Biochemical and Metabolic Changes due to Exercise in Sprint-Racing Sled Dogs: Implications for Postexercise Carbohydrate Supplements and Hydration Management*

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CLINICAL RELEVANCE

Evaluations of biochemical changes associated with spring-style sled dog racing indicate that differences in cortisol, lactate, and serum glucose levels suggest exercise of moderate duration (but high intensity) has metabolic demands that differ from those for typical endurance sled dog racing. Additionally, hematocrit, albumin, sodium, chloride, and blood urea nitrogen levels decreased in one team of dogs, whereas there were mild increases in sodium, chloride, and blood urea nitrogen in the other team. These opposing biochemical findings suggest physiologic changes associated with differences in hydration status, likely attributed to different dietary and hydration strategies used by the respective kennels.

INTRODUCTION

Over the past 30 years, a number of studies have examined the response to exercise in dogs through serum biochemical analyses. Studies evaluating the serum biochemistry of racing greyhounds have shown a consistent response to exercise largely due to the uniformity of the task performed.1-3 In sled dogs, there has been some ambiguity in interpretation of results due to a variety of complicating factors. These variables include diet, time of sampling, ease of obtaining and processing samples, level of fitness of the dogs, and most importantly, the intensity and duration of exercise.

Recently, studies of sled dogs participating in long-distance (<1,600 km) races have led to the publication of hematologic and biochemical parameters for these endurance athletes before and after racing.4,5 These field studies have provided insight into the physiologic responses of sled dogs during this type of endurance racing. Several researchers have previously examined...
the biochemical profiles of sprint-racing sled dogs participating in events of intense exercise with short duration.6–9 These comprehensive studies have been extremely valuable contributors to the understanding of physiology and metabolic responses of high-speed sprint-style racing in sled dogs, yet they did not include field racing situations of high intensity coupled with extended duration (>10 miles).

Premier sprint-racing conditions often consist of three race heats on 3 consecutive days, and mileage varies from 20 to 30 miles each day, with a 30-mile run on the final day. The aim of this study was to examine some of the serum biochemical values before and after exercise during the 1997 Open North American Championship, with the hope of identifying differences in metabolic demands, hydration status, and indicators of muscle-specific damage through creatine kinase (CK) evaluation. These evaluations were similar to earlier field studies evaluating endurance sled dog racing.4,5 Additionally, data were collected to determine whether there were biochemical differences between dogs that completed all 3 days of racing versus dogs that were withdrawn from the race on any of the preceding days.

**MATERIALS AND METHODS**

**Animals and Sampling**

Blood samples were collected from two sled dog teams (26 dogs in Team A, 18 in Team B) that were competing in the 1997 Open North American Championship in Fairbanks, Alaska. All 44 dogs had samples collected before and after racing. Preracing samples were collected 3 days before the race and at least 36 hours after their last low-mileage training run (16 to 20 miles). Dogs in Team A had their prerace blood sample drawn between 12 and 1 PM, about 16 hours after their meal. Dogs in Team B had prerace blood samples drawn between 9 and 10 PM, approximately 4 hours after eating. Subsequent blood samples were drawn approximately 30 to 75 minutes after racing, between 3:30 and 4:15 PM, which was roughly 20 to 24 hours after the dogs were given their meal. Twenty-nine dogs completed the 3 days of racing, and the remaining 15 dogs completed only 1 or 2 days before being withdrawn for various reasons. These 15 dogs had postracing blood samples taken approximately 24 hours after running their last heat. All of the dogs represented the very best canine athletes from two prominent racing kennels. Teams were solicited from kennels with a history of finishing in the top 10 places in this particular race to assure that all dogs were undergoing a similar workload and pace of racing. Thestudy was limited to two teams for logistical reasons, and the first two teams to volunteer from those solicited were enlisted. Team A finished second on each day of racing and finished second overall. Team B finished twelfth on Day 1, sixth on Day 2, fourth on Day 3, and sixth overall.

Whole-blood samples were collected by venipuncture of the cephalic vein into sterile blood collection tubes in the following order: plain glass tube (5 ml for cortisol analysis), lithium heparin tube (5 ml for plasma biochemistry and hematocrit [HCT]), and sodium fluoride tube (3 ml for lactate analysis). Samples were consistently drawn in this order to prevent falsely elevated CK levels. Data from the authors’ laboratory suggest that the action of needle insertion into any muscle may cause enough damage to allow CK release, which may contaminate the first sample collected. The samples collected in lithium heparin and sodium fluoride tubes were then centrifuged immediately (2,000 × g) for 15 minutes for separation of plasma. Just before centrifugation of the lithium heparin tubes, heparinized capillary tubes were filled and simultaneously centrifuged in a separate mi-
crocentrifuge to determine HCT volumes. The delay from the time of venipuncture to the time of centrifugation was less than 10 minutes for all lithium heparin and sodium fluoride tubes, and all samples were kept on ice until centrifugation. The plain glass tubes were allowed to stand refrigerated for 2 to 4 hours to allow clot contraction before centrifugation (2,000 × g) for 15 minutes to obtain the serum needed for cortisol analysis. All samples were frozen at –20˚C, shipped on dry ice to the laboratory, and stored at –80˚C until analysis.

Sample Analyses

Plasma concentrations of CK, glucose, albumin, sodium, chloride, and blood urea nitrogen (BUN) were measured by an automated biochemical analyzer (Hitachi 911). Serum cortisol was measured at the New York State Diagnostic Laboratories by radioimmunoassay, validated for canine serum, with interassay and intraassay coefficients of variation of 7.8% and 6.0%, respectively. Plasma lactate was analyzed using a commercial lactate analyzer (YSI 27) with interassay and intraassay coefficients of variation of 6.2% and 7.2%, respectively.

Statistical Analysis

Values before and after exercise (racing) were compared by paired t-tests, and comparisons between the two teams of dogs were made using Student’s t-test. For both tests, the critical α-value for statistical significance was set at .05. The t-tests were modified using the Bonferroni correction factor for multiple comparisons; thus, the α-value was modified to .025.

RESULTS

Of the 26 dogs in Team A that had blood samples collected 3 days before the race, four were dropped from racing after the first day of competition. Three of these were withdrawn due to poor performance and one was due to stiff gait. Five more dogs were dropped after the second day of competition, all due to poor performance that day.

Of the 18 dogs in Team B sampled 3 days before the race, four were withdrawn from the race after the first day of racing, one due to stiffness, one due to a sore shoulder, and two because of poor performance. Two more dogs from Team B were dropped from the race after the second day due to inexperience in running the distance that the third day entailed.

Both teams exhibited biochemical changes after the third day of racing (Table 1). The most pronounced were significant (P < .025) increases in cortisol and plasma CK and decreases in plasma glucose and lactate concentrations. Additionally, HCT remained unchanged for Team A, whereas Team B showed a pronounced decrease in HCT, with no apparent hemolysis observed in the samples. In light of these findings, albumin, sodium, chloride, and BUN were also assessed to help establish whether this change may have been due to intravascular volume expansion. Albumin, BUN, sodium, and chloride decreased significantly in Team B. Hematocrit and albumin remained virtually unchanged in Team A, but there were mild increases in sodium, chloride, and BUN, which was assumed to be a mild prerenal azotemia due to mild dehydration or fluid shifts from central organs to skeletal muscle. None of the dogs that failed to complete the race exhibited any significant biochemical changes in the blood samples collected 24 hours after their withdrawal from the race, when compared with their prerace values (Table 2).

DISCUSSION

Many of the biochemical findings in this study did not correspond well with previous endurance sled dog studies, which can be
attributed to the metabolic stress and the physiologic response to high-intensity exercise in the sprint-racing sled dog. In both teams, the most noteworthy changes observed were profound increases in plasma cortisol and decreases in plasma glucose concentrations. To date, there has never been a statistically significant rise in cortisol nor a hypoglycemic response to racing documented in racing dogs. During exercise, cortisol will not be released to aid in energy production until a threshold of oxygen consumption above 50% maximal oxygen consumption (VO_{2,max}) has been attained.\textsuperscript{10,11}

Observation of a threefold to fivefold increase in cortisol between samples tested before and after racing in the present study is similar to those reported in previous exercising canine studies when dogs were working at above 50% VO_{2,max} for longer than 30 minutes.\textsuperscript{12,13} Endurance-racing dogs rarely exceed 50% VO_{2,max} during racing, whereas sprint-racing dogs are assumed to maintain a VO_{2,max} between 70% and 90% while racing.\textsuperscript{14} Conversely, greyhounds work at a level greater than 100% VO_{2,max}, but their short duration of exercise is primarily sustained through anaerobic metabolism of glucose and glycogen stores; therefore, the greyhound never undergoes significant negative energy balance facilitating cortisol release for fatty acid utilization and protein catabolism.

Considering Team B was sampled about 30 minutes after racing, and Team A was sampled 45 to 75 minutes after racing, there appears to be a modest difference between postracing cortisol levels between the teams, which is likely due to the difference in the time of blood sampling. Plasma cortisol levels tend to peak between 5 and 30 minutes after exercise when the exercise is at a level of 70% to 90% VO_{2,max}. From 30 to 60 minutes after exercise, renal clearance and liver consumption rapidly rid the

### TABLE 1. Serum Biochemical Values Before and After Day 3 of Racing for Two Sled Dog Teams That Completed 3 Days of Sprint Racing

<table>
<thead>
<tr>
<th>Variable</th>
<th>Reference Range</th>
<th>Team A Before Racing</th>
<th>Team A After Racing Day 3</th>
<th>Team B Before Racing</th>
<th>Team B After Racing Day 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mg/dl)</td>
<td>60–120</td>
<td>100.9 ± 8.4</td>
<td>66.2 ± 23.9*</td>
<td>101.2 ± 14.3</td>
<td>60.8 ± 17.5*</td>
</tr>
<tr>
<td>Lactate (mmol/L)</td>
<td>0.6–1.5</td>
<td>1.44 ± 0.35</td>
<td>1.13 ± 0.23*</td>
<td>2.76 ± 0.55†</td>
<td>1.22 ± 0.21*</td>
</tr>
<tr>
<td>Cortisol (µg/dl)</td>
<td>1–4</td>
<td>2.4 ± 1.5</td>
<td>6.4 ± 2.7*</td>
<td>1.4 ± 0.6</td>
<td>8.7 ± 3.4*†</td>
</tr>
<tr>
<td>Creatine kinase (U/L)</td>
<td>58–241</td>
<td>110 ± 26.6</td>
<td>594.9 ± 461.4*</td>
<td>83.3 ± 40.4</td>
<td>193.2 ± 144.7*†</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>35–56</td>
<td>51.7 ± 5.3</td>
<td>53.1 ± 4.4</td>
<td>53.8 ± 3.1</td>
<td>48.3 ± 4.2*</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>3.0–4.5</td>
<td>4.0 ± 0.4</td>
<td>3.9 ± 0.2</td>
<td>4.4 ± 0.6</td>
<td>3.8 ± 0.2*</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>141–156</td>
<td>148.4 ± 2.3</td>
<td>153.8 ± 4.5*</td>
<td>150.9 ± 3.4</td>
<td>148.0 ± 2.1*</td>
</tr>
<tr>
<td>Chloride (mEq/L)</td>
<td>109–124</td>
<td>114.9 ± 2.2</td>
<td>117.2 ± 3.4*</td>
<td>117.3 ± 2.9</td>
<td>115.6 ± 2.2</td>
</tr>
<tr>
<td>Blood urea nitrogen (mg/dl)</td>
<td>8–30</td>
<td>16.2 ± 4.1</td>
<td>31.9 ± 4.1*</td>
<td>19.5 ± 4.1</td>
<td>18.8 ± 3.6†</td>
</tr>
</tbody>
</table>

*Significantly different from value before racing (P < .025).
†Significantly different from value for Team A at same evaluation (P < .025).
body of cortisol, returning concentrations to baseline resting levels within 2 hours of termination of exercise.\(^{11}\)

Both teams also showed a profound decrease in blood glucose. Blood glucose concentrations at the levels observed 30 to 75 minutes following exercise are highly suggestive of the initiation of glycolysis, gluconeogenesis, or both to sustain blood glucose levels. Fifteen of the 29 dogs that finished the race had blood glucose levels below 60 mg/dl. This degree of hypoglycemia has never been reported in sprint-racing dogs, but in those previously published studies, the distances run were considerably shorter and those dogs were participating in a single exercise episode on a day following one day of rest.\(^ 6\)–\(^ 9\) In one study reported by Reynolds et al,\(^ {15}\) a strenuous 30-km run induced a similar drop in glucose that was observed immediately following exercise, and there was a rapid return to preracing values within 1.5 hours. Results in the present study suggest that hypoglycemia persisted for up to 75 minutes in many of the participating dogs, a finding that is likely due to the intensity of the exercise performed over this 3-day event.

Previous publications have shown that the working sled dog undergoes extensive muscle glycogen depletion during moderately strenuous activity; thus, it is likely that the muscle glycogen reserves have been depleted, and extensive gluconeogenesis is occurring to maintain blood glucose concentrations under these more rigorous conditions.\(^ {15,16}\) Further evidence to support gluconeogenesis is the significant drop in postexercise lactate concentration, which may be due to hepatic scavenging of glucose precursors.

In greyhounds, lactate levels have been shown to increase to 20 times normal levels after racing,\(^ {1,2,3}\) whereas in sprint-racing sled dogs, the degree of hypoglycemia caused by strenuous exercise was much more pronounced.
Lactates have been documented as high as seven times normal. Mean lactate values for both teams of dogs after racing in this study were similar to one another, with no significant increases in either team 30 to 75 minutes after racing. Lactate is rapidly cleared by the liver, and the increases observed after exercise only last 20 to 30 minutes. Blood collection in this study was performed 30 to 75 minutes after racing, which is not within the optimal time frame for the ability to observe transient changes in serum lactate concentrations. Although one would expect that lactate concentrations would have been elevated immediately after exercise, the marked decrease in lactate suggests that hepatic gluconeogenesis via the Cori cycle may be occurring in an effort to increase blood glucose levels. The preracing lactate values for Team B were quite misleading. It was believed that the mild increases in resting lactate values were due to the postprandial lactate spike (samples were taken only 4 hours after a meal). Therefore, changes seen in lactate values for Team A were closer to the physiological changes associated with exercise. It is known that postprandial lactate tends to rise to about 2.5 times normal and does not return to normal levels for about 8 to 12 hours. In light of these findings and other recent literature that discusses the use of postexercise carbohydrate supplements in racing sled dogs, it seems that such supplements may be particularly beneficial for these sprint-racing sled dogs to replenish glycogen stores. Although exercising dogs thrive on anaerobic oxidative fat metabolism for their primary source of energy, it is clear that glucose is being used as an energy source during this high-intensity exercise.

Due to the increased metabolic changes in skeletal muscle during exercise, transient changes in CK were expected to occur. CK is the only muscle-specific enzyme that is used to assess muscle membrane permeability changes (i.e., damage), and CK concentrations showed tremendous variations among dogs within the two kennels in this study. Overall, CK was elevated after racing, but CK values were substantially higher for Team A than for Team B. These CK increases are similar to previously published values by Hinchcliff and colleagues when examining long-distance sled dogs. Unfortunately, no correlation could be made in this study between significantly increased CK concentration and subjective performance ratings of the dogs by the dog driver. It may be that the increases in CK concentration have to be much higher and sustained over longer periods to make these associations between CK and poor performance.

The two groups had remarkably different HCT profiles. HCT remained virtually unchanged in Team A, whereas dogs in Team B showed a decrease, which was hypothetically attributed to an acute shift of fluid into the intravascular compartment in that group of dogs. This hypothesis was further strengthened with albumin, sodium, and chloride also being decreased in Team B, whereas Team A exhibited a mild increase in sodium, chloride, and BUN after racing. These changes observed in Team A are likely due to mild dehydration, preferential blood flow to skeletal muscle during exercise (leading to diminished blood flow to the internal organs resulting in an increase in BUN) or could be related to both of these factors. After lengthy discussion with both kennel managers, the only significant difference observed between the two kennels was dietary and hydration strategies adopted by each kennel. Team B did not receive an appreciable number of calories from meats or oils added to their kibble-based feed, whereas dogs in Team A received about 50% to 60% of their calories from various meat sources (i.e., chicken, beef, tripe) and some oils (i.e., wheat germ, fish).

What may have been more significant were
the hydration strategies adopted by both kennels. All dogs in Team A received roughly 3 to 3.5 L of water daily (120–150 ml/kg/day), whereas dogs in Team B received only 1 to 1.2 L of water daily (40–50 ml/kg/day). During racing, drivers of dogs in Team A did not change the dogs’ water intake; however, drivers in Team B increased water consumption to roughly 2.5 to 3.0 L daily (100–120 ml/kg/day). Although it is speculative, this abrupt change in hydration status may have led to intravascular fluid expansion, which sustained better perfusion of organs and skeletal muscle mass, leading to the lower CK and BUN values for dogs in Team B after 3 days of racing.

In summary, it appears that the effects of sprint racing in these sled dogs can induce a marked increase in plasma cortisol, which may be a response to reduced blood glucose concentrations during racing. The marked depletion of blood glucose shows that the energy demands on sprint-racing sled dogs are very different from those of endurance-racing sled dogs, and these demands are met by significant anaerobic metabolism that likely requires extensive glycogenolysis and gluconeogenesis. Although there are extensive metabolic demands required by skeletal muscle in this situation, there appears to be only a mild increase in CK values, suggesting minimal damage to skeletal muscle overall. Additionally, although it is speculative, the biochemical and HCT profile of Team B in conjunction with CK and BUN values observed after racing suggests a mild volume expansion, which is likely related to the unique hydration management and strategy adopted by this particular kennel.

ACKNOWLEDGMENTS

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REFERENCES


