 min. Respiratory quotient, lactate, glucose, and FFA were measured, and muscle and liver biopsies were taken.

During the high speed exercise test, blood lactate reached 4 mmol/liter after 6 min of exercise in the control group and after 12 min of exercise on the high fat group (Pagan et al., 1987). Blood lactate accumulation was positively correlated with muscle glycogen utilization. Resting levels of blood glucose dropped significantly in all treatment groups through 10 min of exercise. In the high fat group, post exercise and the last four minutes of exercise glucose levels were not significantly different from resting glucose levels. The high protein group had the lowest blood glucose levels after 8 min of exercise. There was no significant difference between treatments in blood glucose or plasma FFA before, during or after exercise. The high fat and high protein diets appeared to spare muscle glycogen utilization during exercise. During the long, slow exercise test, RQs indicated that carbohydrate oxidation was highest for the control horses, and lowest for horses on the fat diets. RQ for the horses on the high protein diet was 54%. As the horses were exercised, the RQ decreased, indicating a shift in substrate use from carbohydrate oxidation to fat oxidation. Concentration of plasma free FA

followed the shift in substrate utilization. Blood glucose levels significantly decreased in the high protein diet after 30 min of exercise indicating a heavy reliance on blood glucose for energy generation. It was concluded that the delay in mobilization and utilization of fat may have occured due to lack of conditioning from the light training during the experimental period.

Anaerobic Work. Added fat has been claimed to improve the performance of sprinting and cutting horses. Such an improvement may be due to lighter weight, but no beneficial effect of dietary fat on anaerobic metabolism has been reported for other species. Two experiments examined effects of 10% added dietary fat on aerobic work and anaerobic work (Webb et al., 1987). In one study, horses were exercised every other day, performing three, 4-minute miles. An initial SET was performed, then the horses were fed the 10% added fat diet. After three weeks another SET was performed. During each SET horses worked on an inclined treadmill at 2.9 m/s, a slow trot, for 10 minutes with 22.73 kg of added weight. No significant change was found in heart rates. Blood lactate levels did not differ significantly, but tended to be elevated when the control ration was fed. Plasma glucose levels dropped significantly when horses were fed the control diet, but remained constant when the fat diet was fed. The authors concluded that the

horses were using more anaerobic metabolism for energy production when they were on the control diet than when they were fed the fat diet. Unfortunately, the effects of diet were confounded by those of training.

In the second experiment, four mature cutting horses were used to examine the effects of added fat on anaerobic work. Before anaerobic work, the horses underwent aerobic conditioning for eight weeks. The aerobic work consisted of a 45 minute workout at a walk, trot and lope for 5 days/week. During the six week anaerobic conditioning period, the horses performed an additional two days of a 30 minute warmup period followed by a 15 minute anaerobic workout using a "mechanical cow". The horses were fed the control diet during the first experimental period. During a second experimental period, fat was added to the control ration at 10%. They found that the horses recovered more slowly when the 10% added fat diet was added and that heart rates were near maximum for both diets. Blood lactate concentrations were greater at the end of exercise and 5 min recovery for the horses on the 10% added fat diet. Authors concluded that the horses on the high fat diet executed more hindquarter turns, worked harder and had more energy for work than the horses on the control diet. Again, the experimental agenda confounded the effects of training and

diet. Moreover, rider bias could have influenced incentive to the horses during each SET.

A switchback experiment on six mature racehorses (three Quarter Horses and three thoroughbreds) determined the effects of a 10 % fat diet on sprinting performance (Oldham et al., 1989). The horses were conditioned by cantering and galloping on a track during the experiment. The horses were given a three week adaptation period on each diet and then subjected to an exercise test. The exercise test included four, 600-m gallops, interspersed with five min partial recovery intervals. Horses on the high fat diet had significantly greater resting muscle glycogen concentrations and mobilized more glycogen than horses on the control diet. Blood lactate tended to be higher for horses on the high fat diet than for the control diet, and most likely resulted from increased glycogenolysis and glycolysis. No significant differences were found in blood glucose, which seemed to indicate that the blood glucose homeostasis was not overloaded. Sprint times tended to be lower for the horses on the high fat diet. It was concluded that the addition of fat improved performance at an exercise intensity above the anaerobic threshold. Also, feeding a moderately high fat diet can increase storage of muscle glycogen and the amount of muscle glycogen utilized during an anaerobic workout. This conflicts with decreased resting

levels of muscle glycogen found by Greiwe et al., (1989). Also, similar effects of dietary fat on training and intense exercise have not been reported for other species.

Training Programs. Training is the physical preparation for a specific activity. It reduces chance of injury, increases fitness of a horse and helps to delay the onset of fatigue. An animal adapts to training by increasing its capacity for energy metabolism, neuromuscular function, or psychological condition, depending on the demands placed on these systems. However, the most basic component of all performance and the limiting factor in equine endurance is the capacity to supply and utilize energy (McMiken, 1983).

The use of treadmills has allowed evaluation of performance in a climate controlled environment. Treadmills allow more precise control of speed, incline, time and heart rate, so that these variables can be kept constant for each horse. However, treadmills don't take into account wind resistance, ventilation problems at higher speeds, thermoregulation by convection and the difference in diaphragm loading that results from treadmill acceleration and deceleration (Hillidge, 1987).

A training program is designed to increase the physiological ability of a horse to accomplish a specific task. This includes psychological and neuromuscular aspects

in addition to the utilization of energy. A lot of emphasis has been placed on energy utilization and it has been assumed that improvement of the energy producing systems also results in improvement of psychological and neuromuscular capabilities (Bayly, 1985). Over the past century much improvement has been made in human performance as a result of refinement in training methods (Fox, Bowers, Foss, 1989). Traditional methods involve continuous or interval exercise and differ depending on intensity, duration and frequency. The contribution of these three variables result in the "training effect" and performance by the horse (Bayly, 1985).

Continuous training involves an increase in fitness from an increase in duration and intensity. This type of training is submaximum by nature and varies from high intensity, continuous activity of moderate duration to low intensity activity of an extended duration. A continuous training program calls on the aerobic energy system for its primary energy source and therefore improves maximum oxygen consumption or endurance capacity. Advantages of continuous exercise include: an effective training method without uncomfortable levels of work and a near work intensity of actual competition levels (Bayly, 1985; Fox, Bowers, Foss, 1989; McArdle, 1986; Snow and Vogel, 1987; Wilmore and Costill, 1988).

Another approach to training is interval training. This method allows work that would normally cause exhaustion after a short time period if performed continuously to be done intermittently with work and rest intervals. In interval training, a short to moderate exercise period is alternated with a short to moderate period of rest or reduced activity. Specific energy producing systems can be emphasized and improved by altering the duration of work and rest intervals, the intensity , length and type of relief interval and the number of work intervals. The principal behind interval training is to allow athletes to perform a greater amount of work by breaking the work up into short periods of intense work alternated with periods of rest or reduced activity (Bayly, 1985; Fox et al., 1989; McArdle, 1986; Snow and Vogel, 1987; Wilmore and Costill, 1988).

Costill (1986) has defined three types of interval training: aerobic, aerobicanaerobic, and anaerobic. Aerobic interval training includes repeated short workouts alternated with short rest intervals. The workouts fall just below competition pace. Oxygen uptake falls around 65 to 75% of VO_2 max and heart rate is 70 to 85% of maximum heart rate. This type of training improves aerobic capacity. Aerobicanaerobic interval training involves working at competition pace at 80 to 95% of VO_2 max and at 85 to 100% of maximum heart rate. This entails shorter work

intervals and is aimed at improving speed and race pace. Anaerobic interval training involves even shorter work intervals than aerobicanaerobic training, but at an intensity higher than that at competition. The oxygen uptake falls around 80 to 95% of VO_2 max and a heart rate of 85 to 100% of maximum heart rate. Anaerobic training is aimed at strength, muscle buffering capacity and lactate clearance from the muscles (Wilmore and Costill, 1988; Costill, 1986).

Interval training allows the aerobic system to replenish the muscular stores of ATP and PC, which were depleted during the previous anaerobic work intervals, and the oxygen myoglobin stores. This averts accumulation of lactic acid and delays fatigue, allowing overall more work. Interval training allows use of ATP and CPr stores over and over again during the workout. It improves the aerobic energy system by stressing the oxygen transport system with longer work intervals, many repetitions and brief relief intervals. Also, anaerobic glycolysis can be improved by regulating the duration and type of relief interval (Fox, Bowers, Foss, 1989).

Bayly (1985) warns that interval training, if not carefully done, has some disadvantages. 1) Training adaptations of the skeleton and supporting structures takes more time than the metabolic improvements. This can result

in musculoskeletal injury if horses are brought along too quickly. 2) The horses, particularly the younger ones, can develop psychological problems. 3) Heat stroke can result from problems with thermoregulation and electrolyte imbalances. This is due to increased fluid loss and the increased amount of heat generated by the muscles.

Mature standardbred horses were trained for 12 weeks in two consecutive years (Gabel et al., 1983). Each year, four horses were trained by the conventional method and four horses were trained by the interval training method. No significant differences were found in lactate, heart rate, cardiac output, rectal temperature or time between horses trained by the conventional method and horses trained by an interval technique. However, horses trained by the interval schedules travelled the same distance as those trained by the continuous method. Also, horses in the interval training regime only went 10 % faster than continuously trained horses. This did not take advantage of the major benefit from the interval technique that allows more fast work per workout than the continuous method.

Objectives

The project was designed to assess the influence of added dietary fat and interval training on metabolic responses of mature Arabian horses to an incremental stress test. More specifically, we wanted to determine blood lactate and glucose thresholds and to see if these were affected by interval training or a high (10%) fat diet. An additional aim was to establish interrelationships of lactate and glucose responses to carbohydrate and fat metabolism.

Materials and Methods

Horses. Eight mature Arabian horses, consisting of 5 fillies and 3 geldings between 36 and 48 months of age were adapted to a mixed ground hay and concentrate diet. The horses were kept in stalls from 4 PM to 10 AM and turned out in a dry lot for 6 h during the day throughout the experiment. They were accustomed to exercise on a high speed treadmill prior to starting the experiment. The horses were weighed weekly. After three weeks of conditioning, all horses were dewormed with ivermectin[1]. All horses had their feet trimmed initially and after 6 and 12 wks of conditioning.

Horses were fed the basal (control) diet for 3 wk before the first standard exercise test (SET). They were paired according to endurance ability in the first SET. Then each horse in a pair was randomly assigned to the control or the fat diet.

Diets. Total mixed diets were used. The ration was adjusted weekly so that the horses were fed 2% of their body weight. Horses were fed individually, twice a day.

The control diet consisted of ground hay, cracked corn, molasses, and limestone (Table 2). In the fat diet, 10 % corn oil was substituted for cracked corn. Corn oil was

chosen as the fat source because it was highly palatable, practical, and easy to add into a completely ground ration. Diets were balanced in accordance with the NRC (1989) requirements for moderate work in young horses, however diets were not isocaloric. Analysis of diets is shown in Tables 2 and 3. The horses were given a five day adaptation period to the fat diet during which the fat was introduced gradually. The horses had access to a free choice mineral supplement[1]. The diets were sampled monthly, and samples were analyzed for dry matter, crude protein, ether extract, ADF and ADL throughout the trial.

A digestibility trial was conducted 10 d before the last SET. A 10 % chromic oxide pellet was added to the diet at each feeding to produce a 0.5 % chromic oxide level. During the last 4 days, fecal samples were collected every 12 h and frozen for analysis. Feed samples were also collected and frozen for analysis.

Conditioning. Horses were exercised 4 days per week (4 days work alternated with 3 days rest) on a high speed equine treadmill[2] in a climate controlled barn at a 6% slope. Temperature and humidity ranged from 55-60 F and 30-50%, respectively. Heart rates were monitered and rectal

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[1]Mustang 2200, Kagra Ag, 5615 Fahrwangen, Suisse
[2]Equi-Choice, Wilson Enterprises Inc., Disputanta, VA 23842

TABLE 2. Composition of Experimental Diets[a]

INGREDIENT (%)	Control	Fat
Orchardgrass hay	48.0	48.0
Cracked corn	43.0	30.0
Molasses	8.0	8.
Limestone	0.5	0.5
Corn oil[b]	-	10.0
44 % Soybean meal	-	3.0
Vitamin/mineral premix	0.5	0.5
	100	100

[a]As fed basis
[b]donated by Corn Products Corp., Best Foods Unit, Union, NJ

TABLE 3. Chemical Composition of Experimental Diets

Ingredient[a]	Control	Fat
Dry Matter (%)	89.3	90.1
Crude Protein[a]	10.0	9.3
Ether Extract[a]	2.3	15.7
Acid detergent fiber[a]	31.8	34.0
Energy Mcal/kg diet[b]	2.3	2.2

[a]Dry matter basis
[b]calculated

TABLE 4. Lameness Scores

Score	Definition	Exercise[a]
0	No visible signs of lameness	Yes
1	Discomfort, slightly uneven gait	Yes
2	Mild lameness, inconsistent signs	Yes
3	Moderate lameness, consistent signs	No
4	Moderate to severe lameness	No
5	Severe lameness	No

[a]Horses with scores of 2 or lower were exercised, and scores of 3 and up were not exercised.

TABLE 5. Interval Training Protocol Between SET 1 and 2

Gait	Time (min)	Speed (m/s)	Cumulative time (min)
Walk	5	1.6	5[a]
Trot	4	3.2	9
Canter	2	7.0	11
Trot	4	4.8	15
Gallop	2	8.0	17
Trot	4	4.8	21
Walk	3	1.6	24

[a]At 2 1/2 min the slope was increased to 6%.

TABLE 6. Interval Training Protocol between 2nd and 3rd SET

Gait	Total min of gait	Speed (m/s)	Cumulative time (min)
Walk	5	1.6	5
Trot	4	3.2	9
Canter	2	7.0	11
Trot	4	4.8	15
Gallop	2	8.0	17
Trot	4	4.8	21
Gallop	2	9.0	23
Trot	4	4.8	27
Walk	3	1.6	29

temperatures were taken before and after exercise. Horses were assigned lameness scores from 0 to 5 at each gait (Table 4).

The horses were exercised in a different order every day to alternate running time. All horses underwent a total of 17 d conditioning between the 1st and 2nd SET, and a total of 16 d conditioning between the 2nd and 3rd SET. The interval training protocol between the first two SET's is shown in Table 5. An extra sprint was included in the protocol between the 2nd and 3rd SET (Table 6). Speeds chosen for gallops to be about 70, 80, and 90% of average maximum speed (approximately 10 m/s on 6% incline).

Standard Exercise Test. An incremental standard exercise test (SET) was performed at 0, 6, and 11 weeks of conditioning. Before each SET horses were fasted 12 h.

Before beginning each SET, a jugular Abbocath-T catheter was inserted. The horses were placed back in stalls and left undisturbed for 1 h. Then, a digital heart rate monitor (Kagra Ag, 5615 Fahrwangen, Suisse) was placed over the heart with a surcingle. A resting heart rate, rectal temperature, and blood sample were obtained. A muscle biopsy was taken from the middle gluteals by means of a Bergstrom needle, and immediately frozen in liquid nitrogen. Then the horse began its SET. During the SET,

TABLE 7. STANDARD EXERCISE TEST PROTOCOL

Speed (m/s)		Cumulative Time (min)
Warm-up:	1.3	3[a]
	1.3	6[b]
Step test:	1.6	9
	2.0	12
	2.5	15
	3.0	18
	3.5	21
	4.0	24
	4.5	27
	5.0	30
	5.5	33
	6.0	36
	6.5	39
	7.0	42
	7.5	45
	8.0	48
	8.5	51
	9.0	54
	9.5	57
	10.0	60

[a]Slope 0'

[b]Slope 6%

TABLE 8. DEFINITION OF FATIGUE

Heavier pounding of front feet
Spreading of hind feet
Uneven gait
Gradual drift rearwards
Occasional stumbling
Anxious expression

the horse walked at 1.3 m/s, for 3 min on the flat then another 3 min at 6% slope. The speed was increased 0.5 m/s every 3 min in small steps as shown in Table 7. Horses were worked until they showed signs of fatigue (Table 8).

Blood samples were taken every 3 min just prior to each speed increment and a second muscle biopsy was taken after exercise. Blood samples were centrifuged, frozen and analyzed for lactate, glucose, cholesterol, triglycerides and total serum protein using an automatic analyzer.[4-8] The muscle biopsy sample was deep frozen in liquid nitrogen and analyzed for glycogen using the procedure of Lo et al. (1970). During this procedure, the glycogen was broken down to glucose in the presence of sulfuric acid.

Digestion Trial. Feed and fecal samples (pooled over days by animal), were dried in a 100 C forced air oven. They were ground through a Cyclone and then a Wiley mill using a 1 mm screen. Feed and feces were analyzed for DM, ether extract, ADF, and lignin, (A.O.A.C., 1980). Crude protein was determined using a Kjehdal Automatic Analyzer. Chromic oxide levels were determined spectrophotometrically

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[4]Proc. 826-UV, Sigma Diagnostics, St. Louis, MO 63178
[5]Proc. 16-UV, Sigma Diagnostics, St. Louis, MO 63178
[6]Proc. 352, Sigma Diagnostics, St. Louis, MO 63178
[7]Proc. 336, Sigma Diagnostics, St. Louis, MO 63178
[8]Proc. 541, sigma Diagnostics, St. Louis, MO 63178

after digestion with acid (A.O.A.C., 1980). Apparent nutrient digestibilities were then calculated.

One horse had to be eliminated from the study after two wks of conditioning due to laryngeal hemiplagia.

Statistical Analysis. Data were analyzed using SAS GLM Procedure for split plot analysis of variance with repeated measures (SAS, 1985). A SAS multi-variate analysis (MANOVA) procedure was used to test for correlation between responses on the same horse. Lactate and glucose data were plotted against speed and fitted to a broken line model using the SAS PROC NLIN program (Robbins, 1986). Resulting parameters for lactate and glucose thresholds were tested for significance by SAS ANOVA. The null hypothesis was tested and probabilities (P) that it is valid have been given to show tendencies ($P<0.15$) even when the normal convention of significance ($P<.05$) has not been met. With any three and four horses in each group, statistical inferences will be more than normally at the discretion of the reader.

Apparent digestibilies were compared using a paired t-test.

The broken line model fits data to two straight lines with different slopes, one of which may be zero (Robbins, 1986). The intersection of these two lines is the breakpoint. The broken line model was developed initially for the determination of nutrient requirements (Robbins and

Baker, 1980). We adapted it to determine lactate thresholds. Previously, visual inspection has been used to estimate the lactate threshold in horses. The broken line model provided a statistically valid method for establishing precisely the lactate threshold in this study. In order to use it, the steps in our SET were made small to yield 7 to 18 points.

Results and Discussion

Apparent Digestibility. Apparent digestibility for DM, CP, and ADF were not significantly different for control and fat fed horses (Table 9). However, apparent digestibility for lignin tended to be slightly higher in horses fed fat ($p = .10$). Ether extract was higher in horses fed added fat than in controls ($p = .000068$). This is in agreement with previous studies where the addition of fat in the form of corn oil, peanut oil, blended fat or tallow increased the apparent digestibility of ether extract (Rich et al.,1982; Kane and Baker, 1977).

Lactate Threshold. Most R^2 values describing the fit were not much different when the initial slope was zero, .93 to .99, than when it was allowed to vary from zero, using the two slope model, .93 to .99 (Table 10).

Lactate threshold ranged from 3.32 to 6.53 m/s (7.5 - 14.7 mph), an extended trot or canter. The overall mean and SE of the lactate y intercepts was 4.89 $\pm$ .36 mg/dl, and the range was 2.39 to 9.35 mg/dl. The lactate y intercept in the Broken Line Model corresponds to the onset of blood lactate accumulation (OBLA). OBLA is the concentration of plasma lactate at the lactate threshold. Our values may be compared to an OBLA of 4 mmole/L used previously for horses by other workers (Thornton, 1985; Persson, 1983; Wilson et

TABLE 9. Apparent digestibilities of diet components.

Component (%)	Fat diet[a]	Control diet[b]
Dry matter	56.5	59.9
Crude protein	54.4	56.1
Acid detergent fiber	47.1	49.3
Permanganate lignin[c]	68.1	59.0
Ether extract[d]	85.8	50.63

[a]Mean of three horses
[b]Mean of four horses
[c]Diet effect (P<.11)
[d]Diet effect (P<.000068)

Table 10. Lactate thresholds determined from plasma lactate concentrations plotted against speed, using the broken line model.

SET		Intercept Lactate (mg/dl)	Intercept Speed (m/s)	Slopes First	Slopes Second	Points (speeds)	R-square
1	Control						
	1	8.81	4.67	0.00	37.2	14	.96
		21.25	5.17	-4.44	41.9		
	2	4.95	4.06	0.00	22.0	13	.99
		4.88	4.06	0.03	22.0		
	3	2.39	3.93	0.00	15.1	15	.96
		2.95	4.00	-0.18	15.3		
	4	5.09	3.32	0.00	10.1	13	.98
		4.00	3.21	0.77	10.1		
	5	3.46	4.47	0.00	23.8	16	.99
		4.80	4.53	-0.67	23.8		
	6	3.50	4.21	0.00	28.2	16	.99
		7.78	4.45	-1.69	29.2		
	7	4.95	4.06	0.00	22.0	13	.99
		4.89	4.06	0.03	22.0		
Mean (1 slope)		4.74	4.10		22.63		
SEM		.78	.16		3.31		
2	Fat						
	1	4.26	4.60	0.00	24.7	16	.99
		7.81	4.74	-1.85	24.7		
	2	5.29	4.86	0.00	21.5	15	.99
		5.22	4.85	0.03	21.5		
	3	4.15	5.15	0.00	26.4	15	.96
		7.11	5.28	-1.29	26.4		
LS Mean		4.57	4.87		24.2		
SEM		.334	.27		3.39		
	Control						
	4	4.49	3.88	0.00	21.8	14	.98
		3.45	3.83	0.58	21.8		
	5	4.92	5.18	0.00	31.4	15	.98
		7.75	5.27	-1.12	31.4		
	6	4.46	4.84	0.00	39.0	15	.99
		7.62	4.92	-1.31	39.0		
	7	3.62	4.77	0.00	34.6	15	.99
		4.85	4.81	-0.54	34.6		

Table 10. Continued.

SET		Intercept Lactate (mg/dl)	Intercept Speed (m/s)	Slopes First	Slopes Second	Points (speeds)	R-square
2	Mean	4.37	4.67	0.00	31.7		
	SEM	.289	.232		2.93		
3	Fat						
	1	9.35	6.53	0.00	40.86	18	.95
		20.46	6.80	-3.37	40.86		
	2	6.02	5.71	0.00	26.7	17	.93
		9.83	5.86	-1.33	26.7		
	3	4.54	6.12	0.00	39.2	17	.97
		9.10	6.23	-1.53	39.18		
	LS Mean	6.64	6.12	0.00	35.6		
	SEM	.990	.275		3.35		
	Control						
	4	5.15	6.01	0.00	17.85	18	.97
		11.65	6.37	-2.09	17.85		
	5	4.14	5.75	0.00	27.08	18	.97
		6.19	5.82	-0.73	27.08		
	6	2.93	4.81	0.00	19.93	16	.99
		4.15	4.87	-0.51	19.93		
	7	4.43	5.51	0.00	22.86	17	.98
		5.41	5.56	-0.35	22.86		
	LS Mean	4.16	5.52		21.93		
	SEM	.857	.238		2.90		
	Fat and Control						
	Mean	5.22	5.78		27.78		
	SEM	.774	.204		3.41		
2,3	Fat						
	Mean	5.61	5.50	0.00	29.89		
	SEM	.803	.307		3.30		
	Control						
	Mean	4.27	5.09		26.81		
	SEM	.251	.238		2.67		
	Fat and Control						
	Mean	4.84	5.27		28.13		
	SEM	.399	.189		2.04		

Table 10. Continued.

SET	Intercept Lactate (mg/dl)	Intercept Speed (m/s)	Slopes First	Slopes Second	Points (speeds)	R-square
1,2,3 All						
Mean	4.89	4.90	0	26.29		
SEM	.360	.182	0	1.79		

al., 1982). This OBLA was established in human athletes (Kindermann et al., 1979). It has been used arbitrarily in horses because no threshold had been determined. We attributed this lack to the small number of points used by other researchers which did not support rigorous curve analysis. The present study shows that 13 to 18 points (or speeds) were sufficient to determine a lactate threshold in the horse using a broken line model (Table 10).

The speed intercepts (mean $\pm$ SE) were $4.10 \pm .16$, $4.67 \pm .23$, and $5.78 \pm .20$ in SETS 1,2, and 3, respectively. The speed, x intercept, corresponds to the common meaning of the lactate threshold, ie, the intersection of each at which plasma lactate concentration begins to rise sharply. These data show an improvement in lactate threshold with conditioning from SET 2 to SET 3 ($P= 0.15$) but no improvement from SET 1 to SET 2 ($P = 0.59$).

Conditioning increased the lactate threshold from 4.67 ± 0.22 m/s in SET-II to 5.78 ± 0.22 m/s in SET-III ($P < 0.05$). The lactate threshold was increased by diet in SET 3 (Figure 2) and SETs 2 and 3 combined (Figure 3), but not SET 2 alone (Figure 4). These results show that a more complete metabolic adaptation to a high fat diet was achieved in 11 wk but not 6 wk, because diet effects were significant in SET 3 but not SET 2. Previously, a period of 12 wk was sufficient to allow horses fed a high fat diet to spare

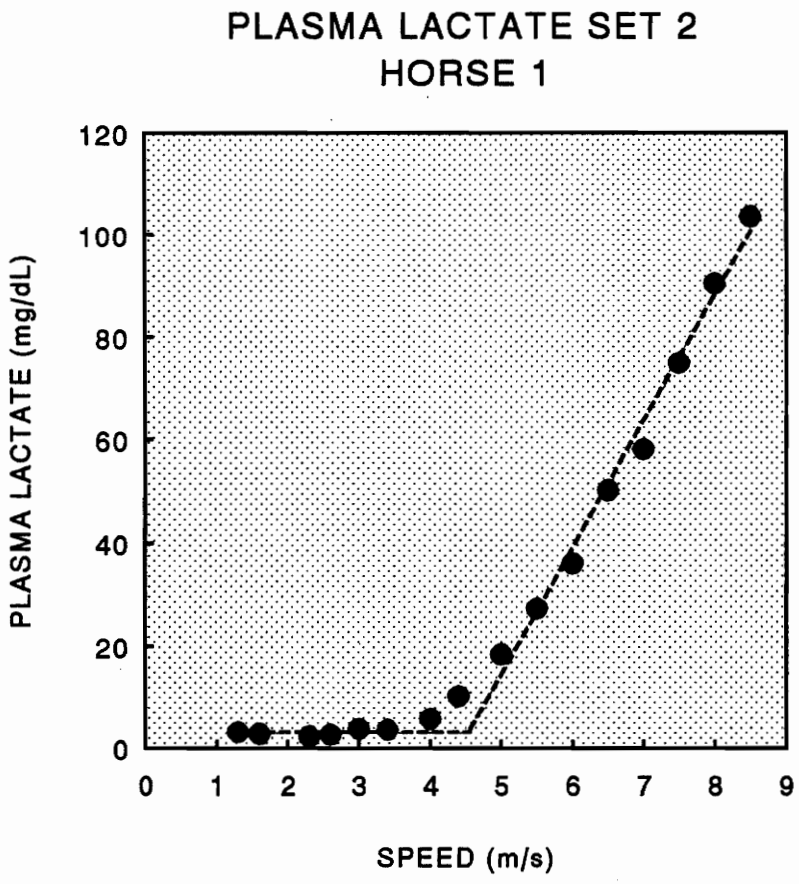

Figure 1. An example of fitting the lactate data (R2 = 0.9932) on one horse in one SET to the broken line model to determine a lactate threshold (y=4.25, x=4.60).

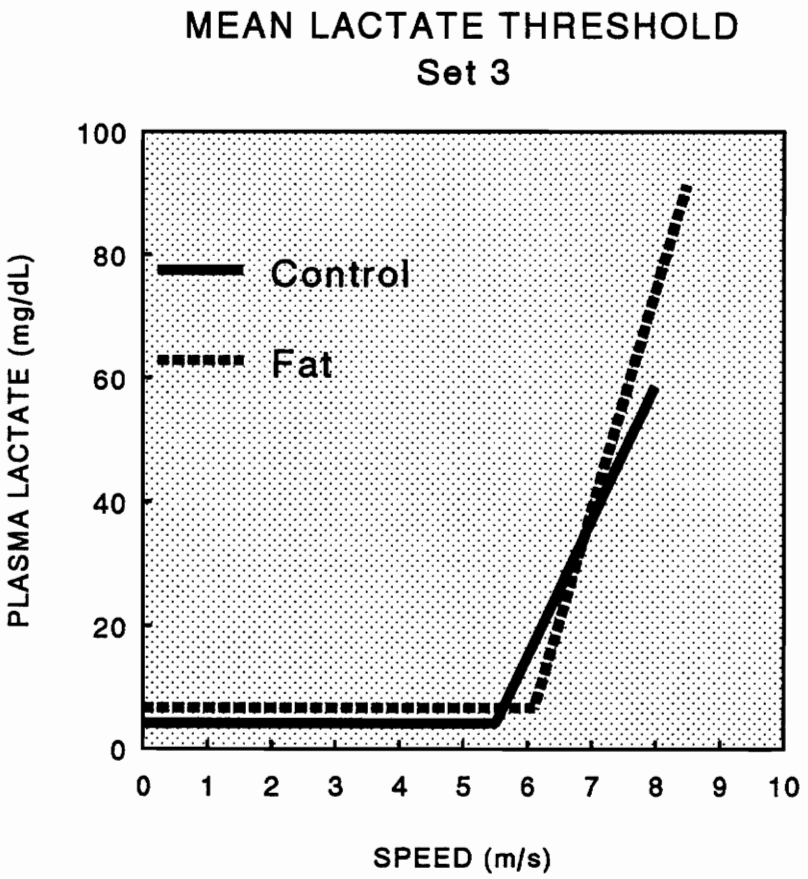

Figure 2. Mean lactate threshold occured at a faster speed in horses fed in SET 3 and determined lactate threshold (y=4.16, x=5.52 for control, and y=6.64, x=6.12 for fat diet).

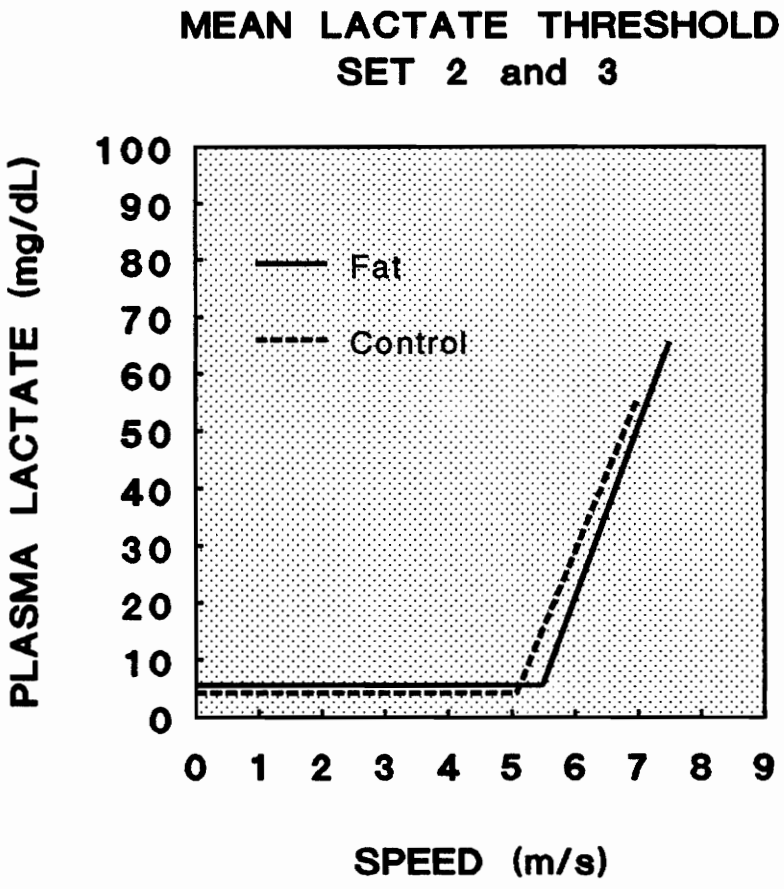

Figure 3. Mean lactate threshold occurred at a faster speed in horses fed fat in SET 2 and 3.

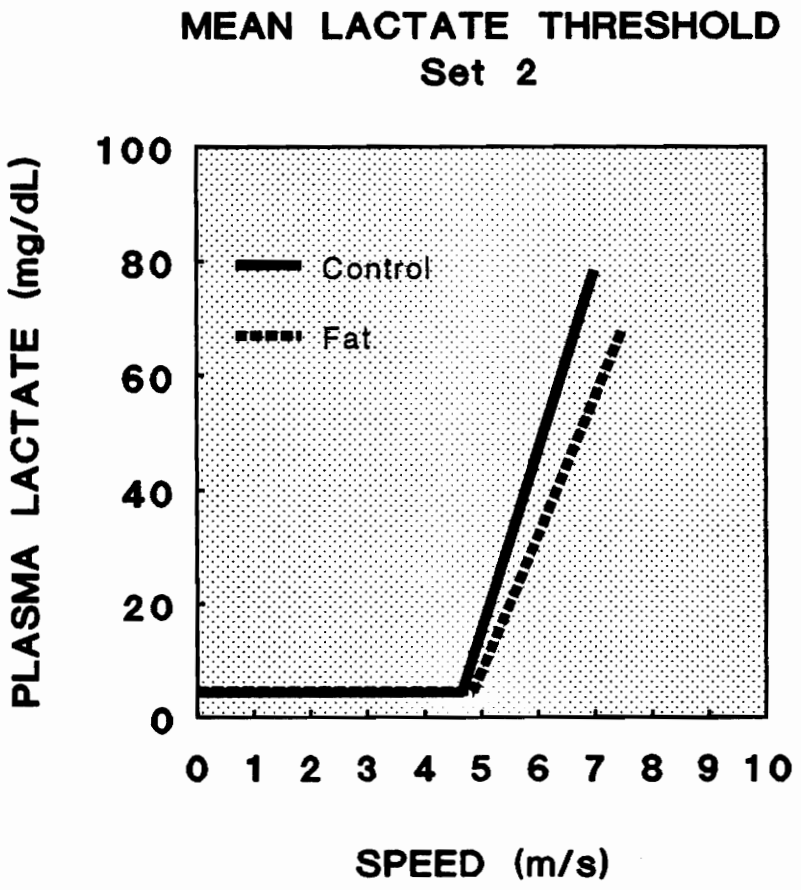

Figure 4. Mean lactate threshold was not significantly different between diets in SET 2 alone.

muscle glycogen during aerobic exercise (Greiwe et al., 1989).

There were three effects of the fat diet on the lactate curve (Figure 1). The high fat diet tended to increase the lactate threshold (x intercept) during SET-III ($P = 0.16$) (Figure 2) and SETs-II-and-III combined ($P = 0.08$) (Figure 3). The effect of this increase in lactate threshold would be to delay fatigue to a higher speed.

Second, the high fat diet tended to increase the lactate (y) intercept (ie, the plasma lactate level up to the lactate threshold) during SET-III ($p = 0.11$) and SETs-II-and-III ($p = 0.10$) (Figure 2 and 3). Similarly, the third effect was an increase in slope after the point of inflection in SET-III ($P = 0.03$). Both the second and third effect indicate that fat adaptation increased the tendency for lactate to accumulate in the blood at all speeds except close to the lactate threshold.

The higher plasma lactate before the threshold, and the steeper slope of plasma lactate after the threshold, are consistent with an increase in lactate production from glycogen breakdown. One possible explanation assumes that the total power output is constant at a given speed. It has been shown that fatty acid oxidation, with its low power output, is increased, while glucose oxidation, with its higher power output, is decreased, by conditioning on a high

fat diet (Pagan, et al., 1987). In this situation, an increase in glycogen breakdown to lactic acid, which has about twice the power output of glucose oxidation, would compensate for the decreased glucose oxidation.

The lactate threshold and heart rate were correlated (R^2= 0.39, $p<0.0023$). In humans, a correlation between heart rate and lactate threshold was 0.60 (Dwyer and Bybee, 1983).

Glucose Threshold. Glucose thresholds were determined initially for each horse in each SET. The broken line model fit the data well (Figure 5). Most R^2 values were slightly better when the initial slope was zero, .52 to .99 , than when it was allowed to vary from zero, .49 to .99 (Table 7).

The overall mean $\pm$ SE across diets of the glucose intercepts were 88.91 $\pm$ 1.57 mg/dl, and the range was 77.37 to 102.59 mg/dl. The broken line model sufficiently determined a glucose threshold from 7 to 18 points (Table 10).

The speed intercepts (mean + SE) were 4.16 $\pm$ .32, 4.08 $\pm$ .28, and 4.51 $\pm$ .24 in SETs 1, 2, and 3, respectively (Table 8).

Horses on the high fat diet tended to have higher glucose y intercept (the concentration up to the point of glucose inflection) during SET 2 and 3 combined (P = .082) and during SET-III (P = .055) (Figure 6 and 7). The high

Table 11. Glucose thresholds were determined from plasma glucose concentrations plotted against speed, using the broken line model.

SET		Intercept Glucose (mg/dl)	Intercept Speed (m/s)	Slopes First	Slopes Second	Points (speeds)	R-square
1	Control						
	1	86.6	2.94	0.00	14.4	8	.93
		82.7	2.67	3.04	14.4		
	2	97.8	3.67	0.00	18.52	13	.98
		96.82	3.62	0.63	18.52		
	3	80.74	5.23	0.00	13.46	15	.49
		89.43	6.39	-2.31	22.67		
	4	88.94	3.66	0.00	16.82	12	.95
		116.20	6.03	-6.49	27.69		
	5	81.75	4.05	0.00	20.32	16	.97
		86.59	4.51	-1.70	22.12		
	6	83.8	5.15	0.00	27.5	8	.95
		90.27	5.39	-1.83	27.50		
	7	85.77	4.44	0.00	14.98	7	.94
		88.88	4.65	-1.28	14.98		
	MEAN[a]	86.49	4.16		18.0		
	SEM	2.17	.32		1.83		
2	Fat						
	1	100.10	4.10	0.00	22.81	16	.93
		111.59	4.75	-5.17	24.07		
	2	100.18	4.07	0.00	24.13	15	.99
		105.06	4.43	-1.85	25.86		
	3	85.55	4.13	0.00	12.09	15	.99
	LS Mean[a]	95.28	4.10		19.68		
	SEM	3.15	.468		3.67		
	Control						
	4	90.29	3.00	0.00	14.07	13	.76
		79.18	2.26	13.07	14.34		
	5	87.94	4.44	0.00	21.35	15	.94
		87.05	4.35	0.37	20.95		
	6	89.36	5.35	0.00	27.96	15	.91
		98.46	5.82	-3.01	31.17		
	7	86.86	3.47	0.00	16.16	15	.97
		89.97	3.67	-1.90	16.16		
	LS Mean[a]	88.61	4.07		19.89		
	SEM	2.73	.405		3.18		

[a]One slope model

Table 11. Continued.

SET	Diet / Horse	Intercept Glucose (mg/dl)	Intercept Speed (m/s)	Slopes First	Slopes Second	Points (speeds)	R-square
2	Control and Fat						
	Mean[a]	91.47	4.08		19.79		
	SEM	2.31	.279		2.19		
3	Fat						
	1	102.59	3.69	0.00	21.96	18	.98
		109.89	4.10	-3.24	22.50		
	2	99.20	4.03	0.00	25.14	17	.99
		106.16	4.44	-2.79	26.26		
	3	86.73	4.73	0.00	23.59	17	.99
		98.90	5.51	-3.75	26.70		
	LS Mean[a]	96.17	4.15		23.56		
	SEM	3.94	.339		1.55		
	Control						
	4	86.38	4.69	0.00	15.85	18	.98
		91.58	5.09	-1.92	16.30		
	5	79.77	4.17	0.00	18.98	18	.99
		82.88	4.43	-1.16	19.61		
	6	89.35	4.65	0.00	20.01	16	.99
		93.81	5.01	-1.53	21.29		
	7	77.37	5.62	0.00	23.65	17	.85
		92.49	6.82	-3.40	35.35		
	LS Mean[a]	83.22	4.77		19.62		
	SEM	3.41	.294		1.34		
	Control and Fat						
	Mean[a]	88.77	4.51		21.31		
	SEM	3.52	.236		1.22		
2,3	Fat						
	Mean[a]	95.73	4.13		21.62		
	SEM	3.07	.137		1.96		
	Control						
	Mean[a]	85.92	4.43		19.76		
	SEM	1.68	.311		1.61		
1,2,3	All						
	Mean[a]	88.91	4.23		19.69		
	SEM	1.57	.158		1.03		

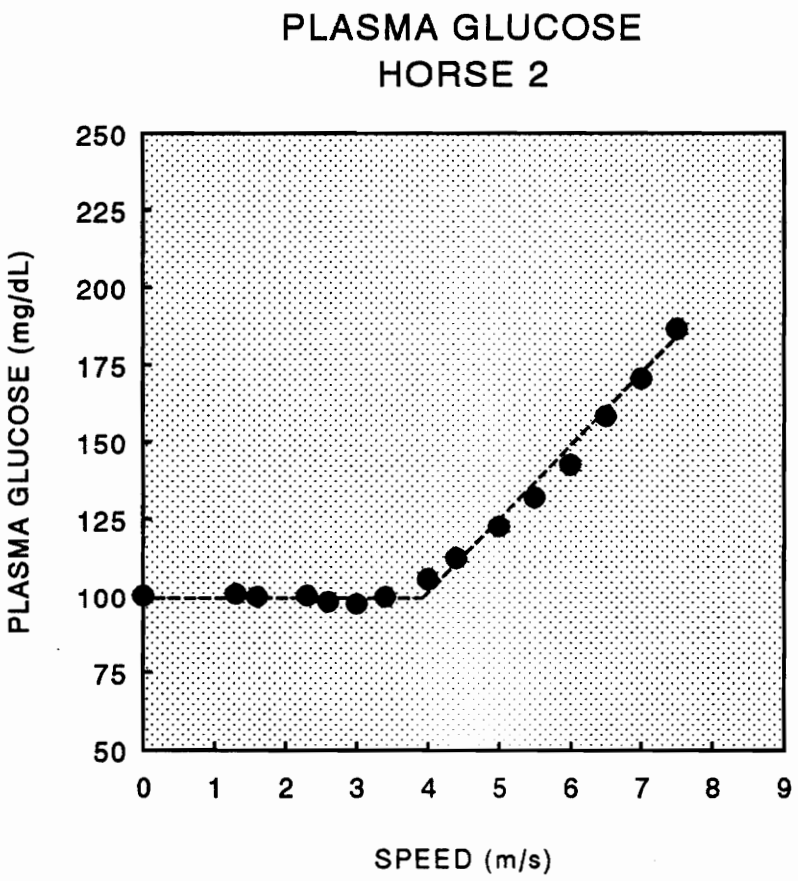

Figure 5. An example of fitting the glucose data of one horse in one SET to the broken line model (R^2 = 0.9922).

fat diet tended to increase the slope in SET-III ($P = .113$) (Figure 6).

The plasma glucose curves paralleled the lactate curves (Figure 8). Plasma glucose and lactate concentrations were correlated ($R^2 = 0.848$, $P < 0.0001$). The glucose threshold preceded the lactate threshold, usually by one or two increments of speed (Figure 8): mean values for SET-II-and-III combined were 4.42 ± 0.29 and 5.09 ± 0.20 m/s (mean $\pm$ SE) for glucose and lactate thresholds, respectively, in the control group, and 4.13 ± 0.33 and 5.50 ± 0.23 m/s, respectively, in the fat group.

The interrelationships between glucose and lactate are numerous and complex. The key here is that the increase in glucose came first. It was most likely due to increased hepatic glycogenolysis, possibly due to epinephrine. When exercise becomes intense epinephrine release might increase blood lactic acid concentration in 3 ways. It can stimulate breakdown of muscle glycogen directly, hence increase lactic acid production in the muscle. Breakdown of liver glycogen might raise blood glucose which increases rate of glucose entry into cells: this could also increase lactic acid production in muscle. Thirdly, epinephrine, by constricting blood vessels in the splanchnic region, mainly the liver may

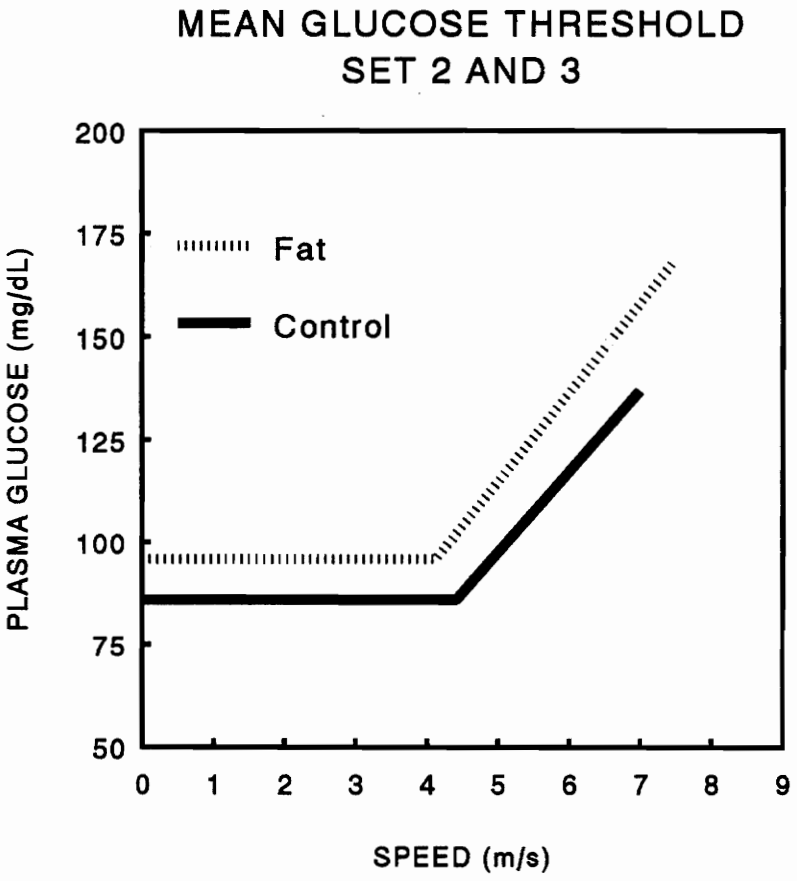

Figure 6. Horses on the high fat diet tended to have higher mean glucose y intercept during SET 2 and 3 (P = 0.113).

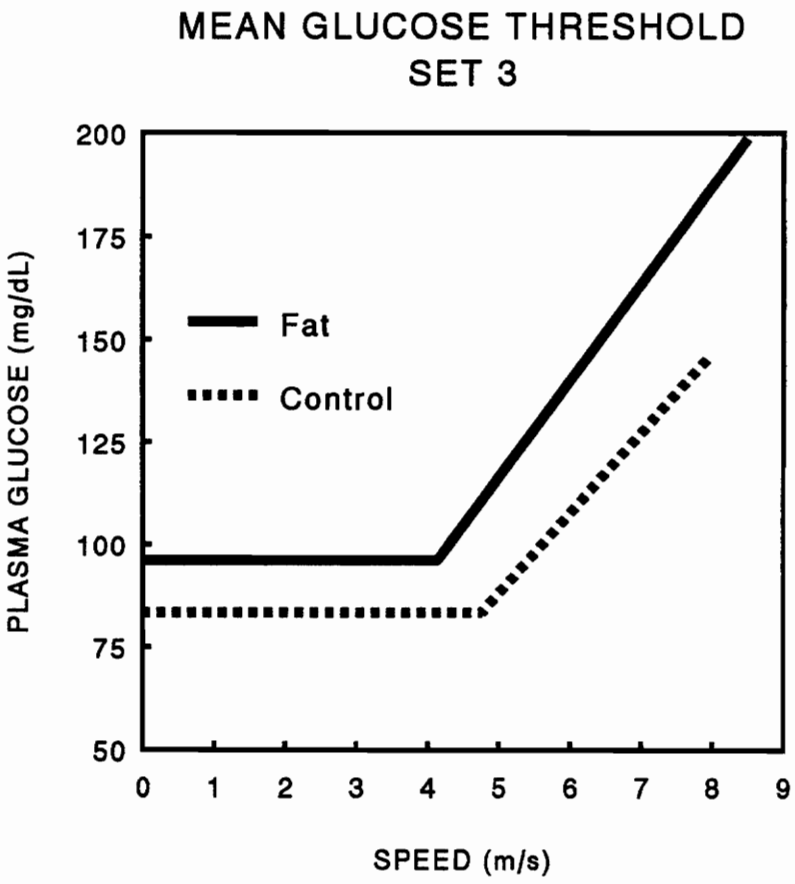

Figure 7. Horses on the high fat diet tended to have higher mean glucose y intercept during SET 3 (P = 0.055).

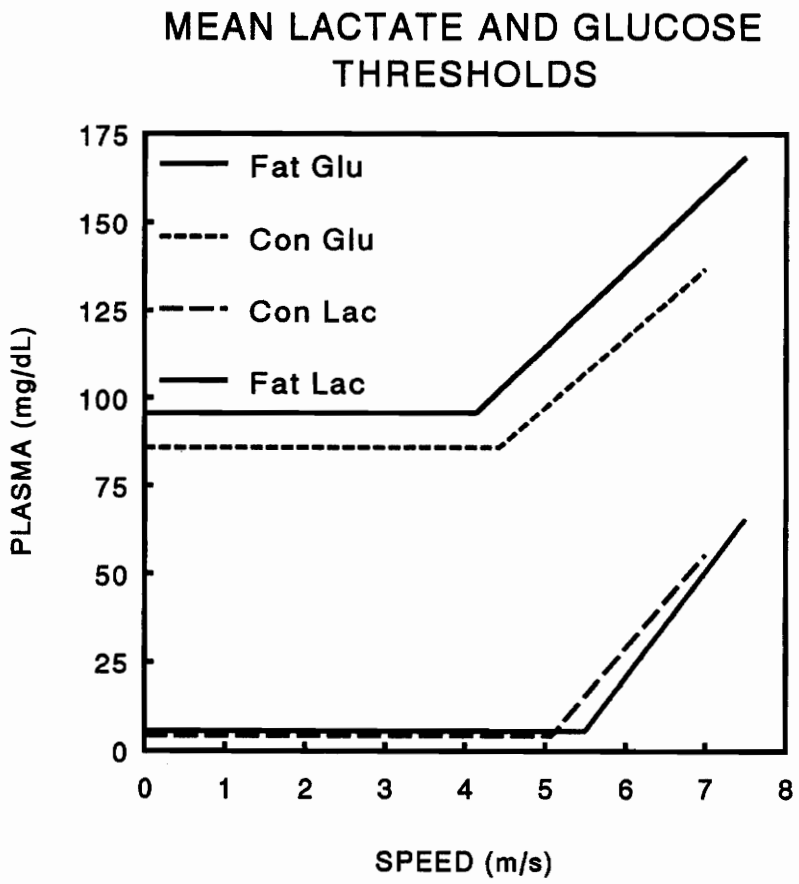

Figure 8. The mean plasma glucose curves paralleled the mean lactate curves.

also reduce the the rate of lactic acid removal from blood.

Other Blood Variables. Plasma protein was not affected by diet, conditioning, or exercise. This has also been observed in horses after trail rides at 6 miles/hour (Hintz et al, 1978) and in total serum protein with increasing exercise (Thomas and Fregin, 1981).

Plasma cholesterol concentrations tended to be higher ($p = 0.15$) in the high fat group than in controls during SET 3 (Figure 9). There was no difference in SET 2, suggesting a need for more than 6 wks for metabolic adaptation to develop fully. Cholesterol has been shown to increase after exercise (Hambleton et al., 1980; Rose et al., 1977), and with introduction of higher protein/fat diets (Kronfeld et al., 1977).

Plasma Triglycerides tended to increase ($p = .1349$) during SET 3 but not SET 2 (Figure 10). There was a strong interaction ($p < 0.0001$) between diet and speed. Triglyceride levels were lower in the fat fed horses than controls in SET 2, but higher in SET 3 (Figure 10). A linear model was fitted to the data and R values were o.90 and 0.68 for the control and fat diets, respectively in SET 2. R values in SET 3 were o.93 and 0.98 for the control and fat diets, respectively (Figure 10). A polynomial equation was fitted to both diets in SET 3, which gave R values of

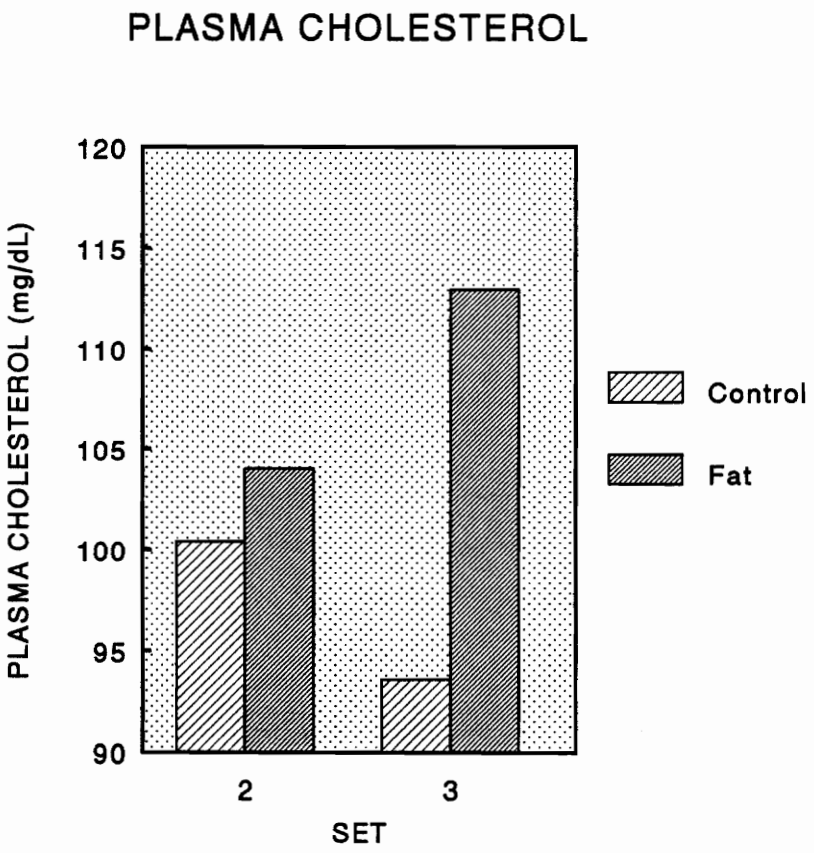

Figure 9. Mean plasma cholesterol concentration tended to be higher (P = 0.15) in the high fat group than in the controls during SET 3.

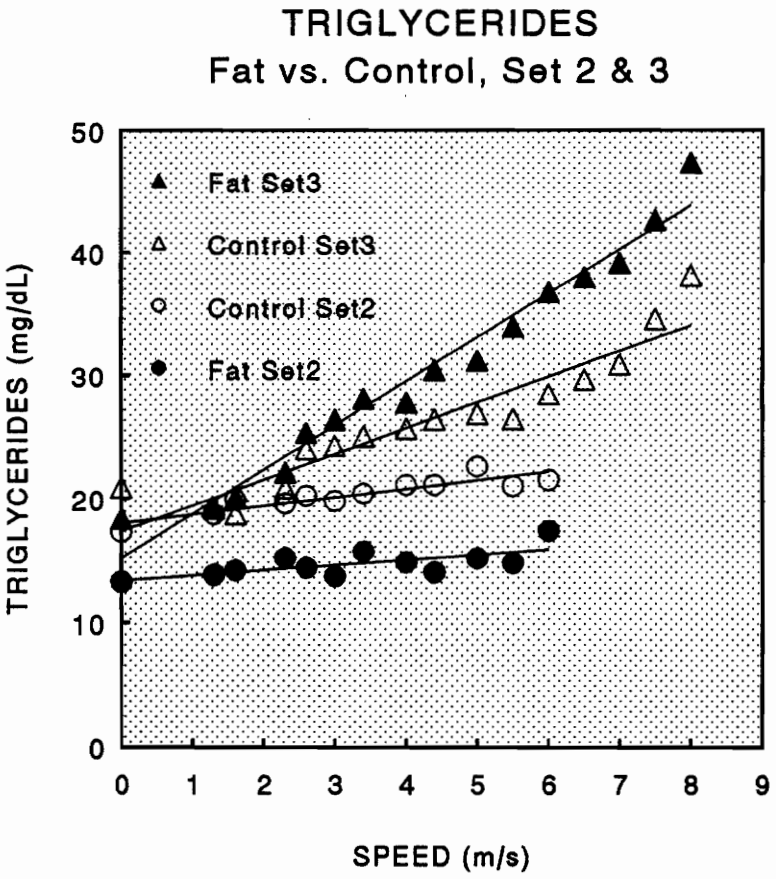

Figure 10. Mean plasma triglycerides tended to increase (P = 0.1349) during SET 3 but not SET 2.

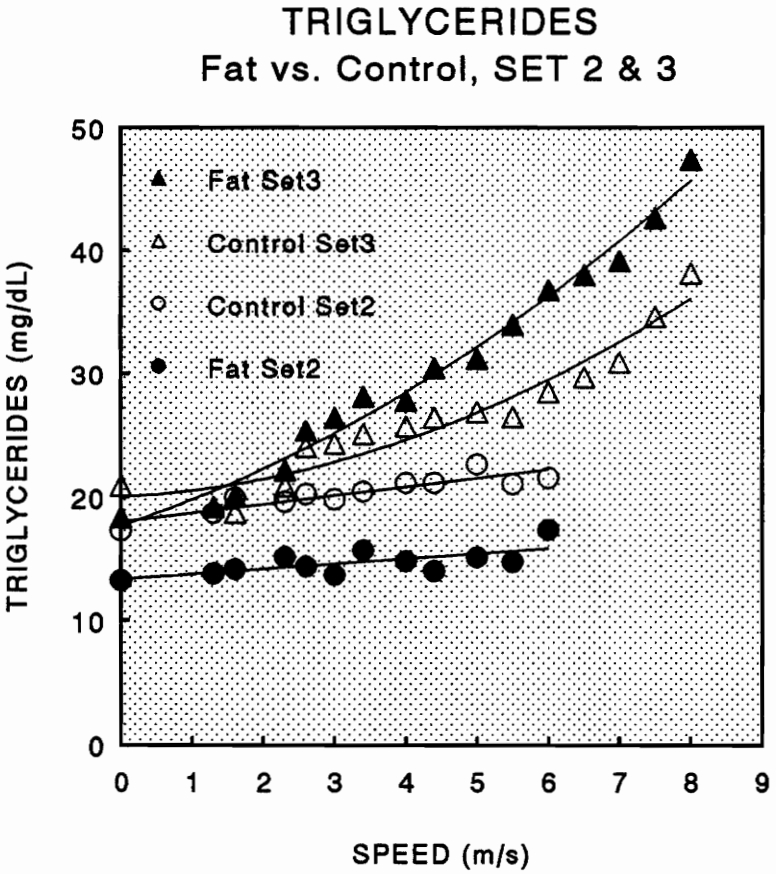

Figure 11. Mean plasma triglycerides in SET 2 were fitted to a linear equation and in SET 3 were fitted to a polynomial equation.

0.96 and 0.99 for the control and fat diets, respectively (Figure 11).

Muscle Glycogen. There were no significant dietary differences in resting concentrations of muscle glycogen or in the amount of muscle glycogen utilized during exercise (Table 12). Thus no sparing effect on muscle glycogen was observed in these SETs, which progressed from aerobic to anaerobic intensities. In contrast, muscle glycogen sparing has been observed during aerobic work in fat adapted horses (Greiwe et al., 1989). Also, muscle glycogen concentration has been shown to be higher in horses adapted to a high fat diet during aerobic exercise (Hambleton et al., 1980; Meyers et al., 1987; Scott et al., 1991). During this study there was no glycogen sparing effect observed because our exercise intensity was anaerobic as well as aerobic.

Resting muscle glycogen tended to be higher ($p<0.10$) for all horses in SET 3 compared to SET 2, which suggests that there was a conditioning effect (Figure 12).

The influence of fat adaptation on muscle glycogen is well understood in regard to prolonged aerobic work. Skeletal muscle undergoes important adaptations to regular endurance exercise which improve performance. These adaptations occur in conjunction with increased mitochondrial content and muscle fiber respiratory capacity.

Table 12. Muscle glycogen concentrations

SET	Control	Fat
1	mg/g tissue	
Resting	43.35	53.35
Post exercise	28.14	37.77
Difference	15.22	15.57
2		
Resting	35.61	39.34
Post exercise	28.33	24.48
Difference	7.28	14.86
3		
Resting	41.94	43.46
Post exercise	27.47	25.22
Difference	14.47	18.24

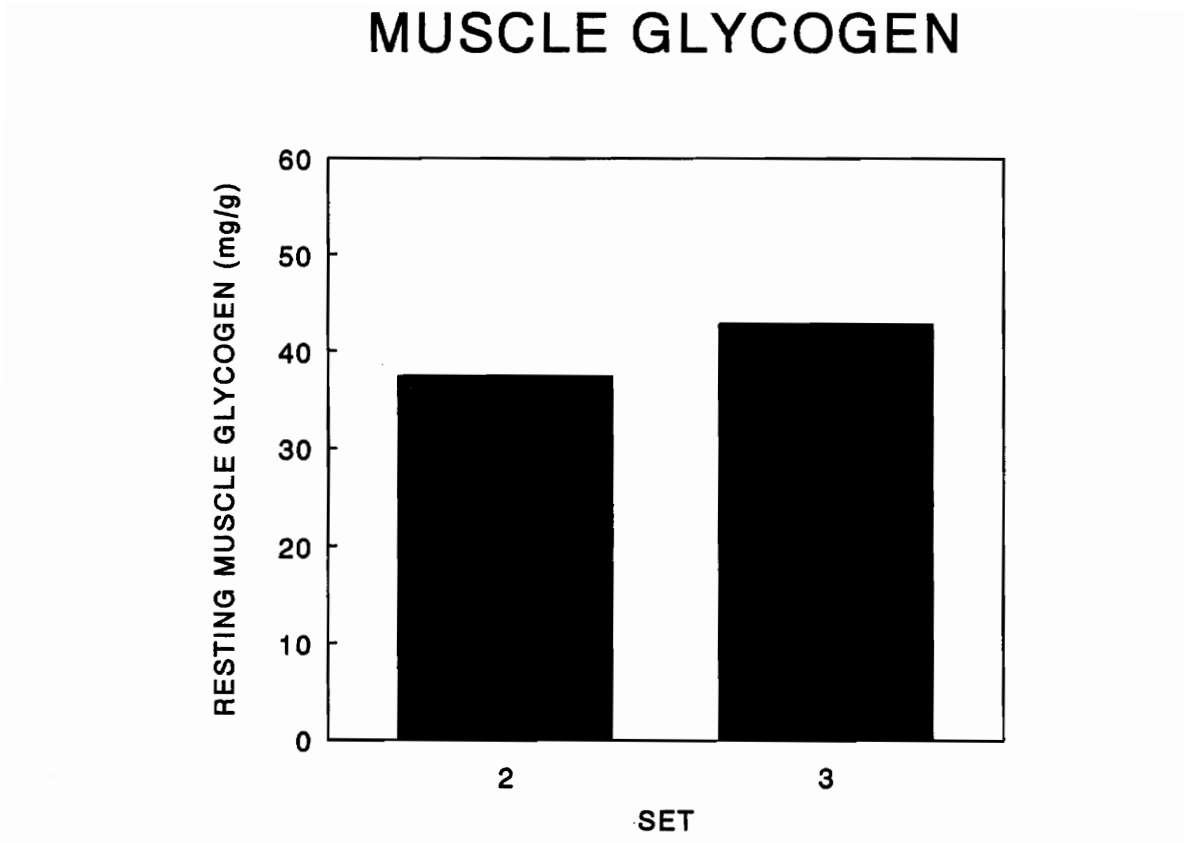

Figure 12. Muscle glycogen concentration tended to be higher (P = 0.10) for all horses in SET 3 compared to SET 2.

They include a slower utilization of muscle glycogen and blood glucose, a greater reliance on fat oxidation, and less lactate production (Holloszy and Coyle, 1984). Training decreases the rate of glycogen utilization (Hodgson et al., 1985;Holloszy et al., 1984).

The principle behind feeding horses a high fat diet is to increase the oxidation of fat and thereby spare the utilization of blood glucose and muscle glycogen. This increases the ability to perform prolonged aerobic exercise without excessive lactate production. However, this project was anaerobic and aerobic, so a sparing effect on muscle glycogen was not observed.

Conclusion

In our study, cholesterol levels were higher in the fat fed horses in SET 3 but not in SET 2, which shows an adaptation of the horses to the fat diet by SET 3 but not SET 2.

Feeding a 13% added fat diet and interval training increased tha lactate threshold of horses, ie, the speed at which plasma lactate sharply increased during an incremental (step) exercise test. Lactate thresholds are valuable in designing interval training programs and in predicting performance.

Enhanced fat oxidation in fat adapted horses may have depressed glucose oxidation, hence required an increase in glycogen conversion to lactic acid. This effect would contribute to the higher lactate (y) intercept and final slope observed in our fat adapted horses.

Parallel changes were observed in plasma glucose, except that the glucose threshold preceded the lactate threshold, usually by only one step. The increase in plasma glucose was probably in response to epinephrine, and this invites further study.

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APPENDIX 1

TABLE 1. EQUI-CHOICE VITAMIN MINERAL SUPPLEMENT
Guaranteed Analysis.

Ingredient	%
Calcium (max)	9.00
Calcium (min)	7.00
Phosphorous	8.00
Magnesium	1.00
Sulfur	1.00
Zinc	0.500
Iron	0.300
Copper	0.120
Manganese	0.100
Cobalt	0.0020
Salt (max)	36.00
Salt (min)	34.00
Sodium	13.65
Chlorine	21.35
Selenium	0.0011
Vitamin A	150,000 IU/lb
Vitamin D	25,000 IU/lb
Vitamin E	300 IU/lb

Appendix Table 2. Feed Intake by Periods

SET	Control[a]	Fat[b]
	kg	
1	8.5	8.0
2	8.4	7.9
3	7.4[c]	7.0[c]

[a]Mean of 4 animals
[b]Mean of 3 animals
[c]Means with superscripts differ significantly ($p<0.05$)

Appendix Table 3. Body Weighy by Periods

SET	Control[a]	Fat[b]
	kg	
1	425	402[c]
2	425	399[c]
3	434	413[d]

[a]Mean of 4 animals
[b]Mean of 3 animals
[cd]Means in the same column with different superscripts differ significantly (p,0.05)

Appendix Table 4. Heart rates and lactate threshold

SET	Horse	Heart Rate	X Intercept	Y Intercept
1	1	143	4.67	8.81
	2	137	4.06	4.95
	3	125	3.93	2.39
	4	128	3.32	5.09
	5	128	4.47	3.46
	6	147	4.21	3.50
	7	140	4.06	4.95
2	1	145	4.60	4.26
	2	148	4.86	5.29
	3	141	5.15	4.15
	4	130	3.88	4.49
	5	132	5.18	4.92
	6	150	4.84	4.46
	7	165	4.77	3.62
3	1	153	6.53	9.35
	2	155	5.71	6.02
	3	155	6.12	4.54
	4	154	6.01	5.15
	5	138	5.75	4.14
	6	140	4.81	2.93
	7	155	5.51	4.43

VITA

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LACTATE AND GLUCOSE RESPONSES TO EXERCISE IN THE HORSE: INFLUENCE OF INTERVAL TRAINING AND DIETARY FAT

by

Susan E. Custalow

Committee Chairman: Thomas N. Meacham
Animal Science

(ABSTRACT)

Eight mature Arabian horses were assigned randomly to two groups. The two groups were fed either a control diet or a diet similar in which 10 % corn oil was substituted for cracked corn after a baseline SET.

Horses were interval trained in a climate controlled barn 4 days per week on a high speed equine treadmill at a slope of 6 %. An SET was performed at 0, 6 and 11 wk of training. The horse walked at 1.6 m/s, then speed was increased every 3 min until the horse showed signs of fatigue.

Blood samples were taken every 3 min prior to each speed increment and analyzed for glucose, lactic acid, cholesterol, triglycerides, total protein, and muscle glycogen. Muscle biopsies were taken before and after exercise and analyzed for glycogen.

Lactate and glucose were plotted against speed and fitted to a broken line model. Resulting parameters were tested for significance by analysis of variance.

Diet had an effect in SET 3 and SETs 2 and 3 combined, but not SET 2 alone. There were 3 effects of the fat diet on the lactate curve. The high fat diet tended to increase the lactate threshold (x intercept at point of inflection) during SET 3 ($P = 0.16$) and SETs 2 and 3 combined ($P = 0.08$). Lactate threshold ranged from 3.88 to 6.53 m/s.

The high fat diet also increased the plasma lactate level prior to the lactate threshold (y intercept) during SET 3 ($P = 0.11$) and SETs 2 and 3 ($P = 0.10$). The third effect was an increase in slope after the point of inflection in SET 3 ($P = 0.03$).

Training increased the lactate threshold from 4.77 $\pm$ 0.22 m/s in SET 2 to 5.82 $\pm$ 0.22 m/s in SET 3. Plasma glucose and lactate concentrations were correlated ($R^2 = 0.848$, $P < 0.0001$). Mean values for SET 2 and 3 combined were 4.42 $\pm$ 0.29 and 5.09 $\pm$ 0.20 m/s for glucose and lactate thresholds, respectively, in the control group, and 4.13 $\pm$ 0.33 and 5.50 $\pm$ 0.23 m/s, respectively, in the fat group.

There was a strong diet * speed interaction for triglycerides ($P < 0.0001$). Cholesterol tended to be higher in the high fat group during SET 3 ($P = 0.15$).

Muscle glycogen decreased with exercise, however there was no significant difference between diets.

These results show that full metabolic adaptation to a high fat diet was achieved in 11 wk but not 6 wk. The higher speeds of the lactate thresholds with both training and high fat diet were small but consistent.