

INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps. Each original is also photographed in one exposure and is included in reduced form at the back of the book.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

U·M·I

University Microfilms International
A Bell & Howell Information Company
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
313/761-4700 800/521-0600



Order Number 9121490

**Differences in the effects of carbohydrate food form on
endurance performance to exhaustion**

Murdoch, Scott David, Ph.D.

The University of North Carolina at Greensboro, 1990

U·M·I
300 N. Zeeb Rd.
Ann Arbor, MI 48106



**DIFFERENCES IN THE EFFECTS OF CARBOHYDRATE FOOD FORM
ON ENDURANCE PERFORMANCE TO EXHAUSTION**

by

Scott David Murdoch

A Dissertation Submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Greensboro
1990

Approved by


Dissertation Advisor

APPROVAL PAGE

This dissertation has been approved by the following committee of the faculty of the Graduate School at the University of North Carolina at Greensboro.

Dissertation Advisor *Jerry A. Bazzane*

Committee Members *Allan H. Goldfarb*

Helen A. Shaw

David L. Ludwig

November 9, 1990
Date of Acceptance by Committee

August 2, 1990
Date of Final Oral Examination

MURDOCH, SCOTT DAVID, Ph. D. Differences in the Effects of Carbohydrate Food Form on Endurance Performance to Exhaustion. (1990) Directed by Terry L. Bazzarre. 125 pp.

High carbohydrate intake is essential to maintaining prolonged endurance performance (Coyle et al., 1986). The form in which carbohydrate food is ingested alters the glycemic response to that food (Crapo and Henry, 1988). Therefore, the purpose of this investigation was to examine the metabolic and performance effects of ingesting solid compared to slurried carbohydrate food (bananas) between two prolonged exhaustive exercise bouts.

Eight highly trained male triathletes participated in this study. Subjects mean (\pm S.E.M.) age, weight, percent body fat, running VO_2 max, and cycling VO_2 max were 25.7 ± 1.1 years, 68.3 ± 3.0 kilograms, 9.8 ± 1.5 %, 68.1 ± 1.9 and 67.1 ± 2.6 ml/kg/min, respectively. Subjects performed three exhaustive endurance tests (ET), each separated by at least two weeks. Each ET consisted of a 90 minute run followed by 90 minutes of cycling, both at 70% VO_2 max. Workloads were then gradually increased on the cycle, such that by 15 minutes the total increase in work equaled 200 kpm. Subjects continued to cycle until exhausted. Subjects were then given a 20 minute rest during which they ingested one of the following: a) an artificially sweetened placebo drink (P), b) slurried bananas (SL), or c) solid bananas (SO).

Bananas were given in equal portions relative to each subject's body weight (1.1 g of carbohydrate/kg body weight; total volume = 6.8 ml per kg of body weight). Subjects then cycled to exhaustion a second time at 70% of their VO_{2max} . Mean (\pm S.E.M.) times for the second exhaustion period were 28.2 ± 4.3 , 44.1 ± 8.7 , and 46.4 ± 13.2 minutes for P, SL, and SO, respectively. Due to the large variances, these times to exhaustion were not significantly different ($p=0.2699$). The mean blood glucose concentration for the combined carbohydrate treatments at the end of the second exhaustion period (4.4 ± 0.2 mmol/L) was significantly higher than the P treatment (3.5 ± 0.3 mmol/L). At the second exhaustion period, the mean glucose concentration from the SL treatment (4.2 ± 0.3 mmol/L) did not differ significantly from that of the SO treatment (4.6 ± 0.2 mmol/L). Mean glucose concentrations were maintained throughout the second exhaustion ride for the two carbohydrate feedings, but decreased for the P treatment from 4.3 ± 0.3 to 3.5 ± 0.3 mmol/L. Plasma free fatty acid and lactate concentrations were not significantly different between the three treatments at any time. These data demonstrated that the ingestion of solid bananas was equally effective as that of slurried bananas in maintaining plasma glucose concentration toward the end of prolonged, exhaustive exercise at moderate intensities.

ACKNOWLEDGEMENTS

Sincere thanks are offered to the members of my committee, Dr. Terry L. Bazzarre, Dr. Allan H. Goldfarb, Dr. Helen A. Shaw, and Dr. David A. Ludwig, for their valuable insight, advise, helpful suggestions, and encouragement. Special appreciation is expressed to my advisor, Dr. Terry L. Bazzarre, whose optimistic demeanor, enthusiasm, patients, and constant support have made this 4.5 year experience both deeply rewarding and enjoyable. Additional gratitude is offered to Dr. Allan H. Goldfarb who helped assist with several critical aspects of the study at a time when my advisor became unexpectedly ill.

Sincere appreciation is expressed to Mr. David S. Chance, Ms. Rita C. Sigmon, and especially my investigative partner, Mr. I. Phillip Snider, whose unselfish assistance and sense of humor helped to make those many long hours of work from the study much more bearable. I thank Mrs. Shih-min L. Wu for her technical assistance in teaching me many laboratory techniques and assays while always maintaining a pleasant, refreshing disposition. I offer recognition and thanks to the 21 potential subjects, and especially the 11 that participated in the study, whose courage and endurance made this challenging type of investigation possible.

I am very grateful to Tracy Ward, liaison for Dole Fresh Fruit, for donating fresh bananas throughout the studies duration; Hansen & Frank, creators of CAPS™ Enerzymes™, for their financial contribution to the study; and to Dr. Terry L. Bazzarre for his contributions of equipment and supplies, via his RESHAPE and NCARS accounts, that were crucial for this investigation.

And to my parents, Miriam E. and David D. Murdoch, for their unconditional love and support, and for instilling in me a desire to learn along with the confidence needed to do so, I offer a very special thank you!

TABLE OF CONTENTS

	Page
APPROVAL PAGE.....	ii
ACKNOWLEDGMENTS.....	iii
LIST OF TABLES.....	viii
LIST OF FIGURES.....	x
CHAPTER	
I. INTRODUCTION.....	1
Purpose of the Investigation.....	5
Hypotheses.....	5
Definitions.....	5
II. REVIEW OF LITERATURE.....	7
Effects of Prolonged Exercise on Muscle and Liver Glycogen.....	7
Factors Affecting Gastric Emptying of Carbohydrate.....	12
Carbohydrate Ingestion During Prolonged Exercise.....	16
Effects of Carbohydrate Food Form on Glycemic Response.....	21
Summary.....	25
III. METHODOLOGY.....	26
Subjects.....	26
Anthropometric Measurements.....	27
VO ₂ max Testing.....	27
Trial Performance Test.....	30
Endurance Performance Tests.....	32
Blood Analysis.....	37
Statistical Analysis.....	40

IV.	RESULTS.....	42
	Subject	42
	Statistical Analyses.....	43
	Plasma Glucose Concentrations.....	45
	Time To Exhaustion-2.....	50
	Plasma Free Fatty Acid Concentrations.....	50
	Plasma Lactate Concentrations.....	58
	Plasma Volume Shifts.....	58
	Body Weight Changes.....	58
	Run and Bike VO ₂ max Changes.....	67
V.	DISCUSSION.....	69
	Blood Glucose.....	69
	Time To Exhaustion-2.....	76
	Other Variables.....	79
	Limitations.....	86
	Recommendations For Future Research.....	86
VI.	SUMMARY AND CONCLUSIONS.....	89
	BIBLIOGRAPHY.....	91
APPENDIX A.	Consent Form.....	102
APPENDIX B.	Medical History Questionnaire.....	105
APPENDIX C.	Skinfold Conversion Table.....	107
APPENDIX D.	Underwater Weighing Procedures.....	108
APPENDIX E.	Assay Procedures For Plasma Glucose, FFA, And Lactate.....	112
APPENDIX F.	Plasma Volume Determination Procedures.	115
APPENDIX G.	Descriptive Data for Preliminary Sample (n=21).....	116
APPENDIX H.	Individual Performance Data of Potential Subjects (n=21).....	117
APPENDIX I.	Individual Descriptive Data of Potential Subjects (n=21).....	118
APPENDIX J.	Individual Performance Test Times (minutes) From RIDE-1 (n=8).....	119

APPENDIX K.	Individual Run and Bike VO_{2max} Values (ml/kg/min) at Weeks 0, 5, and 11 (n=8).....	120
APPENDIX L.	Raw Data For Each Subject (n=12).....	121

LIST OF TABLES

TABLE		Page
1.	Summary Of Research Demonstrating Improved Performance From Liquid Carbohydrate Feedings.....	18
2.	Mean Descriptive And Preliminary Performance Data For Experimental Subjects (n=8).....	44
3.	Mean Plasma Glucose Concentrations (mmol/L) During The Performance Tests For Each Treatment (mean \pm S.E.M.)	46
4.	Statistical Analyses On Plasma Glucose Data Collected At The Exhaustion-2 Time Period...	47
5.	Statistical Analyses On Plasma Glucose Data Collected At The Post Recovery Time Period..	49
6.	Statistical Analyses Of The Exhaustion-2 Riding Times.....	52
7.	Individual Performance Test Times (minutes) From Each Treatment From RIDE-2.....	53
8.	Mean Plasma Free Fatty Acid Concentrations (mmol/L) During The Performance Tests For Each Treatment (mean \pm S.E.M.).....	54
9.	Statistical Analyses On Plasma FFA Data Collected At The Post Recovery Time Period..	56
10.	Statistical Analyses On Plasma FFA Data Collected At The Exhaustion-2 Time Period...	57
11.	Mean Plasma Lactate Concentrations (mmol/L) During The Performance Tests For Each Treatment (mean \pm S.E.M.).....	59
12.	Statistical Analyses On Plasma Lactate Data Collected At The Post Recovery Time Period..	61
13.	Statistical Analyses On Plasma Lactate Data Collected At The Exhaustion-2 Time Period...	62

14.	Percent Plasma Volume Shifts During The Performance Tests For Each Treatment (mean \pm S.E.M.).....	63
15.	Changes In Mean Body Weight (kg) During The Performance Tests For Each Treatment (mean \pm S.E.M.).....	65
16.	Mean (\pm S.E.M.) Run and Bike $VO_{2,max}$ Values (ml/kg/min) at Weeks 0, 5, and 11 (n=8).....	68

LIST OF FIGURES

FIGURE		Page
1.	Time Periods For Max Tests And Performance Tests During The 12-Week Study.....	29
2.	Trial Performance Test.....	31
3.	Performance Test.....	34
4.	Mean Plasma Glucose Concentrations At Exhaustion-1, Post Recovery & Exhaustion-2.....	48
5.	RIDE-2 Mean Times (\pm S.E.M.) To Exhaustion...	51
6.	Mean Plasma FFA Concentrations At Exhaustion-1, Post Recovery & Exhaustion-2.....	55
7.	Mean Plasma Lactate Concentrations At Exhaustion-1, Post Recovery & Exhaustion-2.....	60
8.	Mean Plasma Volume Shifts Throughout Each Performance Test.....	64
9.	Changes In Mean Body Weights Throughout Each Performance Test.....	66

CHAPTER I
INTRODUCTION

Many factors such as dehydration (Buskirk, Iampietro, & Bass, 1958; Nielsen, 1984) and the depletion of endogenous carbohydrate stores (Ahlborg, Bergstrom, Eklund, & Hultman, 1967; Bergstrom & Hultman, 1967; Bergstrom, Hermansen, Hultman, & Saltin, 1967; Coggan & Coyle, 1987; Coyle, Coggan, Hermert, & Ivy, 1986; Hermansen, Hultman, & Saltin, 1967) contribute to fatigue during prolonged exercise. Dehydration may be avoided by consuming fluids throughout exercise. Assuming adequate hydration, the depletion of endogenous carbohydrate stores (muscle glycogen, liver glycogen, and blood glucose) becomes the primary limiting factor in endurance events of moderate to high intensity (60%-85% of VO_{2max}), and prolonged duration (two hours or more) (Bergstrom et al., 1967; Coggan & Coyle, 1989; Coggan & Coyle, 1988; Coggan & Coyle, 1987; Coyle et al., 1986). A series of studies by Coggan and Coyle (Coggan & Coyle, 1989; Coggan & Coyle, 1988; Coggan & Coyle, 1987; Coyle et al., 1986) have demonstrated that plasma glucose is the predominant carbohydrate energy source during the latter stages of prolonged exercise; and, that a decline in plasma glucose during prolonged exercise is associated with

fatigue. Glucose uptake by the working skeletal muscle is linearly related to exercise intensity (Katz, Broberg, Sahlin, & Wahren, 1986; Saltin, Wahren, & Pernow, 1974; Wahren, Felig, Ahlborg, Jorfeldt, 1971), and increases in proportion to the extent of muscle glycogen depletion (Coggan & Coyle, 1988; Coyle et al., 1986; Gollnick, Pernow, Essen, Jansson, & Saltin, 1981). Plasma glucose utilization may exceed 1.5 g/minute during prolonged cycling at 70% of VO_2 max (Coggan & Coyle, 1988). Late in exercise, an estimated 50% of the total energy needed was supplied by carbohydrate, with approximately 76% of this carbohydrate being supplied from plasma glucose (Coggan and Coyle, 1987).

Carbohydrate intake of certain solid foods elevated plasma glucose levels in healthy resting subjects (Jenkins, Wolever, Taylor, Baker, Fielden, Baldwin, et al., 1981). Sixty minutes after the ingestion of solid carbohydrate food, plasma glucose concentrations in resting subjects were higher than when the same food was ingested in liquid form (Haber, Heaton, Murphy, and Burroughs, 1977). Therefore, carbohydrate intake of solid food between two exhaustive exercise bouts may supply enough fuel later in exercise to delay exhaustion during the second exercise bout, as compared to carbohydrate food in slurried (liquid) form.

Concentration of Exogenous Carbohydrate

There are numerous studies (Coggan & Coyle, 1987; Coggan & Coyle, 1988; Coggan & Coyle, 1989; Coyle et al., 1986; Davis, Lamb, Pate, Slentz, Burgess, & Bartoli, 1988) which demonstrate that carbohydrate drinks enhance endurance performance. However, there is considerable controversy regarding the optimal concentrations and composition of carbohydrate drinks. In particular, fluids of high carbohydrate concentrations (10% or more; weight per unit volume) are thought to compromise fluid replacement via slower rates of gastric emptying (Costill & Saltin, 1974; Coyle, Costill, Fink, & Hoops, 1978; Fordtran & Saltin, 1967; Foster, Costill, & Fink, 1980; Ryan, Bleiler, Carter, & Gisolfi, 1989). Coggan and Coyle (1987, 1988, and 1989) recently reported significant increases in time to exhaustion of cyclists ingesting a 50% carbohydrate solution. Their work suggests that the intake of high concentrations of carbohydrate may be the most effective means of supplying enough blood glucose for prolonged, high intensity activity.

Food Form: Solid vs Slurried

There are no known studies that have compared the effects of solid versus liquid carbohydrate food intake during prolonged exercise on endurance performance. Yet, solid foods are often ingested during training and racing

events that last longer than 2 hours. In healthy resting subjects, food form (i.e., solid vs slurried) significantly alters the glycemic response to that food (Bolton, Heaton, & Burroughs, 1981; Crapo & Henry, 1988; Haber, Heaton, Murphy, & Burroughs, 1977). These investigators hypothesized that carbohydrate foods presented to the gastrointestinal tract in slurried form will give all nutrient molecules equal availability to the digestive enzymes and absorptive processes, thereby increasing the delivery of glucose into the circulation. On the other hand, the ingestion of solid carbohydrate foods may be of greater benefit as a source of fuel (blood glucose) than liquid carbohydrate foods because of the following:

- a) solid food may stay in the GI tract longer, theoretically providing a continuous supply of carbohydrate for the blood,
- b) solid food provides more carbohydrate per unit weight,
- c) solid food may have the advantage of greater availability/access to the athlete (may be carried by the athlete during the event),
- d) solid food may be more psychologically and physiologically pleasing/satisfying,

Purpose of the Investigation

The purpose of this study was to examine the metabolic and performance effects of ingesting either solid or slurried carbohydrate food between two prolonged exhaustive exercise bouts.

Hypotheses

Hypothesis 1: The ingestion of solid carbohydrate will result in higher mean plasma glucose concentration at the end of the second exhaustion period, than when the same carbohydrate is ingested in slurried form.

Hypothesis 2: The ingestion of solid carbohydrate will increase the time to exhaustion during the second exhaustion ride, as compared to the same carbohydrate ingested in slurried form.

Definitions

VO₂max: Maximal rate at which an individual can consume oxygen, measured in ml/kg of body wt/minute.

Exogenous carbohydrate: Newly obtained dietary carbohydrate via ingestion.

Gastric emptying: The rate at which the contents within the stomach move into the small intestines.

Biathlon: Endurance event consisting of consecutive bicycling and running.

Triathlete: An individual who participates in triathlons, which consist of swimming, bicycling, and running, consecutively.

Volitional fatigue: Failure to maintain the prescribed work output required at a prescribed workload.

CHAPTER II

REVIEW OF LITERATURE

This review of the literature will address four specific areas: the metabolic limitations of prolonged exercise; factors affecting gastric emptying rate of carbohydrates; the effects of ingesting carbohydrate during prolonged activity; and the glycemic response to altered food form.

Effects of Prolonged Exercise on Muscle and Liver Glycogen

Data from many studies have clearly demonstrated that prolonged exercise at 60%-85% of VO_2 max substantially lowered both muscle (Ahlborg et al., 1967; Bergstrom & Hultman, 1967; Bergstrom et al., 1967; Coyle et al., 1986; Hermansen et al., 1967; Essen, 1977; Karlson & Saltin, 1971; Baldwin, Reitman, Terjung, Winder, & Holloszy, 1973; Hickson, Rennie, Conlee, Winder, & Holloszy, 1977; Jansson & Kaijser, 1987; Richter & Galbo, 1986) and liver (Baldwin et al., 1973; Hickson et al., 1977) glycogen concentrations. In these studies, fatigue correlated highly with low muscle and liver glycogen concentrations, suggesting that glycogen was an important source of energy during exercise at these moderate intensities.

Blood glucose is produced mainly from hepatic glycogen stores, and, to a smaller extent, from hepatic gluconeogenic precursors supplied from the periphery (Laundu & Wahren, 1988; Kirland & Pilakis, 1989). Only the liver and kidneys have the enzyme glucose-6-phosphatase, needed for the release of glucose into the blood stream. Yet, blood glucose is the primary, and to a large extent, the exclusive fuel utilized by the central nervous system (Cryer, 1985). Once muscle glycogen concentration falls to a critical level, blood glucose becomes the primary carbohydrate source needed to continue the same exercise intensity. The increased need for glucose during exercise results in the rapid utilization and subsequent lowering of blood glucose concentrations, since the liver has a limited supply of glycogen. Signs and symptoms of low blood glucose concentrations (i.e., hypoglycemia) are lethargy, confusion, changes in behavior, visual impairment, impaired performance of routine tasks, seizures, coma, and death (Cryer, 1985). Prolonged, moderately intense exercise increases blood glucose utilization. Since hepatic glycogen concentration is in limited supply, prolonged exercise can lower blood glucose concentrations to a hypoglycemic level. Fatigue is likely to occur under these conditions.

In man, muscle glycogen can be sampled using needle biopsy procedures (Bergstrom and Hultman, 1967).
Determining the effect of prolonged exercise on liver

glycogen concentrations has primarily been studied in animals (Baldwin et al., 1973; Hickson et al., 1977). Baldwin et al. (1973) ran rats at a moderately high intensity (approximately 27 meters/minute up an 8% grade) for two hours in order to determine the extent of muscle and liver glycogen (and endogenous triglyceride) depletion. Muscle glycogen content was determined in three different fiber types; red, intermediate, and white. Muscle glycogen depletion for both the red and intermediate fibers was greatest during the first 15 minutes of exercise. There was little change in muscle glycogen content of the white fibers. At the end of the exercise period, red and intermediate fibers were 42% and 72% lower in muscle glycogen concentration, respectively. Liver glycogen decreased approximately 85% from the exercise bout. These data emphasize the substantial contribution of both muscle and liver glycogen to prolonged exercise at a moderately high intensity of exercise.

Hickson et al. (1977) ran rats to exhaustion at a moderately high intensity (22-27 meters/minute up a 15% grade), and determined the extent of liver and muscle glycogen depletion. Mean running time to exhaustion was 118 minutes at which time muscle glycogen concentration was approximately 73% depleted in all three muscle fiber types. Mean liver glycogen was approximately 97% depleted. At exhaustion, the mean blood glucose concentration had fallen

to a hypoglycemic level (from an average of 8.5mM to less than 3.0mM). It is interesting to note that liver glycogen concentration decreased to a greater extent than muscle glycogen concentration as a result of prolonged exercise. Thus, depletion of liver glycogen concentration may be the primary cause of exhaustion from prolonged, moderately intense exercise.

Dietary manipulation of both muscle and liver glycogen has a direct effect on prolonged exercise (Bergstrom et al., 1967; Costill, Bowers, Branam, & Sparks, 1971; Costill, Sherman, Fink, Maresh, Witten, & Miller, 1981; Dohm, Tapscot, Barakat, & Kasperek, 1983; Hermansen et al., 1967; Pruett, 1970; Rodahl, Miller, & Issekutz, 1964; Sherman, Costill, Fink, Hagerman, Armstrong, & Murray, 1983; Sherman, 1987; Vissing, Wallace, & Galbo, 1989). In a classic study by Bergstrom et al. (1967), nine fasted subjects bicycled to exhaustion at a workload corresponding to 75% of their VO_2 max after consuming three different diets in random order: mixed-uncontrolled (M), fat-protein (FP), and high-carbohydrate (HC). Subjects consumed each of these diets for three consecutive days. The composition for the M diet was not reported. The FP diet provided 1300 kcals as fat and 1500 kcals as protein; and the HC diet contained 2300 kcals carbohydrate and 500 kcals protein. The actual composition (ie. food sources) of these diets was not reported. Muscle biopsies were taken before and after the

three rides to exhaustion. Mean pre-exercise muscle glycogen concentration was highest after consuming the HC diet (3.31 g/100g muscle), lowest after the FP diet (0.63 g/100g muscle), and intermediate on the M diet (1.75 g/100g muscle). Mean post-exercise muscle glycogen concentrations were 0.43, 0.13, and 0.17 g/100g muscle for diets HC, FP, and M, respectively. The mean work times to exhaustion were 57, 114, and 167 minutes for the FP, M, and HC diets, respectively. Work time to exhaustion and initial muscle glycogen concentration were highly correlated ($r=0.92$). Liver glycogen can be manipulated by diet (Vissing, Wallace, & Galbo, 1989), suggesting that habitual and acute dietary intake contribute to pre-exercise liver and muscle glycogen content.

Prolonged exercise also requires the oxidation of fat in order to maintain muscular activity and to spare the limited stores of endogenous carbohydrate. Triglyceride depots are found predominantly in adipose and skeletal muscle tissue. Endogenous muscle triglyceride concentrations decrease throughout prolonged exercise (Baldwin et al., 1973; Hoppeler, Howald, Conley, Lindstedt, Claassen, Vock, & Weibel, 1985; Jansson & Kaijser, 1987), indicating their role as a fuel for muscular work. Free fatty acids from adipose tissue are released into the circulation and oxidized by the working muscles for energy. In addition to providing fuel for exercise, plasma free

fatty acids help inhibit plasma glucose utilization. Ultimately, blood glucose provides much of the additional energy that cannot be met by free fatty acids, muscle triglycerides, and muscle glycogen (Newsholme & Start, 1981).

The elevation of plasma free fatty acids prior to exercise can increase the time to exhaustion, and decrease the rate of both liver and muscle glycogen utilization (Costill et al., 1971; Hickson et al., 1977). Free fatty acid utilization by skeletal muscle is directly related to its concentration in the plasma (Gollnick, 1977; Newsholme & Start, 1981). Studies documenting increased plasma free fatty acids during prolonged exercise are numerous (Gollnick, 1977; Hickson et al., 1977; Newsholme, 1977; for example), indicating free fatty acids function as an important fuel source.

Factors Affecting Gastric Emptying of Carbohydrate

The rate of gastric emptying is influenced by several factors including caloric density, volume, composition, metabolic status of the individual, temperature of the ingested feeding, and environmental conditions (Minami & McCallum, 1984; Murray, 1987). Among these factors, caloric density and volume are the predominant factors which determine gastric emptying, during both rest (Brener, Hendrix, McHugh, 1983; Foster et al., 1980; Hunt, Smith, &

Jiang, 1985; McCann & Stricker, 1986; Minami & McCallum, 1984; Murray, 1987) and exercise of moderate intensity (Minami & McCallum, 1984; Murray, 1987).

Osmoreceptors in the stomach were hypothesized to be regulators which control the gastric emptying rate of highly concentrated carbohydrate feedings (Minami & McCallum, 1984). Although recent evidence has demonstrated that caloric density, not osmolarity (Murray, 1987) determines the rate of gastric emptying, this conflict has not been resolved.

Carbohydrate solutions whose concentrations exceed 10% (weight/volume) decreased the rate of gastric emptying, when compared to solutions of less than 10% (Foster et al., 1980). Even though gastric emptying was slowed using the more concentrated glucose solutions of similar volumes, the duodenum received greater absolute amounts of carbohydrate (Foster et al., 1980; Hunt, Smith, & Jiang, 1985; McCann & Stricker, 1986). Foster et al. (1980) had 15 fasted subjects ingest 400 ml glucose solutions containing one of the following concentrations: 5%, 10%, 20%, and 40%, weight per volume of water. Using a nasogastric tube for aspirating the gastric residue 30 minutes after ingestion, the gastric emptying rate decreased in the more concentrated glucose solutions. However, absolute carbohydrate delivery to the duodenum 30 minutes after ingestion, increased directly with the concentration of the solution: i.e., 12g,

23g, 45g, and 68g for the 5%, 10%, 20%, and 40% glucose solutions, respectively. Thus, for these glucose solutions, carbohydrate delivery was 0.4 g/min, 0.8 g/min, 1.5 g/min, and 2.3 g/min, respectively. Hunt et al. (1985) found that after administering glucose polymer solutions of varying volumes (300, 400, and 600 ml) and carbohydrate concentrations (12.5%, 17.5%, 25%, 32%, 37.5%, and 50%, weight per volume of water) via nasogastric tube, gastric emptying rate decreased at the higher concentrations. After 30 minutes, the glucose solutions at 400ml with higher concentrations of carbohydrate delivered more calories to the duodenum (i.e., the 12.5%, 25%, and 50% glucose solutions delivered 0.8 g/min, 1.0 g/min, and 1.4 g/min, respectively). Increasing the volume at similar carbohydrate concentrations resulted in a greater volume of emptying, therefore, delivering more energy substrate to the duodenum. Thus, the volume emptied (per unit of time) was directly related to the volume present in the stomach (Minami & McCallum, 1984); while carbohydrate concentration was directly related to the rate of gastric emptying at concentrations that deliver less than 2.3g/min. to the duodenum.

The composition of the gastric contents will affect gastric emptying response rate. Meals containing fat (determined by the fatty acid chain length) or amino acids (particularly L-tryptophan) will slow gastric emptying

(Minami & McCallum, 1984).

The metabolic state of the individual alters gastric emptying rate. Conditions of insulin-induced hypoglycemia increase the rate of glucose gastric emptying in rats (McCann & Strickner, 1986), while stimulating gastrointestinal contractions in man (Fellows, Evans, Bennet, MacDonald, Clark, & Bloom, 1987). Given this information, it is conceivable that the gastric emptying of carbohydrate food ingested toward the latter stages of prolonged moderately intense exercise could be increased due to lower blood glucose concentrations. In a study by Cammack, Read, Cann, Greenwood, & Holgate (1982), seven fasted subjects ate a meal consisting of three small sausages (20g each), baked beans (120g), labelled (technetium sulphur colloid) mashed potatoes (150g), and a pineapple dessert (75g); totaling 630 kcal. Shortly after ingesting the meal, subjects began cycling at a constant rate for five out of every ten minutes, for six hours. The intensity was light, producing a mean heart rate of 119 beats/minute. The same subjects performed a non-exercising control experiment for the six hour period. The half time for gastric emptying was significantly shorter during the exercise period (1.2 hours) as compared to the control period (1.5 hours). Although the reasons for this difference in gastric emptying time cannot be elucidated from this study, it appears that the internal energy status

of an individual is in communication with the gastrointestinal tract, signaling faster gastric emptying for the purpose of increasing fuel availability for continued exercise. Neuffer, Costill, Fink, Kirwan, & Fielding (1986) also demonstrated an increase in the rate of gastric emptying after 15 minutes of running at light and moderate intensities (between 50% and 70% of VO_{2max}). However, other research suggests that exercise at intensities below 80% VO_{2max} has no influence on gastric emptying or intestinal absorption of carbohydrate solutions (Murray, 1987). These discrepancies need further clarification. In addition, diurnal fluctuations will alter gastric emptying (Murray, 1987). Therefore, under conditions of prolonged moderately intense exercise where endogenous carbohydrate can become low, the stomach and small intestines are capable of delivering significant amounts of carbohydrate to the circulation for fuel. Thus, one limiting factor of exhaustive exercise may be the amount of carbohydrate ingested during the activity.

Carbohydrate Ingestion During Prolonged Exercise

Endurance performance is frequently measured by one of the following three methods: the quantity of work performed within a given time period; the quantity of time taken to perform a set amount of work; and, the quantity of time subjects can perform work at a given intensity before

exhaustion is reached. Using all of these methods, the ingestion of carbohydrate solutions during prolonged exercise have been repeatedly shown to improve endurance performance (see references in Table 1.). The research demonstrating performance improvements from liquid carbohydrate feedings during exercise is summarized in Table 1.

In the studies where no improvement in endurance performance from ingesting carbohydrate solutions was observed, either the total amount of carbohydrate ingested was low (less than 60 grams) (Flynn, Costill, Hawley, Fink, Neuffer, Fielding, & Sleeper, 1987; Maughan & Whiting, 1983), the intensity was under 60% of their VO_2 max (Ahlborg & Felig, 1976), and/or the time period of exercise was under 90 minutes (Bonen, Malcolm, Kilgour, MacIntyre, & Belcastro, 1981; Brodowicz, Lamb, Bauer, & Conners, 1984). Endogenous carbohydrate stores are capable of supplying enough fuel to support moderately intense exercise for up to 2 hours (Newsholme & Start, 1981). Additionally, carbohydrate ingestion may only benefit trained athletes that are accustomed both physiologically and biomechanically to exhaustive exercise (Felig, Cherif, Minagawa, & Wahren, 1982; Noakes, Koeslag, McArthur, 1983).

Table 1. Summary of Research Demonstrating Improved Performance from Liquid Carbohydrate Feedings During Exercise.

References	Mode of Exer.	Time (min)	Intensity (%VO ₂ max)	Type CHO*	Total CHO (g) Intake	Solution Conc. (wt/vol)	Volume of Feeding in ml*Number Feedings
Brooke et al. (1975)	bike	214	67%	GS	890	36%	250 * 10
Ivy et al. (1979)	bike	120	72%	GP	90	---	--- * 7
Coyle et al. (1983)	bike	157	74%	GP	123	50%	140 * 1
						6%	300 * 3
Ivy et al. (1983)	walk	299	45%	GP	120	20%	150 * 4
Coyle et al. (1986)	bike	241	71%	GP	448	50%	280 * 1
						10%	280 * 11
Foster et al. (1986)	soccer	160	---	GP	75	25%	300 * 1
Seifert et al. (1986)	bike	241	64%	GP+F	123	7%	260 * 7
Coggan & Coyle (1987)	bike	184	70%	GP	210	50%	420 * 1
	bike	215	70%	DI	49	20%	continuous
Mitchell et al. (1987)	bike	96	70%	GP	100	8%	167 * 8
Coggan & Coyle (1988)	bike	205	60%-85%	GP	322	50%	140 * 1
						20%	210 * 6
Davis et al. (1988)	bike	160	75%	S+G	149	6%	275 * 9
Coggan & Coyle (1989)	bike	205	70%	GP	210	50%	420 * 1
Range		96-205	45-85%		49-890	6-50%	140-420

* Abbreviations: GS=glucose syrup; GP=glucose polymer; F=fructose; DI=dextrose infusion; S=sucrose.

Several studies in the literature have investigated the effects of feeding solid carbohydrate foods during exercise on endurance performance (Brooke, Davis, & Green, 1975; Fielding, Costill, Fink, King, Hargreaves, & Kovaleski, 1985; Hargreaves, Costill, Coggan, Fink, & Nishibata, 1984). In a unique investigation by Brooke et al. (1975), an unprecedented quantity of carbohydrate (totalling 890 grams) was ingested by eight highly trained European cyclists, during exhaustive cycling at 67% of their VO_{2max} . The ingested carbohydrate was either glucose syrup (GS) in a 36% solution or canned rice pudding (RP) with added sucrose. These treatments were ingested every 20 minutes (totaling 10 feedings) until exhaustion. Thus, 89 grams of carbohydrate were ingested every 20 minutes. Control treatments were a low energy (LE) drink of equal volume as the glucose syrup treatment (250 ml every 20 minutes), and a treatment where nothing (NO) was given (although not stated, it appears that no water was given in this last treatment). The times to exhaustion for the GS, RP, LE, and NO treatments were 214, 200, 180, and 148, respectively. The exhaustion times of the GS and RP were not significantly ($p < 0.05$) different from each other but were significantly different from the NO and LE treatments. The consistency of rice pudding can be classified as a semi-solid, which may be why no statistical differences were observed between the RP and the GS treatments. These data suggest that massive

carbohydrate feedings can improve prolonged performance to exhaustion.

Hargreaves et al. (1984) and Fielding et al. (1985) used a similar protocol in testing the effects of ingesting solid mixed food during 4 hours of intermittent cycling. The protocol consisted of 30 minute intervals of cycling 20 minutes at 50% of their $VO_2\text{max}$, followed by 10 minutes of intermittent cycling (30 seconds at 100% $VO_2\text{max}$ followed by 2 minutes of rest). This 30 minute regime was repeated 8 times, with the final sprint bout of each trial being continued until exhaustion. Hargreaves et al. (1984) had ten well trained men (mean $VO_2\text{max}$ of 62 ml/kg/min) ingest either a solid feeding consisting of 43 g of sucrose, 9 g of fat, and 3 g of protein along with 400 ml of water, or an artificially sweetened drink (placebo) at 0, 1, 2, and 3 hours of cycling. Actual compositions (food sources) were not given. The ingestion totaled 1060 kcal, consisting of 172, 36, and 12 g from carbohydrate (64%), fat (31%), and protein (5% of total kcal), respectively. The cyclists on the solid feeding treatment cycled significantly longer on the mean final sprint ride to exhaustion compared to the placebo treatment; 127 vs 87 seconds, respectively. Fielding et al. (1985) had nine moderately trained men (mean $VO_2\text{max}$ of 49 ml/kg/min) ingest a solid feeding consisting of 86 g of sucrose, 18 g of fat, and 6 g of protein over the 4 hour period, altering the frequency (F) or dosage (D) of

each ingestion. In the F trial, the portions of the total feeding (10.8, 2.3, and .8 g of sucrose, fat, and carbohydrate, respectively), along with 200ml of water, were ingested at 0, 30, 60, 90, 120, 150, 180, and 210 minutes of exercise. In the D trial, the portions of the total feeding (21.5, 4.5, and 1.5 g of sucrose, fat, and carbohydrate, respectively), along with 400 ml of water, were ingested at 0, 60, 120, and 180 minutes of exercise. In the control (C) trial, subjects ingested 400 ml of an artificially sweetened drink at 0, 60, 120, and 180 minutes of exercise. Mean (\pm S.E.M.) sprint riding times were significantly longer with treatment F (121 ± 10 seconds) compared to the C treatment (81 ± 7 seconds). Sprint riding time for treatment D (110.6 ± 13.9 seconds) did not differ from either treatments F or C. The data from these investigations demonstrate the beneficial effects of ingesting solid food during prolonged performance. However, the mechanisms involved in improving exhaustive sprint performance cannot be elucidated by these data since more than one type of fuel was ingested, and because the extreme variation in the intensity of work performed (50% to 100% of VO_2 max) result in different metabolic fuel requirements.

Effects of Carbohydrate Food Form on Glycemic Response

Despite similarities in macronutrient composition, different carbohydrate foods elicit different glycemic

responses, which can vary greatly (Crapo, Reaven, & Olefsky, 1976; Crapo, Insel, Sperling, & Kolterman, 1981; Jenkins et al., 1981). It was suspected, and later confirmed (Jenkins, Ghafari, Wolever, Taylor, Baker, Fielden, et al., 1982), that differences in the rates of digestion were, in part, responsible for the variable postprandial blood glucose response to ingesting carbohydrate foods. Measuring the extent to which various carbohydrate foods raise blood glucose concentrations brought about the development of the "glycemic index (GI)" (Jenkins et al., 1981).

The GI has been measured for approximately 120 carbohydrate foods and sugars in both diabetic and nondiabetic subjects at rest (Thorburn, Brand, & Truswell, 1986). Factors known to affect the glycemic response of foods include food form (Bolton et al., 1981; Crapo & Henry, 1988; Haber et al., 1977; O'Dea, Nestel, & Antonoff, 1980; Wong & O'Dea, 1983), the type and amount of starch and fiber (Behall, Scholfield, & Canary, 1988; Goddard, Young, & Marcus, 1984; Vinik & Jenkins, 1988), the methods of cooking and food processing (Brand, Nicholson, Thorburn, & Truswell, 1985; Collings, Williams, & McDonald, 1981), the rate of meal ingestion (Jenkins et al., 1982), the quantity of phytic acid, lectins, and tannins (Snow & O'Dea, 1981; Thorburn, Brand, & Truswell, 1986; Yoon, Thompson, & Jenkins, 1983), liquid/solid food combinations (Schusdziarra, Dangel, Klier, Henrichs, & Pfeiffer, 1981),

macronutrient interaction (Thorburn, Brand, & Truswell, 1986), psychological state (Morse, Schacterle, Furst, Zaydenberg, & Pollack, 1989), and salt content (Thorburn, Brand, & Truswell, 1986). This review is limited to alterations in carbohydrate food form.

Disruption of the food form in a single carbohydrate food will alter the metabolic response from the ingestion of that food (Bolton et al., 1981; Crapo & Henry, 1988; Haber et al., 1977; O'Dea et al., 1980; Wong & O'Dea, 1983). Ground brown and white rice had significantly higher glycemic responses than the whole rice form (O'Dea et al., 1980). When whole lentils were ground, the results observed were similar (Wong & O'Dea, 1983). Additionally, when apples were pureed, and grapes were squeezed into juice, glycemic responses to their ingestion were increased (Bolton et al., 1981; Haber et al., 1977). Recent work by Crapo and Henry (1988) further demonstrated that the glucose and insulin responses to ingestion of a particular starch was affected by the form in which it was introduced into the gastrointestinal tract. A test load of whole and blended (slurried) rice and potato was consumed by each of 12 non-diabetic subjects. The lower postprandial glycemia of whole rice was eliminated when slurried, which was similar to the glycemic response of slurried potatoes. The low surface area:starch ratio of whole rice and potato may limit access of the intestinal enzymes to the rice starch, resulting in

the low blood glucose response seen after ingestion. Yet, between 2 and 3 hours after ingestion, the serum glucose concentrations were higher from ingesting the solid foods, than the slurried foods. These results are limited to foods that have significant amounts of fiber as part of the food structure, which can limit the complete breakdown of all the available carbohydrate.

Of the few studies in the literature investigating the topic of liquid vs solid foods and their postprandial glycemic responses (Fox, Bartels, Keller, & Vivian, 1983; Schusdziarra et al., 1981), the foods tested were mixed with other macronutrients. Thus, comparisons of single liquid and solid foods, or of a single macronutrient were not made. Sugar ingested in liquid form (15 g of sucrose in 200 ml of water = 7.5% carbohydrate solution) with a solid meal (50 g rice, 150 g steak, and 100 g peas = 600 kcal) increased blood glucose response, throughout the 60 minute post ingestion period, more than the same meal in liquid form (via blender) with the sugar included (Schusdziarra et al., 1981). These data suggest that a mixture of solid food and carbohydrate solutions may produce a higher glycemic response than liquid foods of similar composition. This conclusion is speculative, at best, since meal composition included all three macronutrients.

Summary

Successful prolonged endurance training and racing requires the intake of large amounts of fuel, primarily in the form of carbohydrates, both during and after the exercise period. The practice of ingesting solid carbohydrate foods (fresh and dried fruits, candy, cookies, energy bars, and sandwiches) during prolonged training and racing is commonplace among endurance athletes (Brouns, 1988; Burke & Read, 1987; DeMoss, 1989). The advantages of ingesting solid carbohydrates, when compared to liquid carbohydrates, appear to be related to the high absolute amounts of carbohydrate ingested, as well as the convenience of transporting the highly concentrated, light weight fuel. The performance and metabolic differences observed when ingesting the same carbohydrate food in a solid or liquid form during exhaustive exercise have not been investigated. The need to identify metabolic and other factors that regulate fuel absorption and utilization under the conditions of prolonged stress is essential to our understanding of nutrition during prolonged endurance performance.

CHAPTER III

METHODOLOGY

Subjects

Subjects, between the ages of 18-35, were solicited from an advertisement in the Carolina Triathlon Club News Letter. Twelve male biathletes/triathletes were chosen from a pool of 21 volunteers. All 21 potential subjects gave written, informed consent to participate (Appendix A), and were required to complete a medical history questionnaire (Appendix B) to certify that they had no known medical problems, and no history of heart disease, hypertension, hyperlipidemia, diabetes or other known medical disorders that might compromise their full participation in this study.

Selection of the final 12 subjects required normal blood levels of total cholesterol and HDL-cholesterol. Hemoglobin, hematocrit, and ferritin were analyzed for the purpose of identifying potential iron abnormalities in these athletes. Subjects were required to have averaged, during the previous month, a minimum of 30 miles/week running and 100 miles/week cycling. These requirements were verified from their training logs. A VO_{2max} of at least 50 ml/kg/min on both the treadmill and the cycle, was also required to

participate in this study. Additionally, all subjects had to complete at least three hours of an endurance trial performance test. The endurance trial performance test was used to determine the final 12 participants. This test included 90 minutes of running immediately followed by 90 minutes (or longer) of cycling at 70% VO_{2max} .

Anthropometric Measurements

Height (cm) and weight (kg) were measured using a Detecto™ beam scale. Percent body fat was determined using the sum of four skinfold measures and by hydrostatic weighing. The Durnin & Womersley (1974) procedure was used to determine body fat based on the sum of four skinfold measurements (triceps, biceps, suprailiac, and subscapular). The sum of four skinfolds was converted to percent body fat from the conversion table by Durnin & Womersley (1974) (Appendix C). Percent body fat was also assessed by hydrostatic weighing from the procedure described by Sinning (1975) (Appendix D).

VO_{2max} Testing

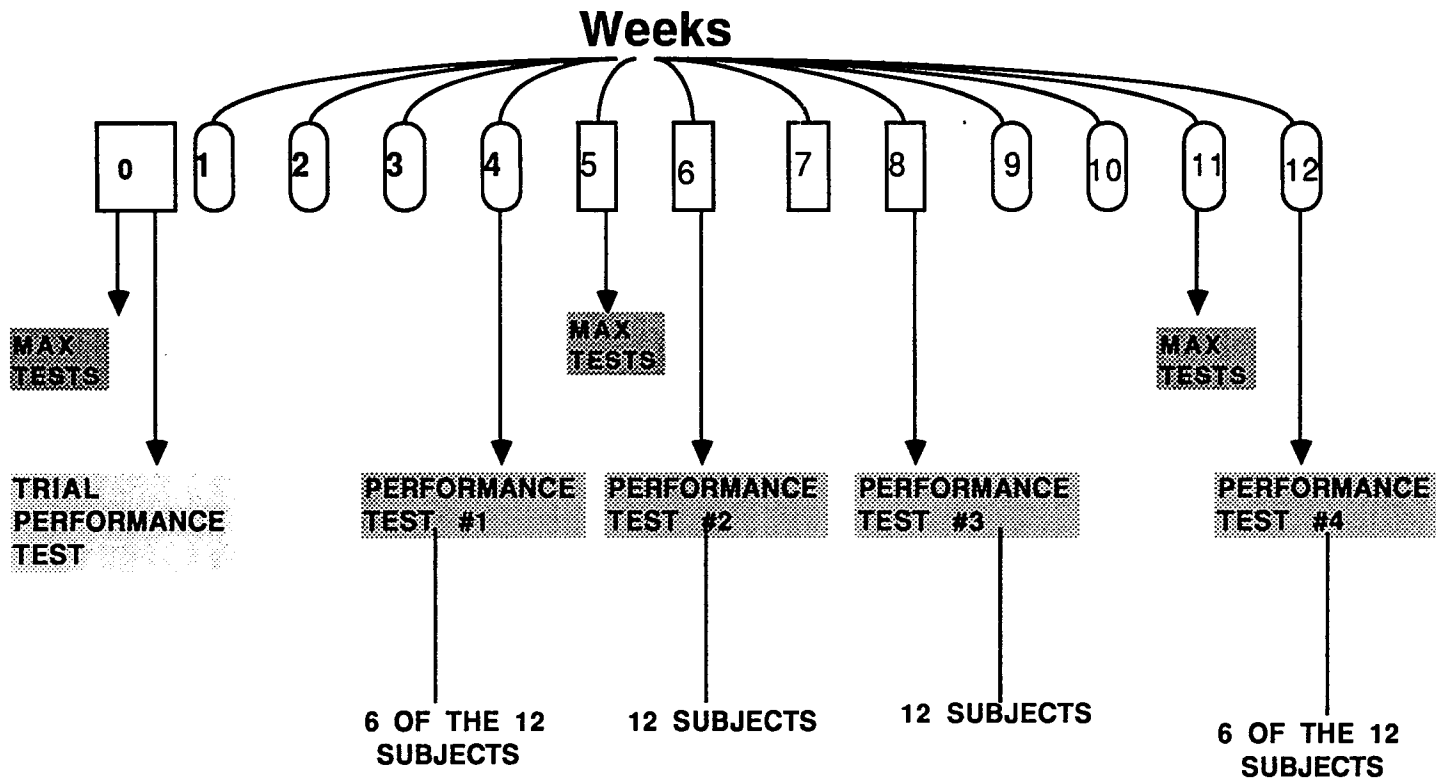
Cycling VO_{2max} 's were determined using a graded maximal exercise test performed on a modified BodyGuard cycle ergometer (BodyGuard 990, Jonas Ogaend A.s, Sandnes, Norway) with racing toe clips. The protocol began with a 5 minute warmup at a workload that produced a heart rate of

approximately 120 beats per minute. The workload, at 90 rpm, was increased every 2 minutes by 200 kpm. Subjects cycled until volitional fatigue was reached with the VO_2 max being identified as the highest VO_2 observed during any full minute of the test.

Running VO_2 max was determined using a graded maximal exercise test performed on a treadmill (Quinton Q55, Quinton Instrument Co., Seattle, WA). The protocol began with a 5 minute warmup at a workload that elicited a heart rate of approximately 120 beats per minute. Treadmill speed was increased every two minutes until 10 miles per hour was reached; after which the grade was increased 1% every two minutes (unless subjects indicated an increase of 2% was tolerable) until volitional fatigue was reached. VO_2 max was identified as the highest VO_2 recorded during any full minute of the test. Both a cycling and a running max test were performed on weeks 0, 5, and 11, totaling 6 max tests/subject (3 on the cycle and 3 on the treadmill) throughout the 12-week study (see Figure 1). These tests were performed to ensure that no significant changes in aerobic fitness level occurred throughout the 12 week study.

Oxygen and CO_2 were monitored throughout each max test using an automated gas analysis system that employed a two-way non-rebreathing valve (Rudolph Valve, Hans Rudolph, Inc., Kansas City, MO). Expired air volumes were measured using a turbine flow meter (Ametek, Thermo Instruments

FIGURE 1
Time Periods For Max Tests And Performance Tests During The 12-week Study.

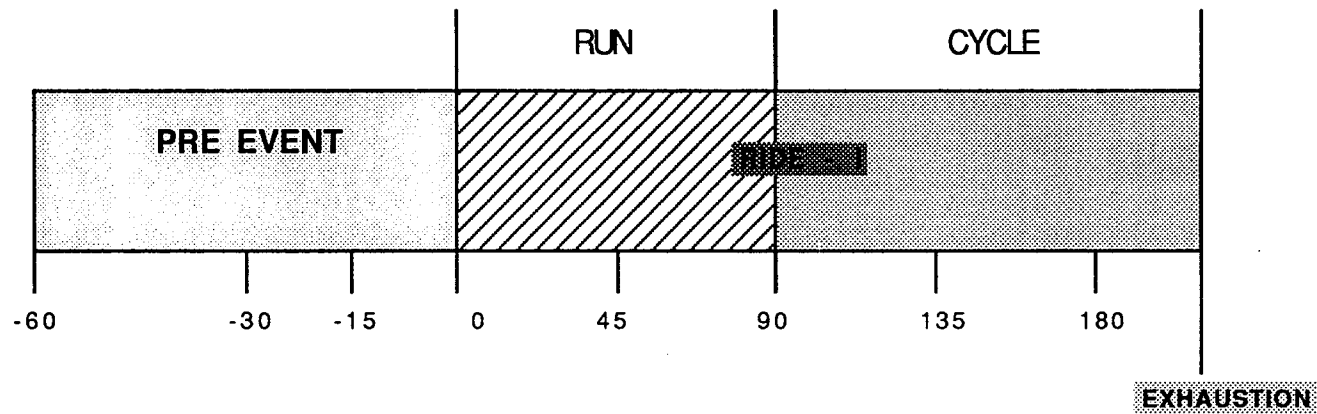


Division, Pittsburgh, PA). Expired gases were continuously sampled from a mixing chamber and analyzed for O₂ (Applied Electrochemistry S3-A) and CO₂ (Applied Electrochemistry SD-3A). Software (Ametek's Stress Program) and box interface (Ametek-71911KE) were programmed to calculate ventilation rate (L/min BTPS), VO₂ (ml/kg/min), VCO₂ (ml/kg/min), and the ventilatory exchange ratio (R) every 30 seconds. All values were corrected for STPD. Before each test, the O₂ and CO₂ were calibrated with gas (Air Products Industrial Gas Division) composed of 4.72 moles % carbon dioxide, 15.8 mole% oxygen. The balance consisted of nitrogen.

Trial Performance Test

The trial performance test consisted of running and cycling until exhaustion was reached (Figure 2). No blood was taken during this test. After an overnight fast, subjects reported to the laboratory and had four ECG electrodes (three leads and one ground) placed on their chest area. Subjects warmed up on both the treadmill (before the run portion) and cycle ergometer (before the ride portion) at their chosen workload for 5 minutes. After the warmup, subjects ran on the treadmill for 90 minutes at 70% of their running VO₂max, which was followed by 90 minutes of cycling at 70% of their cycling VO₂max (Figure 2). Percent VO₂max was determined within the first 10-20 minutes of each mode of exercise. At the end of the 180

FIGURE 2
Trial Performance Test



NOTE: Cold water was given ab libitum; and every 15 minutes subjects were encouraged to finish 8oz. of water, if they haven't already done so.

minutes, the workload on the cycle ergometer was increased every 5 minutes to elicit a workload corresponding to approximately 75%, 80%, and finally 85% of their cycling VO_{2max} ; which was maintained until exhaustion (ie, failure to maintain cycling cadence above 70 rpm).

This test was designed to help us select those subjects who could/would go longer, to assist in determining the workloads that approximately elicit 70% of their VO_{2max} for the four performance tests, to familiarize each of the subjects with the test protocol, and to identify any potential problems that might be associated with the collection process.

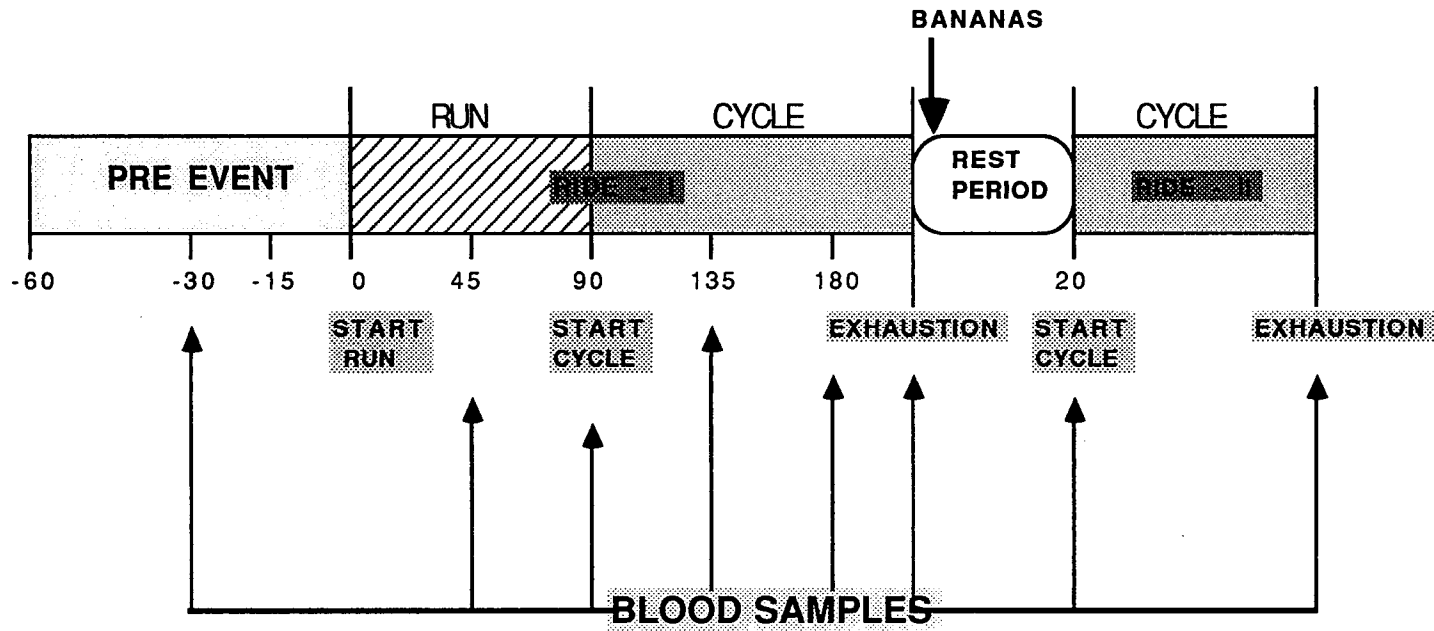
Cool water was provided for the subjects ad libitum, in water bottle containers. Each 15 minute period during exercise subjects were encouraged to drink 8 ounces of the water, if they had not already done so.

Endurance Performance Tests

The room temperature was maintained at approximately 23 degrees Centigrade by an individual air conditioning unit. After an overnight fast, subjects reported to the lab and their body weight was recorded (only shorts were worn). A catheter with a saline lock was placed in a forearm vein to facilitate repeated blood samples. The catheter was kept patent with normal, sterile saline. After resting for 30 minutes, four ECG electrodes (three leads and one ground)

were placed on the subjects chest area. Subjects warmed up on both the treadmill (before the run portion) and cycle ergometer (before the ride portion) at their chosen workload for 5 minutes. After the warmup, subjects ran on the treadmill for 90 minutes at 70% of their running VO_2 max, which was followed by 90 minutes of cycling at 70% of their cycling VO_2 max (Figure 3). Percent VO_2 max was determined within the first 10-20 minutes of each mode of exercise. At the end of the 180 minutes, the workload on the cycle ergometer was increased every 5 minutes by 65 kpm, designed to elicit a workload corresponding to approximately 75%, 80%, and finally 85% of their cycling VO_2 max; which was maintained until exhaustion (i.e., failure to maintain cycling cadence above 70 rpm). The first run-ride (of 180 minutes plus) was defined as RIDE-1. RIDE-1 was designed to bring each subject to a common, glycogen depleted endpoint. Each subject was required to perform at least 180 minutes of RIDE-1 in order for their data to be usable. The final increases in workload were designed to simulate biathlon/triathlon racing. Subjects were verbally encouraged toward the end of the ride. Heart rates were monitored throughout the ride to exhaustion using a 4-lead ECG. Subjects were encouraged to drink 3.4 ml/kg of body weight (ie, 8 oz. for a 70 kg subject) of cold water every 15 minutes during the tests in order to maintain hydration.

FIGURE 3
Performance Test



NOTE: Cold water was given ab libitum; and every 15 minutes subjects were encouraged to finish 8oz. of water, if they haven't already done so.

Ingestion Period. At the end of RIDE-1, subjects rested in a chair for 20 minutes during which they ingested one of the following (based on a 70 kg male): a) a blended mixture of 3 bananas and 4.4 oz. of water (equaling a total of 16 oz.), b) 3 whole bananas and 4.4 oz. of water (equaling a total of 16 oz.), or c) 16 oz. of an artificially flavored placebo drink. The placebo was designed to mimic the taste of an orange flavored carbohydrate drink for the purpose of letting the subjects believe they were getting some energy to help them continue the second part of the performance test. Except for the placebo treatment, carbohydrate intake was 1.1 g/kg of body weight. The total volume for each of the three treatments (placebo, slurried, and solid) was 6.8 ml/kg of body weight. For a 70 kg subject, this volume equaled 16 oz. The banana was chosen as the carbohydrate food in this study because it is essentially all carbohydrate; its carbohydrate concentration is relatively high (23% weight/volume); and, because bananas are a frequently eaten food of biathletes/triathletes during training and races of longer than 2 hours. The carbohydrate concentration of 3 bananas and 4.4 oz of water was approximately 16.4% (weight per volume).

RIDE-2. At the end of the 20 minute rest period, subjects began RIDE-2. Subjects warmed up for three minutes at a workload of their choice, and then cycled at 70% VO_2max

(i.e., the workload used on the cycle ergometer during RIDE-1) until exhaustion (Figure 2). A minimum of 10 minutes riding was required for the ride data to be considered usable. Water (3.4 ml/kg of body weight) was provided at 15 minute intervals throughout exercise.

Assessment of the effectiveness of these supplements (ie, solid or slurried bananas) on endurance performance was quantified by time to exhaustion from RIDE-2. Additionally, total work output of body weight was calculated by recording the number of pedal revolutions during RIDE-2, which was another measure of performance. Plasma glucose, lactate, and free fatty acids were measured throughout the entire exercise test session (i.e., 30 minutes before and at 45 and 90 minutes into the run, at 45 and 90 minutes into the bike, at exhaustion-1, at post recovery, and at exhaustion-2 of each performance test; Figure 3).

Incentives. Each subject was paid, in increasing amounts, for the completion of each of the four performance tests. In addition, monetary rewards were given to those subjects having the best performance (ie, longest time) for both RIDE-1 and RIDE-2 in each of the four performance tests. Thus, for each of the four performance tests, two monetary rewards were given. Subjects were unaware of the other subjects performance times until the end of each performance trial.

Blood Analysis

Blood was sampled from a sterile, radiopaque, teflon catheter (18 gauge, 1 inch; Angio-Set™; Deseret Medical Inc.; Sandy, Utah) inserted into an antecubital vein. The catheter was kept patent with sterile isotonic saline between each blood draw. Blood hematocrit (micro-hematocrit method) and hemoglobin concentrations (cyanmethemoglobin method [Drabkin & Austin, 1935]) were immediately determined in duplicate from each whole blood sample. Whole blood was then centrifuged for 15 minutes at 3000 rpm (at 10 degrees Centegrade), and placed into storage containers and stored at -70 degrees Centigrade for future analyses.

Plasma Glucose. Plasma glucose concentrations were determined using an enzymatic kit from Sigma Chemical Company (1988; 16-UV; St. Louis, MO). This method utilizes the enzymatic action of hexokinase and glucose-6-phosphate dehydrogenase on glucose to ultimately produce NADH. Glucose concentrations were determined in duplicate, from spectrophotometric changes in absorbance at 340 nm. Absorbancies were measured with the Bausch & Lomb Spectronic 2000 spectrophotometer. Detailed procedure steps for this assay are listed in Appendix E.

Plasma Free Fatty Acids. Plasma long-chain free fatty acid concentrations were determined using the colorimetric assay methods of Noma et al. (1973). This method is based on the formation of FFA-Cu soaps based on the following

three steps: extraction of FFA, formation of copper salts of FFA, and color reaction of FFA-Cu. The color solution was spectrophotometrically measured against a blank at 610 nm, using the Bausch & Lomb Spectronic 2000 spectrophotometer. Plasma free fatty acid concentrations were determined in duplicate. The standard curve was produced from various dilutions of 2mmol/L palmitic acid (Sigma Chemical Company, St. Louis, MO). Detailed procedure steps for this assay are listed in Appendix E.

Plasma Lactate. Plasma lactate concentrations were determined using an enzymatic kit by Sigma Chemical Company (1989; 826-UV) modified for use in plasma without deproteinization. The chemical reaction utilized in this method involves the subsequent oxidation of NADH to NAD⁺, which accompanies the reaction of the enzyme lactate dehydrogenase with lactate. The changes in the NADH concentration (reduced to oxidized form) are sensitive at the absorbance of light at 340 nm. Plasma lactate concentrations were determined in duplicate. Absorbancies were read using the Bausch & Lomb Spectronic 2000 spectrophotometer. Detailed procedure steps for this assay are listed in Appendix E.

Total Cholesterol. Total blood cholesterol was determined using the assay method described by Allain et al. (1974). The procedure generated two reactions: the oxidation of cholesterol following cholesterol ester

hydrolysis by the enzyme cholesterol esterase, and the reaction of hydrogen peroxide with 4-aminoantipyrine and p-hydroxybenzenesulfonate in the presence of peroxidase to yield a quinoneimine dye. Thus, the amount of quinoneimine dye is proportional to the amount of cholesterol.

Quantitative determination of the amount of quinoneimine dye was determined from spectrophotometric analysis at an absorbance of 500 nm. All concentrations were determined in duplicate. Absorbancies were determined using the Bausch & Lomb Spectronic 2000 spectrophotometer.

HDL Cholesterol. HDL blood cholesterol determination was based on selective precipitation of low density lipoproteins as described by Lopes-Virella et al. (1977). After precipitation, enzymatic determination of HDL cholesterol was accomplished following the methods of Allain et al. (1974), as stated previously. HDL2 and HDL3 were determined by a simple precipitation procedure as described by Lewis et al. (1982).

Serum Ferritin. Serum ferritin was determined in duplicate using a Ferrizyme kit from Abbott Laboratories (1985; 83-1156/R10). The procedure is based on two chemical reactions: an antibody-antigen reaction between ferritin and anti-ferritin coated beads, and a color producing reaction involving the addition of phenylenediamine to the ferritin/anti-ferritin coated bead complex. The intensity of the solution color is proportional to the amount of

ferritin present. The intensity was quantitatively measured against a standard curve, by the absorbance of light at 492 nm. The Bausch & Lomb Spectronic 2000 spectrophotometer was used to measure the absorbance.

Plasma Volume. All individual plasma substrate concentrations were adjusted for their individual plasma volume shifts, as determined by the methods of Dill & Costill (1974). These adjustments were performed before any statistical analysis. Substrate pre-event concentrations were considered the reference value (ie, plasma volume of 100%); all other concentrations were adjusted to the pre-event reference value. Detailed procedure steps for these calculations are listed in Appendix F.

Statistical Analysis

Statistical analyses were performed with the data collected pre and post RIDE-2 (ie., post recovery and exhaustion-2 time periods) which occurred after the first exhaustion period. Eight subjects completed all experimental tests. The two primary experimental interests were comparing the mean data from the combined carbohydrate treatments with that from the placebo treatment, as well as comparing the mean data from the two carbohydrate treatments with each other. Statistical comparisons were made using two one-way (separate analyses at each of the two time periods) analysis of variance in a randomized block

(subjects) design, with orthogonal polynomial contrasts to further subdivide the three treatment conditions. The level of statistical significance for all comparisons was set at $p=0.05$.

CHAPTER IV

RESULTS

Subjects

All of the 21 subjects had maintained the minimal milage requirements for running and cycling. Mean descriptive data on these 21 subjects are presented in Appendix G. All but one subject had VO_2 max values greater than 50 ml/kg/min (Appendix H). Of the 21 subjects, three had total blood cholesterol concentrations equal to or greater than 240 mg% (Appendix I), which is the high risk level identified by the National Cholesterol Education Program (1987). Two of the subjects were unable to achieve the large time commitment required. Four of the remaining 16 subjects were eliminated from the study due to their lower trial performance times (three were barely able to perform for the required three hours, and another subject had knee problems associated with the protocol). Thus, twelve subjects (ID numbers one through twelve) were chosen for this investigation.

As this study progressed, subject number 7 withdrew due to a severe back injury. Subject number 11 was forced to miss one of the treatment rides due to a cycling-running crash in a triathlon. And finally, subjects number 3 and 4

did not complete all parts of the four performance tests. These four subjects were eliminated from all statistical analysis. The number of subjects who completed all experimental tests in this investigation was eight. There are some missing data points for some variables due to blood draw equipment problems (i.e., clotting of the catheters) and increased discomfort from individual venipunctures of some subjects during the latter stages of the performance tests. Mean descriptive and preliminary performance data on these eight subjects are summarized in Table 2.

Statistical Analyses

Results from the statistical comparisons for each of the four primary variables of interest (plasma glucose, free fatty acids, lactate, and time to exhaustion-2) at the three time periods (i.e., exhaustion-1, post recovery and exhaustion-2) are presented in Tables 4-6, 9, 10, 12, and 13. Each table provides a statistical test of the overall treatment conditions for one variable at one time period, consisting of degrees of freedom, sums of squares, mean square, F-value, and the probability associated with that test statistic. Contrast-1 subdivides the overall treatment effect into a comparison between the placebo treatment data and that of the combined carbohydrate treatments. Contrast-2 subdivides the overall treatment effect into a comparison between the slurried and solid treatments.

Table 2

Mean Descriptive And Preliminary Performance Data For Experimental Subjects (n=8)

Variable	Mean	Standard Deviation	Minimum Value	Maximum Value
Age (yr)	25.7	3.1	22.0	31.0
Height (cm)	178.2	9.4	165.0	191.0
Weight (kg)	68.3	8.5	56.4	82.1
% Body Fat (hydrostatic)	9.8	4.4	4.3	15.7
Total Blood Cholesterol (mg%)	170.0	21.6	140.4	204.9
HDL Cholesterol (mg%)	55.5	13.5	40.0	84.2
Serum Ferritin (ng/ml)	56.8	32.4	15.2	94.2
Bike VO ₂ max (ml/kg/min)	67.1	7.4	55.8	76.4
Run VO ₂ max (ml/kg/min)	68.1	5.4	60.4	74.2
Trial Performance Time (min)	207.8	13.4	195.0	232.0

Contrast-1 of the plasma glucose data at the exhaustion-2 time period was the only statistically significant contrast of all the analyses (Table 4). It should be noted that the large number of statistical analyses performed with these data (a total of eight separate analyses) increases the likelihood of finding a statistical difference by chance alone.

Plasma Glucose Concentrations

Hypothesis 1: The ingestion of solid carbohydrate will result in higher mean plasma glucose concentration at the end of the second exhaustion period, than when the same carbohydrate is ingested in slurried form.

At the exhaustion-2 time period of RIDE-2, the mean (\pm S.E.M.) glucose concentration for the solid treatment (4.6 ± 0.2 mmol/L) was not statistically different from that of the slurried treatment (4.2 ± 0.3 mmol/L) (Table 3, 4 and Figure 4), resulting in the rejection of the first hypothesis. The mean glucose concentration for the combined carbohydrate treatments at the exhaustion-2 time period (4.4 ± 0.2 mmol/L) was statistically higher ($p > 0.0094$) than that of the placebo treatment (3.5 ± 0.3 mmol/L) at the same time period (Table 4). Mean glucose concentrations at the post recovery time period were not statistically different from each other (Table 5).

Table 3

Mean Plasma Glucose Concentrations (mmol/L) During The Performance Tests For Each Treatment (mean \pm S.E.M.)

	PLACEBO (n)	SLURRIED (n)	SOLID (n)
RESTING	4.9 \pm 0.1 (8)	4.9 \pm 0.2 (8)	4.7 \pm 0.1 (8)
45 RUN	5.3 \pm 0.4 (5)	4.9 \pm 0.1 (4)	4.5 \pm 0.3 (7)
90 RUN	4.9 \pm 0.1 (8)	4.9 \pm 0.2 (6)	4.9 \pm 0.3 (8)
45 BIKE	4.5 \pm 0.3 (4)	4.3 \pm 0.4 (3)	4.0 \pm 0.1 (6)
90 BIKE	4.2 \pm 0.2 (2)	4.0 \pm 0.4 (3)	4.1 \pm 0.1 (2)
EXH-1	3.9 \pm 0.3 (8)	4.5 \pm 0.4 (8)	3.6 \pm 0.3 (7)

Treatments Given			

POST RECOVERY	4.3 \pm 0.3 (7)	5.0 \pm 0.3 (7)	4.7 \pm 0.3 (8)
EXH-2	3.5 \pm 0.3 (8)*	4.2 \pm 0.3 (8)	4.6 \pm 0.2 (8)

* statistically lower ($p < 0.0094$) than the combined carbohydrate treatments (see Table 4).

Table 4

Statistical Analyses On Plasma Glucose Data Collected At The Exhaustion-2 Time Period

Source	DF	Sum of Squares	Mean Square	F-Value	Pr > F
Subjects	7	6.0439	0.8634	---	---
Treatment	2	4.8417	2.4208	5.12	0.0214
Contrast-1	1	4.2811	4.2811	9.06	0.0094
Contrast-2	1	0.5606	0.5606	1.19	0.2945
Error	14	6.6172	0.4727	---	---
Total	23	17.5028	---	---	---

NOTE: **Contrast-1** subdivides the overall treatment effect into a comparison between the placebo treatment data and that of the combined carbohydrate treatments.

Contrast-2 subdivides the overall treatment effect into a comparison between the slurried treatment data and that of the solid treatment.

FIGURE 4
MEAN PLASMA GLUCOSE CONCENTRATIONS AT
EXHAUSTION-1, POST RECOVERY & EXHAUSTION-2

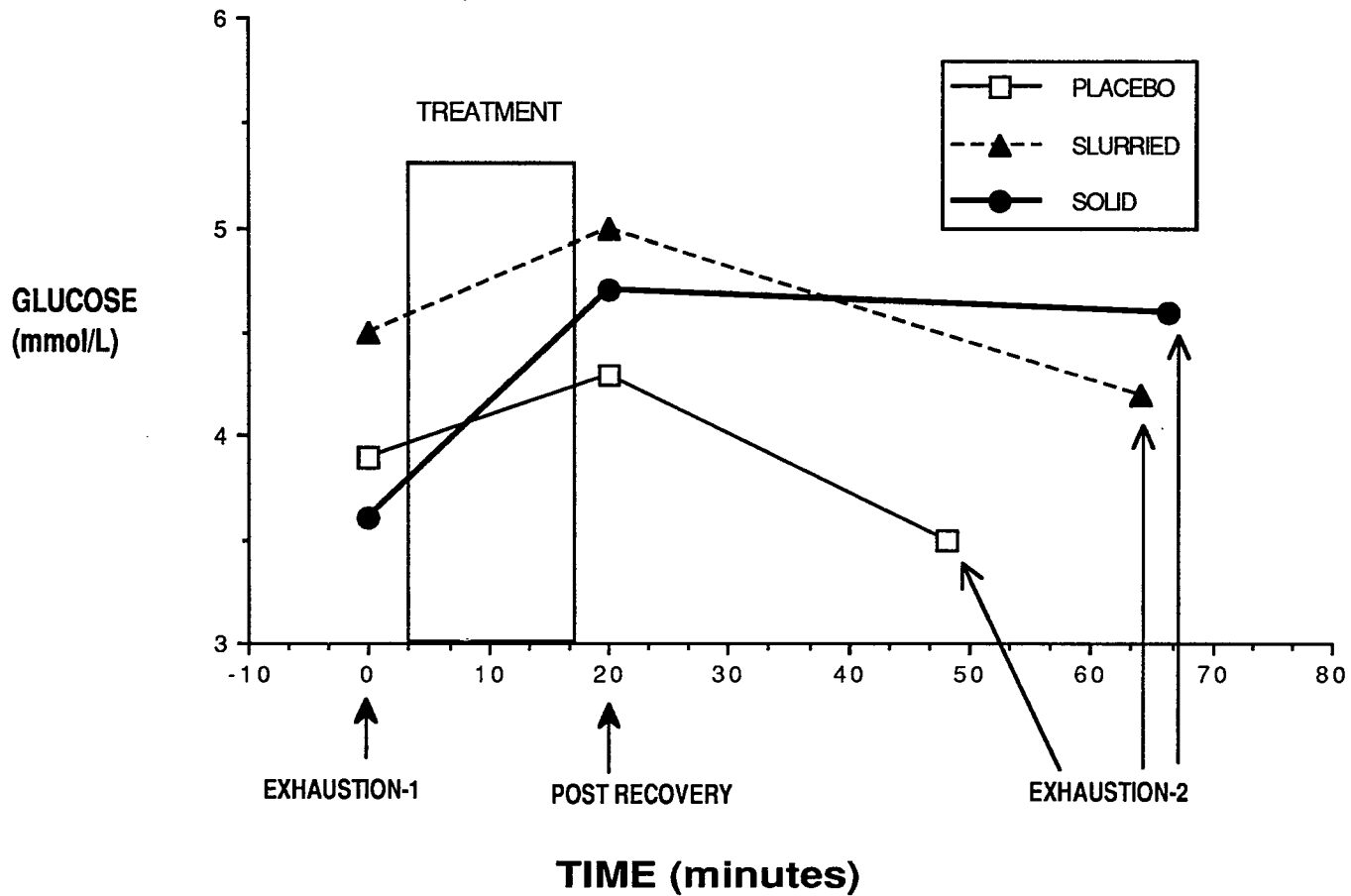


Table 5

Statistical Analyses On Plasma Glucose Data Collected At The Post Recovery Time Period

Source	DF	Sum of Squares	Mean Square	F-Value	Pr > F
Subjects	7	5.5027	0.7861	---	---
Treatment	2	1.9019	0.9509	1.35	0.2958
Contrast-1	1	1.6204	1.6204	2.30	0.1551
Contrast-2	1	0.3701	0.3701	0.53	0.4823
Error	12	8.4487	0.7041	---	---
Total	21	15.6628	---	---	---

NOTE: **Contrast-1** subdivides the overall treatment effect into a comparison between the placebo treatment data and that of the combined carbohydrate treatments.

Contrast-2 subdivides the overall treatment effect into a comparison between the slurried treatment data and that of the solid treatment.

Time To Exhaustion-2

Hypothesis 2: The ingestion of solid carbohydrate will increase the time to exhaustion during the second exhaustion ride, as compared to the same carbohydrate ingested in slurried form.

Mean performance test times from RIDE-2 were 28.2 ± 4.3 , 44.1 ± 8.7 , and 46.4 ± 13.2 for the placebo, slurried, and solid treatments, respectively (Figure 5). These mean performance times were not statistically different from each other (Table 6), resulting in the rejection of the second hypothesis.

Individual performance times from RIDE-2 are listed in Table 7. These times range from 13.0 to 124.2 minutes. Individual performance test times for RIDE-1 are listed in Appendix J. Total mean performance test time for RIDE-1 was 217.3 ± 7.1 minutes from all three treatment groups. The mean trial performance test time (\pm S.E.M.) was 207.8 ± 4.7 minutes.

Plasma Free Fatty Acid Concentrations

Mean plasma free fatty acid concentrations from RIDE-2 for each treatment are reported in Table 8 (Figure 6). Mean plasma free fatty acid concentrations were not statistically different among treatments at both the post recovery and exhaustion-2 time periods (Table 9 and 10).

FIGURE 5
RIDE-2 Mean Time (+ S.E.M.) To Exhaustion

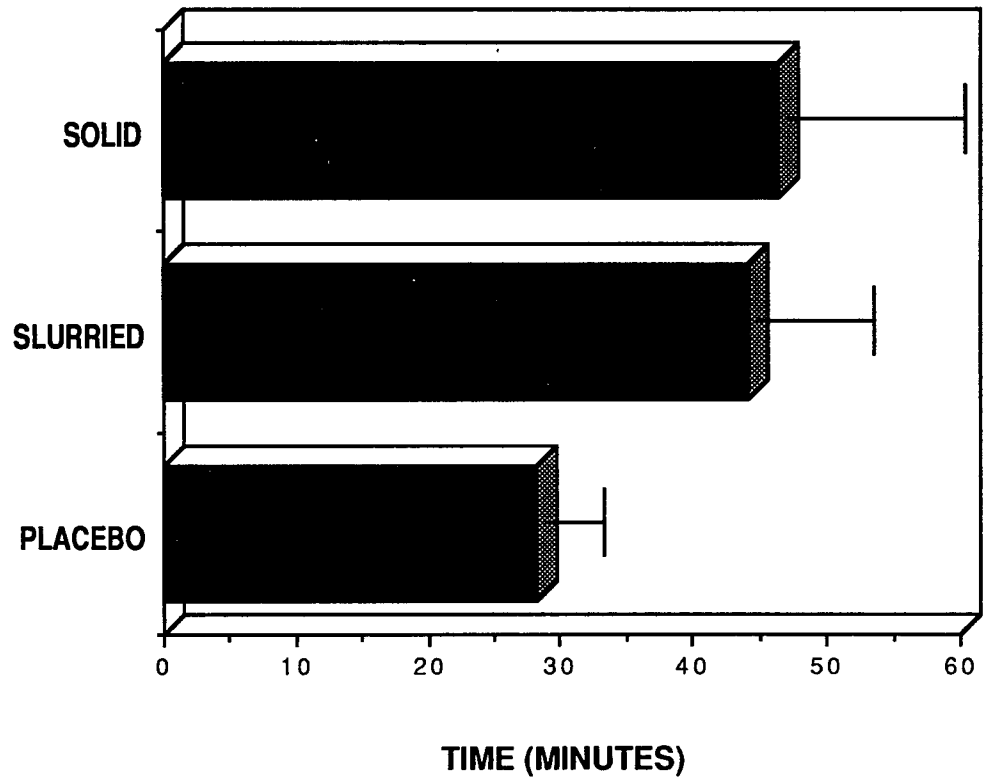


Table 6

Statistical Analyses Of The Exhaustion-2 Riding Times

Source	DF	Sum of Squares	Mean Square	F-Value	Pr > F
Subjects	7	7366.5136	1052.3590	---	---
Treatment	2	1566.1265	783.0633	1.44	0.2696
Contrast-1	1	1546.3240	1546.3240	2.85	0.1137
Contrast-2	1	19.8025	19.8025	0.04	0.8513
Error	14	7605.5865	543.2562	---	---
Total	23	16538.227	---	---	---

NOTE: **Contrast-1** subdivides the overall treatment effect into a comparison between the placebo treatment data and that of the combined carbohydrate treatments.

Contrast-2 subdivides the overall treatment effect into a comparison between the slurried treatment data and that of the solid treatment.

Table 7

Individual Performance Test Times (minutes) from Each Treatment From RIDE-2

ID	PLACEBO	SLURRIED	SOLID
1	15.1	45.1	20.0
2	15.1	62.4	31.0
5	50.7	92.3	78.5
6	31.5	42.2	27.2
8	30.1	26.5	46.3
9	38.1	16.0	30.5
10	20.0	45.1	124.2
12	25.3	23.6	13.0
Mean±S.E.M.	28.2±4.3	44.1±8.7	46.4±13.2
Range	15.1-50.7	16.1-92.3	13.0-124.2

Table 8

Mean Plasma Free Fatty Acids Concentrations (mmol/L) During The Performance Tests For Each Treatment (mean \pm S.E.M.)

	PLACEBO (n)	SLURRIED (n)	SOLID (n)
RESTING	0.27 \pm 0.05 (8)	0.25 \pm 0.03 (8)	0.25 \pm 0.04 (8)
45 RUN	0.37 \pm 0.08 (5)	0.45 \pm 0.06 (4)	0.33 \pm 0.10 (7)
90 RUN	0.72 \pm 0.10 (8)	0.63 \pm 0.10 (6)	0.42 \pm 0.06 (8)
45 BIKE	0.52 \pm 0.11 (3)	0.49 \pm 0.21 (2)	0.46 \pm 0.05 (6)
90 BIKE	0.60 \pm 0.05 (2)	0.66 \pm 0.34 (2)	0.58 \pm 0.16 (2)
EXH-1	0.90 \pm 0.09 (7)	0.88 \pm 0.13 (8)	0.92 \pm 0.05 (7)

Treatments Given			

POST RECOVERY	0.85 \pm 0.08 (7)	0.96 \pm 0.15 (7)	0.80 \pm 0.10 (8)
EXH-2	0.92 \pm 0.06 (8)	0.88 \pm 0.13 (8)	0.83 \pm 0.09 (8)

FIGURE 6
MEAN PLASMA FFA CONCENTRATIONS AT
EXHAUSTION-1, POST RECOVERY & EXHAUSTION-2

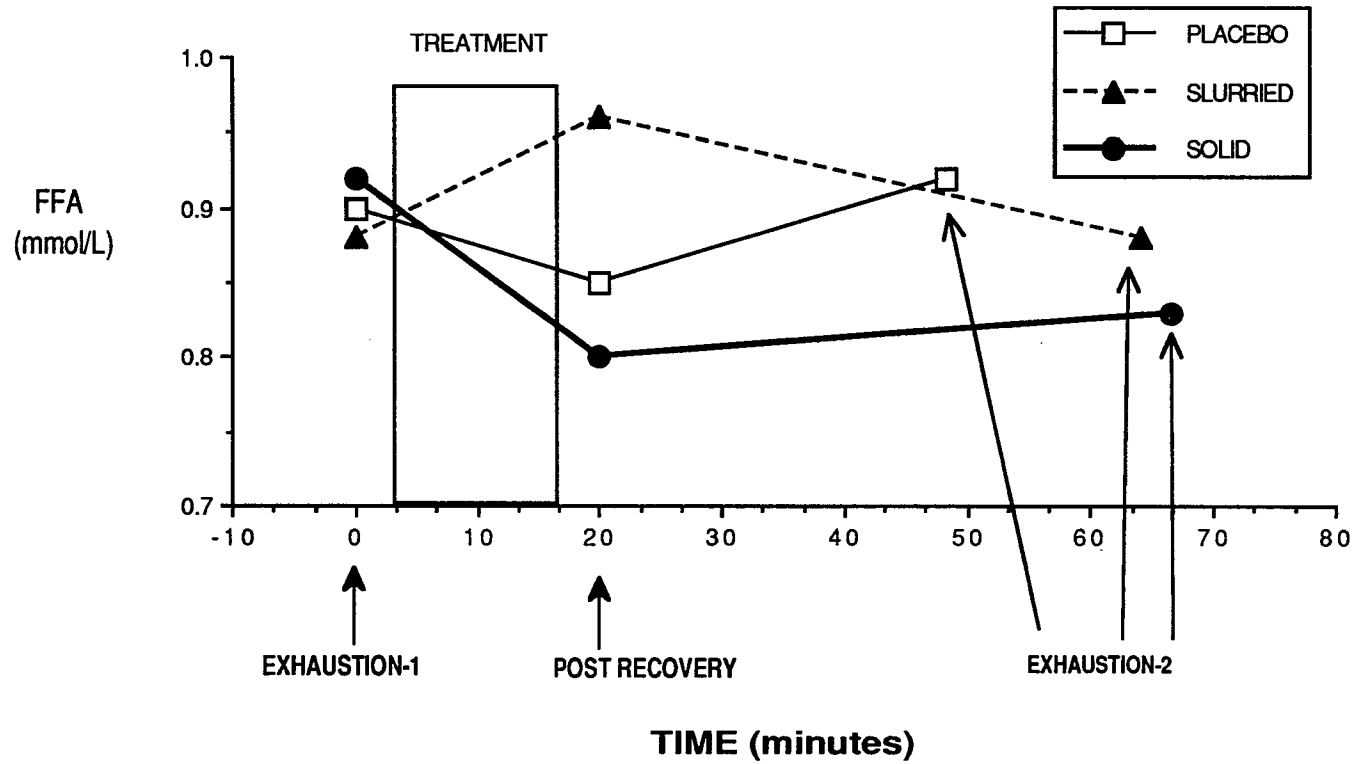


Table 9

Statistical Analyses On Plasma Free Fatty Acid Data
Collected At The Post Recovery Time Period

Source	DF	Sum of Squares	Mean Square	F-Value	Pr > F
Subjects	7	1.3063	0.1866	5.42	0.0054
Treatment	2	0.1133	0.0567	1.65	0.2335
Contrast-1	1	0.0079	0.0079	0.23	0.6390
Contrast-2	1	0.1014	0.1014	2.95	0.1118
Error	12	0.4130	0.0344	---	---
Total	21	1.8065	---	---	---

NOTE: **Contrast-1** subdivides the overall treatment effect into a comparison between the placebo treatment data and that of the combined carbohydrate treatments.

Contrast-2 subdivides the overall treatment effect into a comparison between the slurried treatment data and that of the solid treatment.

Table 10

Statistical Analyses On Plasma Free Fatty Acid Data
Collected At The Exhaustion-2 Time Period

Source	DF	Sum of Squares	Mean Square	F-Value	Pr > F
Subjects	7	0.5759	0.0823	---	---
Treatment	2	0.0307	0.0153	0.26	0.7773
Contrast-1	1	0.0149	0.0149	0.25	0.6250
Contrast-2	1	0.0139	0.0139	0.23	0.6370
Error	13	0.7767	0.0597	---	---
Total	22	1.3818	---	---	---

NOTE: **Contrast-1** subdivides the overall treatment effect into a comparison between the placebo treatment data and that of the combined carbohydrate treatments.

Contrast-2 subdivides the overall treatment effect into a comparison between the slurried treatment data and that of the solid treatment.

Plasma Lactate Concentrations

Mean plasma lactate concentrations from RIDE-2 for each treatment are summarized in Table 11 (Figure 7). Mean plasma lactate concentrations were not statistically different among treatments at both the post recovery and exhaustion-2 time periods (Tables 12 and 13). The mean lactate concentrations range from 0.9 to 2.7 mmol/L. The normal resting range for plasma lactate concentration is 0.5-2.0 mmol/L (Young, 1987).

Plasma Volume Shifts

Mean plasma volume shifts throughout RIDE-1 and RIDE-2 for each treatment are noted in Table 14 (Figure 8). Mean plasma volume shifts at the exhaustion-2 time period were -12 ± 2 , -5 ± 3 , and $-10 \pm 3\%$ for the placebo, slurried, and solid treatments, respectively.

Body Weight Changes

Mean body weight changes for the placebo, slurried, and solid experiment trials were -1.3, 0.6, -4.9 kg, respectively. Changes in mean body weight for each of the three performance tests are presented in Table 15 and Figure 9.

Table 11

Mean Plasma Lactate Concentrations (mmol/L) During The Performance Tests For Each Treatment (mean \pm S.E.M.)

	PLACEBO (n)	SLURRIED (n)	SOLID (n)
RESTING	1.6 \pm 0.2 (8)	1.6 \pm 0.2 (8)	2.0 \pm 0.2 (8)
45 RUN	1.0 \pm 0.1 (5)	1.4 \pm 0.3 (4)	1.3 \pm 0.1 (7)
90 RUN	1.1 \pm 0.2 (8)	1.1 \pm 0.1 (6)	1.5 \pm 0.3 (8)
45 BIKE	1.0 \pm 0.2 (4)	1.4 \pm 0.4 (3)	1.5 \pm 0.2 (6)
90 BIKE	0.9 \pm 0.1 (2)	1.3 \pm 0.3 (3)	1.2 \pm 0.4 (2)
EXH-1	1.9 \pm 0.3 (7)	2.2 \pm 0.3 (8)	2.5 \pm 0.6 (7)

Treatments Given			

POST RECOVERY	1.5 \pm 0.3 (7)	2.0 \pm 0.5 (6)	2.7 \pm 0.2 (8)
EXH-2	1.7 \pm 0.3 (8)	1.4 \pm 0.2 (7)	1.9 \pm 0.2 (8)

FIGURE 7
MEAN PLASMA LACTATE CONCENTRATION AT
EXHAUSTION-1, POST RECOVERY & EXHAUSTION-2

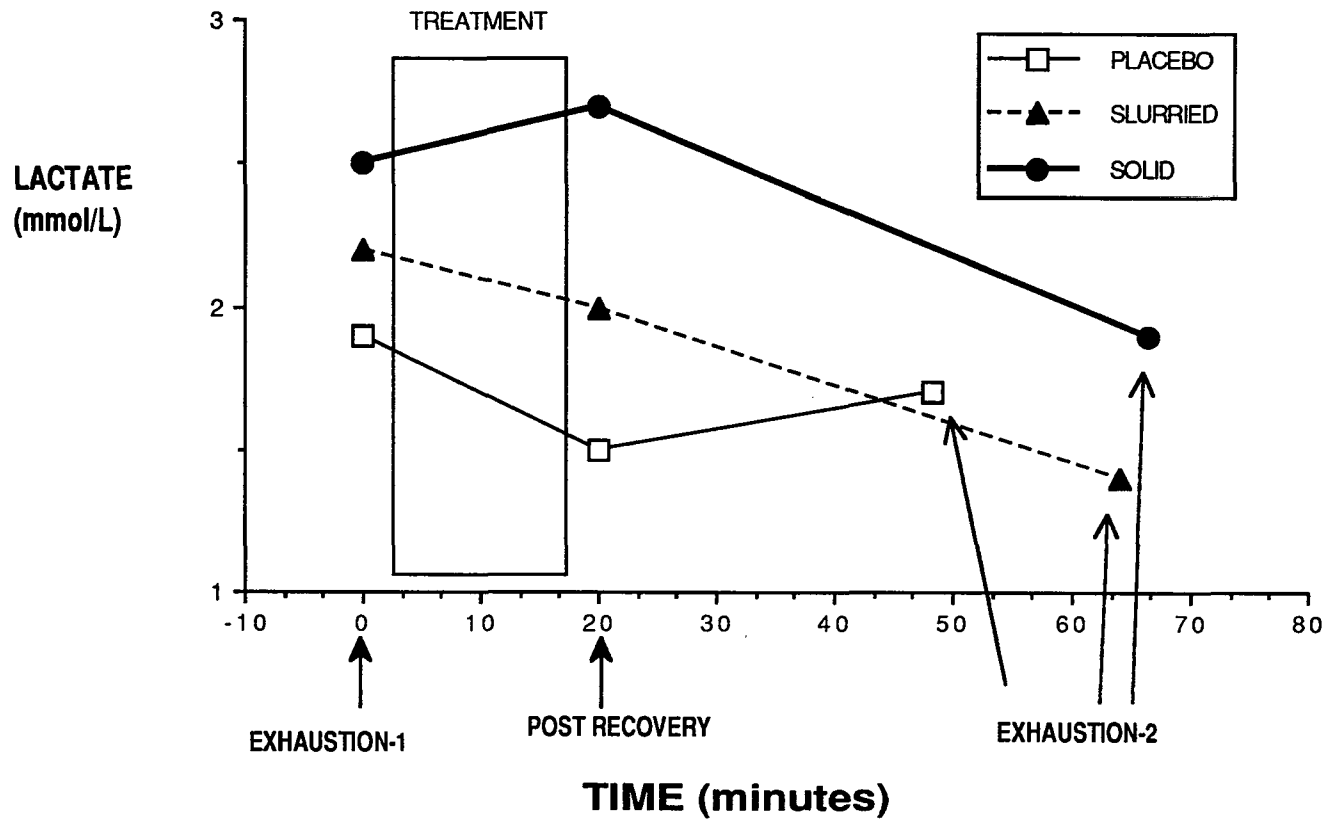


Table 12

Statistical Analyses On Plasma Lactate Data Collected At The
Post Recovery Time Period

Source	DF	Sum of Squares	Mean Square	F-Value	Pr > F
Subjects	7	5.3239	0.7605	---	---
Treatment	2	5.3708	2.6854	3.21	0.0799
Contrast-1	1	3.5911	3.5911	4.29	0.0626
Contrast-2	1	1.1442	1.1442	1.37	0.2671
Error	11	9.2086	0.8371	---	---
Total	20	19.4954	---	---	---

NOTE: **Contrast-1** subdivides the overall treatment effect into a comparison between the placebo treatment data and that of the combined carbohydrate treatments.

Contrast-2 subdivides the overall treatment effect into a comparison between the slurried treatment data and that of the solid treatment.

Table 13

Statistical Analyses On Plasma Lactate Data Collected At The
Exhaustion-2 Time Period

Source	DF	Sum of Squares	Mean Square	F-Value	Pr > F
Subjects	7	2.4278	0.3468	---	---
Treatment	2	0.9156	0.4578	1.04	0.3819
Contrast-1	1	0.0005	0.0005	0.00	0.9724
Contrast-2	1	0.9145	0.9145	2.07	0.1736
Error	13	5.7357	---	---	---
Total	22	9.1492	---	---	---

NOTE: **Contrast-1** subdivides the overall treatment effect into a comparison between the placebo treatment data and that of the combined carbohydrate treatments.

Contrast-2 subdivides the overall treatment effect into a comparison between the slurried treatment data and that of the solid treatment.

Table 14

Percent Plasma Volume Shifts (%) * During The Performance Tests For Each Treatment
(mean \pm S.E.M.)

	PLACEBO (n)	SLURRIED (n)	SOLID (n)
RESTING	0	0	0
45 RUN	-4 \pm 5 (5)	-4 \pm 3 (4)	-8 \pm 3 (7)
90 RUN	-6 \pm 2 (8)	-2 \pm 3 (6)	-7 \pm 6 (8)
45 BIKE	-13 \pm 2 (4)	-5 \pm 1 (3)	-13 \pm 4 (6)
90 BIKE	-17 \pm 1 (2)	-2 \pm 6 (3)	-12 \pm 1 (2)
EXH-1	-14 \pm 2 (8)	-6 \pm 2 (8)	-12 \pm 3 (7)

Treatments Given			

POST RECOVERY	-6 \pm 5 (7)	-1 \pm 2 (7)	-9 \pm 3 (8)
EXH-2	-12 \pm 2 (8)	-5 \pm 3 (8)	-10 \pm 3 (8)

* percentages determined from hemoglobin and hematocrit concentrations (Dill & Costill, 1974).

FIGURE 8
MEAN PLASMA VOLUME SHIFTS THROUGHOUT
EACH PERFORMANCE TEST

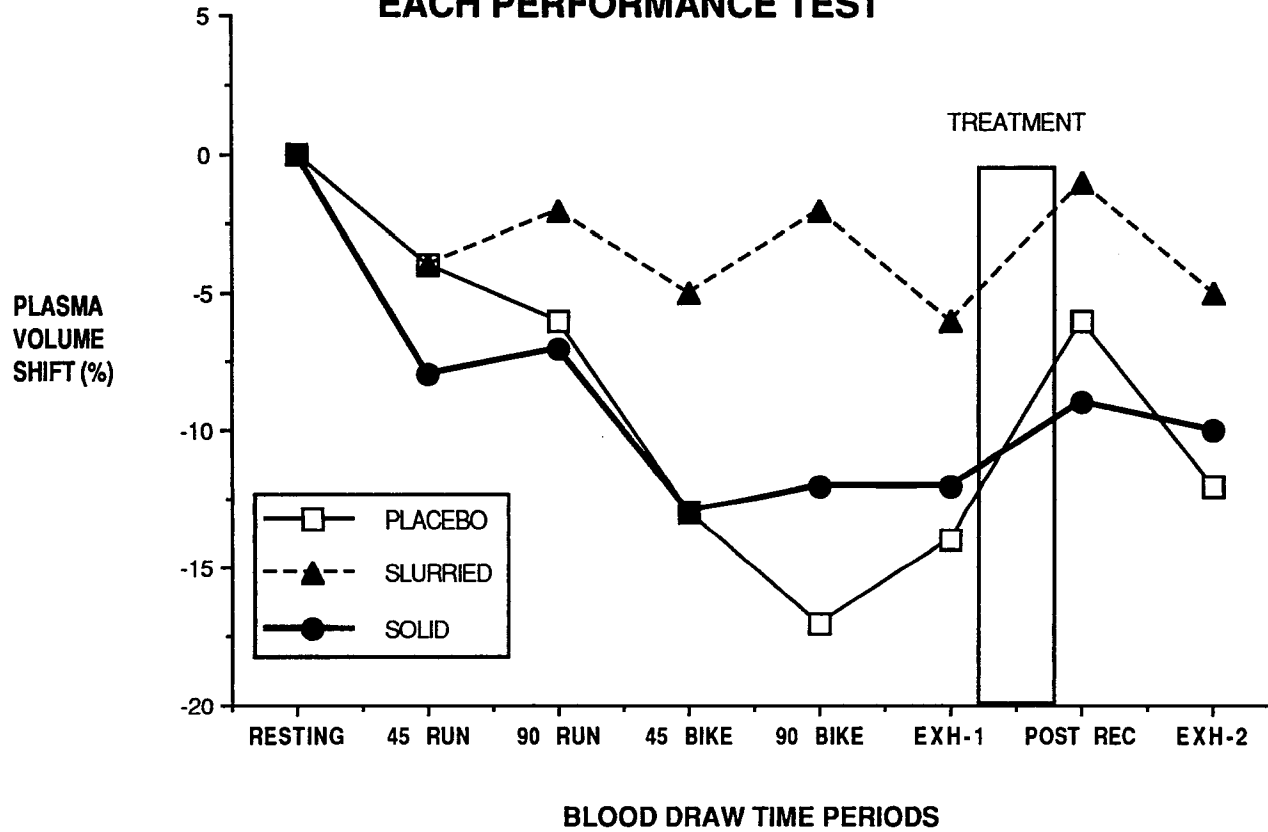


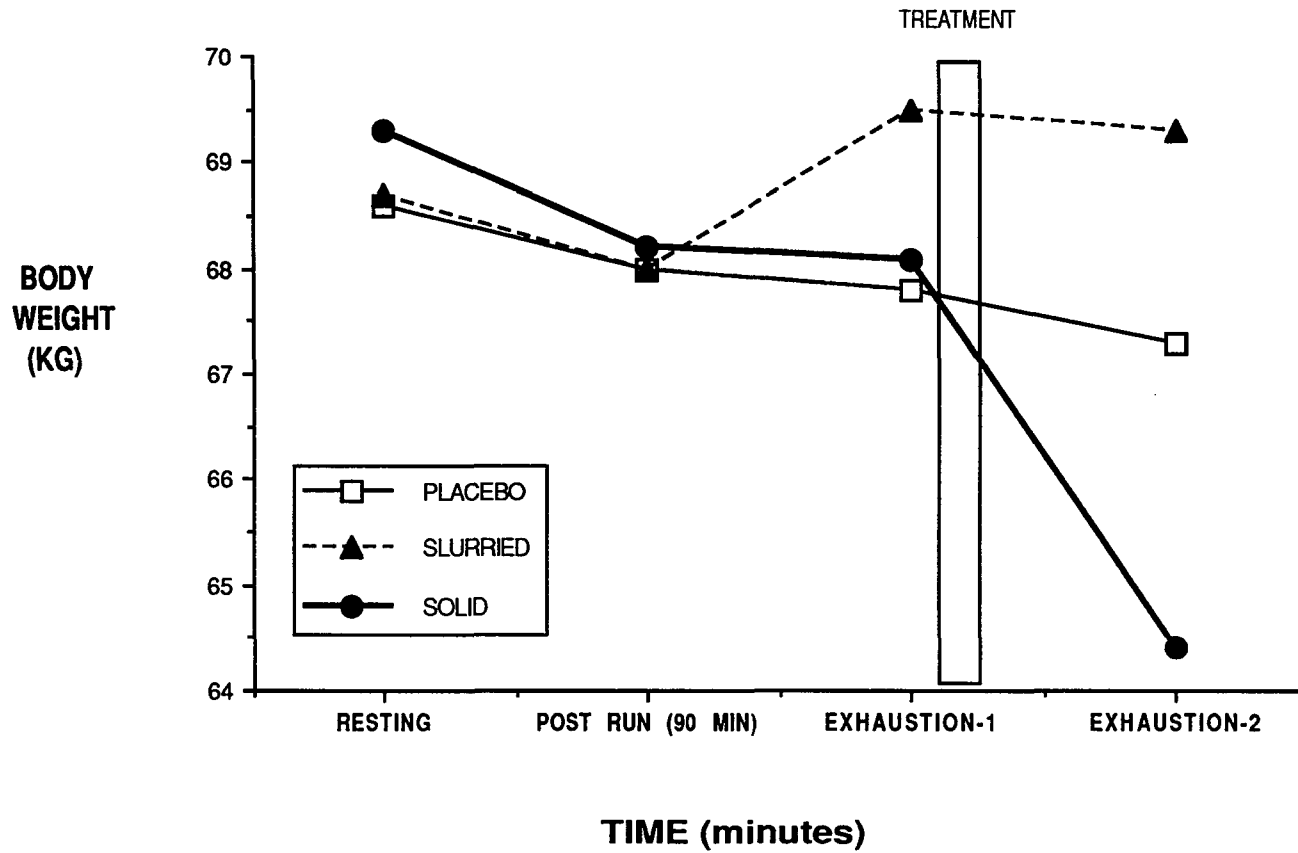
Table 15

Changes In Mean Body Weight (kg) During The Performance Tests For Each Treatment
(mean±S.E.M.)

	PLACEBO (n)	SLURRIED (n)	SOLID (n)
RESTING	68.6±3.1 (8)	68.7±3.1 (8)	69.3±3.1 (8)
90 MINUTE RUN	68.0±3.1 (8)	68.0±3.0 (8)	68.2±3.1 (8)
EXHAUSTION-1	67.8±3.1 (8)	69.5±3.0 (7)	68.1±3.2 (8)
EXHAUSTION-2	67.3±3.5 (7)	69.3±4.2 (5)	64.4±2.6 (6)

TOTAL CHANGE	-1.3	+0.6	-4.9

FIGURE 9
CHANGES IN MEAN BODY WEIGHTS THROUGHOUT
EACH PERFORMANCE TEST



Run & Bike VO₂max Values

Mean run and bike VO₂max values at weeks 0, 5, and 11 were 68.1±1.9, 68.0±2.3, and 69.5±2.1 ml/kg/min for the run; and 67.1±2.6, 67.7±2.5, and 66.9±2.0 ml/kg/min for the bike, respectively (Table 16). These means were not significantly different either between modalities or across time. Individual VO₂max values are presented in Appendix K. Individual VO₂max values range from 55.8 to 78.2 ml/kg/min on the cycle ergometer and treadmill, respectively.

Table 16

Mean (+ S.E.M.) Run And Bike VO₂max Values (ml/kg/min) At Weeks 0, 5, And 11 (n=8)

	RUN-VO ₂ max	BIKE-VO ₂ max
WEEK ZERO	68.1 ± 1.9	67.1 ± 2.6
WEEK FIVE	68.0 ± 2.3	67.7 ± 2.5
WEEK ELEVEN	69.5 ± 2.1	66.9 ± 2.0

CHAPTER V
DISCUSSION

This discussion has been organized with a focus on the results of this dissertation as they pertain to each hypothesis. Comparisons of these data with findings previously reported in the literature, a discussion of the limitations of this dissertation research, and recommendations for future research are also provided.

Blood Glucose

Hypothesis 1: The ingestion of solid carbohydrate will result in higher mean plasma glucose concentration at the end of the second exhaustion period, than when the same carbohydrate is ingested in slurried form.

There was no statistical difference between the solid and slurried mean glucose concentrations at the second exhaustion time period, resulting in the rejection of the first hypothesis. Several studies have demonstrated increased blood glucose concentration as a result of ingesting both liquid and solid carbohydrates during endurance exercise, as compared to water alone. The results of this dissertation are in agreement with these findings, i.e., the mean glucose concentration at the exhaustion-2

time period for the combined solid and slurried carbohydrate treatments (4.4 ± 0.2 mmol/L) was statistically higher than that for the placebo treatment (3.5 ± 0.3 mmol/L).

In resting subjects, the ingestion of 60 grams of carbohydrate (plus 1.5 grams of total fiber) as whole or pureed apples (9, 37, and 14 grams of glucose, fructose, and sucrose, respectively) caused rises in blood glucose and insulin concentrations that peaked between 20-30 minutes after the start of the ingestion period (Haber, et al., 1977). No statistical differences were observed in the blood glucose concentrations between the solid or pureed apple treatments until 50 minutes after the start of the ingestion period; the pureed treatment resulted in a lower mean glucose concentration than the solid treatment. This difference was primarily attributed to the statistically higher insulin response in the pureed apples 30 minutes after the start of the ingestion period.

Ingesting carbohydrate during endurance exercise at an intensity of 70% of the subjects predetermined VO_{2max} prevents the usual hyperinsulinemic response following carbohydrate ingestion, while also preventing the usual decline of the already low plasma insulin concentration seen during exercise performed in a fasted state (Coyle, et al., 1983; Coyle, et al., 1986; Coggan and Coyle, 1987; Coggan and Coyle, 1989). Thus, insulin mediated disposal of plasma glucose appears to be minimal. Increased glucose uptake

into the muscle is more likely a possibility of the disposition of the ingested glucose into the glycogen depleted fibers, via mechanism (s) not completely elucidated (Gollnick, et al., 1981).

The rate of gastric emptying for solid or liquid foods influences the rate of absorption. In resting subjects, solid food emptied from the stomach more slowly than liquid foods (Malagelada, 1977). Ingesting solid bananas results in lumps of the fruit being presented to the stomach. These lumps of banana are, with time, reduced in size and ultimately introduced to the small intestines. The digestive enzymes required to further breakdown the banana into its simplest absorptive components has limited access to all the nutritive constituents in these banana lumps, suggesting that the digestion of solid carbohydrate food is slower when compared to that of liquid carbohydrate food (Bolton et al., 1981). However, no differences were observed in the rate at which blood glucose concentration peaked from the ingestion of solid or pureed carbohydrate (Bolton et al., 1981), suggesting that the delay in gastric emptying that accompanies the ingestion of solid carbohydrate foods occurs only after significant absorption has taken place (i.e., after 60 minutes in resting subjects). Since little is known about the rate of gastric emptying of solid foods ingested during exercise, its consequential effect upon absorption, and the peak time in

blood glucose elevation, further investigation is required.

In a similarly designed study by Coggan & Coyle (1987), seven male cyclists/triathletes cycled at 70% of their VO_2 max until exhaustion was reached (termed BOUT-1). They received one of three treatments during a 20 minute rest period, after which they cycled to exhaustion again (BOUT-2). The three treatments included a placebo drink (420ml), a carbohydrate drink (50% solution; 420ml; equivalent to 3 grams of carbohydrate/kg of body weight), and a euglycemic infusion clamp (which maintained plasma glucose concentrations at 5.1 mmol/L throughout BOUT-2). Mean (\pm S.E.M.) post recovery and exhaustion-2 glucose concentrations from both this dissertation and from the study conducted by Coggan and Coyle (1987) are reported below in tabular form:

TREATMENT	MEAN GLUCOSE (mmol/L)			
	Post Recovery Murdoch	Recovery Coggan	Exhaustion-2 Murdoch	Exhaustion-2 Coggan
Placebo	4.3 \pm 0.3	3.9 \pm 0.3	3.5 \pm 0.3	3.1 \pm 0.2
Slurried/Liq.	5.0 \pm 0.3	5.0 \pm 0.6	4.2 \pm 0.3	3.9 \pm 0.3
Solid	4.7 \pm 0.3	---	4.6 \pm 0.2	---
Infused	---	4.6 \pm 0.4	---	5.1 \pm 0.1

As a result of RIDE-2 in the present study, the mean plasma glucose concentration from the placebo group decreased significantly from 4.3 to 3.5 mmol/L at post-recovery and exhaustion-2 time periods, respectively. This mean decrease of approximately 0.8 mmol/L is similar to that

reported by Coggan and Coyle (1987) after the placebo treatment. Mean plasma glucose concentration also fell 0.8 mmol/L in the slurried carbohydrate treatment group. Mean plasma glucose concentrations with the solid treatment were 4.7 and 4.6 mmol/L at post recovery and exhaustion-2 time periods, respectively. Thus, the ingestion of solid bananas after RIDE-1 prevented the rapid decline in plasma glucose that was observed with the placebo and slurried treatments.

The question of how much carbohydrate should be ingested in order to maintain euglycemia (or to avoid hypoglycemia) is, in part, dependent upon the rate of absorption for that carbohydrate. Glucose infusion, for the purpose of maintaining blood glucose within a normal range, during exhaustive cycling at 70% VO_2max required an infusion rate of 1.1 grams of glucose/minute (Coggan and Coyle, 1987). In the latter stages of exhaustive cycling at a similar intensity, carbohydrate oxidation rate is estimated to be 2 grams of carbohydrate/minute (Coyle et al., 1986). Whether the ingestion of a particular amount of solid carbohydrate food can supply 1.1 grams of glucose/minute to the circulation over a prolonged period of time and without compromising hydration, remains to be determined.

The type of carbohydrate ingested influences its digestion and metabolic fate. One average size banana consist of approximately 8.6, 8.8, and 2.1 grams of fructose, glucose, and sucrose, respectively. Thus,

essentially half of the available carbohydrate is fructose. Fructose is primarily phosphorylated in the liver (and to a small extent in the intestines and kidneys) by the enzyme fructokinase, which has a high affinity for fructose (Harper's Biochemistry, 1988). Fructose may be further degraded via the glycolytic pathway or, as is more often the case, converted to glucose. The absorption rate of fructose from the small intestines is slower than that of glucose, with 70-90% of the ingested fructose entering the portal circulation as fructose (Levine, Evans, Cadarette, Fisher, and Bullen, 1983).

Exogenous fructose ingested during prolonged exercise is less readily available for oxidation than glucose, which is likely due to its slower conversion to glucose by the liver, before it can be peripherally oxidized (Massicotte, Peronnet, Brisson, Bakkouch, and Hillaire-Marcel, 1989). The ingestion of fructose does not provoke the rise in plasma insulin concentration like the ingestion of glucose does in resting subjects, since the gastrointestinal and portal glucoreceptors are less sensitive to fructose (Mei, 1985). Thus, the direct supply of glucose from the banana can provide more readily available carbohydrate for oxidation while the fructose supply provides fuel at a slower rate; a seemingly well balanced carbohydrate food for prolonged exercise if taken at the proper times.

Many of the liquid carbohydrate supplements used during

prolonged exercise consisted of a glucose polymer (Table 1). Because glucose polymers (maltodextrins) have a much lower osmolality than an isocaloric solution of glucose, it was originally hypothesized that these polymer solutions empty from the stomach more quickly than glucose solutions (Foster, Costill, & Fink, 1980). Yet, research has demonstrated that rapid or large fluid shifts do not occur across the gastric membrane in response to osmolality, and thus fluids are emptied from the stomach near the same osmolality that they were ingested (Murray, 1987). A practical advantage of ingesting glucose polymers over simple sugars is that highly concentrated glucose polymer solutions are not as sweet tasting as isocaloric solutions made of simple sugars. The bananas (like most other solid carbohydrate foods) offer the same advantage by encasing it's simple sugars within the solid structure (i.e., plant cell walls), making this carbohydrate food pleasantly sweet as opposed to overbearingly sweet.

In the present study, as well as previously reported investigations (Coyle, et al., 1986; Coggan and Coyle, 1987; Coggan and Coyle, 1988; Coggan and Coyle, 1989; Davis et al., 1988), exhaustion did not always coincide with low blood glucose concentration. Hypoglycemia as a result of prolonged exercise was originally suspected as a cause of fatigue due to central nervous system dysfunction (Christensen & Hanson, 1939). Fatigue also occurs when

muscle glycogen is low and blood glucose is unable to offset the reduced glycogen availability (Coyle et al., 1986). Thus, fatigue from prolonged, moderately intense exercise appears to result from an inadequate delivery rate of carbohydrate to the exercising muscle (i.e., muscular fatigue) instead of an inadequate delivery of carbohydrate to the central nervous system (i.e., CNS fatigue).

In this dissertation, the mean pre-event plasma glucose concentration was 4.8 mmol/L and the mean glucose concentration at exhaustion-1 was 4.0 mmol/L (range from 2.5-5.8 mmol/L). Fourteen of 23 values were less than 4.0 mmol/L, while 6 values were less than 3.0 mmol/L. The decrease in blood glucose concentrations are consistent with data from other investigations (Coyle et al., 1983; Coyle et al., 1986; Coggan & Coyle, 1987; Coggan & Coyle, 1988; Coggan & Coyle, 1989). Yet, nine out of 23 glucose concentrations at exhaustion-1 were above 4.0 mmol/L, suggesting that carbohydrate availability is only one of several factors (such as hydration, core temperature, motivation, etc.) that influence the onset of volitional fatigue.

Time To Exhaustion-2

Hypothesis 2: The ingestion of solid carbohydrate will increase the time to exhaustion during the second exhaustion ride, as compared to the same carbohydrate ingested in

slurried form.

No statistically significant differences between the performance times to exhaustion after the administration of a placebo drink, a slurried, or a solid carbohydrate food were observed. Thus, the findings of this dissertation do not support the second hypothesis. Although the mean differences between the performance times of the placebo treatment (28 ± 4 minutes) and both carbohydrate treatments (44 ± 9 and 46 ± 13 for the slurried and solid, respectively) were large, the variances within each treatment were too large to detect significant differences. Having a larger sample size would have provided greater statistical power, helping to identify any metabolic and performance differences, if any existed.

The mean (\pm S.E.M.) times to exhaustion-2 from Coggan and Coyle (1987) BOUT-2 are compared with the times to exhaustion for RIDE-2 in the following table:

TREATMENT	MEAN EXHAUSTION-2 TIMES (min)	
	Murdoch	Coggan
Placebo	28 ± 4	10 ± 1
Slurried/Liq.	44 ± 9	26 ± 4
Solid	46 ± 13	---
Infused	---	43 ± 5

At the same intensity, the subjects in this study appeared to cycle for a considerably longer period of time after both the placebo the slurried/liquid banana feedings, than the subjects evaluated by Coggan and Coyle (1987). The subjects

in both studies had similar cycling VO_2max values (Coggan & Coyle: 66 ml/kg/min; and this study: 67 ml/kg/min). Additionally, both sets of subjects were exhausted/depleted from the previous event (RIDE-1 and BOUT-1).

The mean plasma glucose concentration of the subjects studied by Coggan and Coyle (1987) was 3.1 mmol/L at exhaustion-1, while 4.0 mmol/L was the mean glucose concentration for this investigation. Although blood glucose concentrations were quite variable from subject to subject, the lower average value may explain the shorter mean performance times of BOUT-2 in their study, as compared to this study.

Mean time to exhaustion (\pm S.E.M.) from RIDE-1 for all three performance tests was 217.3 ± 7.2 minutes. Other studies (Coyle et al., 1983; Coyle et al., 1986; Coggan & Coyle, 1987; Coggan & Coyle, 1988; Coggan & Coyle, 1989) have shown cycling to exhaustion, at a similar intensity, without ingestion of carbohydrate, to take between 160-180 minutes. Differences in training status, nutritional status, personal motivation, financial incentives, or the type of exercise modality(s) used in these studies may have contributed to these performance time differences. There are no studies in the literature that have used the run-bike combination protocol to determine the time at which exhaustion is reached.

The performance times for RIDE-2 in this study resulted

in 5 out of 8 subjects on the slurried feedings being faster than those taking the solid feedings. This finding suggests that the presumably quicker absorption of carbohydrate from the slurried banana was better able to supply fuel for exercise. Even more surprising was the fact that in four subjects, either one or both carbohydrate feedings did not improve performance times as compared to the placebo treatment. The reasons for these responses are unknown. The range of RIDE-2 times was quite large (13-124 minutes) suggesting that all the factors influencing performance time to exhaustion were not identified or quantifiable.

Other Variables

As with all plasma substrate measures, interpreting the physiological significance of a change in substrate concentration is difficult without measuring production/liberation and utilization rates. Understanding the mechanisms that influence the production and utilization of these substrates can lend insight into the interpretation of changes in plasma concentrations. Plasma free fatty acids are released into the circulation from adipose tissue as a result of endurance activity via neural, hormonal, and metabolic control (Essen, 1977; Newsholme & Start, 1981; Schnabel, Kindermann, Schmitt, Biro, & Stegmann, 1982).

Fatty acids are transported in the plasma via albumin because of their low solubility in water. Approximately

0.1% of the total fatty acid concentration in the blood is in the "free" form, that is, without any association with albumin (Newsholme & Start, 1981). It is the free fatty acid (FFA) concentration that regulates the rate of simple diffusion into the muscle (Gollnick, 1977). Thus, only a small percentage of the fatty acids passing through the muscle is taken up. Hence, the rate of FFA uptake by the muscle is not high enough to exclusively supply adequate fuel for sustained moderately intense exercise (Newsholme & Start, 1981). Therefore, in order to supply the energy needed to maintain power output greater than 60% VO_{2max} , carbohydrates are concomitantly oxidized by the exercising muscle. As exercise continues FFA's are increasingly oxidized, resulting in partial conservation of the limited carbohydrate stores (Essen, 1977).

Intracellular control of this FFA-glucose relationship occurs via concentrations of several glycolytic and oxidative allosteric regulators: acetyl-CoA, citrate, and glucose -6-phosphate (Newsholme & Start, 1981). Regulation occurs as follows: increased intracellular acetyl-CoA concentration from FFA oxidation will inhibit pyruvate dehydrogenase and thus slow down carbohydrate oxidation; increased intracellular citrate concentration from FFA oxidation will inhibit 6-phosphofructokinase resulting in an increased glucose-6-phosphate concentration, thereby inhibiting both hexokinase and glycogen phosphorylase

(Newsholme & Start, 1981).

On the other hand, a decreased rate of FFA oxidation during exercise would require greater carbohydrate oxidation in order to meet the energy demands, causing early fatigue and hypoglycemia (Carlson, Hovel, & Ekelund, 1963). Thus, FFA's regulate, to some extent, carbohydrate utilization and oxidation.

Hormonal changes that accompany prolonged exercise and facilitate FFA utilization include decreased plasma insulin concentration and increased plasma catecholamines, growth hormone, and corticosteroids (Galbo, Richter, & Hilsted, 1977). Plasma insulin inhibits lipolysis, while catecholamines, growth hormone, and corticosteroids promote lipolysis. On the other hand, insulin stimulates glucose uptake, while catecholamines, growth hormone and corticosteroids stimulate glucose production (Cryer, 1985).

As exercise continues, plasma FFA concentration increases. Since their uptake/utilization is directly dependent upon their concentration in the circulation, they become an increasingly important fuel source in the latter stages of exhaustive exercise when carbohydrate is in limited supply. Hence, without the increased availability of free fatty acids as a fuel supply, endogenous carbohydrate concentration would be depleted sooner, resulting in a more rapid onset of hypoglycemia.

As expected, plasma free fatty acid concentration

throughout RIDE-1 increased. The mean concentration increased gradually from .26 mmol/L at the pre-event time period to .90 mmol/L by exhaustion-1. These concentrations and their responses to moderately intense exercise are similar to what was previously reported (Coyle et al., 1983; Coyle et al., 1986; Coggan & Coyle, 1987; Coggan & Coyle, 1988; Coggan & Coyle, 1989).

Although plasma free fatty acid concentrations remained higher at post recovery and exhaustion-2, compared to pre-event time period values, they did not differ from the other treatments at the post recovery and exhaustion-2 time periods. These results were similar to those of Coggan & Coyle (1987), except that their post recovery concentrations were higher. The mean (\pm S.E.M.) FFA concentrations at the post recovery and exhaustion-2 time periods are presented in the following table:

TREATMENT	MEAN PLASMA FFA (mmol/L)			
	Post Recovery		Exhaustion-2	
	Murdoch	Coggan	Murdoch	Coggan
Placebo	0.85 \pm 0.08	1.47 \pm 0.29	0.92 \pm 0.06	0.74 \pm 0.09
Slurried/Liq.	0.96 \pm 0.15	1.33 \pm 0.18	0.88 \pm 0.13	0.53 \pm 0.06
Solid	0.80 \pm 0.10	---	0.83 \pm 0.09	---
Infused	---	1.15 \pm 0.11	---	0.63 \pm 0.34

Once again, the lower mean plasma glucose concentration at the end of BOUT-1 in Coggan & Coyle's (1987) study, as compared to that of the present investigation (3.1 and 4.0 mmol/L, respectively), may have resulted in greater

lipolysis in an attempt to sustain the required energy output that carbohydrates (via blood glucose) were unable to meet. As a result, greater concentrations of lipolytic stimulating hormones may have been present in the circulation resulting in higher FFA concentrations.

Lactate is formed as a result of glycolysis, hence it is a product of carbohydrate metabolism (Cryer, 1985) Lactate can be taken up by the liver as a gluconeogenic precursor. Increased plasma lactate concentration may be loosely interpreted to mean increased lactate production. However, plasma concentration is a result of both production and uptake (i.e., turnover) making it equally as possible that increased lactate concentrations results from decreased uptake (Brooks & Fahey, 1984).

Plasma lactate concentrations were not statistically different from one another at the post recovery or exhaustion-2 time periods. Similar results were reported by Coggan & Coyle (1987). The mean (\pm S.E.M.) lactate concentrations at the post recovery and exhaustion-2 time periods are presented in the following table:

TREATMENT	MEAN PLASMA LACTATE (mmol/L)			
	Post Recovery		Exhaustion-2	
	Murdoch	Coggan	Murdoch	Coggan
Placebo	1.5 \pm 0.3	1.5 \pm 0.1	1.7 \pm 0.3	1.5 \pm 0.1
Slurried/Liq.	2.0 \pm 0.5	1.6 \pm 0.2	1.4 \pm 0.2	1.6 \pm 0.1
Solid	2.7 \pm 0.2	---	1.9 \pm 0.2	---
Infused	---	1.5 \pm 0.1	---	1.2 \pm 0.2

All these mean plasma lactate concentrations are within the normal resting range of .5-2.0 mmol/L (Young, 1987). These data suggest that either plasma lactate is being utilized as fuel or minimal production of lactate is occurring.

Subjects developed significant decreases in plasma volume (up to 12%) throughout exercise. Davis et al. (1988) demonstrated a similar pattern of plasma volume loss with cyclists during a 2 hour ride at 70% of their VO_2 max. The mean plasma volume shifts for each treatment were variable at both the post recovery and exhaustion-2 time periods; however, no significant differences were detected. This plasma volume loss represents either a shift of the fluid into other areas of the body, or an inability to completely replace all the fluid lost during exercise. The subjects were encouraged to drink at least 8 oz. of cool water every 15 minutes, minimizing the effects of dehydration to affect their performances. Nevertheless, the mean weight loss at exhaustion-2, when compared to mean pre-event weight, was 2 kilograms. This weight/fluid loss may have contributed to the volitional fatigue in some of these subjects.

The banana was selected as the experimental solid food because its caloric composition is almost exclusively carbohydrate, and because it is one of the most popular foods ingested during exhaustive endurance events. Bananas provide carbohydrate in both simple and complex forms. The banana is approximately 75% water, which softens its form.

The magnitude of both the metabolic and performance differences observed between the ingestion of solid and slurried foods may have been greater if a carbohydrate food with a lower water/moisture content was used (i.g., cookies, figs, apples, tootsie rollsTM, etc.).

Jenkins et al. (1981) determined the glycemic response from ingesting 2 solid bananas (exactly 50 grams of carbohydrate), as compared to 50 grams of glucose (reference value equal to 100%). They found a moderate glycemic response equal to 62% of the reference glucose value. The primary assumption of this dissertation was that the glycemic response of ingesting solid bananas would be increased when the banana was consumed in slurried form. This assumption appears reasonable since the literature clearly demonstrates that the disruption of food form (via mixing, cooking, blending pureed, etc.) increases the glycemic response to that food in healthy resting subjects (Bolton et al., 1981; Crapo and Henry, 1988; Harber et al., 1977). Yet, statistical differences in plasma glucose concentrations were not seen at either the post recovery or exhaustion-2 time periods between the solid and slurried banana treatments. These results suggest that the glycemic effect of food may be altered during exercise. The following limitations may account for the results obtained.

Limitations

Three limitations with this dissertation were the small sample size, the type and the amount of carbohydrate used. The small sample size was the result of subject injury (n=2) and the extreme physical demand of the protocol that resulted in failure of 2 subjects to complete all parts of the performance tests.

The banana was selected as the solid form of carbohydrate for its carbohydrate composition as well as its frequent use during prolonged endurance training and racing events. However, because of its moisture content (approximately 75% water) it is more semi-solid than solid. This semi-solid carbohydrate food may not have been structurally different enough from the slurried form to elicit the metabolic and performance responses of other solid carbohydrate foods. Carbohydrate foods with less moisture content may increase the magnitude of any metabolic and performance differences that exist between solid and slurried carbohydrate food ingestion during prolonged, moderately intense exercise.

Recommendations For Future Research

The data from this dissertation provide information regarding some of the metabolic differences resulting from the ingestion of either solid or slurried carbohydrate food (1.1g/kg of body wt.), during prolonged exercise to

exhaustion. Although the ingestion of both solid and slurried bananas significantly increased mean plasma glucose concentration (compared to the placebo) at the final exhaustion period, its effect on increasing endurance performance was not demonstrated. A similar study with more subjects, lower moisture content in the solid carbohydrate food, and greater absolute amount of carbohydrate feedings (approximately 3 grams of carbohydrate per kg of body weight) may help to elucidate any performance enhancing effects of solid carbohydrate feedings and the differences in food form.

Future studies are needed to answer the following questions:

- (1) Is there a different glycemic response to a carbohydrate food ingested during exercise, as compared to resting conditions?
- (2) How quickly during exercise do fuel substrates (ie, glucose, lactate, free fatty acids, pyruvate, glycerol, alanine, and branch chain amino acids) enter the circulation after the ingestion of solid carbohydrates?
- (3) Which hormones are affected by these various carbohydrate ingestions, and how?
- (4) Are glycemic responses affected by gender, age, and fitness status?

- (5) Can one alter his/her glycemic response through short or longterm dietary manipulation/exercise training?
- (6) What is the optimal amount of solid carbohydrate needed to sustain prolonged, moderately intense exercise?
- (7) Which solid carbohydrate foods (food form) provide the most effective exogenous fuel source for enhancing prolonged endurance performance?
- (8) How does the addition of other macronutrients to a solid carbohydrate food affect endurance performance?
- (9) What is the gastric emptying rate of solid carbohydrate food ingested during exercise; and how does this effect digestion and absorption of the carbohydrate itself?

Future research designed to answer these questions can increase our understanding of the underlying mechanisms affecting human endurance performance. This research will support the efforts of competitive endurance athletes to overcome performance variables associated with fuel depletion, and thereby expand the boundaries of human performance.

CHAPTER VI
SUMMARY AND CONCLUSIONS

This dissertation examined the performance and metabolic effects of ingesting either a solid or slurried carbohydrate food, during prolonged, exhaustive exercise. Eight well trained male triathletes/biathletes performed three separate randomized exhaustive endurance tests, each separated by at least 2 weeks. Each test consisted of the following consecutive events: 90 minutes running at 70% VO_{2max} ; 90 minutes cycling at 70% VO_{2max} ; continued cycling at an increased intensity until exhaustion was reached; 20 minute rest during which one of the three treatments was ingested; and finally, cycling at 70% VO_{2max} until exhaustion was reached a second time. The three treatments included the ingestion of either a flavored placebo drink, solid bananas, or slurried bananas.

It was hypothesized that the slower digestion / absorption of carbohydrates from ingesting solid bananas, as compared to slurried bananas, would provide higher plasma glucose concentrations during the latter stages of the endurance exercise protocol. Theoretically, the slower carbohydrate absorption from ingesting the solid bananas would decrease the insulin response, helping to deliver more blood glucose to the working muscles. The steady supply of

glucose during the latter stages of prolonged exercise would thereby supply enough carbohydrate to prolong performance time to exhaustion.

The mean glucose concentration from the combined solid and slurried banana treatments, at the final exhaustion period, was significantly higher than that of the placebo treatment. Mean plasma lactate and free fatty acid concentrations were not significantly different between the three treatments, at the end of the final exhaustion ride.

The increased glucose concentration observed with the combined banana treatments, at the final exhaustion period, did not statistically increase mean performance times to exhaustion. Mean (\pm S.E.M.) performance times for RIDE-2 were 28.2 ± 4.3 , 44.1 ± 8.7 , and 46.4 ± 13.2 minutes for the placebo, slurried, and solid treatments, respectively. Although mean time from the carbohydrate treatments was more than 15 minutes longer than that of the placebo treatment, the variation around the mean was too large to reject the null hypothesis. Future research is needed to determine if different amounts and forms of carbohydrate food could enhance endurance performance.

BIBLIOGRAPHY

- Ahlborg, G., Bergstrom, J., Eklund, L. G., & Hultman, E. (1967). Muscle glycogen and muscle electrolytes during prolonged physical exercise. Acta Physiologica Scandinavica, 70, 129-142.
- Ahlborg, G., & Felig, P. (1976). Influence of glucose ingestion on fuel-hormone response during prolonged exercise. Journal of Applied Physiology, 41(5), 683-688.
- Ahlborg, G., & Felig, P. (1982). Lactate and glucose exchange across the forearm, legs and splanchnic bed during and after prolonged leg exercise. Journal of Clinical Investigation, 69, 45-54.
- Allain, C. A., Poon, L. S., Chan, C. S. G., Richmond, W., & Fu, P. C. (1974). Enzymatic determination of total serum Cholesterol. Clinical Chemistry, 20, 470.
- Baldwin, K. M., Reitman, J. S., Terjung, R. L., Winder, W. W., & Holloszy, J. O. (1973). Substrate depletion in different types of muscle and in liver during prolonged running. American Journal of Physiology, 225(5), 1045-1050.
- Behall, K. M., Scholfield, D. J., & Canary, J. (1988). Effects of starch structure on glucose and insulin responses in adults. American Journal of Clinical Nutrition, 47, 428-432.
- Bergstrom, J., Hermansen, L., Hultman, E., & Saltin, B. (1967). Diet, muscle glycogen, and physical performance. Acta Physiologica Scandinavia, 71, 140-150.
- Bergstrom, J., & Hultman, E. (1967). A study of the glycogen metabolism during exercise in man. Scandinavian Journal of Clinical and Laboratory Investigation, 19, 218-228.
- Bolton, R. P., Heaton, K. W., & Burroughs, L. F. (1981). The role of dietary fiber in satiety, glucose and insulin: studies with fruit and fruit juices. American Journal of Clinical Nutrition, 34, 211-217.

- Bonen, A., Malcolm, S. A., Kilgour, R. D., MacIntyre, K. P., & Belcastro, A. N. (1981). Glucose ingestion before and during intense exercise. Journal of Applied Physiology, 50, 766-771.
- Brand, J. C., Nicholson, P. L., Thorburn, A. W., & Truswell, A. S. (1985). Food processing and the glycemic index. American Journal of Clinical Nutrition, 42, 1192-1196.
- Brener, W., Hendrix, T. R., & McHugh, P. R. (1983). Regulation of the gastric emptying of glucose. Gastroenterology, 85, 76-82.
- Brodowicz, G. R., Lamb, D. R., Bauer, T. S., & Connors, D. F. (1984). Efficacy of various drink formulations for fluid replenishment during cycling exercise in the heat. Abstract. Medicine and Science in Sports and Exercise, 16(2), 138.
- Brooke, J. D., Davis, G. J., & Green, L. F. (1975). The effects of normal and glucose syrup work diets on the performance of racing cyclists. Journal of Sports Medicine and Physical Fitness, 15, 257-265.
- Brooks, G. A., & Fahey, T. D. (1984). Exercise Physiology. John Wiley & Sons, Inc., 202-203.
- Brouns, F. (1988). Food and fluid related aspects in highly trained athletes, Uitgeverij De Vrieseborch - Haarlem.
- Burke, L. M., & Read, R. S. D. (1987). Diet patterns of elite Australian male triathletes. The Physician and Sportsmedicine, 15(2), 140-155.
- Buskirk, E. R., Iampietro, P. F. & Bass, D. E. (1958). Work performance after dehydration: effect of physical conditioning and heat acclimatization. Journal of Applied Physiology, 12, 189-194.
- Cammack, J., Read, N. W., Cann, P. A., Greenwood, B., & Holgate, A. M. (1982). Effects of prolonged exercise on the passage of a solid meal through the stomach and small intestine. Gut, 23, 957-961.
- Carlson, L. A., Hovel, R. J., & Ekelund, L. G. (1963). Effect of nicotinic acid on the turnover rate and oxidation of the free fatty acids of plasma in man during exercise. Metabolism, 12, 837-845.

- Christensen, E. H., & Hansen, O. (1939). Hypoglykamie, Arbeitsfähigkeit und Ermüdung. Skandinavian Archives of Physiology, 81, 172-179.
- Coggan, A. R., & Coyle, E. F. (1987). Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. Journal of Applied Physiology, 63, 2388-2395.
- Coggan, A. R., & Coyle, E. F. (1988). Effect of carbohydrate feedings during high-intensity exercise. Journal of Applied Physiology, 65, 1703-1709.
- Coggan, A. R., & Coyle, E. F. (1989). Metabolism and performance following carbohydrate ingestion late in exercise. Medicine and Science in Sports Exercise, 21, 59-65.
- Collings, P., Williams, C., & McDonald, I. (1981). Effects of cooking on serum glucose and insulin responses to starch. British Medical Journal, 282, 1032.
- Costill, D. L., Bowers, R., Branam, G., & Sparks, K. (1971). Muscle glycogen utilization during prolonged exercise on successive days. Journal of Applied Physiology, 31(6), 834-838.
- Costill, D. L., & Saltin, B. (1974). Factors limiting gastric emptying during rest and exercise. Journal of Applied Physiology, 37, 679-683.
- Costill, D. L., Sherman, W. M., Fink, W. J., Maresh, C., Witten, M., & Miller, J. M. (1981). The role of dietary carbohydrates in muscle glycogen resynthesis after strenuous running. American Journal of Clinical Nutrition, 34, 1831-1836.
- Coyle, E. F., Coggan, A. R., Hemmert, M. K., & Ivy, J. L. (1986). Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. Journal of Applied Physiology, 61, 165-172.
- Coyle, E. F., Costill, D. L., Fink, W. J., & Hoopes, D. G. (1978). Gastric emptying rates for selected athletic drinks. Research Quarterly, 49, 119-124.
- Coyle, E. F., Hagberg, J. M., Hurley, B. F., Martin, W. H., Ehsani, A. A., & Holloszy, J. O. (1983). Carbohydrate feeding during prolonged strenuous exercise can delay fatigue. Journal of Applied Physiology, 55(1), 230-235.

- Crapo, P. A., & Henry, R. R. (1988). Postprandial metabolic responses to the influence of food form. American Journal of Clinical Nutrition, 48, 560-564.
- Crapo, P. A., Insel, J., Sperling, M., & Kolterman, O. G. (1981). Comparison of serum glucose, insulin, and glucagon responses to different types of complex carbohydrate in non-insulin-dependent diabetic patients. American Journal of Clinical Nutrition, 34, 184-190.
- Crapo, P. A., Reaven, G., & Olefsky, J. (1976). Plasma glucose and insulin responses to orally administered simple and complex carbohydrates. Diabetes, 25, 741-747.
- Cryer, P. E. (1985). Glucose homeostasis and hypoglycemia. Textbook of Endocrinology, Seventh Edition, 989-1017.
- Davis, J. M., Lamb, D. R., Pate, R. R., Slentz, C. A., Burgess, W. A., & Bartoli, W. P. (1988). Carbohydrate-electrolyte drinks: effects on endurance cycling in the heat. American Journal of Clinical Nutrition, 48, 1023-1030.
- DeMoss, V. (1989). Fuel to go. Cyclist, April, 34-38.
- Dill, D. B., & Costill, D. L. (1974). Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. Journal of Applied Physiology, 37, 247-248.
- Dohm, G. L., Tapscot, E. B., Barakat, H. A., & Kasperek, G. J. (1983). Influence of fasting in rats on glycogen depletion during exercise. Journal of Applied Physiology, 55, 830-833.
- Drabkin, O. L., & Austin, J. H. (1935). Spectrophotometric studies. II Preparation from washed cells; nitric oxide, hemoglobin, and sulfhemoglobin. Journal of Biological Chemistry, 112, 51-55.
- Durnin, J. V., & Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: Measurements on 481 men and women aged from 16-72 years. British Journal of Nutrition, 32, 77-96.
- Essen, B. (1977). Intramuscular substrate utilization during prolonged exercise. Annals New York Academy of Sciences, 301, 30-44.

- Felig, P., Cherif, A., Minagawa, A., & Wahren, J. (1982). Hypoglycemia during prolonged exercise in normal men. New England Journal of Medicine, 306, 895-900.
- Fellows, I. W., Evans, D. F., Bennet, T., MacDonald, I. A., Clark, A. G., & Bloom, S. B. (1987). The effect of insulin-induced hypoglycemia on gastrointestinal motility in man. Clinical Science, 72, 743-748.
- Fielding, R. A., Costill, D. L., Fink, W. J., King, D. S., Hargreaves, M., & Kovalski, J. E. (1985). Effects of carbohydrate feeding frequencies and dosage on muscle glycogen use during exercise. Medicine and Science in Sports and Exercise, 17(4), 472-476.
- Flynn, M. G., Costill, D. L., Hawley, J. A., Fink, W. J., Neuffer, P. D., Fielding, R. A., & Sleeper, M. D. (1987). Influence of selected carbohydrate drinks on cycling performance and glycogen use. Medicine and Science in Sports and Exercise, 19(1), 37-40.
- Fordtran, J. S., & Saltin, B. (1967). Gastric emptying and intestinal absorption during prolonged severe exercise. Journal of Applied Physiology, 23, 331-335.
- Foster, C., Costill, D. L., & Fink, W. J. (1980). Gastric emptying characteristics of glucose and glucose polymer solutions. Research Quarterly for Exercise and Sport, 51, 299-305.
- Foster, C., Thompson, N. N., Dean, J., & Kirkendall, D. T. (1986). Carbohydrate supplementation and performance in soccer players. Abstract. Medicine and Science in Sports and Exercise, 18(2), S12.
- Fox, E. L., Bartels, R. L., Keller, J. E., & Vivian, V. M. (1983). Solid versus liquid diets: Effects on multiple daily exercise performance. Nutrient Utilization During Exercise, Report of the Ross Symposium, 119-125.
- Galbo, J., Richter, E. & Hilsted, J. (1977). Hormonal regulation during prolonged exercise. Annual NY Academy of Sciences, 301, 72-79.
- Goddard, M. S., Young, G., & Marcus, R. (1984). The effect of amylose content on insulin and glucose responses to ingested rice. American Journal of Clinical Nutrition, 39, 388-392.

- Gollnick, P. D., (1977). Free fatty acid turnover and the availability of substrates as a limiting factor in prolonged exercise. Annals New York Academy of Sciences, 301, 64-71.
- Gollnick, P. D., Pernow, B., Essen, B., Jansson, E., & Saltin, B. (1981). Availability of glycogen and plasma FFA for substrate utilization in leg muscle of man during exercise. Clinical Physiology (Oxford), 1, 27-42.
- Haber, G. B., Heaton, K. W., Murphy, D., & Burroughs, L. F. (1977). Depletion and disruption of dietary fiber. Effects on satiety, plasma glucose, and serum insulin. Lancet, 2, 679-682.
- Hargreaves, M., Costill, D. L., Coggan, A., Fink, W. J., & Nishibata, I. (1984). Effect of carbohydrate feedings on muscle glycogen utilization and exercise performance. Medicine and Science in Sports and Exercise, 16(3), 219-222.
- Hermansen, L., Hultman, E., & Saltin, B. (1967). Muscle glycogen during prolonged severe exercise. Acta Physiologica Scandinavia, 71, 129-139.
- Hickson, R. C., Rennie, M. J., Conlee, R. K., Winder, W. W., & Holloszy, J. O. (1977). Effects of increased plasma fatty acids on glycogen utilization and endurance. Journal of Applied Physiology, 43(5), 829-833.
- Hoppeler, H., Howald, H., Conley, K., Lindstedt, S. L., Claassen, H., Vock, P., & Weibel, E. R. (1985). Endurance training in humans: aerobic capacity and structure of skeletal muscle. Journal of Applied Physiology, 59(2), 320-327.
- Hunt, J. N., Smith, J. L., & Jiang, C. L. (1985). Effect of meal volume and energy density on gastric emptying of carbohydrates. Gastroenterology, 89, 1326-1330.
- Ivy, J. L., Costill, D. L., Fink, W. J., & Lower, R. W. (1979). Influence of caffeine and carbohydrate feedings on endurance performance. Medicine and Science in Sports and Exercise, 11(1), 6-11.
- Ivy, J. L., Miller, W., Dover, V., Goodyear, L. G., Sherman, W. M., Farrel, S., & Williams, H. (1983). Endurance improved by ingestion of a glucose polymer supplement. Medicine and Science in Sports and Exercise, 15(6), 466-471.

- Jansson, E., & Kaijser, L. (1987). Substrate utilization and enzymes in skeletal muscle of extremely endurance-trained men. Journal of Applied Physiology, 62(3), 999-1005.
- Jenkins, D. J., Ghafari, H., Wolever, T. M., Taylor, R. H., Baker, H. M., Fielden, H., Jenkins, A. L., & Bowling, A. C. (1982). Relationship between the rate of digestion of foods and post-prandial glycemia. Diabetologia, 22, 450-455.
- Jenkins, D. J., Wolever, T. M., Taylor, R. H., Baker, H. M., Fielden, H., Baldwin, J. M., Bowling, A. C., Newman, H. C., Jenkins, A. L., & Goff, D. V. (1981). Glycemic index of foods: a physiological basis for carbohydrate exchange. American Journal of Clinical Nutrition, 34, 450-455.
- Karlsson, J., & Saltin, B. (1971). Diet, muscle glycogen, and endurance performance. Journal of Applied Physiology, 31(2), 203-206.
- Katz, A., Broberg, S., Sahlin, K., & Wahren, J. (1986). Leg glucose uptake during maximal dynamic exercise in humans. American Journal of Physiology (Endocrinol. Metab. 14), 251, E65-E70.
- Kurland, I. J., & Pilkis, S. J. (1989). Indirect versus direct routes of hepatic glycogen synthesis. FASEB Journal, 3, 2277-2281.
- Landau, B. R., & Wahren, J. (1988). Quantification of the pathways followed in hepatic glycogen formation from glucose. FASEB Journal, 2, 2368-2375.
- Levine, L., Evans, W. J., Cadarette, B. S., Fisher, E. C., & Bullen, B. A. (1983). Fructose and glucose ingestion and muscle glycogen use during submaximal exercise. Journal of Applied Physiology, 55(6), 1767-1771.
- Lewis, I., Miller, G., Burstein, M., Slagle, S., & Eder, H. (1982). Separation and quantitation of subclasses on human plasma high density lipoproteins by a simple precipitation procedure. Journal of Lipid Research, 23, 1207-1223.
- Lopes-Virella, M. F., Stone, P., Ellis, S., & Colwell, J. A. (1977). Cholesterol determination in high density lipoproteins separated by three different methods. Clinical Chemistry, 23, .

- Malagelada, J. R. (1977). Quantification of gastric solid-liquid discrimination during digestion of ordinary meals. Gastroenterology, 72, 1264-1271.
- Massicotte, D., Peronnet, F., Brisson, G., Bakkouch, K., & Hillaire-Marcel, C. (1989). Oxidation of a glucose polymer during exercise: comparison with glucose and fructose. Journal of Applied Physiology, 66(1), 179-183.
- Maughan, R. J., & Whiting, P. H. (1983). Hypoglycemia in marathon runners: effects of glucose ingestion. Medicine and Science in Sports and Exercise, 15, 128.
- McCann, M. J., & Strucker, E. M. (1986). Gastric emptying of glucose loads in rats: effects of insulin-induced hypoglycemia. American Journal of Physiology, 251, R609-R613.
- Mei, N. (1985). Intestinal chemosensitivity. Physiological Review, 65, 211-237.
- Minami, H., & McCallum, R. W. (1984). The physiology and pathophysiology of gastric emptying in humans. Gastroenterology, 86, 1592-1610.
- Mitchell, J. B., Costill, D. L., Houmard, J. A., Flynn, M. G., Fink, W. J., & Beltz, J. D. (1987). Ingestion of carbohydrate solutions: effects on gastric emptying and performance during prolonged exercise. Medicine and Science in Sports and Exercise, 19(2), S56.
- Morse, D. R., Schacterle, G. R., Furst, L., Zaydenberg, M., & Pollack (1989). Oral digestion of a complex-carbohydrate cereal: effects of stress and relaxation on physiological and salivary measures. American Journal of Clinical Nutrition, 49, 97-105.
- Murray, R. (1987). The effects of consuming carbohydrate-electrolyte beverages on gastric emptying and fluid absorption during and following exercise. Sports Medicine, 4, 322-351.
- Murray, R. K., Granner, D. K., Mayes, P. A., & Rodwell, V. W. (1988). Harper's Biochemistry, Twenty-first Edition, 182-183.
- National Cholesterol Education Program Detection, Evaluation and Treatment of High Blood Cholesterol in Adults, (1987). NIH Publication. No 88-2926, November.

- Neufer, P. D., Costill, D. L., Fink, W. J., Kirwan, J. P., & Fielding, R. A. (1986). Effects of exercise and carbohydrate composition on gastric emptying. Medicine and Science in Sports and Exercise, 18, 658-662.
- Newsholme, E. A. (1977). The regulation of intracellular and extracellular fuel supply during sustained exercise. Annal New York Academy of Sciences, 310, 81-91.
- Newsholme, E. A., & Start, C. (1981). Regulation in Metabolism. John Wiley & Son, New York.
- Nielsen, B. (1984). Dehydration, rehydration and thermoregulation. Medicine and Sport Science, 17, 81-96.
- Noakes, T. D., Koeslag, J. H., & McArthur, P. (1983). Correspondence: Hypoglycemia during exercise. New England Journal of Medicine, 308(5), 279-280.
- Noma, A., Okabe, H., & Kita, M. (1973). A new colorimetric micro-determination of free fatty acids in serum. Clinica Chimica Acta, 43, 317-320.
- O'Dea, K., Nestel, P. J., & Antonoff, L. (1980). Physical factors influencing postprandial glucose and insulin responses to starch. American Journal of Clinical Nutrition, 33, 760-765.
- Pruett, E. D. R. (1970). Glucose and insulin during prolonged work stress in men living on different diets. Journal of Applied Physiology, 28(2), 199-208.
- Richter, E. A., & Galbo, H. (1986). High glycogen levels enhance glycogen breakdown in isolated contracting skeletal muscle. Journal of Applied Physiology, 61, 827-831.
- Rodahl, K., Miller, H. I., & Issekutz, B. Jr. (1964). Plasma free fatty acids in exercise. Journal of Applied Physiology, 19(3), 489-492.
- Ryan, A., Bleiler, T., Carter, J., & Gisolfi, C. (1989). Gastric emptying during prolonged cycling exercise in the heat. Medicine and Science in Sports and Exercise, 21, 51-58.

- Saltin, B., Wahren, J., & Pernow, B. (1974). Phosphagen and carbohydrate metabolism during exercise in trained middle-aged men. Scandinavian Journal of Clinical Laboratory Investigation, 33, 71-77.
- Schnabel, A., Kindermann, W., Schmitt, W. M., Biro, G., & Stegmann, H. (1982). Hormonal and metabolic consequences of prolonged running at the individual anaerobic threshold. International Journal of Sports Medicine, 3, 153-168.
- Schusdziarra, V., Dangel, G., Klier, M., Henrichs, I., & Pfeiffer, E. F. (1981). Effects of solid and liquid carbohydrate upon postprandial pancreatic endocrine function. Journal of Clinical Endocrinology and Metabolism, 53(1), 16-20.
- Seifert, J. G., Langemfeld, M. E., Rudge, S. L., & Bucher, R. J. (1986). Effects of glucose polymer ingestion on ultra-endurance bicycling performance. Abstract. Medicine and Science in Sports and Exercise, 18(2), S5.
- Sherman, W. M. (1987). Carbohydrate, muscle glycogen, and improved performance. The Physician and Sportsmedicine, 15(2), 157-164.
- Sherman, W. M., Costill, D. L., Fink, W. J., Hagerman, F. C., Armstrong, L. E., & Murray, T. F. (1983). Effect of a 42.2-km footrace and subsequent rest or exercise on muscle glycogen and enzymes. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 55(4), 1219-1224.
- Sinning, W. E. (1975). Experiments and demonstrations in exercise physiology. Springfield, MA: W. B. Saunders.
- Snow, P., & O'Dea, K. (1981). Factors affecting the rate of hydrolysis of starch in food. American Journal of Clinical Nutrition, 34, 2721-2727.
- Thorburn, A. W., Brand, J. C., & Truswell, A. S. (1986). The glycaemic index of foods. Medical Journal of Australia, 144, 580-582.
- Thorburn, A. W., Brand, J. C., & Truswell, A. S. (1986). Salt and the glycaemic response. British Medical Journal, 292, 1697-1699.
- Vinik, A. I., & Jenkins, D. J. A. (1988). Dietary fiber in management of diabetes. Diabetes Care, 11, 160-173.

- Vissing, J., Wallace, J. L., & Galbo, H. (1989). Effect of liver glucogen content on glucose production in running rats. Journal of Applied Physiology, 66(1), 318-322.
- Wahren, J., Felig, P., Ahlborg, G., & Jorfeldt, L. (1971). Glucose metabolism during leg exercise in man. Journal of Clinical Investigation, 50, 2715-2725.
- Wong, S., & O'Dea, K. (1983). Importance of physical form rather than viscosity in determining the rate of starch hydrolysis in legumes. American Journal of Clinical Nutrition, 37, 66-70.
- Yoon, J. H., Thompson, L. U., & Jenkins, D. J. A. (1983). The effect of phytic acid on in vitro rate of starch digestibility and blood glucose response. American Journal of Clinical Nutrition, 38, 835-842.
- Young, D. S. (1987). Implementation of SI units for Clinical Laboratory Data. Annals of Internal Medicine, 106(1), 114-129.

Appendix A

CONSENT FORM FOR HUMAN SUBJECTS**THE UNIVERSITY OF NORTH CAROLINA AT GREENSBORO
SCHOOL OF HUMAN ENVIRONMENTAL SCIENCES
DEPARTMENT OF NUTRITION**

**Project Title: An Evaluation of the Ergogenic Effects of
CAPS on Endurance Performance.**

**Principal Investigators: Terry Bazzarre, Ph.D.
Phillip Snider
Scott Murdoch**

PURPOSE

I understand that the purpose of this 13-week study is to investigate the effects of ingesting Coenzyme Athletic Performance System (CAPS) supplements on overall endurance performance as well as metabolic measures (VO_2 , CO_2 , ventilation, respiratory quotient, heart rate, % body fat, blood lactate, glucose, and free fatty acids) throughout each of 4 performance tests. Also the effect of ingesting either solid or liquid (slurried) carbohydrate food (banana) between two exercise periods on both performance and metabolic variables will be assessed.

EXPLANATION OF STUDY

I understand that I will be required to complete a personal and family medical history in order to participate in the study, and that if I have a medical history of coronary heart disease, hypertension, or diabetes that I will be screened from participation in the research. I understand that during this study I will report to the Human Performance Laboratory (Stone 302) on 11 occasions; I will perform 6 maximal exercise stress tests (3 on a motor driven treadmill and 3 on a stationary cycle ergometer), and 5 visits for the purpose of performing two exercise bouts/visit at a moderate intensity (70% of VO_2 max) to exhaustion. During each of the six maximal exercise stress tests electrodes will be attached to my torso so that cardiac responses to exercise may be monitored. Also ventilatory gases will be continuously monitored while breathing through a one-way mouth piece.

I also understand that each of the 5 exercise bouts consists of: 1) running for 90 minutes at a moderate

Appendix A

intensity on a motor driven treadmill, 2) immediately cycling at a moderate intensity until I cannot continue to cycle at a cadence of 50 revolution per minute, 3) resting for 15 minutes while ingesting 2-3 bananas and water in either solid or slurried form, or just a liquid beverage, and 4) cycle at the same moderate intensity until I cannot continue to cycle at a cadence of 50 revolutions per minute. I understand that before each of these 5 exercise bouts I will enter the lab after an overnight fast. Upon entering the lab 60 minutes before each of the 5 exercise tests, I will rest in the supine position while a catheter is inserted into my forearm vein. The catheterization will be performed by a trained phlebotomist, Dr. Terry Bazzarre. I understand that 10-15 ml of blood will be drawn at the following 11 time periods during 4 of the 5 exercise tests; 30 minutes and 1 minute before the test begins, 45, 90, 135, 180 minutes, at the point of exhaustion from the first ride, 1 minute before the second ride, at 10 minute intervals and at the point of exhaustion from the second ride. The catheter will then be removed from my arm, and I will hold direct pressure on the insertion site until the researchers confirm that the bleeding has stopped. I understand that I will be given 6-8 oz. water at every 15 minute interval throughout all exercise bouts.

EXPLANATION OF RISKS

I understand that during and/or after each of the maximal stress tests and the exercise tests that I may become dizzy, tired or weak as a result of fatigue. There is a rare possibility of heart attack (<3 in 10,000) or death (<1 in 10,000) as a result of the maximal exercise stress tests, however the researchers will minimize such risk by using subjects between the ages of 18 and 35, by preliminary screening with a medical history form, and continuous monitoring throughout the test as specified by the American College of Sports Medicine Guidelines. I understand that the insertion of the catheter may be painful, but that the pain will subside soon after insertion. All precautions associated with venipuncture have been taken in order to reduce the risks of venipuncture (i.e., air emboli, infection, bruising and fainting). To prevent clotting in the catheter it will be flushed periodically with "physiological saline" while it is inserted in the arm.

Appendix A

I understand that all data collected on my physical characteristics are considered confidential and will be kept in a file in Dr. Bazzarre's office with controlled access to each subject's file. Research analyses on all subjects' data will be performed using code numbers rather than subjects' names.

EXPLANATION OF BENEFITS

The benefits I may gain from participation in this study include: evaluation of my risk of developing heart disease, hypertension or nutritional problems; evaluation of my endurance capacity, measurements of my body fat and lean body mass, a free 4-week period of CAPS supplements, monetary rewards for 4 of the 5 exercise tests plus monetary bonus opportunities for peak performances relative to the other subjects. I understand that a summary of the results of this study will be made available to me **after** the completion of this study.

I understand that Dr. Terry Bazzarre, Scott Murdoch, and Phillip Snider will be available to answer any questions I have. They can be reached at 919-334-5313 on weekdays.

I confirm that my participation in this study is completely voluntary, and that no coercion of any kind has been used to obtain my cooperation. I also understand that I can withdraw my consent and terminate my participation in this study at any time without prejudice. I confirm that I have been informed of the procedures that will be used in this study. I understand what is required of me as a subject. I agree that any questions I have regarding this study and the procedures have been answered to my satisfaction. I give my voluntary cooperation as a participant.

Signature of Subject _____

Date _____

Signature of Witness _____

Appendix B

MEDICAL HISTORY QUESTIONNAIRE

THE UNIVERSITY OF NORTH CAROLINA AT GREENSBORO
 SCHOOL OF HUMAN ENVIRONMENTAL SCIENCES
 DEPARTMENT OF NUTRITION

Name: _____,

Date: _____

I, _____, hereby authorize the release of and aspect of my medical history which may be necessary for my participation in this project by the Department of Human Environmental Sciences at the University of North Carolina at Greensboro.

.....

Please respond "Yes" or "No" to the following health data.

1. Heart attack, coronary bypass, or cardiac surgery
2. Chest discomfort - especially with exertion
3. High blood pressure
4. Extra, skipped or rapid heart beats/palpitations
5. Heart murmurs, clicks, or unusual cardiac findings
6. Rheumatic fever
7. Ankle swelling
8. Peripheral vascular disease
9. Phlebitis, emboli
10. Unusual shortness of breath
11. Lightheadedness or fainting
12. Pulmonary disease including asthma, emphysema and bronchitis
13. Abnormal blood lipids
14. Diabetes
15. Stroke
16. Emotional disorders
17. Medications of all types
18. Recent illness, hospitalization or surgical procedures
19. Orthopedic problems, arthritis

.....

Appendix B

20. Family (granparents, parents, aunts, uncles, and siblings)

- Coronary disease
- Sudden death
- Congenital heart disease

21. Other habits

- Caffeine including cola drinks
- Alcohol
- Tobacco
- Other unusual habits or dieting

.....

Exercise Informantion

Years of running experience _____

Number of miles averaged per week for the past month _____

Marathon personal best _____

10 km personal best _____

.....

If you have any other medical problems and/or current medications please list below.

*** adapted from Guidlines for Exercise Testing and Perscription (3rd ed.), Lea & Febiger, Philadelphia, PA. 1986.

Appendix C

Skinfold Conversion Table

Table 9. The equivalent fat content, as a percentage of body-weight, for a range of values for the sum of four skinfolds (biceps, triceps, subscapular and supra-iliac) of males and females of different ages

Skinfolds (mm)	Males (age in years)				Females (age in years)			
	17-29	30-39	40-49	50+	16-29	30-39	40-49	50+
15	4.8	—	—	—	10.5	—	—	—
20	8.1	12.2	12.2	12.6	14.1	17.0	19.8	21.4
25	10.5	14.2	15.0	15.6	16.8	19.4	22.2	24.0
30	12.9	16.2	17.7	18.6	19.5	21.8	24.5	26.6
35	14.7	17.7	19.6	20.8	21.5	23.7	26.4	28.5
40	16.4	19.2	21.4	22.9	23.4	25.5	28.2	30.3
45	17.7	20.4	23.0	24.7	25.0	26.9	29.6	31.9
50	19.0	21.5	24.6	26.5	26.5	28.2	31.0	33.4
55	20.1	22.5	25.9	27.9	27.8	29.4	32.1	34.6
60	21.2	23.5	27.1	29.2	29.1	30.6	33.2	35.7
65	22.2	24.3	28.2	30.4	30.2	31.6	34.1	36.7
70	23.1	25.1	29.3	31.6	31.2	32.5	35.0	37.7
75	24.0	25.9	30.3	32.7	32.2	33.4	35.9	38.7
80	24.8	26.6	31.2	33.8	33.1	34.3	36.7	39.6
85	25.5	27.2	32.1	34.8	34.0	35.1	37.5	40.4
90	26.2	27.8	33.0	35.8	34.8	35.8	38.3	41.2
95	26.9	28.4	33.7	36.6	35.6	36.5	39.0	41.9
100	27.6	29.0	34.4	37.4	36.4	37.2	39.7	42.6
105	28.2	29.6	35.1	38.2	37.1	37.9	40.4	43.3
110	28.8	30.1	35.8	39.0	37.8	38.6	41.0	43.9
115	29.4	30.6	36.4	39.7	38.4	39.1	41.5	44.5
120	30.0	31.1	37.0	40.4	39.0	39.6	42.0	45.1
125	30.5	31.5	37.6	41.1	39.6	40.1	42.5	45.7
130	31.0	31.9	38.2	41.8	40.2	40.6	43.0	46.2
135	31.5	32.3	38.7	42.4	40.8	41.1	43.5	46.7
140	32.0	32.7	39.2	43.0	41.3	41.6	44.0	47.2
145	32.5	33.1	39.7	43.6	41.8	42.1	44.5	47.7
150	32.9	33.5	40.2	44.1	42.3	42.6	45.0	48.2
155	33.3	33.9	40.7	44.6	42.8	43.1	45.4	48.7
160	33.7	34.3	41.2	45.1	43.3	43.6	45.8	49.2
165	34.1	34.6	41.6	45.6	43.7	44.0	46.2	49.6
170	34.5	34.8	42.0	46.1	44.1	44.4	46.6	50.0
175	34.9	—	—	—	—	44.8	47.0	50.4
180	35.3	—	—	—	—	45.2	47.4	50.8
185	35.6	—	—	—	—	45.6	47.8	51.2
190	35.9	—	—	—	—	45.9	48.2	51.6
195	—	—	—	—	—	46.2	48.5	52.0
200	—	—	—	—	—	46.5	48.8	52.4
205	—	—	—	—	—	—	49.1	52.7
210	—	—	—	—	—	—	49.4	53.0

In two-thirds of the instances the error was within $\pm 3.5\%$ of the body-weight as fat for the women and $\pm 5\%$ for the men.

Reference:

Durnin, J. V., & Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: Measurements on 481 men and women aged from 16-72 years. British Journal of Nutrition, 32, 77-96.

PLEASE NOTE

Copyrighted materials in this document have not been filmed at the request of the author. They are available for consultation, however, in the author's university library.

pp. 108-111

University Microfilms International

Appendix E

Assay Procedures For Plasma Glucose, FFA, And LactatePlasma Glucose

This procedure is described in the Sigma Diagnostics Instruction Booklet (Procedure No. 16-UV, Quantitative, Enzymatic [Hexokinase] Determination of Glucose in Plasma at 340 nm), P.O. Box 14508, St. Louis, MO 63178, 1988. The enzymatic reaction involved in the assay begins when glucose is phosphorylated by adenosine triphosphate (ATP), in the reaction catalyzed by hexokinase (HK). The glucose-6-phosphate (G-6-P) is then oxidized to 6-phosphogluconate (6-PG) in the presence of nicotinamide adenine dinucleotide (NAD). This reaction is catalyzed by glucose-6-phosphate dehydrogenase (G-6-PDH). During this oxidation, an equimolar amount of NAD is reduced to NADH. The consequent increase in absorbance at 340 nm is directly proportional to glucose concentration.

Reagents: The Glucose (HK) reagent, when reconstituted according to the directions (deionized water), contains non reactive stabilizers and fillers plus the following concentrations of active ingredients:

NAD	1.5 mmol/L
ATP	1.0 mmol/L
Hexokinase (yeast)	1000 units/L
G-6-PDH (L.m.)	1000 units/L
Magnesium ions	2.1 units/L
Buffer	pH 7.5±0.1

Procedure:

1. Prepare Glucose (HK) reagent according to the instructions.
2. Add 1.5 nL of Glucose (HK) reagent into a properly labeled cuvet.
3. Read and record absorbance (A) at 340 nm vs water as reference. This is INITIAL A.
4. Add 0.01 nL (10 uL) of sample. Mix by gentle inversion.
5. Incubate cuvetts for 5 minutes at room temperature (23 degrees centigrade). Read and record the absorbance (A) at 340 nm vs water as a reference. This is FINAL A. Following the completion of reaction, FINAL A remains constant for 60 minutes.
6. Subtract INITIAL A from FINAL A to obtain change in absorbance (change A).
7. To determine glucose concentration of sample, use the following calculations based on a glucose standard (standard cat. no. 16-100; which provides 100 mg/dL standard):

$$\frac{\text{FINAL } A_{\text{sample}} - \text{INITIAL } A_{\text{sample}}}{\text{FINAL } A_{\text{standard}} - \text{INITIAL } A_{\text{standard}}} \quad * \text{ Concentration of Standard}$$

Example:

$$\begin{aligned} \text{INITIAL A (sample and standard)} &= 0.106 \\ \text{FINAL } A_{\text{sample}} &= 0.362 \\ \text{FINAL } A_{\text{standard}} &= 0.415 \\ \text{Glucose CONcentration} &= 0.362 - 0.106 \\ \text{(mg/dL) of sample} &= \frac{0.362 - 0.106}{0.415 - 0.106} * 100 = 83 \end{aligned}$$

Values were then converted to S.I. Units by multiplying the glucose value (mg/dL) by 0.05551.

Appendix E**Free Fatty Acids**

The following reference was used to determine the procedures for this colorimetric free fatty acid assay:

Noma, A., Okabe, H., & Kita, M. (1973). A new colorimetric micro-determination of free fatty acids in serum. Clinica Chimica Acta, 43:317-320.

Reagents:

Extraction solvent: Chloroform-heptane (I:I/V/V) containing 2% methanol reagent grade.

Copper reagent: 10 ml of 10 Ml aqueous $\text{Cu}(\text{NO}_3)_2$ and 5 ml of triethanolamine are diluted with saturated sodium chloride solution to 100 ml. The pH is adjusted to 8.3 with 1 N sodium hydroxide.

TAC solution: 10 mg of 2-(thiozolyazo)-p-cresol (TAC) is dissolved in 100 ml of ethanol and is filtered (coffee filter).

Procedure:

100 ul of plasma is transferred to a glass-stoppered test tube containing 3.0 ml of extraction solvent and followed by 1.0 ml of copper reagent. The tube is immediately stoppered and shaken mechanically for 2 minutes, and centrifuged at 3000 rev./min. for 5 minutes. 2 ml of the upper phase are transferred to a second test tube containing 0.5 ml of TAC solution. After careful mixing, the greenish blue colour developed immediately is measured at 610 nm against a reagent blank. Only quartz cuvetts can be used.

Calculations:

A standard curve was made using palmitic acid (molecular weight = 256.4 g/mol). The standard reference line was determined from the following standard concentrations: .25, .5, .75, 1.0, 1.25, 1.50, 1.75, and 2.00 mmol/L.

Appendix E

Plasma Lactate

The plasma lactate assay utilized the Sigma Diagnostics kit (826-UV) with modified (1989) instructions. The Sigma technique utilizes the enzyme lactate dehydrogenase (LDH), which catalyzes the following reaction: nicotinamide adenine dinucleotide (NAD) and lactate produce pyruvate and the reduced form of NAD (NADH) via the enzyme LDH. In order to force the reaction to completion, it is necessary to trap formed pyruvate with hydrazine. The increase absorbance at 340 nm due to NADH formation becomes a measure of lactate originally present.

Procedure:

1. Reconstitute appropriate number of NAD vials, Catalog No. 260-11-, required by pipeting into each of the following:
 - 2.0 mL Glycine Buffer, Cat No. 826-3
 - 4.0 mL deionized waterCap and invert the vials to mix.
2. Pipet 1.45 mL into Blank and Test cuvetts.
3. To Blank cuvet add 0.025 mL deionized water.
To Test cuvet add 0.025 mL plasma sample.
Mix gently inverting against Parafilm[®].
4. Incubate Blank and Test cuvetts for approximately 5 minutes. Read and record absorbance of Test at 340 nm vs Blank as reference. This is the INITIAL A.
5. Add 0.25 mL Lactate Dehydrogenase, Cat. No. 826-6, to Blank and Test cuvetts. Mix gently by inversion
6. Incubate Blank and Test cuvetts for 30 minutes at 25 degrees Centigrade.
7. Read and record absorbance of Test at 340 nm vs Blank as reference. Complete reading within 10 minutes. This is the FINAL A.

Calculation Using Lactate Acid Standard:

A standard curve was made using the Lactic Acid Standard, Cat. No. 826-10 = 40 mg/dL. Conversion to S.I. Units was performed by multiplying the mg/dL value by the conversion factor of 0.1110 to get mmol/L.

Appendix F

Plasma Volume Determination Procedures

The indirect determination of plasma volume shifts were determined using the procedures from the following reference:

Dill, D. B., & Costill, D. L. (1974). Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. Journal of Applied Physiology, 37(2): 247-248.

In order to use these calculations, the determination of hemoglobin and hematocrit for each blood sample is required. A reference blood sample is pick to represent a 100% plasma volume, to which all other plasma volumes are compared to (this is usually the resting or pre blood sample). In the following example three blood draws have been taken, with the first draw being used as the reference (resting) sample. For these three blood draws (ie, 1, 2, and 3), we will identify their hemoglobin and hematocrit values with the corresponding draw number at the end of the abbreviation (i.e., hemoglobin value from the first blood draw = HB1; and hematocrit value for the second blood draw = HCT2; etc.). The calculations are as follows:

Blood Volume After the resting..... $BVA1=100*(HB1/HB2)$;
 Blood Volume After number 2..... $BVA2=100*(HB1/HB3)$;

Cell Volume After the resting..... $CVA1=BVA1*(HCT2/100)$;
 Cell Volume After number 2..... $CVA2=BVA2*(HCT3/100)$;

Plasma Volume After the resting..... $PVA1=BVA1-CVA1$;
 Plasma Volume After number 2..... $PVA2=BVA2-CVA2$;

All Plasma Volume Before..... $PVB=(100-HCT1)$;

Plasma Volume next..... $PV11=100*(PVA1-PVB)$;
 Plasma Volume next..... $PV22=100*(PVA2-PVB)$;

Plasma Volume 1...actual shift..... $PV1=(PV11/PVB)+100$;
 Plasma Volume 2...actual shift..... $PV2=(PV22/PVB)+100$;

Plasma Volume Shift 1..... $PVS1=PV1/100$;
 Plasma Volume Shift 2..... $PVS2=PV2/100$;

$PVS1 * \text{blood parameter from the second draw} = \text{blood parameter corrected for plasma volume shift.}$

Appendix G

Descriptive Data for Preliminary Sample (n=21)

Variable	N	Mean	Standard Deviation	Minimum Value	Maximum Value
Age (yr)	21	25.9	4.1	19.0	35.0
Height (cm)	21	179.2	7.8	165.0	195.0
Weight (kg)	21	70.9	10.0	55.8	100.3
% Body Fat (hydrostatic)	21	9.5	3.4	3.6	15.7
Total Blood Cholesterol (mg%)	20	176.0	39.8	111.4	263.0
HDL Cholesterol (mg%)	20	52.2	13.1	32.6	84.2
Serum Ferritin (ng/ml)	20	48.4	38.9	10.0	165.0
Bike VO ₂ max (ml/kg/min)	20	64.4	7.8	47.0	76.4
Run VO ₂ max (ml/kg/min)	19	67.1	4.3	59.0	74.2
Trial Performance Time (min)	19	197.2	20.1	135.0	232.0

Appendix H

Individual Performance Data of Potential Subjects (n=21)

ID	B-VO ₂ max ml/kg/min	R-VO ₂ max ml/kg/min	TRIAL PERFORMANCE TIME minutes
1	56	60	195
2	60	61	200
3	63	69	190
4	64	64	210
5	67	66	210
6	60	65	197
7	66	68	194
8	75	71	218
9	68	71	195
10	73	74	215
11	70	69	200
12	76	74	232
13	56	59	181**
14	52	64	181**
15	69	71	180**
16	47*	na	na
17	74	68	202
18	67	68	200
19	62	66	211***
20	na	na	na***
21	63	66	135****

* eliminated from study because VO₂max < 50 ml/kg/min.

** eliminated from study because of lower trial performance test times.

*** subjects withdrew from the study because of the large time commitment required.

**** eliminated from study because of knee problems associated with test protocol.

na data not available.

Appendix I

Individual Descriptive Data of Potential Subjects (n=21)

ID	AGE yr	HT cm	WT kg	% BF hydro	TC mg%	HDL mg%	FERRITIN ng/ml
1	22	170	71	12.9	174.6	48.8	94.2
2	23	188	82	12.1	195.0	59.2	93.1
3	19	186	73	09.3	158.4	43.1	17.5
4	26	182	72	09.3	178.8	56.4	42.4
5	31	174	68	15.7	168.5	60.0	45.0
6	28	183	65	05.2	158.8	53.3	92.0
7	20	177	62	06.4	166.6	44.0	21.7
8	25	191	71	08.8	150.6	84.2	36.1
9	26	165	56	04.3	167.1	54.4	55.2
10	28	171	58	13.7	204.9	40.0	23.7
11	28	196	85	10.1	160.6	48.4	18.1
12	23	184	75	05.8	140.4	44.0	15.2
13	29	183	72	11.6	111.4	38.2	63.0
14	35	178	72	09.0	166.6	73.9	31.1
15	31	175	73	07.8	257.0*	62.2	74.7
16	31	186	100	13.1	187.9	33.6	165.0
17	21	169	56	07.0	263.0*	63.6	25.6
18	23	181	75	11.2	239.5*	58.4	10.0
19	26	171	71	14.1	129.7	32.6	25.0
20	24	179	63	03.6	140.6	46.0	20.0
21	25	177	69	08.9	na	na	na

* eliminated due to TC > 240 mg%.

na data not available.

Appendix J.

Individual Performance Test Times (minutes) from Each Treatment From RIDE-1 (n=8)

ID	PLACEBO	SLURRIED	SOLID
1	190.4	202.0	210.6
2	180.2	208.0	208.1
5	337.4	195.5	292.2
6	213.5	210.5	233.1
8	212.0	210.7	220.6
9	197.1	180.3	202.9
10	251.2	225.1	180.0
12	210.1	213.4	230.1
Mean±S.E.M.	224.0±17.8	205.7±4.7	222.2±11.6
Overall Mean =	217.3±7.2		

Appendix K

Individual Run and Bike VO₂max Values (ml/kg/min) at Weeks 0, 5, and 11 (n=8)

ID	TREADMILL			CYCLE		
	0	5	11	0	5	11
1	60.4	61.0	62.5	55.8	56.0	57.5
2	61.7	63.0	66.5	61.8	63.0	66.9
5	66.3	70.0	66.7	66.7	68.4	64.0
6	65.0	60.0	61.9	60.6	61.0	62.0
8	72.4	67.5	71.2	75.0	74.0	73.5
9	71.0	69.0	75.0	68.1	74.0	69.6
10	74.2	75.0	74.4	72.7	71.0	68.0
12	73.5	78.2	78.0	76.4	74.3	73.5

Appendix L

Raw Data For Each Subject (n=12)

All of the following data has been corrected for plasma volume shifts. The variables and their units are as follows:

ID=1-12 (subject #7 has no data);
 Performance Test (PT)=1-4;
 Supplement (SUPP)=R(CAPS-ergogenic) or B(placebo);
 Treatment (BAN)=1(placebo), 2(slurried), & 3(solid);
 Glucose (G)=mmol/L;
 Free Fatty Acids (FFA)=mmol/L;
 Lactate(LA)=mmol/L;
 Plasma Volume Shifts (PVS)=%;
 Hemoglobin (HB)=g/dL;
 Hematocrit (HCT)=%;

Row one consists of the ID, PT, SUPP, BAN, and 8 [glucose];
 Row two consists of 8 [free fatty acid];
 Row three consists of 8 [lactate];
 Row four consists of 8 plasma volume shifts;
 Row five consists of 8 [hemoglobin];
 Row six consists of 8 [hematocrit];

Any period (.) indicates no data.

1	1	R	3	4.754	4.496	4.268	3.598	3.684	3.389	4.678	3.808
				0.351	0.504	0.779	0.560	0.809	0.991	1.098	0.744
				1.864	1.171	0.934	0.625	2.211	1.772	1.098	0.985
				0	-16.8	-13.1	-18.4	-9.0	-16.3	0.7	-8.4
				14.0	15.1	15.0	15.4	13.8	15.0	13.9	14.5
				42	48	46	48	48	48	42	45
1	2	N	1	4.754	.	5.145	.	.	3.850	3.704	3.269
				0.406	.	1.022	.	.	1.184	1.023	1.011
				2.214	.	1.334	.	.	2.044	0.993	2.154
				0	.	-10.4	.	.	-16.4	-14.2	-6.3
				14.5	.	16.2	.	.	17.0	16.9	15.8
				50	.	50	.	.	51	50	49
1	3	N	2	4.876	4.657	5.304	.	.	4.096	4.924	3.786
				0.214	0.605	0.911	.	.	0.766	1.308	0.817
				1.597	1.570	1.633	.	.	2.037	1.302	0.965
				0	-8.5	-4.9	.	.	-12.0	0	-10.2
				13.4	14.4	13.6	.	.	14.7	13.4	15.2
				43	44	45	.	.	45	43	42
1	4	B	3	4.076	4.484	4.431	4.215	.	3.961	3.435	4.083
				0.195	0.172	0.323	0.439	.	0.848	1.056	0.423
				2.016	1.298	1.981	1.710	.	1.319	2.396	1.716
				0	-6.62	-9.54	-0.70	.	-1.63	-1.63	-4.9
				14.1	15.1	15.3	14.2	.	14.6	14.6	15.1
				46	46	47	46	.	45	45	45
2	1	B	3	5.191	.	5.407	4.254	4.015	3.427	4.472	5.566
				0.080	.	0.554	0.346	0.736	1.098	1.271	0.953
				1.833	.	1.128	2.262	1.577	3.216	2.217	1.773
				0	.	-8.64	-14.4	-13.3	-12.7	-5.45	-4.7
				14.3	.	14.8	15.5	15.3	14.9	14.3	14.2
				45	.	48	49	49	50	48	48
2	2	N	2	5.021	5.248	5.536	.	.	4.909	4.460	5.369
				0.189	0.358	0.880	.	.	1.656	1.562	1.319
				1.186	0.796	0.838	.	.	1.169	1.422	1.670
				0	4.538	4.696	.	.	2.721	2.721	1.533
				15.1	15.0	14.7	.	.	14.7	14.7	14.3
				48	46	47	.	.	48	48	50

Appendix L

2	3	N	1	4.899	4.835	4.613	4.458	.	3.986	.	3.025
				0.163	0.224	0.791	.	.	1.172	.	0.761
				1.399	1.329	1.141	1.506	.	1.915	.	1.905
				0	-8.13	-3.47	-8.98	.	-6.60	.	-6.20
				13.9	14.6	14.4	14.2	.	14.1	.	14.3
				43	45	43	47	.	46	.	45
2	4	R	3	4.852	4.656	4.784	.	.	4.412	6.123	5.172
				0.180	0.183	0.378	.	.	1.114	1.027	0.563
				0.733	0.898	1.541	.	.	0.987	2.062	1.341
				0	-5.91	-3.78	.	.	3.363	6.045	4.520
				14.0	14.6	15.1	.	.	13.8	13.7	13.9
				47	48	45	.	.	46	45	45
3	1	R	1	4.391	4.876	5.885	4.283	.	4.623	5.488	2.860
				0.193	0.359	0.531	0.375	.	0.631	1.062	0.633
				2.571	0.767	1.319	0.944	.	2.594	1.937	1.004
				0	0	18.91	-8.50	.	-10.0	-3.71	-4.91
				15.5	15.5	14.0	16.0	.	16.6	15.8	16.0
				46	46	42	49	.	48	47	47
3	2	N	3	4.536	4.424	4.461	.	.	3.716	4.954	4.140
				0.233	0.212	0.493	.	.	1.493	1.820	1.293
				2.735	1.155	1.089	.	.	1.463	2.191	1.776
				0	-6.48	-0.04	.	.	1.463	17.37	-7.73
				15.5	17.2	15.8	.	.	14.7	14.7	16.8
				47	45	46	.	.	49	41	47
3	3	N	2	4.561	4.359	4.070	2.566	.	4.480	6.946	5.093
				0.421	0.291	0.381	0.389	.	0.925	1.067	0.408
				3.560	0.753	0.781	0.763	.	0.377	1.831	1.615
				0	-5.40	-4.10	-12.5	.	-17.5	-1.93	-10.2
				14.0	14.8	14.6	15.7	.	16.0	15.1	15.9
				48	48	48	49	.	51	45	47
3	4	B	1	4.439	4.265	4.188	3.517	.	2.867	3.919	2.616
				0.253	0.139	0.263	0.241	.	0.449	1.032	0.971
				1.558	1.418	1.358	1.085	.	2.440	2.013	1.939
				0	4.651	-11.4	-16.6	.	-15.5	3.571	-18.3
				13.5	12.9	14.4	15.3	.	15.1	14.0	15.3
				46	46	49	49	.	49	42	50
4	1	B	2	5.312	6.736	5.935	5.253	4.985	4.429	5.423	4.792
				0.268	0.516	0.651	0.490	0.815	0.807	0.928	0.858
				1.129	0.954	1.090	2.330	1.678	1.061	1.005	1.833
				0	-0.11	-1.34	-9.39	8.727	-6.36	1.621	-4.54
				14.7	15.0	14.9	15.6	14.3	15.7	15.3	15.4
				48	47	48	50	45	48	45	48
4	2	N	1	5.7	6.189	6.129	.	.	5.523	5.568	3.471
				0.430	0.360	0.548	.	.	0.747	0.607	1.044
				1.698	1.368	0.049	.	.	2.873	1.827	1.544
				0	-1.48	1.886	.	.	-20.3	-11.7	-10.5
				14.6	15.1	14.6	.	.	17.3	15.6	15.4
				47	46	46	.	.	50	50	50
4	3	N	3	5.021	5.863	5.717	.	.	4.660	4.941	4.524
				0.824	1.230	1.122	.	.	0.733	0.646	0.849
				2.735	0.810	2.234	.	.	1.029	0.991	0.933
				0	11.92	39.32	.	.	-14.9	-13.3	-14.4
				14.4	13.1	10.7	.	.	15.4	15.1	15.3
				45	44	43	.	.	50	50	50
4	4	R	2	5.094	5.086	4.673	4.676	4.520	3.998	5.723	4.796
				0.274	0.237	0.360	0.519	0.605	0.848	1.012	0.871
				3.145	0.482	0.607	0.656	0.903	1.084	1.057	1.005
				0	-4.69	-13.2	-9.91	-4.91	-15.4	4.411	-4.00
				14.2	14.9	14.9	15.2	15.2	15.6	13.6	14.0
				44	44	49	46	43	48	44	47

Appendix L

5	1	B	3	4.852	2.788	4.560	3.647	4.229	2.475	4.000	3.556
				0.270	0.876	0.437	0.385	0.413	0.947	0.765	0.749
				1.158	1.668	1.131	0.653	0.806	1.453	2.850	2.628
				0	-23.8	-24.8	-25.1	-10.6	-20.2	-21.8	-22.8
				12.5	15.8	15.7	16.4	14.5	15.1	15.7	15.3
				46	48	49	47	44	48	47	49
5	2	N	2	5.312	.	5.005	.	.	5.791	5.958	4.487
				0.159	.	0.309	.	.	0.532	0.594	0.697
				1.490	.	1.105	.	.	2.989	4.430	1.509
				0	.	-15.4	.	.	-12.2	-9.35	-9.32
				14.7	.	16.4	.	.	15.8	15.3	15.6
				47	.	50	.	.	50	50	49
5	3	N	1	4.706	4.877	5.337	4.183	.	3.889	4.623	3.834
				0.184	0.212	0.281	0.293	.	.	0.761	0.805
				0.709	1.069	1.112	0.744	.	2.334	1.178	1.331
				0	-10.6	-3.5	-17.46	.	-10.3	-0.71	-16.3
				13.8	14.6	14.3	15.2	.	15.4	13.9	16.2
				45	48	45	50	.	45	45	46
5	4	R	3	5.385	6.056	5.423	.	.	4.467	5.705	4.553
				0.180	0.322	0.366	.	.	0.799	0.776	0.759
				1.433	1.920	1.394	.	.	0.760	1.721	1.451
				0	2.328	0.712	.	.	2.328	-1.18	-10.1
				12.4	11.7	12.1	.	.	11.7	11.9	11.9
				42	44	43	.	.	44	45	50
6	1	R	2	4.657	5.218	4.871	3.380	3.361	3.585	3.275	4.236
				0.521	0.697	0.906	0.892	1.396	1.022	1.556	0.998
				1.090	2.627	1.866	2.518	2.148	2.771	2.156	1.772
				0	-3.97	-0.55	-6.45	13.60	4.814	13.46	-9.51
				14.5	15.1	14.3	15.5	13.5	14.1	14.5	15.1
				48	48	49	48	45	47	41	51
6	2	N	1	4.899	.	5.089	.	.	2.924	4.115	2.928
				0.293	.	0.605	.	.	0.537	0.720	1.128
				1.746	.	1.375	.	.	.	3.256	3.557
				0	.	-9.17	.	.	-21.1	-10.2	-14.4
				13.7	.	14.8	.	.	16.4	14.4	15.1
				47	.	48	.	.	50	50	50
6	3	N	3	4.876	4.824	4.090	3.567	.	2.604	5.506	4.721
				0.444	0.343	0.431	0.624	.	1.026	0.871	1.081
				1.732	1.465	1.325	1.364	.	3.258	2.131	1.293
				0	-3.90	-10.7	-12.4	.	-18.0	2.251	-4.59
				14.2	14.0	14.8	14.8	.	15.2	13.4	14.1
				43	46	47	48	.	50	45	46
6	4	B	2	4.391	4.774	4.895	4.106	4.154	3.674	4.286	4.649
				0.447	0.367	0.739	0.696	0.993	1.025	1.137	0.828
				1.708	2.072	0.930	2.156	1.860	3.764	1.976	2.046
				0	-3.04	0.394	-4.90	7.713	-4.14	-2.91	-7.40
				14.7	14.6	14.1	14.6	13.9	14.2	14.3	14.7
				46	48	48	49	45	50	49	50
8	1	B	1	4.731	5.193	5.003	3.970	3.942	3.914	5.160	4.621
				0.327	0.513	1.077	0.630	0.653	0.783	0.811	0.714
				0.989	0.689	0.734	0.676	0.780	0.987	0.948	0.654
				0	-13.3	-6.66	-13.3	-16.6	-15.0	-14.5	-18.2
				14.0	15.0	15.0	15.3	15.6	15.6	15.8	15.9
				44	48	44	47	48	47	46	48
8	2	N	2	5.361	4.413	6.263	5.319
				0.236	1.077	0.459	0.971
				0.931	1.685	1.539	1.191
				0	-9.02	-1.81	-4.64
				13.5	14.3	13.5	13.9
				45	47	46	46

Appendix L

8 3 N 3	4.391	5.139	5.440	.	.	4.473	5.271	5.052
	0.344	0.284	0.772	.	.	0.999	0.547	1.236
	2.803	1.739	3.650	.	.	5.293	3.701	2.276
	0	-0.98	-1.21	.	.	-6.39	-4.69	-1.29
	14.5	14.4	13.7	.	.	14.2	14.2	14.2
	40	41	44	.	.	45	44	42
8 4 R 1	4.391	4.458	4.376	4.059	4.138	3.210	3.768	3.171
	0.263	.	.	0.888	0.697	0.605	0.563	0.814
	0.709	0.638	0.813	0.423	0.596	0.496	0.777	0.718
	0	-15.6	-15.6	-9.55	-14.6	-18.2	-17.8	-17.2
	14.2	15.7	15.7	15.7	15.8	16.2	16.4	16.0
	41	45	45	41	44	45	44	45
9 1 R 3	4.439	4.439	4.504	5.140	4.819	5.192	5.353	.
	0.221	0.172	0.349	0.652	0.172	0.521	0.300	.
	3.469	1.302	1.012	1.196	2.689	2.603	1.650	.
	0	0	-19.6	2.866	-22.6	-0.44	-12.7	.
	15.9	15.9	18.7	16.3	17.2	15.1	16.9	.
	45	45	48	42	54	48	49	.
9 2 N 2	4.682	.	4.818	.	.	4.811	.	3.413
	0.349	.	0.455	.	.	0.662	.	.
	3.049	.	0.878	.	.	2.904	.	.
	0	.	-3.11	.	.	-12.6	.	-12.6
	15.5	.	16.3	.	.	16.4	.	16.4
	47	.	46	.	.	51	.	51
9 3 N 1	4.973	4.688	4.643	.	.	5.435	5.224	4.679
	0.493	0.582	0.884	.	.	0.956	1.179	1.142
	1.717	0.804	0.700	.	.	2.828	1.639	1.281
	0	-1.88	2.976	.	.	-5.45	24.47	-5.45
	15.6	15.9	15.7	.	.	17.1	13.9	17.7
	45	45	43	.	.	43	39	41
9 4 B 3	4.464	4.525	4.436	4.338	.	4.698	3.841	5.091
	0.280	0.238	0.342	0.392	.	0.745	0.785	0.779
	2.619	0.975	0.767	1.348	.	0.487	3.486	2.436
	0	-2.32	-11.6	-5.87	.	-9.06	-15.7	-5.45
	12.6	12.9	14.0	12.9	.	13.1	13.6	12.6
	45	45	46	47	.	48	50	48
10 1 B 1	5.579	6.980	4.908	5.280	4.380	3.285	3.474	3.126
	0.120	0.303	0.444	0.623	0.548	0.892	0.893	0.970
	2.846	1.310	1.777	1.008	0.942	1.746	1.616	1.223
	0	15.09	-6.74	-13.6	-17.9	-19.4	-14.7	-16.3
	13.9	13.1	14.4	15.0	15.5	15.2	15.2	15.2
	41	36	43	45	46	48	45	46
10 2 N 3	5.191	5.048	6.691	.	.	.	6.221	4.180
	0.120	0.126	0.248	.	.	.	0.373	0.851
	2.359	1.116	1.288	.	.	.	2.789	1.769
	0	1.027	27.11	.	.	.	-11.8	-21.2
	14.5	14.6	11.8	.	.	.	15.6	16.2
	42	41	40	.	.	.	45	49
10 3 N 2	5.604	4.975	.	4.987	4.606	5.879	4.962	3.962
	0.182	0.486	.	.	.	0.843	0.793	1.240
	1.553	1.313	.	1.167	1.096	1.488	.	1.341
	0	-10.4	.	-6.55	-13.2	1.825	-1.66	8.184
	15.1	15.2	.	15.1	15.7	14.1	14.6	13.5
	39	45	.	43	45	42	42	41
10 4 R 1	5.7	.	.	.	4.411	.	.	.
	0.123	.	.	.	0.224	.	.	.
	1.490	.	.	.	0.982	.	.	.
	0	.	.	.	-12.9	.	.	.
	14.0	.	.	.	15.5	.	.	.
	45	.	.	.	47	.	.	.

Appendix L

11 2 B 3	4.754	5.618	5.650	.	5.367	4.818	5.104	4.602
	0.208	0.222	0.342	.	0.299	0.528	0.726	0.560
	2.919	0.849	0.883	.	1.686	1.502	1.330	0.983
	0	6.733	10.91	.	-5.03	-4.48	-2.57	-6.93
	16.1	15.7	15.7	.	17.3	17.2	17.2	17.3
	51	49	47	.	50	50	49	51
11 3 N 1	4.439	5.937	5.484	.	4.420	3.610	4.216	3.318
	0.272	0.528	0.289	.	0.924	0.771	0.968	0.987
	1.051	0.441	1.010	.	1.334	0.258	0.473	.
	0	14.38	11.39	.	7.189	1.234	-1.79	-0.13
	16.4	14.9	15.3	.	15.9	16.2	16.7	16.1
	49	47	47	.	47	49	49	50
11 4 R 2	4.512	4.731	4.657	4.104	4.053	3.585	6.393	5.086
	0.323	0.432	0.535	0.493	0.390	0.656	0.670	0.674
	1.244	1.146	2.111	1.622	1.981	2.529	2.801	1.585
	0	-9.69	-2.54	6.410	9.224	-2.74	4.996	16.08
	16.6	17.3	16.7	15.6	14.9	16.4	15.5	14.3
	49	52	50	49	50	51	50	49
12 1 R 2	4.148	5.037	4.671	.	3.871	4.028	4.988	3.786
	0.447	0.563	0.641	.	.	0.632	0.705	0.517
	1.080	0.888	0.751	.	0.775	1.560	1.452	1.019
	0	-2.02	-3.22	.	-12.3	-13.9	0.314	-12.3
	14.5	14.8	14.7	.	15.6	15.9	15.0	15.6
	47	47	48	.	50	50	45	50
12 2 N 1	4.269	.	4.792	.	.	3.509	3.585	2.683
	0.204	.	0.624	.	.	0.779	0.591	0.821
	1.452	.	0.406	.	.	1.123	1.145	1.199
	0	.	-12.1	.	.	-16.8	-10.4	-12.1
	14.7	.	15.5	.	.	16.7	15.5	15.5
	46	.	50	.	.	49	49	50
12 3 N 3	4.633	4.496	3.942	3.668	.	3.624	4.533	4.581
	0.295	0.263	0.279	0.573	.	0.749	0.761	0.600
	1.051	1.093	1.069	1.790	.	2.484	1.849	1.343
	0	-15.7	-17.9	-17.3	.	-14.6	-13.4	-11.3
	13.7	15.1	15.2	15.1	.	14.9	14.7	14.9
	44	48	49	49	.	48	48	46
12 4 B 2	4.172	.	4.066	3.694	3.301	2.601	3.914	2.850
	0.246	.	0.479	0.278	0.321	0.473	0.830	0.317
	1.196	.	1.416	0.866	1.080	1.485	1.175	1.011
	0	.	6.759	-2.37	-1.37	-0.69	8.304	-8.89
	14.3	.	13.9	15.2	14.5	14.4	14.2	15.4
	47	.	45	45	47	47	43	48