THE EFFECT OF A GLUCOSE POLYMER SUPPLEMENT DRINK, INGESTED IMMEDIATELY POST EXERCISE, ON TRAINING VOLUME

by

Philip Joseph Skinder
B.Sc., University of Victoria, 1989

A Thesis Submitted in Partial Fulfillment of the
Requirements for the Degree of

MASTER OF ARTS

in the School of Physical Education

We accept this thesis as conforming to the required standard

[redacted]

Dr. David Docherty, Supervisor (School of Physical Education)

[redacted]

Dr. Howard A. Wenger, Department Member (School of Physical Education)

[redacted]

Dr. John Hayward, Outside Member (Department of Biology)

[redacted]

Dr. David Moyer, External Member (Department of Anthropology)

Supervisor: Dr. David Docherty

ABSTRACT

This study investigated the effects of carbohydrate supplementation ($4 g \cdot kg^{-1}$ b.w. in a 25% solution; 96% polydextran, 4% fructose) immediately after exercise on training volume (TV) and net dietary carbohydrate intake (CI). Three experienced cyclists aged 34, 23 and 21 years ($\dot{V}O_2$ max = 50.9, 66.3 and 65.1 $ml \cdot min^{-1} \cdot kg^{-1}$) trained three cycles of five days (four training days and one rest day) under baseline (B), supplement (S) and baseline (B) conditions followed by three cycles of S alternated randomly with a fruit pectin thickened Placebo (P) in a double blind fashion. The first day of each training cycle commenced with a four hour easy ride (hr below 120 bpm), and was followed by three days on fixed resistance windtrainers. Training volume was defined as the distance cycled at prescribed heart rates corresponding to varying intensities between 60 and 88% of $\dot{V}O_2$max for 125 or 105 min per session. Heart rates were measured at five minute intervals while cycled distance and perceived exertion were measured at the beginning and end of each training interval. Dietary intake was recorded daily and computer analysed for content. Supplementation successfully raised CI above 10 $g \cdot kg^{-1} \cdot d^{-1}$ in 18 out of 21 sessions. Net intake ranged from 9.2 $g \cdot kg^{-1} \cdot d^{-1}$ to 16.6 $g \cdot kg^{-1} \cdot d^{-1}$ with mean carbohydrate intakes of 10.1 (0.6), 12.0 (1.1) and 11.3 (2.5) $g \cdot kg^{-1} \cdot d^{-1}$. Unsupplemented CI ranged from 3.9 to 12.3 $g \cdot kg^{-1} \cdot d^{-1}$ with mean intakes of 6.0 (0.6), 6.8 (1.4) and 8.7 (1.3) $g \cdot kg^{-1} \cdot d^{-1}$ per subject. After data were summarised in two and three day groupings, linear correlations of TV with CI were reported at $r = 0.94$ ($p < 0.01$) and $r = 0.87$ ($p < 0.05$) in two of three subjects. Total TV per cycle increased with supplementation from between 3.8 to 5.5% (4.71 - 5.54 km). No difference in perceived exertion was apparent between treatment groups. Single subject methodology was successfully utilised to demonstrate a causal relationship between dietary CI and TV. This supplement, ingested immediately post exercise, ensured optimal carbohydrate intake in the first hours after exercise and increased dietary intake to levels associated with maximum interworkout glycogen repletion. The resultant increases in dietary intake volume and immediate ingestion permitted these athletes to achieve greater cycling distance.

Examiners:

[redacted]

Dr. David Docherty, Supervisor (School of Physical Education)

[redacted]

Dr. Howard A. Wenger, Department Member (School of Physical Education)

[redacted]

Dr. John Hayward, Outside Member (Department of Biology)

[redacted]

Dr. David Moyer, External Member (Department of Anthropology)

LIST OF CONTENTS

CHAPTER II. METHODS

CHAPTER III. RESULTS

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGEMENTS

I would first like to acknowledge a very patient man, my supervisor Dr. David Docherty. I've learned a great deal from his humble, cheerful and non-judgemental support. Thanks to Gladys and Jill in the office, Wendy in the lab, Dr. Terry Pearson in Biochemistry, and all those others who, with their volunteer support, helped me perform this study successfully. To the men on my committee - thank-you for your most respected advice. Thanks to the sponsors of the study: Richard Pierce (Nutrition Health and Fitness) for the carbohydrates, Dirk Kessler (Intelligent Software) for the nutritional analysis program, Oak Bay Cycle for the help with the heart rate monitors and Procom (Dave Smith) for his cycling background and support with the stationary windtrainers.

Thanks to Martin Beras who's spirit and knowledge enriched the study from both experimental and human perspectives. Thanks to the men and women in my life who understood my struggles and provided whatever encouragement they could. Thanks to my Father and Mother, Peter and Elise who's love was always evident in their caring and concern and their willingness to do whatever made a difference. Most importantly, thanks to both my Creator and to my Lord for being beside me all the way.

Lastly, thanks to the brave men who endured the thirty days of gruelling training. To Peter, Andreas, Jon, Virgil, Paul and Andrew. I'll never forget the relationship we forged under the concrete overhang, on stationary bikes for longer periods of time than any of us ever wanted or will ever want to repeat again. Thank-you for your sweat, your hearts, your positive approach to each new day of training. Thank-you all.

CHAPTER I

INTRODUCTION

Extensive submaximal training is required by athletes to develop the cardiovascular and cellular physiology to meet maximal training and performance demands. In some sports, increasing aerobic capacity to competitive levels requires high training volumes at sub anaerobic threshold intensities. Athletes, such as competitive swimmers, cyclists and rowers, regularly train in excess of 20 hours per week for months at a time. During peak training phases these athletes can require up to 6000 kcal daily just to maintain body weight (Dengal, 1987; Sjogaard, 1986; Wheeler, 1989).

Athletes and coaches are aware of the detrimental effects that inadequate caloric and dietary carbohydrate intake has on training volume, but many athletes do not meet their carbohydrate needs (Brouns, Saris, Stroecken, Beckers, Thijssen, Rehrer, & F. ten Hoor, 1989; Burke & Read, 1988; Clark, Nelson & Evans, 1988; Short, S.H. & Short, W.R. 1983). Time, financial constraints, poor food choices and improper eating behaviours all interfere with achieving proper carbohydrate volume. (Wright, Sherman & Dernbach, 1991). Although adequate dietary carbohydrate can often be provided through correct food choices, athletes have recently been encouraged to supplement their diets with easily digestible carbohydrate drinks, in particular immediately after training, for optimal inter-workout glycogen repletion (Ivy, 1991; Ivy, Katz, Cutler, Sherman & Coyle, 1988; Wheeler, 1989;). Despite these recommendations little research has addressed the effect of such supplementation on training volume in a realistic setting over an extended period of time.

The importance of intramuscular glycogen stores during prolonged sub maximal exercise is well established. Exercise duration at submaximal intensities is directly related to pre-exercise intramuscular glycogen content and the rate at which glycogen is depleted (Bergstrom, Hermansen, Hultman, & Saltin, 1967). Fatigue results from depleting stores of muscle glycogen at intensities between 60 and 90% of $\dot{V}O_2$ max (Saltin & Karlsson, 1971).

Glycogen is the predominant catabolic substrate when training above 90 % of maximum aerobic output. At this intensity exhaustion occurs when lactate and subsequent proton generation impede sustained work which is often before full tissue glycogen depletion (Hermansen & Osnes, 1972; Sahlin, Edstrom, & Sjoholm, 1983). At exercise intensities less than 50% $\dot{V}O_2$ max, lipid stores provide enough energy to maintain exercise for hours without glycogen depletion. Between these two intensities, as exercise intensity increases linearly, muscle glycogen utilisation increases exponentially. The highest depletion rate attainable for sustained sub maximal work occurs about the anaerobic threshold (Saltin, 1971). Training continuously at this level results in the greatest tissue glycogen depletion before exhaustion.

Intense training can significantly deplete both liver and muscle glycogen. Unlike some animals, gluconeogenesis from lipid and protein stores does not generate a significant amount of glycogen in humans. Athletes depend on a daily dietary consumption of carbohydrates to provide the necessary substrate. Studies have shown that full inter-workout glycogen repletion can occur in 24 hours provided sufficient dietary carbohydrate is consumed in that time (Bergstrom, Hermansen, Hultman, & Saltin 1967; MacDougall, Ward, Sale, & Sutton, 1977) . Bergstrom et al., (1967) demonstrated that the ability to sustain prolonged exercise is directly related to muscle glycogen content and the pre exercise diet.

The "post workout" physiological environment is ideally suited for glycogen repletion because the post depletive muscle cell is in an optimal state for glucose absorption and glycogen synthesis. Cardiac output decreases after exercise but remain high relative to resting levels in the first hour after exercise thus maximising glucose delivery to the muscle. Muscle cells display an increased affinity for blood borne glucose due to increased membrane sensitivity and the local dilatory effect around active muscle (Ivy, Katz, Cutler, Sherman & Coyle, 1988; Piehl, 1974). Muscular contractions increase muscle permeability to glucose and increases insulin sensitivity. Irrespective of glycogen levels, increases in blood insulin levels increase both cellular glucose absorption and the percentage of glycogen synthase(I) levels (Lohmann, Liebold & Heilmann, 1978). Delaying carbohydrate intake after continuous exercise results in increases in free fatty acids from fatty acid breakdown, impairs glucose tolerance, and reduces insulin sensitivity (Ivy et al., 1988a). Conversely, carbohydrate intake maintains the desired environment by increasing plasma insulin and glucose concentration. Elevated splanchnic glucose output (SGO) increases and remains elevated to two times the resting levels during the recovery period immediately after exercise (Maehlum, Felig, & Wahren, 1978). Activity of the rate limiting enzyme for glycogenolysis, glycogen synthase, is inversely proportional to the degree of glycogen depletion and decreases after cessation of exercise.

A glycogen storage rate of 7.7 $\mu mol \cdot g \cdot wet\ wt^{-1} \cdot hr^{-1}$ has been observed to occur in glycogen depleted muscle with carbohydrate ingestion immediately after exercise (Ivy, et al., 1988a). Delaying ingestion two hours post exercise decreased the rate to 4.1 $\mu mol \cdot g \cdot wet\ wt^{-1} \cdot hr^{-1}$. Because the body regains homeostasis relatively soon after the cessation of exercise, carbohydrate ingestion has been recommended immediately after a glycogen depletive workout for optimal glycogenesis (Friedman, Neufer & Dohm, 1991).

If sufficient carbohydrate is not ingested between daily workouts, consistent daily declines in glycogen stores and subsequent decrements in training volume can develop (Costill et al., 1988; Costill, Bowers, Branam & Sparks, 1971). Sufficient carbohydrate intake has been expressed in terms of absolute mass, relative caloric value and as absolute mass per body weight. Costill et al., (1981) found that carbohydrate intakes for elite athletes in the range of 550 - 650 $g \cdot day^{-1}$, equating to roughly 70% of the daily caloric intake, are sufficient to increase muscle glycogen to pre exercise levels within 24 hours. They suggested that resynthesis quantity might be increased by intake of carbohydrate greater than this maximal level. Other studies (Bergstrom, Hultman, & Roch-Norland, 1972; Kochan, Lamb, Lutz, Perrill, Reimann & Schlender, 1977; Sherman & Wimer, 1991; Simonsen, Sherman, Lamb, Dernbach, Doyle & Strauss, 1990) reported that, over 7 days of aerobic training, athletes ingesting a diet including a carbohydrate intake of at least 10 $g \cdot kg^{-1}$ $bw \cdot d^{-1}$ prevented pre exercise glycogen levels from declining and in some cases allowed for increasing levels over the course of the study. An intake amount of 8 $g \cdot kg^{-1}$ $bw \cdot d^{-1}$ resulted in a slow progressive decline in muscle glycogen and intakes of 5 $g \cdot kg^{-1}$ $bw \cdot d^{-1}$ or less resulted in significantly faster declines.

Maximal glycogen synthesis rates have been attained at ingestion volumes of between 1.0 and 3.0 $g \cdot kg^{-1}$ immediately after and every 2 hours after exercise. Blom, Hostmark, Vaage, Kardel, and Maehlum (1987) suggested adequate carbohydrate intake as 0.7g glucose $\cdot kg^{-1}$ bw immediately and every two hours for the first 4 to 6 hours after exercise (500 - 700g carbohydrate total per 24 hours) enabled full glycogen repletion within 24 hours. Doubling the amount of carbohydrate consumed immediately after and every 2 hours for six hours following exhaustive exercise from 0.7 to 1.4 g glucose per kg body weight had minimal effect on glycogen storage, and decreasing intake to 0.35 $g \cdot kg^{-1}$ reduced the storage rate 50%. Ivy et al. (1988b) fed subjects either 0, 1.5 or 3.0 g. glucose polymers after depleting muscle glycogen with intermittent cycling exercise. After four hours muscle biopsies determined muscle glycogen synthesis rates of 0.5, 4.5 and 5.1 $mmol \cdot kg^{-1} \cdot hr^{-1}$ respectively. Blom (1989) fed or infused subjects with approximately 3.0 $g \cdot kg^{-1}$ carbohydrate evenly within 3 hours of exercise and found similar glycogen storage rates of 8.8 $mmol \cdot kg^{-1} \cdot h^{-1}$. In general, post exercise supplementation strategies designed to maximize glycogen repletion suggest intake of up to 6 $g \cdot kg^{-1}$ spaced every two hours immediately and until 4 hours after exercise.

Other consequences occur when athletes do not consume the daily required dietary volume of carbohydrate. Performance begins to plateau and the symptoms of fatigue (such as weight loss, loss of energy and predisposition to illness) begin to develop (Costill et al., 1988; Williams, 1985). Decrements in skill and coordination have also been attributed to the depletive state of glycogen reflecting the dependence of the central nervous system on adequate blood glucose (Kujala, Heinonen, Kuist, Karkkainen, Marniemi, Nittymaki & Havar, 1989). Chronic glycogen depletion can also interfere with anaerobic work because creatine phosphate and adenosine triphosphate regeneration is partially dependent upon adequate glycogen stores (Costill, 1988). Heigenhauser, Sutton & Jones (1983) investigated changes in the ventilatory response during exercise due to glycogen depletion. At any given power output, depleted state O_2 intake, heart rate, blood pH and ventilation were significantly higher than during the fully repleted condition. These results suggest that while training at a set heart rate, power output decreases with muscle glycogen depletion.

Millard - Stafford, Cureton, and Ray (1988) investigated the use of a carbohydrate supplement during eight days of training prior to a test triathalon (30 min of swimming, cycling and running at 70% $\dot{V}O_2$ max followed by a run to exhaustion at 90% $\dot{V}O_2$ max). This study attempted to provide an indication of how routine training and competition might be affected by a carbohydrate supplement but only measurements during the pre and post week criterion performances were taken. The supplement mass was 230 g irrespective of body weight, the time of ingestion was not mentioned, and neither training protocols nor training results were documented. Significant differences in performance were observed only in the run to exhaustion at the end of the supplemented eight training days relative to the run at the end of the unsupplemented eight days. While an effect on performance appeared evident the study did not directly determine an effect of carbohydrate supplementation on training.

Sherman and Wimer (1991) examined recent studies into the effects of carbohydrate intake on muscle glycogen and performance. Of the seven studies identified, four claimed to address the effect on training. No study showed a decrease in the ability to train when moderate ($8g \cdot kg^{-1} \cdot d^{-1}$) amounts of carbohydrate were ingested. They postulated that potential design shortfalls in the studies might not have allowed small dietary changes in carbohydrate intake to effect performance. Training protocols in these studies might not have been rigorous or long enough to provide the opportunity to observe an effect. They recommended further research to clarify the significance of short term deficits in carbohydrate intake on training and performance.

Instances of inadequate dietary intake for athletes are frequently reported. Clark, Nelson and Evans (1988) identified that a significant proportion of elite female runners believed they ate "wisely" but actually had suboptimal intakes. Costill (1988) described four swimmers who failed to recover between workouts as "insensitive to changes in their daily energy expenditure and had difficulty in maintaining a caloric and carbohydrate balance." A need exists to assist some elite athletes in meeting their dietary requirement of energy. Education of the athlete and continual dietary monitoring provides the best solution but a more pragmatic approach exists. Ingestion of a liquid carbohydrate supplement immediately post exercise to assist in optimal glycogen repletion between workouts offers a quick, easy method of ensuring adequate carbohydrate intake without any detrimental consequences.

Supplementing the diet of an athlete with a carbohydrate solution has been recommended as a viable method to assist the achievement of optimal dietary carbohydrate intake (Brouns et al., 1989a; Keizer, Kuipers, van Kranenburg and Geurten, 1987) . While acute studies suggest supplementation, none have tested the daily efficacy of such a drink in realistic training environments over a prolonged span of time. The design imposed a protocol of sufficient volume and intensity to detect differences in training volume due to changes in dietary carbohydrate intake. Based on the direct relationship between training heart rate and glycogen depletion mentioned earlier, training volume at set heart rates was chosen as the main dependent variable. Using training volume at set HR enabled continuous long-term data collection in a realistic setting and maximized the external validity of the study. A single - subject design was chosen for analysis for many reasons, most importantly since it closely represents the coach - athlete relationship present in the field. Complications experienced by athletes in training were permitted in this format since single subject design permits documentation and explanations of the individual case. Such flexibility in methodology closely mirrors the flexibility required in the training field. It was also a method to validate recommendations derived from group analysis to individual situations. Data collection was performed with equipment commonly utilised by elite cyclists in training. The glucose polymer supplement ($4\ g \cdot kg^{-1}\ bw \cdot d^{-1}$) was administered in one dose immediately post exercise instead of many doses distributed equally in the six hours post exercise. The single dose was chosen to ensure compliance and for that reason, considered the method of choice in the field.

Therefore, the purpose of this study was to investigate the effects of a liquid carbohydrate supplement ($4g \cdot kg^{-1}\ bw \cdot d^{-1}$), ingested immediately post exercise, on the quantity of submaximal exercise produced by trained cyclists in a successive series of six training cycles (5 days per cycle) comprising a rigorous 30 day training program.

Research Questions:

A. What is the effect of dietary carbohydrate intake on distance cycled:

1. Is there a difference in total cycling distance achieved during periods of dietary carbohydrate supplementation and no supplementation?

2. Is the effect of carbohydrate supplementation more pronounced:
   - on the second, third or fourth successive day of training?
   - over a greater span of days?

3. Irrespective of supplementation, is there a relationship between total dietary carbohydrate intake and cycled distance?

4. If supplementation raises carbohydrate intake over and above the suggested maximum amount (650g - according to Costill, 1988 or $10 g \cdot kg^{-1} \cdot day^{-1}$ according to Sherman & Wimer, 1991) will the relationship between carbohydrate intake and cycled distance change?

B. What is the effect of dietary supplementation on net dietary intake volumes and composition:

1. Will carbohydrate supplementation change overall dietary intake volume and/or composition?

2. Will a supplement of $4\ g \cdot kg^{-1} \cdot d^{-1}$ carbohydrate ensure that each athlete reaches an intake criterion of $10\ g \cdot kg^{-1} \cdot d^{-1}$.

C. Are there changes in perceived exertion with supplementation:

1. Are there differences in perceived exertion between training under dietary supplementation and training without supplementation?

Operational Definitions

$\dot{V}O_2$ max (maximal aerobic power) : the highest rate of oxygen uptake the individual can attain during physical work while breathing air at sea level (Astrand & Rodahl 1977), reflecting the ability of the oxygen transport system to take up, transport and utilise oxygen (Wenger & Bell, 1986).

Anaerobic Threshold (AT) : a value (gas exchange and /or blood - borne) which marks the level of exercise $\dot{V}O_2$ above which aerobic energy production is supplemented by anaerobic mechanisms, thus causing an increased lactate production relative to the rate of glycolysis (Wasserman, 1986).

Ventilatory Threshold (VT) : a gas exchange parameter used to mark A.T. V.T. is achieved at the point when minute ventilation (Ve - the volume of expired air per unit min ($L \cdot min^{-1}$)) accelerates from its continuous and exponential relationship with increasing power outputs (Janssen, 1989).

Sectional distances: the distances cycled within individual time segments occurring at quarter marks within the hour long ride.

Total Work: the sum of elapsed distance throughout the extent of a day within each training cycle.

Rate of Perceived Exertion (R.P.E.): - an indicator of physical strain on a ten point scale from 1-10 (Borg, 1982).

Training Drink: the liquid ingested during the measured training sessions (2.5% carbohydrate: 4% fructose, 96% glucose polymer).

Carbohydrate Supplement (Treatment Drink): the glucose polymer - fructose mixture (25% carbohydrate - 4% fructose, 96% glucose polymer) ingested post exercise at a volume based on a subject's weight (4.0 $g \cdot kg^{-1}$ b.w.)

Placebo Drink: a artifically sweetened and flavoured solution thickened with pectin to the same consistency as the treatment drink.

Training Segments: a block of time within a training session, from five to sixty min denoting training intensity.

Delimitations

1. All subjects were experienced cyclists with a minimum of two years training greater than 200 km per week for more than six months per year.

2. All subjects participated in the pre-test laboratory session, a three day pre-test dietary analysis and three trial training runs to familiarize them with the procedures used within the study .

3. All subjects participated fully over the 30 days encompassing the training period.

Limitations

1. The results of this study are applicable only to the individual subjects selected.
2. There may have existed inter - sessional differences in response to motivation during the training tests.
3. Training glycogen depletion and repletion were not assessed and could only be presumed based on comparisons with literature.

Assumptions

1. The subjects understood and followed all instructions during the testing period.

2. The participants continued with their normal diet.

3. There was a training effect over the testing period.

4. Differences in environmental and motivational conditions were uniform across the sample set and therefore did not interfere with the results.

5. The dietary log assumed equal proficiency in identification and recording across the sample pool.

6. Each athlete had an equal potential to improve over the training period.

7. No athlete suffered any clinical ailment which might prevent him from achieving a training effect or accomplishing the training protocol.

8. Each subject completed the training protocol to the best of his ability on each given day.

9. Subjects did not participate in any excess physical activity over and above the prescribed training program during the course of the experiment.

Statement of Variables and Controls:

Independent variables	Two treatments, one consisting of all data collected when the carbohydrate supplement was administered and the second consisting of all data collected when the placebo drink was administered. Training data were also collected during baseline periods.
Dependent variables:	1. Volume of work represented as distance cycled over set time periods and at set heart rate intensities near ventilatory threshold. 2. Daily psychological survey results (rate of perceived exertion - R.P.E.).
Placebo	- An artificially sweetened solution thickened with pectin to a consistency similar to the treatment solution (placebo). - With a single subject design the control group was internal - each subject acted as their own control.
Control variables:	- Restriction of exercise to treatment regimen. - Restricting subject pool to elite or experienced athletes familiar with high volume work. - Training intensities were monitored by a heart rate monitor throughout each training session. - Each ride was supplemented *ad libitum* with the same ergogenic drink (2.5% carbohydrate).
Extraneous variables:	- Dietary intake (addressed by recording daily dietary intake) - Motivational differences (reported in log entries).

CHAPTER II

METHODS

2.1 SUBJECTS

Seven competitive road and mountain bike riders were selected from an initial prospective sample pool of over forty local cyclists. Subjects were screened to include trained competitive cyclists between 20 and 35 years of age with at least two training and competitive seasons immediately preceding the study. Only three participants completed the full 30 day protocol and produced results acceptable enough for experimental purposes. Of the seven who started, two subjects missed a number of sessions due to sickness and two failed to meet all the requirements of the study. Results from these four subjects are not included in the report. The experimental protocol, with possible accompanying risks and discomforts, were explained to the volunteer subjects prior to signing the informed consent forms (Appendix F). Physical characteristics are summarised in Table 2.1. Experimental procedures were approved by the Human Subjects Committee at the University of Victoria. In the week prior to testing subjects were familiarised with procedures during a three day mock run.

2.2 EXPERIMENTAL DESIGN

A "realistic" training protocol was designed to establish the external validity in this study. The study was designed to validate recommendations in the literature for individual cases and to produce evidence beneficial to both coach and athlete. Because no invasive technique to validate tissue glycogen levels was used, the protocol had to be demanding enough to significantly deplete mucle glycogen on a daily basis. Training standards and the compliance level were stringently applied. Because of these factors and the limitations inherent in long term, relatively uncontrolled studies, it was difficult to obtain and retain a

Table 2.1
Means (SD) of individual physical characteristics, pre-training (n = 3)

Subject	Age (yrs.)	Ht. (cm)	Wt. (kg)	$\dot{V}O_2max$ (ml·kg^{-1}·min^{-1})	VT (ml·kg^{-1}·min^{-1})	S.S.F. (mm)	*Comp.Ex. (yrs.)
P.N.	35	164.8	70.5	51.3	39.7	40.0	3
A.H.	22	181.5	77.4	66.3	45.2	35.3	3
J.T.	24	182.0	82.3	65.1	52.2	38.5	5
Mean (SD)	27 (7)	176.1 (9.8)	76.7 (5.9)	60.9 (8.3)	45.7 (6.3)	37.9 (2.4)	4 (1)

*Comp. Ex. = competitive experience

large study group. Group analysis was not possible and did not suit the nature of the study. Single subject analyses best suited the format and intent of the design as it best mirrored the coach - athlete relationship.

Changes in the original proposed training protocol were made in response to the result of the three day "mock run". The following is a description of the adapted design.

A single subject, alternating treatment, baseline, placebo pattern (Figure 2.1) consisting of six separate training cycles of five days for a total of 30 days outlined the training format. A training cycle included four days of training (one day of recovery cycling on the road and three days of data collection on a stationary bicycle) followed by one day of complete rest. For the first three cycles all subjects participated in a single - blind, ABA design (baseline - treatment - baseline). During the next three cycles, subjects were randomly assigned treatment or placebo in a double-blind crossover design (Table 2.2). The treatment or placebo drink was administered immediately after each training session except for the last training session within each training cycle (Figure 2.2).

Ten days before starting the training sessions anthropometric data, body composition, maximal oxygen uptake ($\dot{V}O_2max$) and ventilatory threshold (VT) were assessed (Table 2.1). Target training heart rates were determined from the metabolic data. Training heart rates corresponding to 20%, 10% and 0% decrements below the volume of oxygen consumed at VT were interpolated from the graph of heart rate versus oxygen uptake (Table 2.3). Subjects were familiarised with the dietary recall process, including identification and recording procedures, by participating in a three day dietary assessment one week prior to the experiment. During this time a four day mock run of the four training protocols occurred (Table 2.4). As well as the implicit personal fitness gains over the test period, a cash incentive system provided motivation to promote maximum effort. Subjects were paid ten dollars per successful training session. A session was determined successful if target heart rates were reached in each training segment.

Figure 2.1

Daily description of training protocols - by cycle

		Cycles (consisting of 5 days each)					
Training Day	Description	A	B	C	D	E	F
1	Long & easy	→	→	→	→	→	→
2	Front push	→	→	→	→	→	→
3	Specific endurance	→	→	→	→	→	→
4	End push	→	→	→	→	T.T.	T.T.
5	Rest day	→	→	→	→	→	→

T.T.= Time Trial

A = baseline, B = treatment and C = baseline
Cycles D, E & F are conducted double-blind treatment or placebo depending on the subject.

Figure 2.2

Training Cycle Design

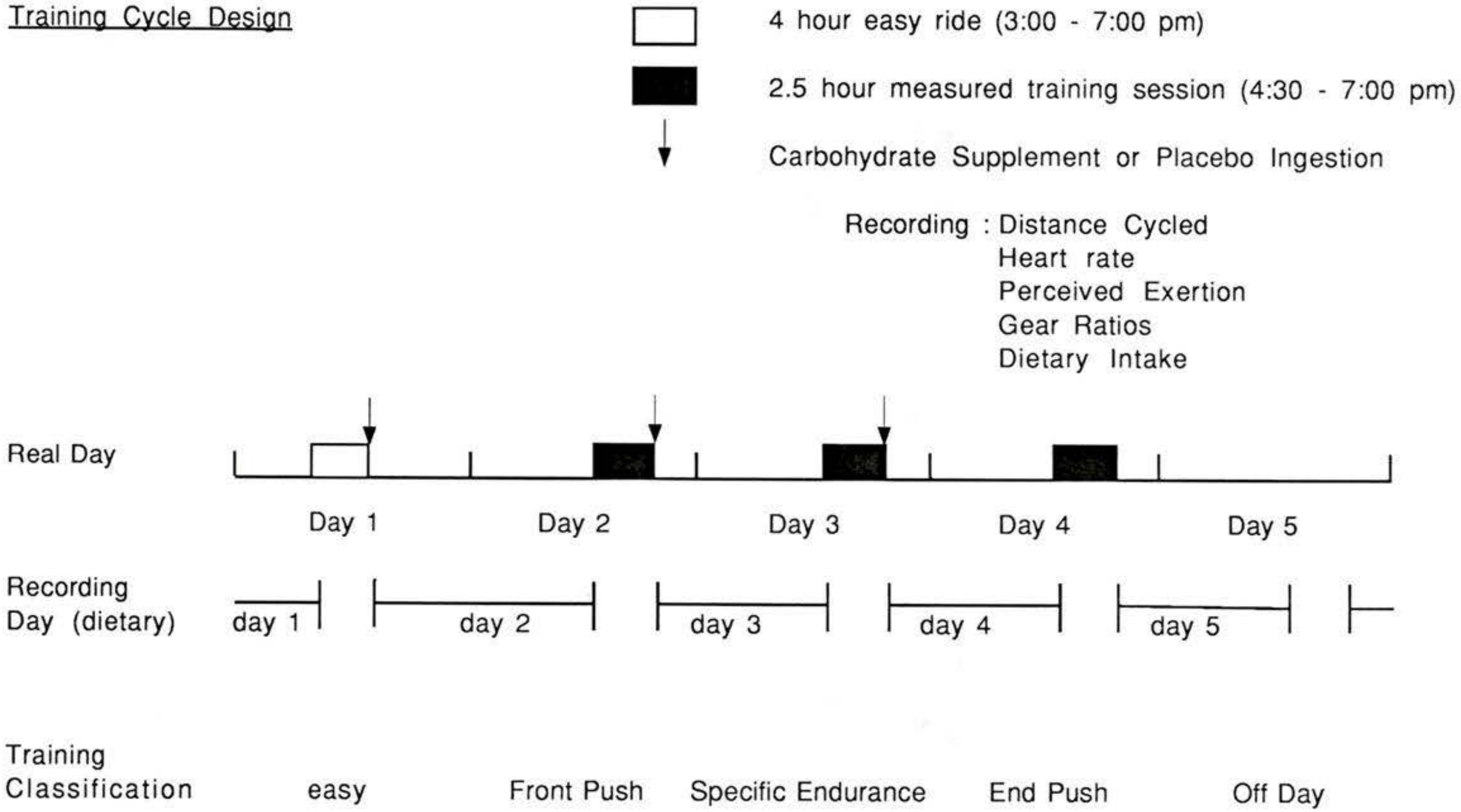


Table 2.2

Study design (by cycles) for each subject

Subject	Cycles					
	1	2	3	4	5	6
P.N.	B	S	B	P	S	P
A.H.	B	S	B	S*	S	P
J.T.	B	S	B	P	S	P

B = Baseline
S = Post exercise supplemented
P = Placebo
* = missed at least one workout

Table 2.3
Training heart rates corresponding to the volume of oxygen consumed at the ventilatory threshold (VT), and at decrements of 20% and 10% below VT.

Subject	Condition				
	Resting (HR)	VT - 20% (HR)	VT -10% (HR)	VT (HR)	Maximum (HR)
P.N.	54	149	156	163	184
A.H.	48	149	158	171	187
J.T.	50	150	160	171	194

Table 2.4
Time (minutes) at each training intensity in the four training protocols

Intensity Range	Training Session			
	Front Push Day 2	Specific Endurance Day 3	End Push Day 4 (first 4 cycles)	Time Trial Day 4 (last 2 cycles)
Spin	25	25	30	10
Build to VT-20%	5	5	5	5
VT - 20%	50	120	50	30
Build to VT-10%	15	-	20	5
VT - 10%	10	-	-	-
Choice	-	-	-	10
VT.	45	-	30	-
Maximal Effort (Time Trial)	-	-	-	60
Total Time	150	150	135	120

2.3 EXPERIMENTAL PROTOCOL

2.3.1 LABORATORY PROTOCOL

Laboratory tests were performed under standardised environmental conditions both before and after the study. Anthropometric measures included stretched height, weight and the sum of six skin folds (tricep, bicep, subscapular, suprailiac, front thigh and medial calf). Body fat was estimated using thigh and subscapular skin folds with the Sloan - Weir formula (Sloan & Weir, 1970).

$\dot{V}O_2$ max and VT were determined on a Monarch cycle ergometer using a continuous exercise protocol. Work load increased incrementally every 3 min by 0.5 - 0.25 kp until VT had been achieved, then by 0.5 kp every min. Cycling speed was set between 80 and 90 rpm. The criteria used to determine achievement of VO_2 max were: no further increase in VO_2 despite a further increase in work load; a respiratory quotient (RER) > 1.1; or voluntary cessation of the test. VT was determined by the method of Wasserman (1986) using the following criteria : i) the point at which the VE / VO_2 max curve, having been flat or decreasing, begins to rise disproportionately while the VE / V_{CO_2} curve remains constant or decreases; ii) the slope of the R - work rate curve, having been flat or rising slowly, becomes more positive. Respiratory gases were collected and analysed every 30 sec using a Beckman Metabolic Measurement Cart (MMC). Prior to, and following each test, the MMC was calibrated, and reassessed for drift using a commercially prepared primary standard gas mixture. Heart rate (HR) was recorded every 30 sec throughout the test and during the 3 - 5 min recovery period using a PE 3000 sports tester (heart rate monitor).

2.3.2 FIELD PROTOCOL

The route on the recovery day covered a variable terrain but was not physiologically demanding. Riding technique was single line, alternating leader. Liquid ingestion occurred *ad libitum*. Riding intensity was demonstrated when led by the principal investigator for the first three rides. The intensity criterion was four hours below 130 bpm, occasionally exceeding 130 bpm but never exceeding 150 bpm when traversing hills. For the training sessions stationary, fixed resistence, wind trainers were positioned in the identical place each day in a covered outside area. Monitored training sessions commenced with initial stretching and relaxation while the bicycles were being fastened and adjusted onto the windtrainers. Types of wind trainers used included magnetic and non-magnetic controlled resistance rollers. Applied resistance and tire pressure were calibrated before and after each session to ensure standardization and reliability of the resistance. Applied resistance was standardized before and checked after each training session using a method similar to the "coast-down test" used by Argentieri, Ennis and Piper (1986). Stopwatches measured time (+/- 0.1 sec) required for the resistance wheel to stop from a speed of 20 +/- 0.1 $km \cdot hr^{-1}$. Resistance was modified at the beginning of each session to maintain consistency in stopping time between sessions. An initial five min "spin" was conducted to warm the machinery before each calibration ("spinning" is a term referring to the process of pedalling against minimum resistance).

Many of the factors which can influence heart rate were controlled through the methodology of single subject design. The athletes were their own control. There was onsisitency in the clothes worn, perspiration rates and individual drinking patterns between sessions. Pre and post session body weight measurements were obtained to ensure adequate hydration levels. While the weather did not range more than 7° C and was consistently overcast, outside temperature and humidity were not controlled and may have impacted on some of the data by influencing training heart rate.

Summaries of the three recorded training days in Tables 2.4 and 2.5 indicate athletes trained at greater than 60% of $\dot{V}O_2max$ for 125, 125, and 105 min, during day 2, day 3, and day 4 training (first four cycles) respectively. Cycling between 80 and 88% of $\dot{V}O_2max$ occurred for 45 and 30 min and an estimated average intensity of between 69 and 77% of $\dot{V}O_2max$ for 25 and 20 min in day 2 and day 4 training (first four cycles), respectively. An intensity level of between 59 and 77% of $\dot{V}O_2max$ was maintained for a little more than 120 min during day 3 training. Day 4 training during the last 2 cycles included 40 min of an average intensity of between 69 and 77% of $\dot{V}O_2max$ and 60 min of maximal effort.

Training volume was measured as the distance cycled during timed intervals at the previously determined target heart rates (Table 2.3). Training distances were recorded to within 10 m with the use of standard cycling computer odometers . Heart rates, gear settings, ratings of perceived exertion for both muscular and cardiovascular effort (termed: "legs" and "lungs"), as well as subjective comments during and after the ride, were also recorded throughout the sessions, whenever changes occurred, or at five min intervals.

The decision to use distance cycled at set heart rates as the dependent variable was justified for a number of reasons. The subjects were accustomed to timing distances cycled and measuring heart rates using the same heart rate monitors and odometers utilized in the study. This maximized the external validity of the study. Options including cycling to exhaustion for many sessions over the 30 days, would be unrealistic and would invariably include more subject performance variations due to mood changes. In addition, because glycogen depletion is associated with increased heart rates and decrements in skill and coordination (Heigenhauser, Sutton & Jones, 1983), it was hypothesised that athletes training for the same time at the same heart rate in a depleted condition would cycle less distance than in a more repleted condition. Therefore, if the post workout carbohydrate supplement made a difference in inter-workout glycogen repletion that difference would be measurable as a change in distance cycled to the extent that the designed training protocol depleted muscle glycogen.

While subjects were requested to train at their target heart rates, the intensity criterion was plus or minus two beats. Failure to reach this target zone within one min was noted as a training segment failure. During the "buildup" segments the riders were asked to gradually increase their work load by increasing pedalling frequency and gear ratio so that they reached target heart rates during the time allotted for build-up.

During the last five min of warm-down subjects began drinking their test solutions (placebo or carbohydrate). Subjects were not permitted to leave their bicycles and were asked to continue cycling until the first 800 ml was ingested even if the five min of warm-down had elapsed. Complete ingestion occurred within 20 min of exercise cessation. Carbohydrate content in the supplement drink was 4 $g \cdot kg^{-1}$ bw in a 25% solution (Table 2.5). During the last three cycles the treatment and placebo drinks were placed into numbered bottles and administered double-blind. The carbohydrate treatment drink, 4 $g \cdot kg^{-1}$ body weight in a 25% solution contained 96% polymer, 4% fructose and artificial flavours. The treatment drinks consisted predominantly of polymer and was of a high concentration for a number of reasons. Starches maintain plasma insulin levels longer than glucose or sucrose (Hodges & Krehl, 1965) and produce greater increases in muscle glycogen than any simple sugar. The use of a more concentrated solution ensured a large but slow, steady supply of blood glucose due to a decreased gastric emptying time and increased gastric emptying rate (Blom, Hostjmar, Odd Vaage Kristin & Maehlum, 1987; Hunt, Smith, & Jiang, 1985). Fructose was used as a sweetener for its ability to selectively increase liver glycogen and for its greater sweetness qualities. The placebo drink consisted of a mixture of fruit pectin and the same flavouring agents. Drink flavours were randomly selected each day. A 2.5% mixture of fructose and glucose polymers (1:4) was ingested *ad libitum* during the training sessions. Flavouring agents were developed by Universal Flavours Inc. (Rexdale, Ontario) and met F.D.A. guidelines.

The level of fatigue and health for each cyclist was monitored through the use of a daily questionnaire and waking heart rate measurements. A registered physiotherapist was used to administer advice and therapy for overuse injuries.

Table 2.5
<u>Supplement drink constituents</u>

Subject	Weight (kg)	Constituents			
		Carb. Polymer (g)	Fructose (g)	Total Carb. (g)	Total Vol. (L)
P.N.	70.5	261	10.9	272	1.09
A.H.	77.4	303	12.6	316	1.26
J.T.	82.3	315	13.1	328	1.31
Means	76.7	293	12.2	305	1.22
SD	5.9	28	1.2	30	0.12

Note: Carbohydrate content was 4 $g \cdot kg^{-1}$ at a concentration of 25% by weight.

2.4 ANALYSIS

2.4.1 DIETARY ANALYSIS

Dietary recording occurred on a daily basis, starting 22 hours before training (i.e. the 22 hour recording day encompassed the time immediately after the previous day's training session and continued until the next training session began). Therefore, the dietary survey commenced 22 hours before the day of the first ride and ended before the last ride in the 30 day schedule (Figure 2.1). Each subject maintained a complete dietary log for the course of the study. Dietary analysis was conducted on an IBM computer using the "Food Perfect" dietary analysis program designed and donated by Intelligent Software Engineering (© 1990).

2.4.2 EXPERIMENTAL ANALYSIS

Results were treated and analysed using single subject methodology. Graphs generated for each subject were visually inspected for patterns and trends. Individual results were compared for commonalities and differences that would help to answer the research questions.

CHAPTER III

RESULTS

Individual dietary intake and training volume results are reported in a series of tables and figures. Supporting results, figures, tables and documents are included in the Appendices B - E. For visual analysis individual summaries of carbohydrate intake and training volume are reported in a series of three figures for each of three subjects. The three graphs contained in the first figure represent training volume and pre-workout (22 hours) carbohydrate intake recorded during the three different training protocols across the six training cycles. Training volume and net daily carbohydrate intakes are presented together to illustrate the relationship between dietary carbohydrtate intake in the previous 22 hours and training volume. Daily results were blocked into summaries to account for interactions between successive days of training and carbohydrate intake. Two and three day summaries of training volumes and their corresponding pre-workout carbohydrate intakes are presented graphically as the second of the three figures. Each graph includes two dashed lines, denoting the desired maximum and minimum carbohydrate intake levels for unsupplemented and supplemented conditions, respectively. The third figure includes correlations between total dietary intake and total training volume for two (day 2 & day 3) and three day (day 2, day 3 and day 4) summaries. These correlations were calculated by employing the linear least-squares regression method to determine the best curve fit (Apple Macintosh LC 475).

Tables 3.5 and 3.6, 3.8 and 3.9 and 3.11 and 3.12 describe dietary carbohydrate intakes for subjects P.N., A.H. and J.T. Complete dietary profiles are found in Appendix C. Work performed during individual training sessions and corresponding cycle summaries were tabulated in Tables 3.7, 3.10 and 3.13. Daily training volume and carbohydrate intake are presented in Figures 3.2, 3.5 and 3.8. Two and three day distance summaries were depicted in Figures 3.3, 3.6 and 3.9. Correlations between carbohydrate intakes and distance cycled are displayed in Figures 3.4, 3.7 and 3.10.

Comparisons of day four totals during the last two cycles with totals from the first four cycles were not performed because the training protocol on day 4 of cycles 5 and 6 was different than on the same day in the previous four cycles. Consequently, comparisons between the last two cycles and the first four are not possible in the 3 day summaries.

Mean (SD) physical characteristics are displayed in Tables 2.1 and 3.1. The mean training experience at a volume of 200 km per week, six months per year was 3.3 years. Between 2000 and 2100 km were cycled during the training sessions by each of the three subjects selected for discussion (Table 3.2). Of this, approximately two thirds were performed on a fixed wind trainer apparatus and one third on the open road (Table 1.0).

Initial $\dot{V}O_2$max scores for P.N., A.H. and J.T. were measured at 51.3, 66.3 and 65.1 $ml^{-1} \cdot min^{-1} kg^{-1}$, respectively. VT was estimated at 39.7, 45.2 and 52.2 $ml^{-1} \cdot kg^{-1} \cdot min^{-1}$. Changes in VO_2max and VT after one month of training are summarised in table 3.17. Increases in VT for two of the three subjects were far greater than increases in $\dot{V}O_2$max: P.N. VT.(+14.3%), $\dot{V}O_2$max (-1.1%) and the VT for A.H. (+10.6%), $\dot{V}O_2$max (+2.9%). For J.T., VO_2max increases (+3.9%) exceeded an increase in VT (+2.3%). Heart rates at VT were 9 and 3 bpm higher in post training for P.N. and A.H., respectively but 11 bpm lower for J.T. (Table 3.2). Subject weight and body fat remained consistent over the thirty days of training (Table 3.1). Subject P.N. was 35 years old and as such, aerobic fitness levels for P.N. are lower than A.H. or J.T. Despite the low scores, P.N. fit the training criteria.

Training intensities were intially determined on the basis of $\dot{V}O_2$ - heart rate graphs developed in the pre-study VO_2max tests. Since subjects trained at defined target heart rates throughout the thirty days, physiological responses to thirty days of training in effect altered the net training intensities cycled. Comparisons of $\dot{V}O_2$ - heart rate graphs before and after the study reveal that for any specific heart rate, training intensities, defined as a percentage of $\dot{V}O_2max$, changed (Table 3.3 and 3.4). In all cases, the lower training heart rates increased the greatest, and the higher training heart rates increased the least (Figure 3.1). In one case (A.H.) the post study measurement of training $\dot{V}O_2$, estimated against the highest training heart rate, actually decreased .

Subjects familiarized themselves with measuring and recording techniques within an independent prestudy dietary assessment and training trial. Individual dietary records from subjects P.N., A.H. and J.T. were reported together for comparison as percentages of the recommended energy intake (% REI), in absolute terms (calories and grams) and compositionally (as a percentage of the total caloric intake). Post exercise supplementation during the supplement condition increased mean caloric intakes 50%, 40% and 54% to levels of 4078, 5585 and 4842 calories (Appendix C). Mean dietary carbohydrate intakes increased between 70 and 80% to 10.2, 12.0 and 11.3 $g \cdot kg^{-1} \cdot d^{-1}$ for P.N., A.H. and J.T. respectively (Tables 3.5, 3.8 and 3.11). Thus, post exercise carbohydrate supplementation effectively increased dietary intake above the 10 $g \cdot kg^{-1} \cdot d^{-1}$ level suggested to prevent glycogen depletion during successive days of intense aerobic training. However, during unsupplemented conditions, average intake per training cycle did not always remain under the study target of 8 $g \cdot kg^{-1} \cdot d^{-1}$ and in fact surpassed 10 $g \cdot kg^{-1} \cdot d^{-1}$ on one occassion . Average unsupplemented intake ranges were 5.6 - 7.3, 7.7 - 10.2 and 6.6 - 9.6 $g \cdot kg^{-1} \cdot d^{-1}$ while average carbohydrate intake ranges under supplemented conditions were 9.8 - 10.6, 11.0 - 13.1 and 11.1- 15.3 $g \cdot kg^{-1} \cdot d^{-1}$ for subjects P.N., A.H. and J.T. respectively (Table 3.5, 3.8 and 3.11).

Supplementation effected dietary composition by increasing carbohydrate intake and decreasing both fat and protein intake to mean percentage levels of 71-20-10, 66-24-10 and

78-17-5 respectively (Appendix C). Although the effect of carbohydrate supplementation on carbohydrate, fat and protein intake was not investigated, none was suspected since pre-supplement intake levels during supplement conditions were similar to intake levels during placebo and baseline conditions. Changes in relative dietary composition (as a % of total caloric intake) were more reflective of the increase in supplemented dietary carbohydrate intake rather than the decrease in dietary intake of fats and proteins.

Figure 3.1

Comparison of training intensities at specific heart rates - before and after training.

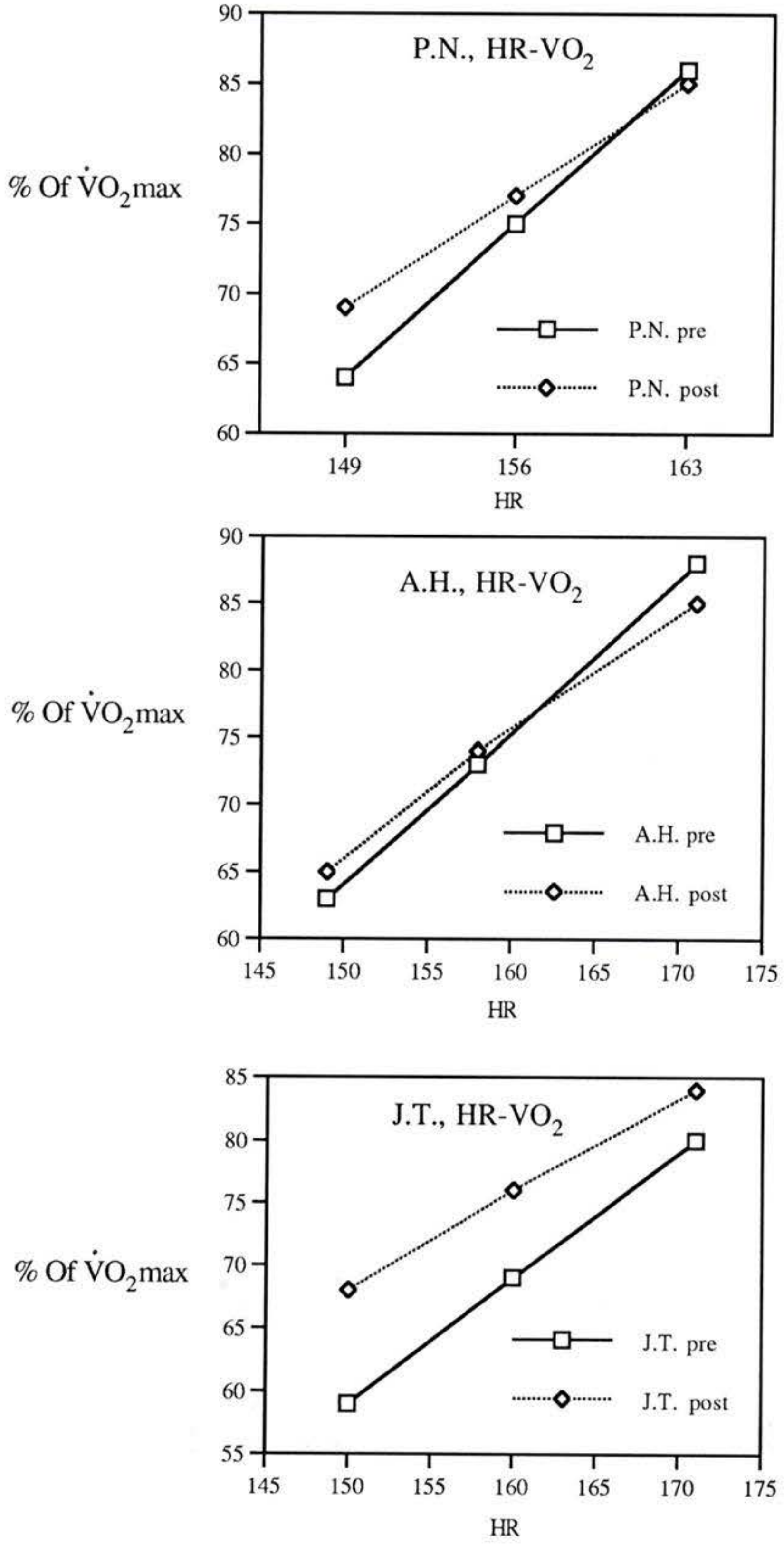


Table 3.1
Body composition summary before and after the study. Means (SD) and differences

Subject		Weight +/- 0.1kg	D	S.O.S. +/- 0.1mm	D	S.O.E. +/- 0.1mm	D	% b.f.(1) (predicted)	D
P.N.	pre	70.5		40.0		52.8		8.8	
	post	70.8	0.3	38.8	- 1.2	50.5	- 2.3	8.4	- 0.4
A.H.	pre	77.4		35.3		47.5		7.8	
	post	78.3	0.9	35.5	0.2	48.5	1.0	7.5	- 0.3
J.T.	pre	82.3		38.5		52.3		7.8	
	post	82.7	0.4	37.3	- 1.2	51.0	- 1.3	7.5	- 0.3
	Mean		0.5		-0.7		-0.9		-0.3
	SD		0.3		0.8		1.7		0.1

(1) Sloan and Weir, 1970

Table 3.2
Pre and post training $\dot{V}O_2$ max, VT and heart rate at VT

Subject	Sessions Attended	Volume Trained (+/-50 km)	Test	Before	After	Change	Relative change %
P.N.	24	2039	$\dot{V}O_2$ max (L.min-1)	3.64	3.60	- 0.04	- 1.1
			VT (L.min-1)	2.80	3.20	+ 0.40	+ 14.0
			hr (@ VT.)	160	169	+ 9	
A.H.	23	2089	$\dot{V}O_2$ max (L.min-1)	5.13	5.28	+ 0.15	+ 2.9
			VT (L.min-1)	3.50	3.87	+ 0.37	+ 10.6
			hr (@ VT.)	160	163	+ 3	
J.T.	24	2087	$\dot{V}O_2$ max (L.min-1)	5.36	5.57	+ 0.21	+ 3.9
			VT (L.min-1)	4.30	4.40	+ 0.10	+ 2.9
			hr (@ VT.)	171	160	- 11	

Table 3.3
Training intensities described as percentage of $\dot{V}O_2$max

Subject		$\dot{V}O_2$max		Percentage of $\dot{V}O_2$max		
		($L \cdot min^{-1}$)	($ml \cdot kg^{-1} \cdot min^{-1}$)	VT - 20%	VT -10%	VT
P.N.	Pre	3.62	50.9	64	75	86
	Post	3.60	50.8	69	77	85
A.H.	Pre	5.13	66.3	63	73	88
	Post	5.28	67.4	65	74	85
J.T.	Pre	5.36	65.1	59	69	80
	Post	5.57	68.6	68	76	84

Table 3.4
Training intensities and durations

Intensities (% of $\dot{V}O_2$max)*			Time (min.)			
P.N.	A.H.	J.T.	Day 2	Day 3	Day 4	Day 4*
< 50 % of VO max			30	30	35	25
64 - 69	63 - 65	59 - 68	65	120	70	35
75 - 77	73 - 74	69 - 76	10	-	-	-
86 - 85	88 - 85	80 - 84	45	-	30	60*
			150	150	135	120

* Training intensity ranges are calculated from aerobic power tests conducted both before and after the study.

P.N.

Mean caloric intake during supplementation increased 33% from 3073 to 4078 (kcal and mean carbohydrate intake increased 64% from 463 to 720 g. (or 6.0 to 10.1 $g \cdot kg^{-1} \cdot d^{-1}$) when compared against combined mean intakes during placebo and baseline conditions (Appendix B and C). Dietary composition changed from 58, 28 and 14% carbohydrate, fat and protein to 71, 20 and 10%, respectively (Appendix C). Daily fluctuations in dietary carbohydrate intake are recorded in Table 3.5 and shown in Figures 3.2 and 3.3. Hydration was maintained with the ingestion of 1.8 - 2.4 L of the training solution per session.

Post exercise carbohydrate intake successfully increased dietary carbohydrate levels above non-supplemented levels. Expected variation in carbohydrate intake occured with *ad libitum* dietary intake. Supplementation raised dietary CHO intake above 8 $g \cdot kg^{-1} \cdot d^{-1}$ for all six supplemented sessions and above 10 $g \cdot kg^{-1} \cdot d^{-1}$ three times (day 2; S1 and S2 and day 3; S2; Table3.5). Net carbohydrate intake during supplementation ranged from 9.2 to 11.3 $g \cdot kg^{-1} \cdot d^{-1}$ and during placebo or baseline conditions from 4.5 to 7.6 $g \cdot kg^{-1} \cdot d^{-1}$. This was consistently under the desired non-supplemented maximum of 8 $g \cdot kg^{-1} \cdot d^{-1}$. When displayed as 3 day summaries, dietary intake during supplementation ranged from 9.8 to 11.2 $g \cdot kg^{-1} \cdot d^{-1}$ and during placebo or baseline conditions ranged from 5.2 to 6.8 $g \cdot kg^{-1} \cdot d^{-1}$, well below the 8.0 $g \cdot kg^{-1} \cdot d^{-1}$ criterion (Figure 3.3).

Distance travelled during baseline and placebo conditions together can indicate training or learning effects within the study. "Unsupplemented" training volume showed a variable but declining trend for day 2 results, and increasing trends for day 3 and day 4 data (Figure 3.2). Summarised into 2 and 3 day blocks, training volume declined and increased, respectively (Figure 3.3). Day two distance values followed the predicted pattern except the distance measured during the first placebo condition was more comparable to the other two supplement values than achieved during baseline or subsequent placebo conditions. For day three results all but the last placebo distance value (P2) followed the expected pattern with distance exceeding the previous supplement data. Day 4 training volume under P2 was also higher than in the previous supplemented condition (S2). As well, no increase in training volume was observed during the first supplemented condition (S1). Consequently, day four training volume progression generally opposed expected performance. Training volume deviated once from expected results during day 2 training, once during day 3 training and three times during day 4 training. However, when blocked into two and three day distance summaries, distance graphing patterns verify without exception the expected trends for differences with supplementation. Linear correlations of dietary carbohydrate intake with distance cycled produced r values of 0.87 for two day means and 0.55 for the three day means (Figure 3.4).

Table 3.5

P.N. Means (SD) for daily and cycle summaries of total recorded carbohydrate intake (g, g.kg-1 and g.kg-1.d-1)

Cycle:		1	2	3	4	5	6
Condition:		B	S	B	P	S	P
Daily results							
Day 2	(g)	489	767	477	367	796	275
	(g/kg)	6.9	10.9	6.8	5.2	11.3	3.9
Day 3	(g)	444	656	315	537	771	376
	(g/kg)	6.3	9.3	4.5	7.6	10.9	5.3
Day 4	(g)	513	649	457	395	680	447
	(g/kg)	7.3	9.2	6.5	5.6	9.6	6.3
Cycle totals							
Mean	(g)	482	691	416	433	749	366
	SD	35	66	88	91	61	86
Mean	(g/kg/d)	6.8	9.8	5.9	6.1	10.6	5.2
	SD	0.5	0.9	1.3	1.3	0.9	1.2
Grand totals							
	(g)	1446	2072	1249	1299	2247	

Table 3.6

P.N. Day means (SD) for carbohydrate intake (g & g.kg -1.d-1)

Condition	n (days)	Mean (SD) (g)	(g.kg-1.d-1)
Before supplementation:			
Baseline	2	449 (47)	6.4 (0.7)
Placebo	2	400 (47)	5.7 (0.7)
Supplement	2	402 (43)	5.7 (0.6)
Rest days	10	411 (98)	5.8 (1.4)
After supplementation:			
Supplement	2	720 (43)	10.2 (0.6)

Table 3.7

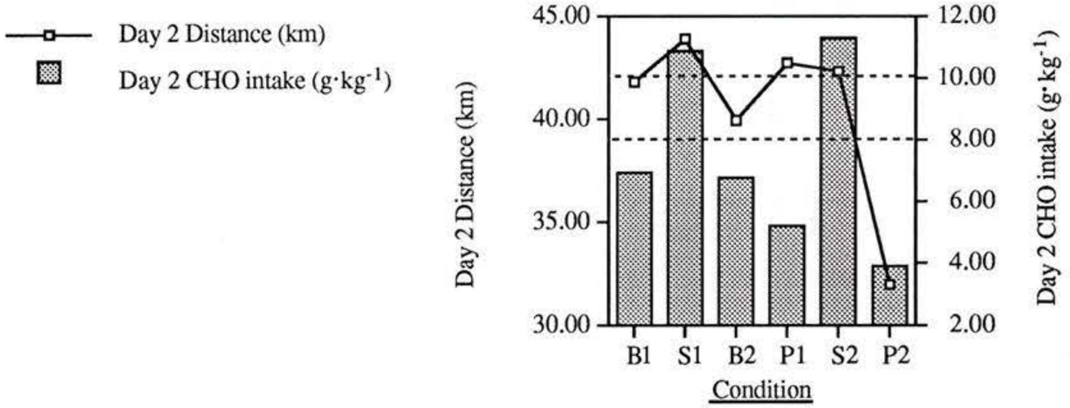
P.N. Means (SD) for daily and cycle totals, distances measured and trained (km)

Cycle: Condition:	1 B1	2 S1	3 B2	4 P1	5 S2	6 P2
Daily Totals:						
Day 2 (0.01km)	41.80	43.90	39.94*	42.73	42.32	31.96*
Day 3 (0.01km)	72.75*	77.35	72.71*	71.95*	75.42**	75.78
Day 4 (0.01km)	29.39	29.30	34.04	34.38	(38.64)	(43.04)
3 Day Totals: (0.03 km)	144.04	150.45	146.69	149.06	(156.38)	(150.78)
2 Day Totals: (0.02 km)	114.55	121.25	112.65	114.68	117.74	107.74

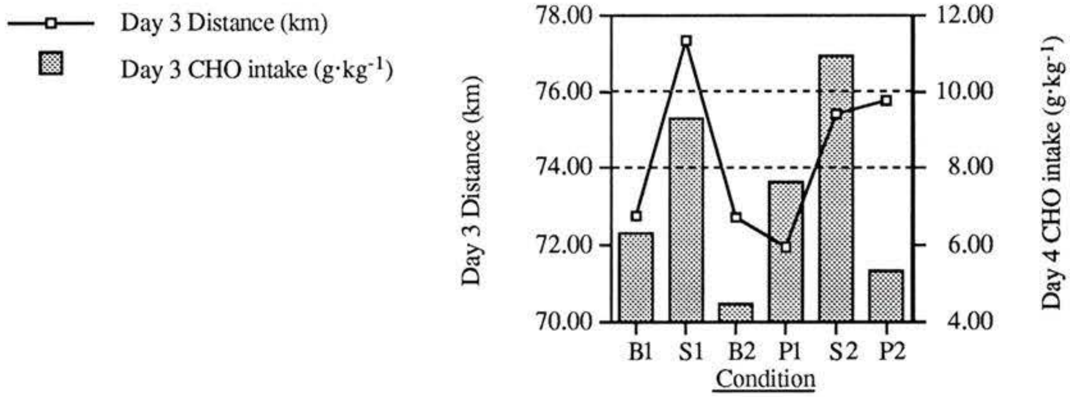
bracketted distances recorded during day 4 of cycles 5 and 6 are not comparable to the previous four cycles due to the change in protocol.

* below target hr due to claims of low energy.
** below target hr due to claims of stomach cramp.

P.N. Day 2: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)



P.N. Day 3: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)



P.N. Day 4: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)

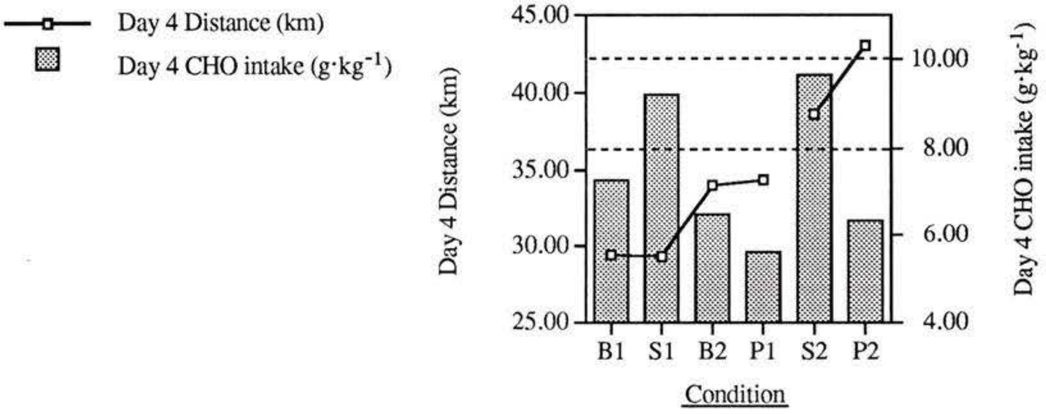
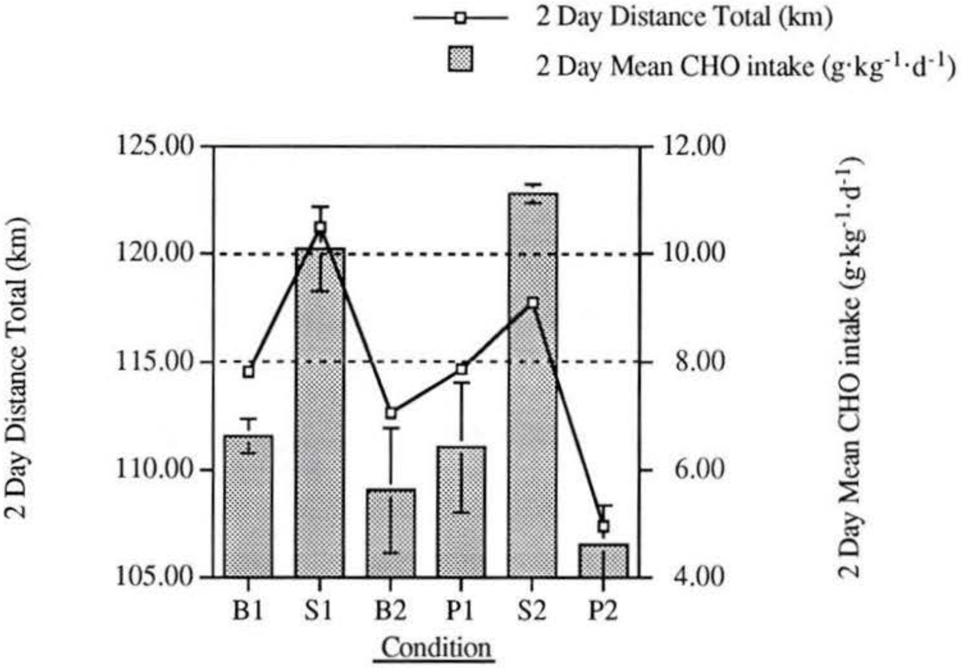


Figure 3.2 P.N. CHO intake ($g \cdot kg^{-1}$) and cycled distance (km) for the three training protocols

P.N. 2 day mean (SD) CHO intake ($g \cdot kg^{-1} \cdot d^{-1}$) and cycled distance (km)



P.N. 3 day mean (SD) CHO intake ($g \cdot kg^{-1} \cdot d^{-1}$) and cycled distance (km)

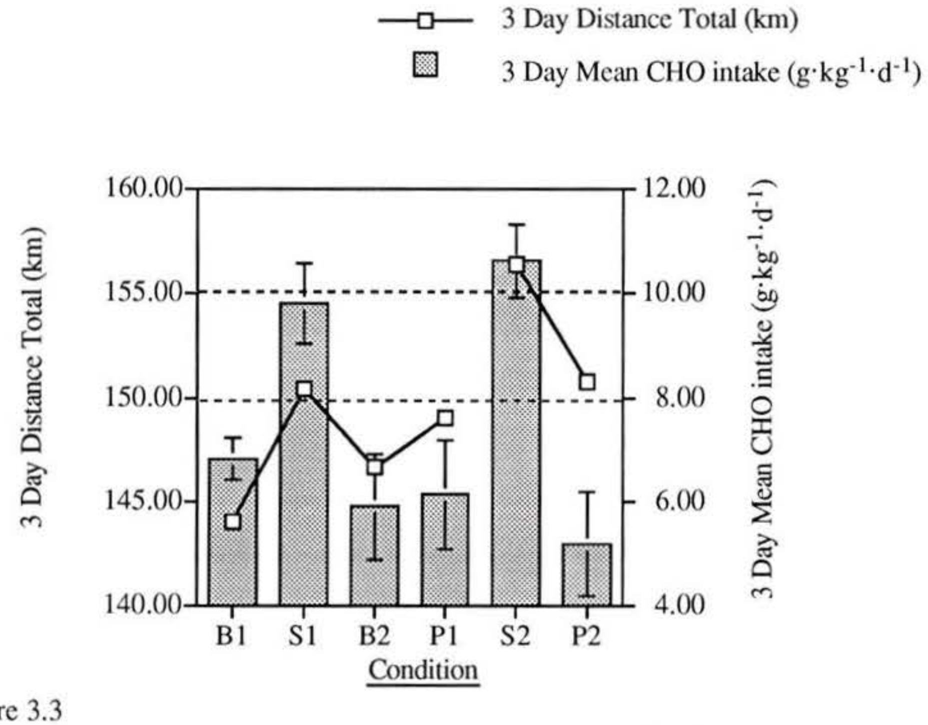


Figure 3.3
P.N. 2 and 3 day summaries of mean (SD) CHO intake ($g \cdot kg^{-1} \cdot d^{-1}$) and cycled distance (km)

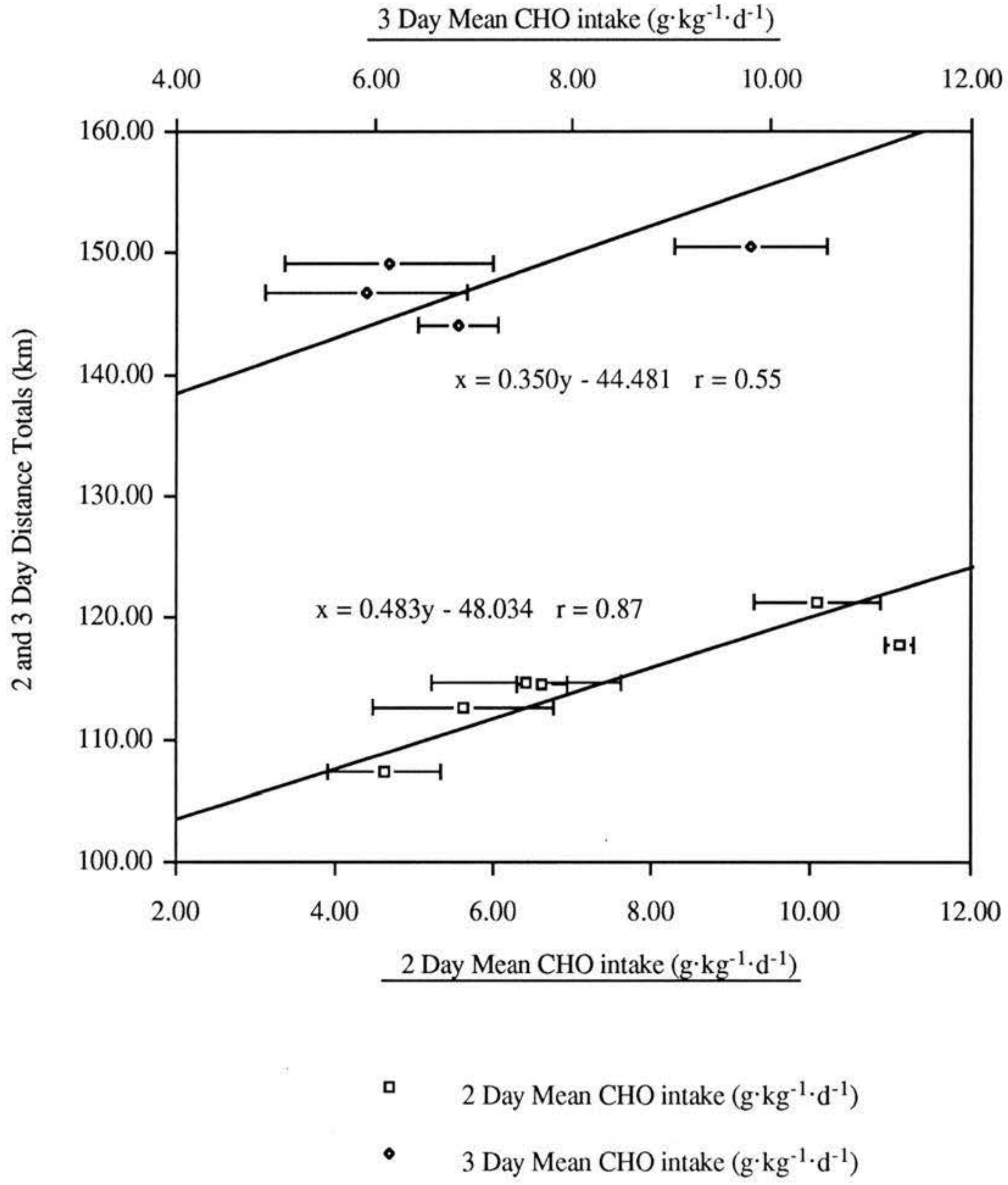


Figure 3.4

P.N. The relationship between mean (SD) CHO intake ($g \cdot kg^{-1} \cdot d^{-1}$) and total distance cycled (km)

A.H.

Mean dietary caloric intake during supplementation increased 27% from 4389 to 5585 kcal and mean carbohydrate intake increased 39% from 8.6 to 12.0 $g \cdot kg^{-1} \cdot d^{-1}$ when compared against combined mean intakes during placebo and baseline conditions (Appendix B and C). Dietary composition changed from 54, 33 and 14% carbohydrate, fat and protein to 66, 24 and 10%, respectively (Appendix C). Daily fluctuations in dietary carbohydrate intake are recorded in Table 3.8 and shown in Figs. 3.5 and 3.6.

Dietary carbohydrate intake increased with post exercise supplementation. *Ad libitum* intake was successful in generating the desired intake during day 2 and day 4 training (Table 3.8). Carbohydrate intake during baseline conditions of day 3 training were higher than desired with intake before the second baseline condition (B2) exceeding the intake before the subsequent two supplemented conditions (S2 and S3). Carbohydrate intake summaries before the second baseline condition (B2) were more similar to the subsequent two supplemented conditions than the previous baseline (B1) or subsequent placebo (P1) intake levels. Carbohydrate intake was calculated above 10 $g \cdot kg^{-1}$ before each supplemented session and ranged from 10.9 to 16.2 $g \cdot kg^{-1} \cdot d^{-1}$. Unfortunately, *ad libitum* intake of dietary carbohydrate during baseline and placebo conditions increased net dietary intake to more than 8 $g \cdot kg^{-1} \cdot d^{-1}$ in all but two situations (day 2;P and day 4;B1) and exceeded 10 $g \cdot kg^{-1} \cdot d^{-1}$ three times (day 2;B1 and B2 and day 3;B2). Blocked into 2 and 3 day summaries, dietary intake followed the desired trends under supplemented conditions with intakes all greater than 12 $g \cdot kg^{-1} \cdot d^{-1}$ (Figure 3.6). Intake before baseline and placebo conditions exceeded 8 $g \cdot kg^{-1} \cdot d^{-1}$ and twice passed the 10 $g \cdot kg^{-1} \cdot d^{-1}$ level (2 day summary; B1 & B2 and 3 day summary; B2). During placebo or baseline conditions CHO intake ranged from 6.54 $g \cdot kg^{-1}$ to 12.27 $g \cdot kg^{-1} \cdot d^{-1}$. This exceeded the desired non-supplemented maximum of 8 $g \cdot kg^{-1} \cdot d^{-1}$, 6 out of 9 times. Presented as 3 day summaries, dietary intake during supplementation ranged from 12.0 (2.0) to 14.4(2.7) $g \cdot kg^{-1} \cdot d^{-1}$ and during placebo or baseline conditions it ranged from 8.4(0.7) to 11.2(1.8) $g \cdot kg^{-1} \cdot d^{-1}$, consistently above the desired maximum for unsupplemented conditions (Figure 3.6).

Training volume recorded during baseline and placebo conditions defined the "non-supplemented" learning (or conditioning) trends. These trends decreased during day 2 and day 3 training and increased during day 4 (Figure 3.5). The corresponding 2 day summary trend decreased while the 3 day summary was stable (Figure 3.6). Day 2 total distance values were as predicted except during the fourth cycle where S2 distance did not increase relative to baseline distance (B2) of the previous cycle. On this day the subject complained of low energy levels and admitted having cycled harder than prescribed on the previous "easy ride" day. Day three distance total values changed according to prediction. Trained distance during day 4 of cycle two was greater than the previous cycle (B1) but lower than the subsequent cycle (B2). The subject did not perform during day 4 on the third cycle due to debilitating knee pain.

Therefore, training volume deviated from the experimentally expected pattern only twice. This was verified in the three and two day summaries and supported with particularily high correlation coefficients (Figure 3.7). Two day distance totals from cycle 4 (S2) were less than the initial levels attained during the first supplement cycle. Calculated training distance during cycle 4 in the 3 day summary graph contains no contribution from day 4 training due to an ankle problem, therefore, it is not comparable to the other day 3 summary results. The subject had a great deal of difficulty maintaining target heart rate during the last day of training (day 4; P1) due to apparent muscular fatigue. Linear correlations between dietary carbohydrate intake and distance cycled generated r values of 0.88 for two day means and 0.98 for three day means (not including results from days 4,5 and 6)(Figure 3.7)

Table 3.8

A.H. Means (SD) for daily and cycle summaries of total recorded carbohydrate intake (g, g.kg-1 and g.kg-1.d-1)

Cycle:		1	2	3	4	5	6
Condition:		B	S	B	S	S	P
Daily results:							
Day 2	(g)	608	1145	859	1002	1011	554
	(g/kg)	7.9	14.8	11.1	12.9	13.1	7.2
Day 3	(g)	808	1098	865	829	768	581
	(g/kg)	10.4	14.2	11.2	10.7	9.9	7.5
Day 4	(g)	461	797	644	779	765	643
	(g/kg)	6.0	10.3	8.3	10.1	9.9	8.3
Cycle totals							
	Mean (g)	626	1013	789	870	848	593
	SD	174	189	126	117	141	46
	Mean (g/kg/d)	8.1	13.1	10.2	11.2	11.0	7.7
	SD	2.3	2.4	1.6	1.5	1.8	0.6
Grand totals							
	(g)	1877	3040	2368	2610	2544	1778

Table 3.9

A.H. Day means (SD) for carbohydrate intake (g & g.kg-1.d-1)

Condition	n (days)	Mean (SD) (g)	 (g.kg-1.d-1)
Before supplementation:			
Baseline	2	707 (116)	9.1 (1.5)
Placebo	1	554 (n.a.)	7.2 (n.a.)
Supplement	3	543 (85)	7.0 (1.10)
Rest days	10	514 (126)	6.6 (1.6)
After supplementation:			
Supplement	3	910 (85)	12.5 (1.1)

Table 3.10

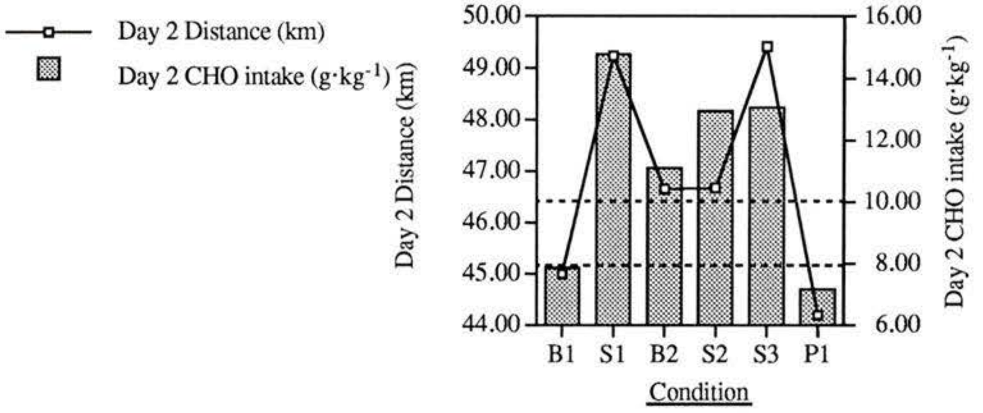
A.H. Means (SD) for daily and cycle totals, distances measured and trained (km)

Cycle: Condition:	1 B1	2 S1	3 B2	4 S2	5 S3	6 P1
Daily Totals:						
Day 2 (0.01km)	45.00**	49.24	46.65	46.67	49.42**	44.20**
Day 3 (0.01km)	83.69	89.97	84.01	87.08	87.97	78.00
Day 4 (0.01km)	36.41	38.18	38.24	-	(47.24)	(44.06)**
3 Day Totals: (0.03 km)	165.10	177.39	168.90	133.75*	(184.63)	(166.26)
2 Day Totals: (0.02 km)	128.69	139.21	130.66	133.75	137.39	122.20

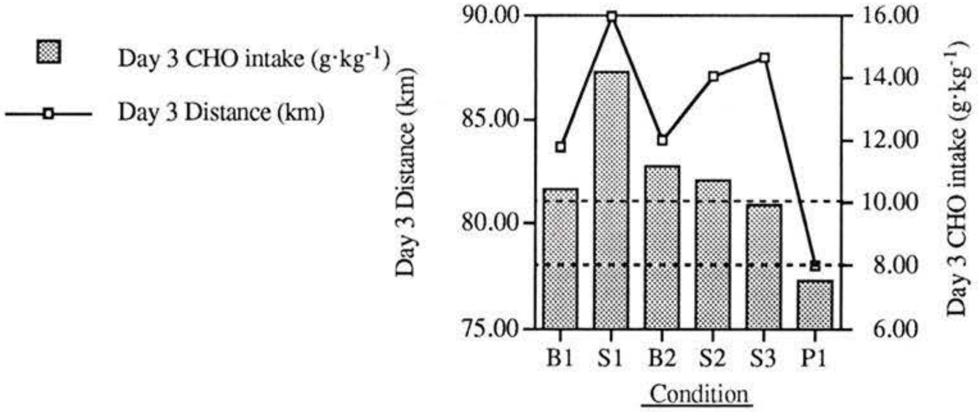
bracketed distances recorded during day 4 of cycles 5 and 6 are not comparable to the previous 4 cycles due to a change in protocol.

* did not complete the entire week.
** below target hr due to claims of low energy.

A.H. Day 2: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)



A.H. Day 3: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)



A.H. Day 4: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)

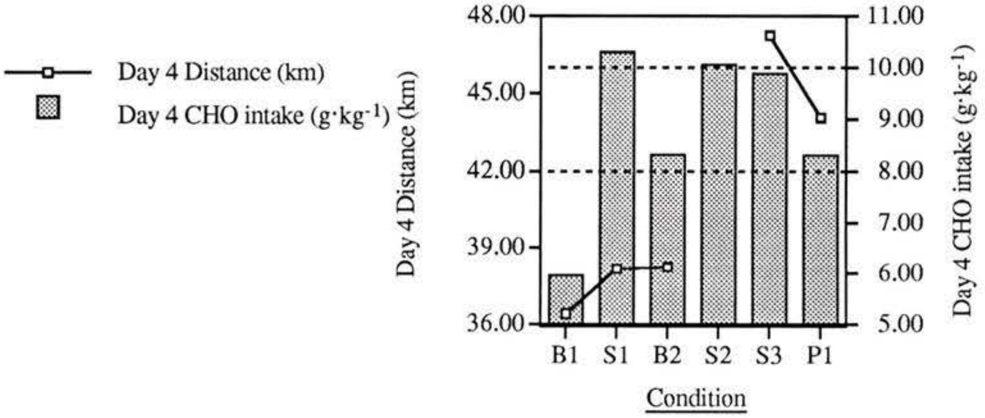
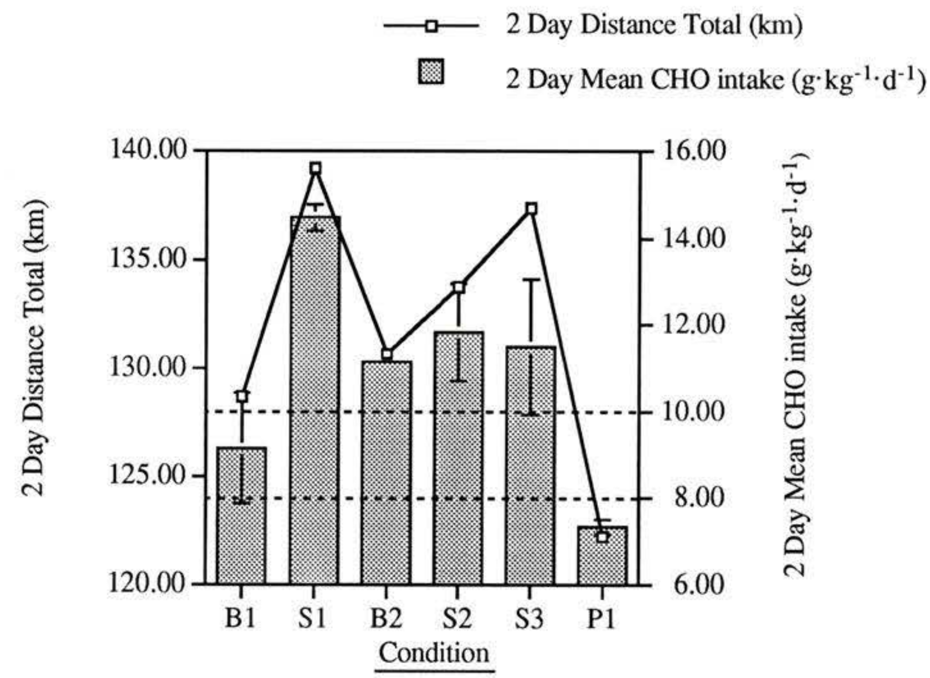


Figure 3.5
A.H. CHO intake ($g \cdot kg^{-1}$) and cycled distance (km) for the 3 training protocols

A.H. 2 day mean (SD) CHO intake (g·kg^{-1}·d^{-1}) and cycled distance (km)



A.H. 3 day mean (SD) CHO intake (g·kg^{-1}·d^{-1}) and cycled distance (km)

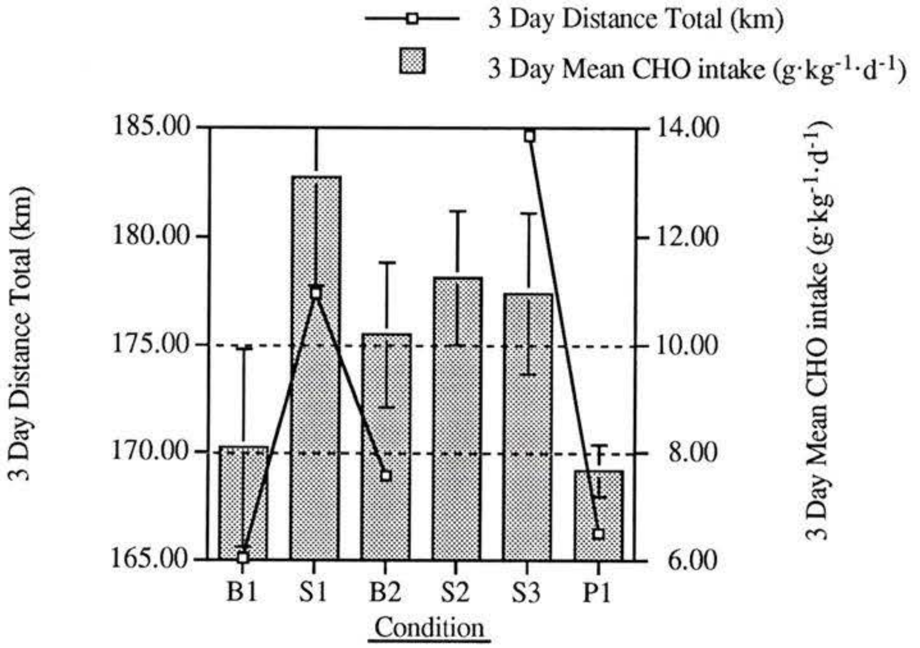


Figure 3.6

A.H.2 & 3 day summaries of mean (SD) CHO intake (g·kg^{-1}·d^{-1}) and cycled distance (km)

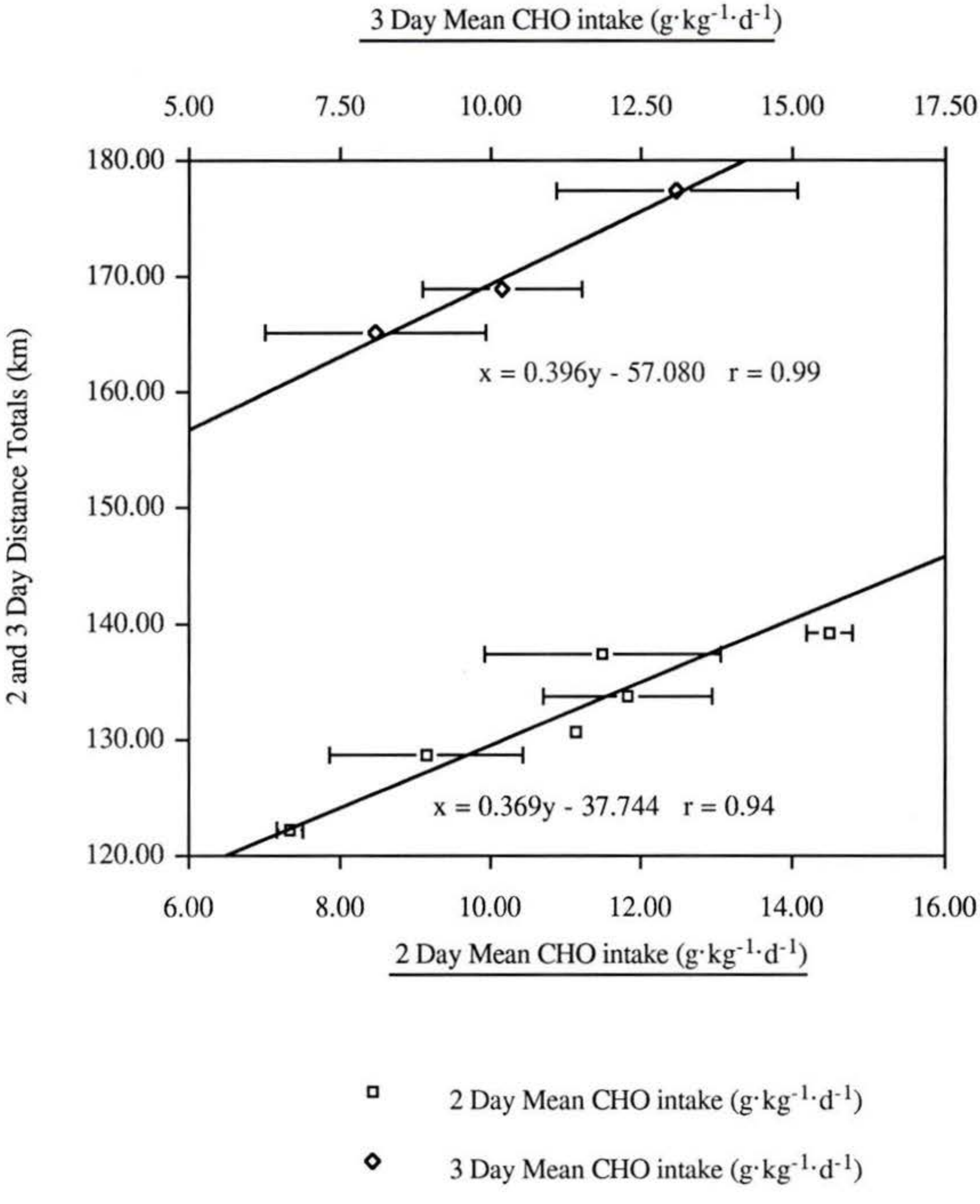


Figure 3.7

A.H. The relationship between mean (SD) CHO intake ($g \cdot kg^{-1} \cdot d^{-1}$) and total distance cycled (km)

J.T.

Mean caloric intake during supplementation increased 31% from 3696 to 4842 kcal and mean carbohydrate intake increased 69% from 6.69 to 11.29 $g \cdot kg^{-1} \cdot d^{-1}$ when compared against combined mean intakes during placebo and baseline conditions (Appendix B and C). Dietary composition changed from 64, 27 and 9% carbohydrate, fat and protein to 78, 17 and 5%, respectively (Appendix C). Weekly carbohydrate totals followed the desired experimental order but the difference between the first two cycles was not great (Table 3.11 and Figs. 3.8 - 3.9). Otherwise, post exercise supplementation produced the desired trend in dietary carbohydrate intake. Intake during supplemented conditions exceeded 10 $g \cdot kg^{-1}$ in each of the six cases. A very high intake value of 19.3 $g \cdot kg^{-1}$ was recorded before the last supplemented ride of day 2 training. Otherwise, dietary intake during supplementation ranged from 10.1 to 13.5 $g \cdot kg^{-1} \cdot d^{-1}$. The maximum non supplemented carbohydrate intake criterion of 8 $g \cdot kg^{-1} \cdot d^{-1}$ was surpassed in 5 out of 9 occasions - twice in cycle one (B1) (day 3 and 4; 10.7 and 10.9 $g \cdot kg^{-1}$), once in cycle three (B2) (day 4; 11.1 $g \cdot kg^{-1}$) and twice in cycle four (P1) (day 2 and day 4; 8.2 and 8.2 $g \cdot kg^{-1}$). Net carbohydrate intake ranged from 5.7 to 10.9 $g \cdot kg^{-1} \cdot d^{-1}$ during either baseline or placebo conditions. Blocking of data into summaries of 2 and 3 days demonstrated that post exercise supplementation succeeded in bringing mean dietary carbohydrate intake levels above 10 $g \cdot kg^{-1} \cdot d^{-1}$ in each cycle (Figure 3.9). Mean intake remained under the maximum unsupplemented criterion of 8 $g \cdot kg^{-1} \cdot d^{-1}$ during cycles 3 and 4 but exceeded the criterion during cycle 1. No records of dietary intake were obtained for the subject's last three days.

Day 2 baseline and placebo training results produced a negative trend in training volume while day 3 and day 4 baseline and placebo training volume showed increasing trends (Figure 3.8). Both summary graphs reflect the increasing trends in volume of training. Day two distance totals followed the expected pattern although variability was less than 330 metres among the first four cycles (Figure 3.8). Training volume for cycle 2 (S1) was lower than predicted as compared to the volume attained in cycle 5 (S2) and against baseline and placebo data. Even though distance trained during the second cycle exceeded distances trained in the first, compared against day three results, cycle 1 and especially cycle 2 totals appear unexpectedly low. Training volume did not follow the predicted outcome during day 4 as P2 volume exceeded the training volume attained in the previous supplemented condition (day 4; S2). The subject's logs and morning heart rate data suggested he was experiencing symptoms of a "flu-like" condition.

The most visible result of the summary graphs was the increase in training volume occurring during the second of two supplemented conditions (S2)(Table 3.13; Fig. 3.8 and 3.9). Volume attained on either side of the spike during the placebo conditions was more than 5 km lower than the volume attained during the supplemented condition. Of note is the relative degree of increase in training volume occuring during the supplemented conditions. The slope of the line leading into S1 and S2 increases are both steeper than the slope leading into the B2 and P1 conditions. This subject did not reach target heart rates for greater than 30 min. during two sessions of cycle 6 training (day 2 and day 3). Correlations between dietary carbohydrate intake and distance cycled generated a positive correlation of 0.49 for two day means and a negative correlation (-0.44) for the three day means not including days 5 and 6 (Figure 3.10). Both correlations failed to attain significance at the 0.05 level. The most severe symptoms of sickness were noted on day 3 of cycle 2. The symptoms began to abate after day 3 of cycle 2.

Table 3.11

J.T., Means (SD) for daily and cycle summaries of total recorded carbohydrate intake (g, g.kg-1 and g.kg-1.d-1)

Cycle:		1	2	3	4	5	6
Condition:		Baseline	Sup.	Baseline	Placebo	Sup.	Placebo
Daily results							
Day 2	(g)	505	709	403	579	1363	-
	(g/kg)	6.1	8.6	4.9	7.0	16.6	-
Day 3	(g)	756	848	406	490	952	-
	(g/kg)	9.2	10.3	4.9	6.0	11.6	-
Day 4	(g)	765	785	578	575	915	-
	(g/kg)	9.3	9.5	7.0	7.0	11.1	-
Cycle totals							
	Mean (g)	675	781	462	548	1077	
	SD	148	70	100	50	249	
	Mean (g/kg/d)	8.2	9.5	5.6	6.7	13.1	
	SD	1.8	0.8	1.2	0.6	3.0	
Grand totals							
	(g)	2026	2342	1387	1644	3230	

Table 3.12
J.T., Day means (SD) for carbohydrate intake (g and g.kg -1.d-1)

Condition	n (days)	Mean (SD) (g)	Mean (SD) (g.kg -1.d-1)
Before supplementation:			
Baseline	6	569 (162)	6.9 (2.0)
Placebo	1	490 (n.a.)	6.7 (n.a.)
Supplement	6	530 (209)	6.4 (2.5)
Rest Days	9	446 (147)	5.4 (1.8)
After supplementation:			
Supplement	2	929 (209)	11.3 (2.5)

Table 3.13

J.T. Means (SD) for daily and cycle totals, distances measured and trained (km).

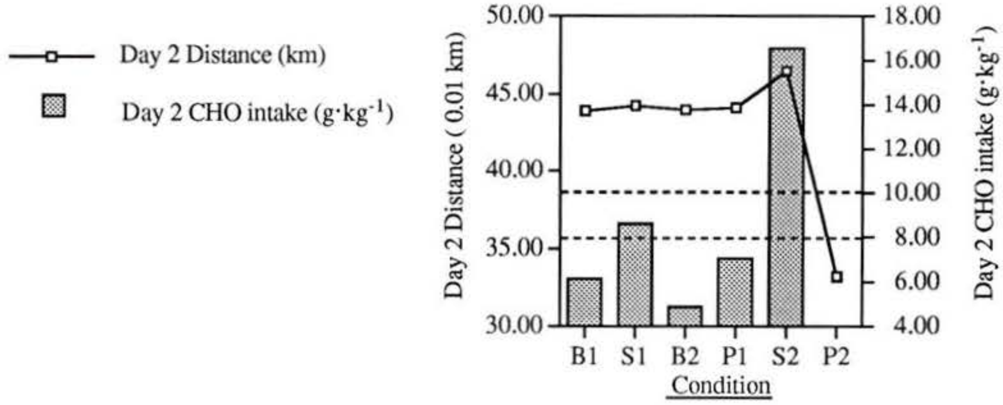
Cycle: Condition	1 B1	2 S1	3 B2	4 P1	5 S2	6 P2
Daily Totals:						
Day 2 (0.01km)	43.90**	44.23*	43.97**	44.10**	46.48	33.22**
Day 3 (0.01km)	72.59	78.89*	81.22	81.82	84.88	78.75**
Day 4 (0.01km)	32.80	35.56	34.98	36.82	(44.55)	(46.44)
3 Day Totals: (0.03 km)	149.29	158.68	160.17	162.76	(175.91)	(158.41)
2 Day Totals: (0.02 km)	116.49	123.12	125.19	125.92	131.36	111.97

bracketed distances recorded during day 4 of cycles 5 and 6 are not comparable to the previous four cycles due to the change in protocol.

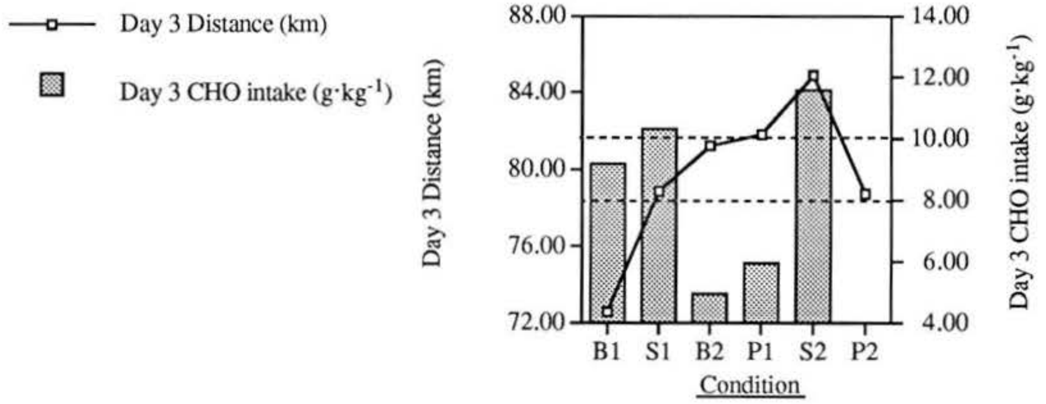
** below target hr due to claims of low energy.

* Feeling sick - seldom reaching target hr

J.T. Day 2: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)



J.T. Day 3: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)



J.T. Day 4: CHO intake ($g \cdot kg^{-1}$) and cycled distance (km)

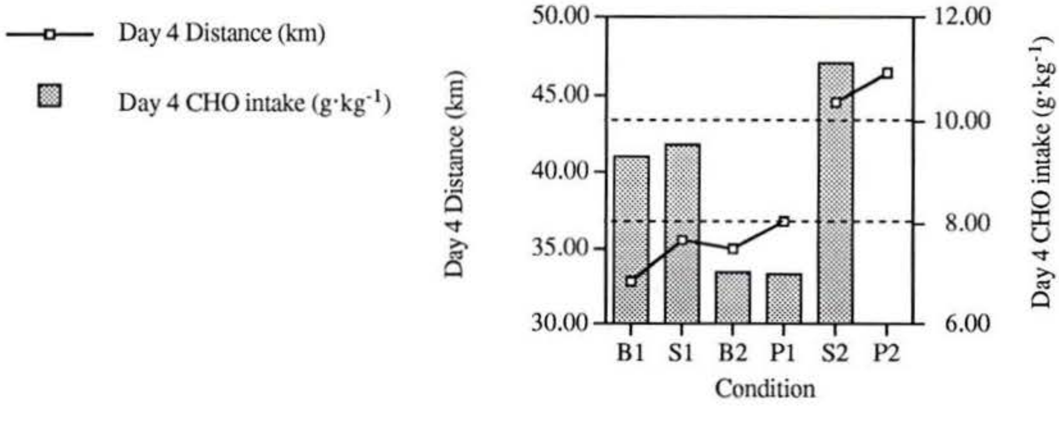
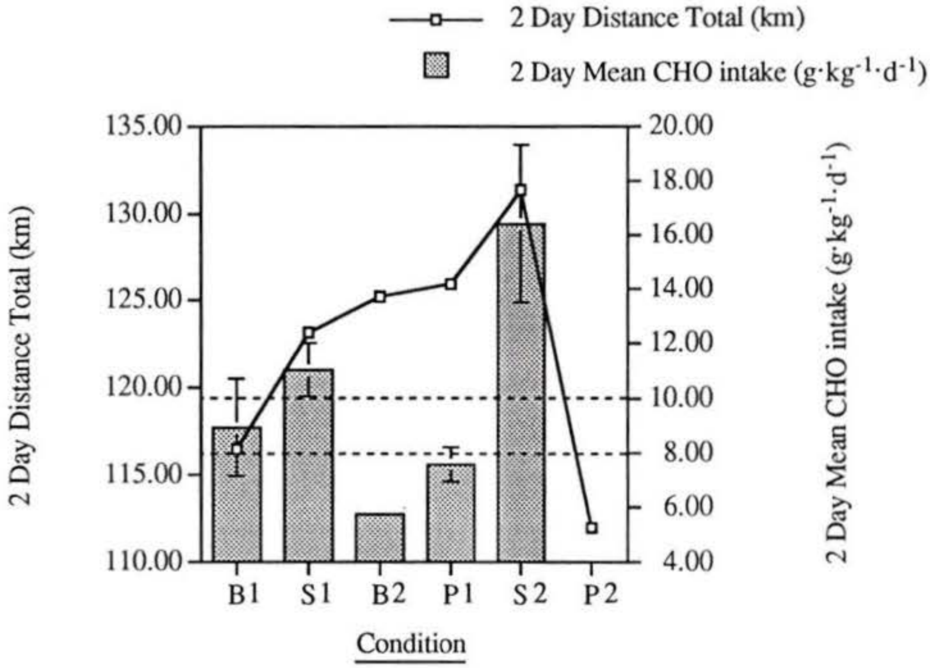


Figure 3.8
J.T. CHO intake ($g \cdot kg^{-1}$) and cycled distance (km) for the 3 training protocols

J.T. 2 day mean (SD) CHO intake ($g \cdot kg^{-1} \cdot d^{-1}$) and cycled distance (km)



J.T. 3 day mean (SD) CHO intake ($g \cdot kg^{-1} \cdot d^{-1}$) and cycled distance (SD)

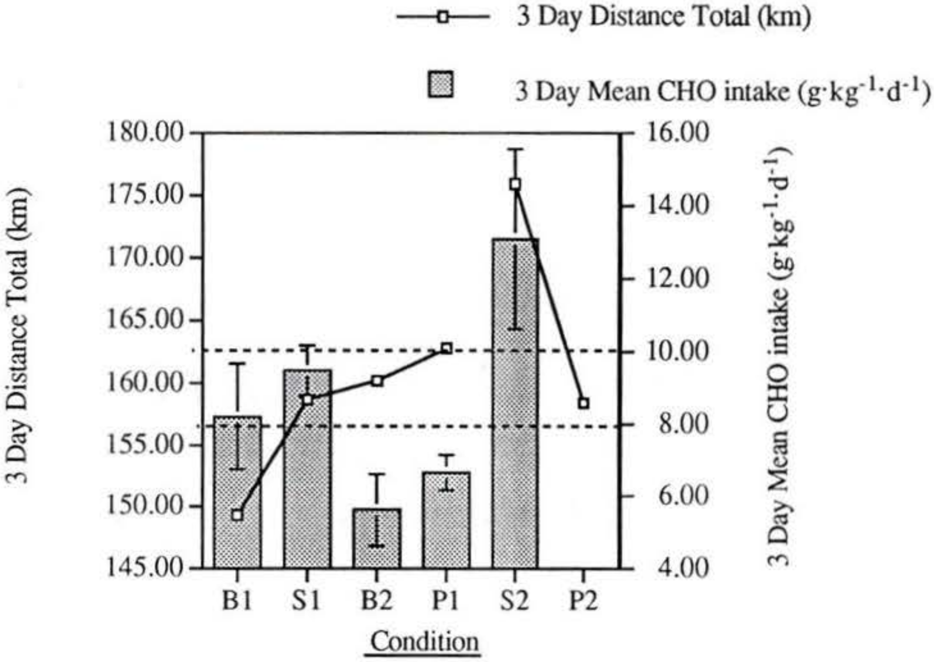


Figure 3.9
J.T. 2 & 3 day summaries of mean (SD) CHO intake ($g \cdot kg^{-1} \cdot d^{-1}$) and cycled distance (km)

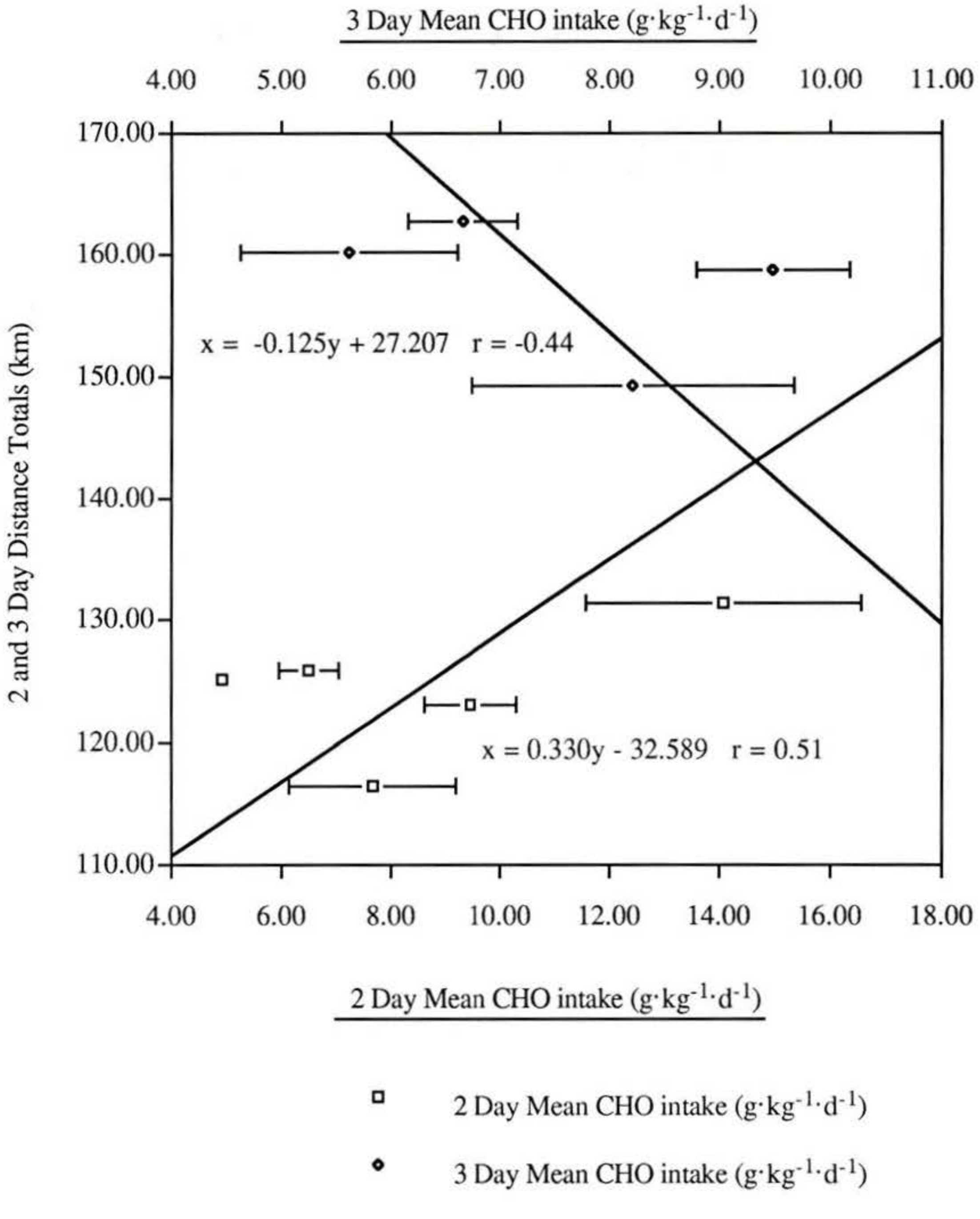


Figure 3.10

J.T. The relationship between mean (SD) CHO intkake ($g \cdot kg^{-1} \cdot d^{-1}$) and total distance cycled (km)

Table 3.14 summarises the number of session segments and training sessions when target heart rates were not attained within 10 min of training. None of the three subjects participated without failing to reach their target heart rate in at least seven segments throughout the training program. Only 5 of 28 failures occurred during the supplement condition - the rest occurred during either baseline (11) or placebo (12) conditions. Failures were characterised by "a general lack of energy" or muscle fatigue in the 23 unsupplemented occurrences. The 4 of the five failures recorded during the supplement condition occurred due to sickness or injury as documented on the training log sheets. None of the failures occurring during unsupplemented conditions appeared to be due to either sickness or injury. For J.T. sickness was verified by increases in waking heart rates of 15 beats.min^{-1}. Subjects struggled to finish cycling three times complaining of "a complete lack of energy". Each of these occurred during the placebo condition, in the last cycle of the study.

The number of sessions in which a segment failure occurred were recorded with the following results: failure occurred in 6 of 18 (33.3%) baseline sessions, 7 of 15 (46.7%) placebo sessions and in 4 of 20 (20%) supplement sessions.

Mean (SD) total training volume shows that for each subject, more distance was cycled during supplemented phases than during unsupplemented phases. Increases were 5.54, 7.10 and 4.71 km for a combined mean increase of 5.78 km per subject (Table 3.15).

There was no apparent relationship between perceived exertion and carbohydrate content (Appendix C and D).

Table 3.14

Target heart rate failure frequency totals for the three subjects A.H., P.N. & J.T.

Condition		Sessions Trained		Failures		Did not finish
			segments failed*	sessions failed**	failure frequency (session)-1	
Baseline (B)	A.H.	6	1	1		0
	P.N.	6	5	3		0
	J.T.	6	5	2		0
	Total:	18	11	6	33%	0
Placebo (P)	A.H.	3	2	2		1
	P.N.	6	4	2		0
	J.T.	6	6	3		2
	Total:	15	12	7	47%	3
Total (B & P)	A.H.	9	3	3		1
	P.N.	12	9	5		0
	J.T.	12	11	5		2
	Total:	33	23	13	39%	3
Supplement	A.H.	8	1	1***		0
	P.N.	6	1	1****		0
	J.T.	6	3	2*****		0
	Total:	20	5	4	20%	0

* training segments in which target heart rate was not achieved within 10 min. of training.

** number of sessions where subject failed to reach target hr in at least one segment.

***entire session missed due to swollen ankle.

**** reason for missing h.r. was a stomach cramp.

*****sick (15 beat higher greater than normal morning hr) with accompanying flu-like symptoms: fever, general non-exercise associated fatigue, stomach ache

Table 3.15

Mean (SD) change in training volume (km) per training cycle (day 2 and day 3 training) under supplemented and unsupplemented conditions

Subject	Condition				
	Unsupplemented		Supplemented		Change
	n	(km)	n	(km)	(km)
P.N.	3	113.96 (1.14)	2	119.50 (2.48)	+ 5.54
A.H.	2	129.68 (1.39)	3	136.78 (2.78)	+ 7.10
J.T	3	122.53 (5.25)	2	127.24 (5.83)	+ 4.71

Mean Change in training volume + 5.78 (1.21)

CHAPTER IV

DISCUSSION

This study was designed to verify recommendations in the current literature regarding inter-workout dietary supplementation for optimal glycogen repletion under realistic training conditions. Specifically, the study examined the effects of a complex carbohydrate supplement drink ($4g \cdot kg^{-1}bw. \cdot d^{-1}$), ingested immediately after each workout on training volume in a series of three training sessions performed at intensities from approximately 60 - 88% of $\dot{V}O_2max$. Training volume was defined as the total distance cycled at prescribed heart rates. Single subject design was selected to provide detailed analysis of the individual cases.

The validity of the study protocol will be discussed and followed by descriptions of how dietary supplementation effected net dietary intake in the three subjects. Individual dietary and training results, both daily and summarised over 2 and 3 days, will be examined for evidence to support or refute the premise that changes in dietary intake due to supplementation correspond with changes in training volume. Results from three cases will be discussed individually and then as a group before general conclusions and recommendations will be presented.

Choosing single subject design methodology instead of group design in a study of training performance is relatively unique. No single subject research was obtainable for comparison in the field of sport physiology. However, given the nature of this study, single subject format has proven an effective method of analysing previously validated data in field application.

One of the limitations of group analysis is the inability to focus and answer questions and problems for specific individuals (Kazdin, 1982). Results obtained through group analysis may or may not apply to an individual due to specific variables unique to that individual or population subset he/she represents. Repeated individual case testing of conclusions derived from group analysis can be performed to determine a clinical (or applied) significance. Single subject studies present opportunities to discover and document results defined by the individual athlete in his/her particular mental and physical environment by including more detail in results than is acceptable in group design studies. Unexpected results or individual peculiarities can develop more questions suggesting foci for further studies. Both group and single subject designs can logically complement each other because they seek to answer different questions relating to the same issue. In fact, regarding any single research question, a natural experimental feedback loop can develop in which results obtained from one design method develop questions and research options for the other design method. Each cycle of the loop alternatively validates results from either a more therapeutic or experimental perspective.

In the field of application, results are discussed within the context of the individual subject and determined significant only within the perspective of that individual. Single subject methodology mirrors the interdependent relationship between cause and effect that defines the foundation of coaching technology. Therefore results obtained through single subject design may be more applicable to the field.

Classical single-subject design methodology requires a stable baseline developed from a minimum of five data points before an intervention can be introduced (Kazdin, 1982). This criteria may be difficult to meet in the field of exercise physiology. Asking the subjects to perform the same training protocol repeatedly could introduce staleness and complicate the analysis with motivational problems. A stable baseline may not be possible if a training effect develops. It this case, five days of exhaustive, sub maximal training could also produce debilitating depletion levels (Costill, 1988; Costill, 1971). Instead, a repeating baseline was relied upon to perform the same function.

Considering the subjects' extensive experience, little variability due to a learning effect was expected. However, it was possible that some variability occurred due to social, health, and motivational reasons - factors present under normal training conditions. Training protocol changed over the three successive recording sessions encompassing each training condition. Because these three data points were not comparable, variability in performance within a training cycle was not measurable. It is possible that some variability due to individual factors did occur so athletes recorded anything they thought may impact on performance in their training logs. These entries were used as ancillary evidence and as collaberative information in the discussion. Blocking of data into groups of 2 and 3 consecutive days was done to correct for variability and to provide a more representative effect. Given the lack of constraints in this study, greater validity is attributed to blocked results of 2 and 3 days. Any extraneous factors impacting on a daily training session were partially muted when summarised over the days encompassing the treatment cycle.

Costill, Bowers, Branam and Sparks (1971) demonstrated that glycogen depletion in subsequent days of training could be cumulative. Over three days of training an average of 72 min at 80% of $\dot{V}O_2max$, mean pre- exercise muscle glycogen concentration progressively decreased approximately 45% despite the consumption of mixed diets (40 - 60% carbohydrate, 30 - 40% fats, and 10 - 15% protein). Within the current study, training volume occurring in subsequent days within the same training cycle were also totalled to account for any variability in training due to possible decreasing muscle glycogen levels. If training was sufficiently vigorous to produce a cumulative depletion effect from successive days of training, as in the previously mentioned research, the glycogen stores not depleted in a previous session due to any reason would be more available and allow for greater training volume in the subsequent session. For example, referring to the daily performance graphs for P.N., neither day 2, day 3 nor day 4 training produced ideal visual evidence to support the thesis premise. However, when blocked into two and three day summaries, data points produced changes in means which follow the expected pattern. Blocking of two and three sessions effectively accounted for any variability effecting performance in addition to supporting the presumption that the training was intense enough to cause cumulative depletion. While daily results did not correlate as strongly as in the summary graphs, this does not exclude the possibility that dietary intake 24 hours prior to exercise may effect training in that specific workout. Either the study design was not sensitive enough or was not constructed in a way to identify inter-workout variability in training performance. Supportive evidence was most pronounced in the summaries due to a cumulative effect of depletion on training performance over the three days.

Study protocol was designed as rigorously as possible so that the desired effects of supplementation would be apparent, without the use of parametric statistics, eliciting obvious and significant therapeutic benefits to the subject. The ability to demonstrate reversal is a strong indicator of a dependent relationship (Kazdin, 1982). Causal inferences were made as application and subsequent withdrawl of the intervention repeatedly reversed the direction of results consistent with expectations based on the thesis hypothesis. Differences in training performance were identified through visual analysis of the graphed results.

Experienced cyclists were chosen as subjects for a number of reasons. They were familiar with and gained the greatest conditioning benefit from training continuously at or about their anaerobic threshold. They were personally motivated for consistent effort to improve their competitive racing abilities. A less skilled or less trained group would be more influenced by learning and training effects. Typical instruments of the sport including a stopwatch, odometer, heart rate monitor, wind trainer and bicycle were utilised to measure changes in training volume. The use of practical instruments and the design of the study attempted to obtain optimal internal and ecological validity. To ensure the appropriateness of the training program, an elite cycling coach was consulted in the design of the three individual sessions.

The protocol was designed to evoke the greatest glycogen depletion and optimise the desired training effects necessary for cycling racing. While measuring cycling time to exhaustion at between 70 - 80% of $\dot{V}O_2max$ each day would provide the best indicator of fatigue based on tissue glycogen stores (Saltin & Karlsson, 1967) the thought of exercising at such intensities and durations without some variation in training protocol was unrealistic and could have caused motivational problems. Therefore, training protocols were developed to: (a) resemble normal training, (b) provide enough variation to keep the athletes from losing motivation, (c) be rigorous enough to achieve daily depletion of muscle glycogen and (d) provide enough opportunities to collect data and record changes in training performance due to the difference in dietary carbohydrate intake and its effect on tissue glycogen stores.

A summary of the three recorded training days were presented in Tables 2.4 and 2.5. Measured against pre- and post study $\dot{V}O_2$max tests and interpolated on the basis of target heart rates, sub maximal training intensities expressed as a percentage of $\dot{V}O_2$max increased but remained within the range associated with glycogen depletion (Tables 3.3 and 3.4). In addition to changes in V.T. and $\dot{V}O_2$max scores after training, these results strongly support the inductive claims of the training protocols and suggest that training volume and intensity was sufficient to regularily deplete muscle glycogen stores.

Saltin and Karlsson, (1971) showed that depletion of glycogen in working muscle was the limiting factor for exhaustion at work rates of between 60 and 89% of $\dot{V}O_2$max for durations of 45 - 200 min. Work loads below 60% $\dot{V}O_2$max permitted continuous effort, (unlimited by substrate) and above 90% VO_2max were limited by the accumulation of anaerobic by-products and a depletion of high-energy phosphates. Subsequently, exercise protocols designed to deplete muscle glycogen have varied amounts of training within this range (Ivy , Katz, Cutler, Sherman & Coyle, 1988a; Ivy, Lee, Brozinick & Reed, 1988b; Sahlin, Katz & Broberg, 1990). Similarly, the four protocols employed in this study were comprised of 20 - 30 min of free cycling and 100 - 125 min of cycling at between 59 and 88% of $\dot{V}O_2$max. (Table 2.4). Biopsies were not performed to verify degrees of glycogen depletion. Training times, intensities and volumes comprising the individual protocols resemble the methods designed to deplete glycogen within a single session (Ivy et al., 1988a,1988b; Sahlin et al., 1990). Coupled with the initial 100 km ride on day one of each cycle, it is possible that during unsupplemented conditions, four successive workouts induced a cumulative depletive state similar to that produced by Costill (1971). Of particular note was the occurrence of failure due to muscular fatigue at least once in each of the four protocols, predominantly during placebo and baseline conditions (Table 3.14) and subjective comments from the athletes of lower energy levels during the same conditions. No significant difference in perceived exertion or expected performance scores were noticed.

It is reasonable to assume that a failure to achieve target heart rates was partially due to decreased muscular glycogen. It is unlikely that failures were due to a buildup of the metabolic end products of anaerobic metabolism (i.e. lactate, H^+, P_i, ADP) because the failures occurred during training in a range of intensities from between 59% and 88% of $\dot{V}O_2max$ - within the range of aerobic work intensities for these athletes (Saltin & Karlsson, 1972; Hermansen et al., 1976). Furthermore, subjects demonstrated the ability to successfully complete each of the different protocols at other times in the study.

Defining the dependent variable as training volume at set heart rates was based on the indirect relationship between training heart rate and oxygen consumption. Based on that relationship, it was also expected to allow for adjustment because of increased aerobic conditioning. Because heart rate (HR) at any specific power output decreases after training (Ekblom, Astrand, Saltin, Stenberg & Wallstrom, 1968; Winder, Hagberg, Hickson Ehsani & McLane, 1978), exercising at a fixed HR throughout the course of the study should have increased the cardiovascular and muscular stress on the subjects comparable with their increasing cardiovascular and muscular fitness. This criterion was also reflective of muscle glycogen levels. It has been determined that training heart rate increases with decreasing muscle glycogen content (Heigenhauser, Sutton & Jones, 1983; Schwellnus et al., 1990; Sahlin & Katz, 1990). Therefore, to train at a specified HR, athletes with muscle glycogen levels compromised by decreases in dietary carbohydrate intake should cycle at a lower intensity and cover less distance.

Training intensity was calculated as a percentage of the individual's V.T. to standardise intensity between subjects because the relationship between power output and VO_2 is subject-specific. Alternatively, choosing a range of training intensities based on percentages of VO_2 max may have been above the V.T. and limited performance due to build up of the end products of anaerobic metabolism.

Training heart rates also increase with dehydration levels in what is called the "cardiovascular drift". Therefore, maintaining tissue hydration during training was a priority. Cardiovascular drift was minimized by ingesting enough water to maintain training body weight (Hamilton, Gonzalez-Alonso, Montain & Coyle, 1991; Montain & Coyle, 1992). This was accomplished throughout the study by ingesting a 2.5% complex carbohydrate liquid mixture at the rate of 600 - 800ml per hour and validated with before and after training body weight. Subjects were able to maintain a stable body weight with *ad libitum* liquid intake for the first cycle and kept ingestion volume constant in subsequent cycles. Based on the literature, it is assumed that this hydration strategy effectively standardised and minimised the degree of drift within training sessions.

Training heart rates can also be influenced by emotions of apprehension and excitement, diet, muscle temperature, environmental temperature, air humidity and sickness (Bergstrom, Hermansen Hultman & Saltin, 1967; Janssen, 1989; McCafferty, 1978,). Apart from sickness, which in one case was reflected in resting heart rates, the other factors remained constant throughout the study and, as a result, were presumed to have negligible effects.

Bergstrom and Hultman (1967) determined a correlation of 0.91 between pulse rate and glycogen utilisation. They concluded that the exercise heart rate was a good measure of relative work load. Therefore, cycling greater distances at a set heart rate can be considered a performance advantage since greater power outputs are generated at a set intensity level.

Conversely, training set power outputs could be generated at a lower heart rate and, consequently, with a lower cost to muscle glycogen stores.

The effect of carbohydrate supplementation may be more noticeable in day 3 because the training volume (by design) was greater than during day 2 and day 4. No weighting formula was introduced in the results to account for this difference although individual session results were presented for comparison. Since training volume generated during day 4 training was the lowest of the three sessions, day 4 training protocol was altered before the fifth cycle to potentially increase the demands on muscle glycogen in the expectation of a greater effect. During the altered protocol training volume was measured while subjects trained as close to their maximum heart rate level as possible (equating to 80 - 90%of VO_2max) for one hour after a one hour, progressive warmup. At this point in the study subjects appeared capable of adjusting to an increase in training demands. Such a change in protocol would not be acceptable in a study employing a between - group design.

Case Discussions:

P.N.

Subject P.N. recorded pre-study oxygen utilisation values of 3.64 L·min^{-1} (51.3 ml·kg^{-1}·min^{-1}) and 2.80 L·min^{-1} (39.7 ml·kg^{-1}·min^{-1}) at $\dot{V}O_2$max and VT. The training was demanding but provided the stimulus to significantly increase his VT 14% to 3.20 L·min^{-1} and maintain $\dot{V}O_2$max at 3.60 L·min^{-1} (50.9 ml·kg^{-1}·min^{-1}). Based on pre and post study submaximal $\dot{V}O_2$max tests, training intensities at the three established training heart rates increased from 64 to 69% and 75 to 77% of $\dot{V}O_2$max, while VT remained stable at 86 and 85% of $\dot{V}O_2$max (Fig 3.1 and Tables 3.3 & 3.4). Therefore, submaximal training heart rates decreased as a result of training. While tissue glycogen levels were not assessed, based on the changes in oxygen utilization after training, the program appeared strenuous enough to develop a certain amount of depletion on a regular basis.

Increasing carbohydrate intake by 4 g·d^{-1} per day effectively generated the experimentally desired intake patterns for P.N. in each of the three day-graphs. Visual analysis of carbohydrate intake across baseline and placebo conditions in the 2 and 3 day summaries show slightly declining levels suggesting that the introduction of the placebo had no effect on carbohydrate intake. Therefore, no dietary adjustments due to a learning effect were anticipated for this subject. Dietary supplementation at 4 g·kg^{-1}·d^{-1} successfully increased mean caloric and carbohydrate intakes 33% and 64% to 4078(414) kcal and 720(41)g·d^{-1} or expressed in relative terms carbohydrate intake increased to 10.1(0.6) g·kg^{-1}·d^{-1}. The supplement also altered dietary composition to 71, 20 and 10% carbohydrate, fat and protein from non supplemented levels of 58, 28 and 14%. During training phases in which the supplement was not provided P.N. consistently ingested less than recommended nutrient intakes of dietary carbohydrate. Since *ad libitum* dietary intake for P.N. produced the desired variation it was anticipated that training volume would follow as expected. Over the period of the study, the subject's weight remained constant whereas the sum of six skin-folds decreased 1.2 mm over the thirty days of the study suggesting mean caloric intake was adequate. Prior to the study P.N. experienced difficulty reaching the 70% carbohydrate intake level. In a post study questionaire P.N. wrote "the supplement drinks most definitely had an effect on my riding, my day-to-day recovery and how well I felt each day". He did not know what he was drinking and felt both the supplement and placebo drinks "tasted awful" and "worked as appetite suppressors" for between 2 - 3 hours. This may have been a concern because, in effect, the placebo trial may have prevented P.N. from metabolising any foods in the first three hours after training. However, he consistently ate his first post exercise meal within 3 hours of training regardless of the treatment phase. Time taken to clean-up, dress, travel and prepare food was the same and determined the time from exercise cessation to the first meal.

As mentioned earlier, graphical representation of the daily training performance by condition often differed from the expected pattern. However, grouped into 2 and 3 day totals, the graphs showed the expected pattern of response. Changes in total training volume corresponded identically with changes in mean carbohydrate intake when summarised in the 2 day graph. Correlation coefficients were calculated at 0.87 and 0.54 for the 2 and 3 day summaries. When observed as summaries, training volume under supplementation consistently exceeded training volume without supplementation. Graphically, peaks and valleys corresponded as expected to training either with or without supplementation indicating that for this subject, increased carbohydrate intake due to supplementation positively effected training volume.

In the training and competitive season after finishing this study P.N. increased his racing performance category to one higher level. He was convinced that the supplement had a positive effect on his ability to recover from training during periods of actual supplementation. The effect was most pronounced in the first 3-4 hours after ingestion. He mentioned that he "felt considerably fresher" a few hours after exercise instead of still feeling fatigued as he would have without the supplement. The supplement allowed him to efficiently increase his dietary intake because time to eat was a challange in his life. He continued to supplement his diet during successive days of high volume training and learned to adjusted the flavour with fruit juices to suit his personal taste.

A.H.

Subject A.H. was a professional mountain-bike rider with maximum aerobic power of 5.13 $L\cdot min^{-1}$ (66.3 $ml\cdot kg^{-1}\cdot min^{-1}$) and a VT at 3.50 $L\cdot min^{-1}$ (45.2 $ml\cdot kg^{-1}\cdot min^{-1}$). His $\dot{V}O_2max$ fell slightly below the range of 67 - 77 quoted by Lamb et al. (1984) defining elite cyclists. Training was rigorous enough to induce physiological changes over the thirty days of training, as evidenced by a stable sum of six skin-folds coupled with an increase in body weight (77.4 to 78.3 kg), a 3% increase in $\dot{V}O_2max$ (5.28 $L\cdot min^{-1}$), a 11% increase in VT (3.87 $L\cdot min^{-1}$), and increases in training intensity at given submaximal heart rates. Based on pre- and post study submaximal $\dot{V}O_2max$ tests, training intensities at the two of the three lower established training heart rates increased from 63 to 65% and 73 to 74% of $\dot{V}O_2max$ whereas the highest, decreased from 88 to 85% of $\dot{V}O_2max$ (Fig 3.1 and Tables 3.3 & 3.4). A.H. failed to reach target heart rates only once during supplemented conditions due to complaints of a swollen ankle and 3 times during unsupplemented conditions (Table 3.14). These results suggest the training significantly challenged the subject through successive cycles of work just below the anaerobic threshold. Therefore, some inter-workout glycogen depletion is suggested to have occurred. This subject did not participate in one session (cycle 3, day 4) during the study due to a debilitating knee problem. Rest and physiotherapy enabled his return.

Dietary supplementation at 4 $g\cdot kg^{-1}\cdot d^{-1}$ successfully increased mean caloric and carbohydrate intakes 27% and 39% to 5585(545) kcal and 926(83)$g\cdot d^{-1}$ (or 12.0 (1.1) $g\cdot kg^{-1}\cdot d^{-1}$. It also altered dietary composition to 66, 24 and 10% carbohydrate, fat and protein from 55, 33 and 14%. During training phases in which the supplement was not provided, carbohydrate intake for A.H. averaged 8.6 (1.34) $g \cdot kg^{-1} \cdot d^{-1}$, exceeding the $8g \cdot kg^{-1} \cdot d^{-1}$ standard five out of nine times, and the $10g \cdot kg^{-1} \cdot d^{-1}$ standard twice. While supplementation successfully increased intake past the $10g \cdot kg^{-1} \cdot d^{-1}$ standard, the subject independently increased dietary intake so that a large difference in net dietary intake did not develop between the different phases.

Visually, the daily performance graphs followed the expected pattern except for two sessions. Training volume recorded during the second supplemented session was lower than expected and possibly attributed to a harder than prescribed ride on the preceding day. Cumulative fatigue from the previous two sessions may have negatively affected training volume in the first supplement condition of day 4 training.

The effect of the supplement could be seen in the analysis of daily results as well as in the summaries. Distance cycled during supplemented conditions were consistently higher than all total distances cycled during non-supplemented conditions in both the 2 and 3 day summaries. Including the non-overlapping data, changes in mean, level and trend were as expected and the high correlation coefficients ($r = 0.94$ for the 2 day summary and $r = 0.99$ for the 3 day summary) suggest that dietary carbohydrate intake had an effect on training volume. Correlations may have been even higher had A.H. followed the intensity criterion on the first day of the fourth cycle so that his performance on day 2 of that cycle was not compromised.

Although A.H. was a professional mountain-bike rider he had previously followed a self - directed training program and had never used a carbohydrate supplement to augment dietary intake. Since the study he continues to use the carbohydrate supplement during periods of heavy training. The dietary analysis ensured an adequate carbohydrate intake but encouraged supplementation during training of high volume. In the season after the study he improved from top 10 Provincial standing to a top 10 National standing and signed a professional contract with a sponsor. He attributed a great deal of his success to the training and dietary behaviours he learned while participating in this study.

J.T.

J.T. was another professional mountain-bike rider with an initial $\dot{V}O_2max$ of 5.36 L·min^{-1} (65.1 ml·min^{-1}·kg^{-1}) and a VT at 4.30 L·min^{-1} (52.2 ml·kg^{-1}·min^{-1}). After 30 days of training $\dot{V}O_2max$ increased 4% to 5.57 L·min^{-1} (68.6 ml·kg^{-1}·min^{-1})and VT increased 3% to 4.40 L·min^{-1} (53.2 ml·kg^{-1}·min^{-1}). Based on pre- and post study sub maximal $\dot{V}O_2max$ tests, training intensities at the established training heart rates increased from 59 to 68%, 69 to 76% and 80 to 84% of $\dot{V}O_2max$ (Fig 3.1 and Tables 3.3 & 3.4). These physiological changes suggest training was sufficient to deplete muscle glycogen on a daily basis. Unfortunately, this subject suffered through a mild sickness, verified by a change in waking heart rates of 10 bpm for approximately one week in the early portion of the study. He was permitted to continue because he met all other training criteria.

Dietary supplementation at 4 g.kg-1.d^{-1} successfully increased mean caloric and carbohydrate intakes 31% and 69% to 4842(922) kcal and 929(209)g.d^{-1} or 11.3 (2.5) g.d^{-1}. It also altered dietary composition to 78, 17 and 5% carbohydrate, fat and protein from non supplemented levels of 64, 27 and 9%. During training phases when the supplement was not provided, carbohydrate intake for J.T averaged 6.7 (1.4) g·kg^{-1}.d^{-1}, below the 8g·kg^{-1}.d^{-1} standard seven out of nine times, and never over 10g.kg^{-1}.d^{-1}. Supplementation successfully increased intake past the 8g.kg^{-1}.d^{-1} standard in every case and above 10g.kg^{-1}.d^{-1} four of six times. The calculation for mean intake with supplementation was effected by an abnormally high dietary intake of 16.6 g.kg^{-1}. No effect of sickness on dietary intake was apparent. J.T. felt satiated in the first hour after drinking the supplement. At times he also felt nauseas and had a headache after the drink. Otherwise he did not mind the "after-effects" and appreciated the difference it made to his training.

J.T. did his best through his sickness which peaked during the first supplemented phase of his program. Visual analysis of daily results shows the effect of the sickness on performance in many ways. In the day 2 training graph, training performance in the first supplemented phase was indistinguishable from training performance attained during adjacent baseline and placebo conditions. The change in resting heart rate occurring with the sickness appeared to impact on training volume by allowing him to train at lower intensities to elicit the same heart rate (Janssen,1989). This conclusion was supported in the day 3 training graph where the athlete's improving condition throughout the study was evident as training volume increased but started leveling off up until the second supplemented phase. At this point the slope of the performance line increased against the developing trend and then decreased rapidly on the other side when the supplement was withdrawn. Day 4 performance was more supportive of the premise and again indicated an athlete experiencing an increase in fitness level.

It is suggested that J.T. started the study sick, reached a peak in sickness during or around the first supplemented phase (as supported by elevated morning heart rates) and progressively increased in health as the study continued. Since the slope of the performance line was greater before than after the first supplemented phase it is suggested that carbohydrate supplementation positively effected training volume despite the sickness. This is supported by the summary graphs as well. In all but the last session, training volume attained during the second supplemented phase was the highest of the six phases. Training volume attained in the previous two days were significantly lower than in previous cycles perhaps reflecting a larger volume of available muscle glycogen. Additionally, the athlete may have been motivated towards finishing the study.

Little support is derived from the coefficients obtained in the 2 and 3 day correlations ($r = 0.49$ and $r = -0.44$) of dietary intake with training volume. This was probably due to the problems that accompanied his sickness. Had training volume measured in the first two training phases been higher the correlations would have been considerably stronger. The presence of a negative correlation was an accurate indicator of sickness and may possibly merit some further investigation as an indicator of health status in athletes.

This case included two supplemented phases and three baseline or placebo phases with complete data. Although the subject's sickness complicated interpretation, visual analysis still supports the study premise. Evidence is primarily derived from the change in slope on either side of the volume peaks obtained in the two supplemented conditions and the sharp change in the mean of the second supplemented condition.

In a post study questionaire, J.T. stated feeling more motivated during his last training cycle (placebo) than in the previous cycle (supplement). Despite that, he still produced a higher volume of work in the supplement cycle compared to the placebo cycle. This subject was convinced the supplement aided in recovery after training and continued using it to supplement his diet during heavy training. He had difficulty ingesting enough carbohydrates due to time and financial challenges and appreciated the difference the supplement made. Like the previous mountain bike rider, J.T. acquired a major riding sponsor after graduating into the National elite level of riding in the season after the study. Instead of supplementing his diet with a premixed drink he now places a greater emphasis on eating as soon after training as possible.

General Discussion and Conclusions

Regardless of the effect *ad libitum* intake had on dietary carbohydrate intake, training volume was consistently higher during periods of post exercise supplementation than during either placebo or baseline phases. The strength of the effect is supported by repeated performance reversals in response to the four or five changes in condition that occurred for each athlete and the strong correlation coefficients obtained in two of three cases (low correlations in the third case were due to sickness). A placebo effect was discounted since mean training volumes obtained during placebo phases were similar to those obtained during baseline phases. Post experiment questionares indicate the subjects were unaware of drink compositions. Placebo trials were of particular significance from a therapeutic as well as experimental perspective because in field application, athletes are aware of the contents of the drinks.

Carbohydrate supplementation raised carbohydrate intakes, as expected by increasing both caloric and carbohydrate intakes in each case to 147%, 150% and 160% of the recommended intake levels and changed dietary composition as expected by increasing relative carbohydrate and decreasing relative fat and protein intake. Athletes increased carbohydrate intake percentages of 66, 71 and 78% from 54, 58 and 64%. These values are a little lower than the 80% carbohydrate intake level recorded by cyclists in two days of a mock Tour de France race to reach energy balance (Brouns, Saris, Stroecken, Beckers, Thijssen, Rehrer & F.ten Hoor, 1989). For two of the athletes, dietary composition increased into the range of 60 - 70%, reportedly necessary by Costill et al., (1988) to permit daily training at intensities which deplete muscle glycogen. Greater increases in relative carbohydrate and protein intakes would occur if A.H. decreased his fat intake. Referring to relative intake as percentages of caloric intake does not seem valid in this context. Indeed muscle glycogen content is correlated most strongly to dietary carbohydrate volume than relative intakes.

Supplement drinks were designed to provide enough carbohydrate to meet substrate needs in the first three to four hours after training and to increase total intake past 10 g·kg^{-1}. Intakes past 10 g·kg^{-1}·d^{-1} were successfully obtained in 18 of 21 supplemented sessions with net intake ranging from 9.2 g·kg^{-1}·d^{-1} to 16.6 g·kg^{-1}·d^{-1}. Expressed as 3 day averages, mean dietary carbohydrate intakes for the three subjects were consistently over 10 g·kg^{-1}·d^{-1} at 10.1 (0.6), 12.0 (1.1) and 11.3 (2.5) g·kg^{-1}·d^{-1}, suggesting that the 4 g·kg^{-1}·d^{-1} supplementation level was effective in raising carbohydrate intake to a range (9.3

to 11.3 $g\cdot kg^{-1}\cdot d^{-1}$) associated with full glycogen repletion within twenty four hours after training of this intensity and duration (Bergstrom, Hultman, & Roch-Norland, 1972; Kochan, Lamb, Lutz, Perrill, Reimann & Schlender, 1977; Sherman & Wimer, 1991 and Simonsen, Sherman, Lamb, Dernbach, Doyle & Strauss, 1990). Supplementation less than 4 $g\cdot kg^{-1}$ may not have been sufficient to ensure dietary intake levels of 10 $g\cdot kg^{-1}$ level in these three athletes. All three subjects maintained their body weight over the 30 days suggesting this supplementation level more than fullfilled their energy needs.

Uncontrolled dietary intake produced a range of net intake quantities. Daily carbohydrate intake without supplement ranged from 3.90 to 12.27 $g\cdot kg^{-1}$ with mean intakes for the three athletes averaging 6.02 (0.64), 6.83 (1.40) and 8.65 (1.35)$g\cdot kg^{-1}\cdot d^{-1}$. These intakes were above the 5.0 $g\cdot kg^{-1}\cdot d^{-1}$ intake level reported by Costill et al., (1981) for swimmers who could not tolerate an increase in training volume. They reported 20% and 15% decreases in pre-exercise muscle glycogen content over the ten days of training at average intakes of 5.3 $g\cdot kg^{-1}\cdot d^{-1}$ and 8.2 $g\cdot kg^{-1}\cdot d^{-1}$. Simonsen et al., (1990) showed that training 4 weeks under diets of either 5 or 10 $g\cdot kg^{-1}\cdot d^{-1}$ resulted in pre-exercise muscle glycogen increases from 94 to 155 $mmol\cdot kg^{-1}$ with the high carbohydrate diet and a maintenance of pre-exercise muscle glycogen at 119 $mmol\cdot kg^{-1}$ with the low carbohydrate diet. The intake values recorded in this current study are in a range reported by these and other authors (Sherman, 1991; Keizer, 1987; Sherman, Doyle & Lamb, 1991) to have no detrimental effect on training but can permit declines in muscle glycogen with repeated days of exhaustive exercise. This suggests that if the training was demanding enough on muscle glycogen stores, intake volumes without supplementation were insufficient to fully replete muscle glycogen between workouts.

While no research could be found to conclusively demonstrate that a moderate carbohydrate diet (5 - 8 $g\cdot kg^{-1}\cdot d^{-1}$) impairs the ability to complete the required task, intake volumes greater than 10 $g \cdot kg^{-1} \cdot d^{-1}$ have been proven to facilitate greater performance gains over an equal amount of time (Sherman et al., 1991; Simonsen et al., 1990). Simonsen et al., (1990) compared changes in power output for three 2,500m time trials for rowers training 4 weeks under diets of either 5 or 10 $g \cdot kg^{-1} \cdot d^{-1}$. After training, power output increased 10.7% with the high carbohydrate diet whereas the moderate carbohydrate diet increased power output by only 1.6%. Intake levels recorded in this study during periods of supplementation exceeded the 10 $g \cdot kg^{-1} \cdot d^{-1}$ associated with greater performance gains with training. Thus, the increase in dietary intake volume may have contributed to the higher training volume under supplemented conditions in the present study.

Athletes reported "feeling fresher" or "more energised" 3 - 6 hours after supplementation than they did without. This may have been a result of the carbohydrate supplement. Costill (1990) reported that the effects of muscle damage can be partially overcome by ingestion of increased amounts of carbohydrates. Muscle weakness and fatigue after exhaustive endurance exercise have also been reported to be related to insufficient repletion of the glycogen stores (Hultman, 1967; Young & Davies, 1984).

Ideally, and for experimental purposes, carbohydrate intake should have remained above 10 $g \cdot kg^{-1}$ during supplementation and well below 8 $g \cdot kg^{-1}$ without supplementation. The decision to protect external validity by permitting *ad libitum* intake of dietary carbohydrate may have seriously complicated the study by not providing enough variance in carbohydrate intake between the treatment and baseline conditions to effect changes in training performance. Also, satiation during placebo conditions may have deterred athletes from ingesting foods which may have confounded the comparison between baseline and placebo trials.

Correlation coefficients of 0.87 and 0.94 were obtained from the 2 day summary graphs of dietary intake and training volume for subjects P.N. and A.H. These correlations are higher than those reported by Costill (1981) ($r= 0.84$, $p < 0.05$) for the relationship between increasing amounts of dietary carbohydrate and glycogen repletion within 24 hours of depletive training. These correlations were unexpectedly high given that the clinical approach to this study permitted the inclusion of a wide range of uncontrolled variables. Additionally, such strong correlations were not expected as dietary carbohydrate intake exceeded 10 $g \cdot kg^{-1} \cdot d^{-1}$. In particular, for A.H., correlation coefficients of 0.98 and 0.88 were calculated for training volume and total dietary carbohydrate intake despite recording intakes ranging from 7.5 to 15.5 $g \cdot kg^{-1}$. Ingesting more than 10 $g \cdot kg^{-1}$ appeared to benefit this athlete.

The relationship between dietary CHO intake and maximal glycogen storage (and indirectly, training volume) has been suggested to exceed 10 $g \cdot kg^{-1} \cdot d^{-1}$. Costill et al., (1981) investigated the effect of dietary carbohydrate volume on muscle glycogen storage by progressively increasing dietary carbohydrate intake and determined that consumption of 525 to 648 g (for a 72 kg man) carbohydrate during the 24 hours after strenuous running resulted in normal muscle glycogen levels. But because no plateauing of intake volume occurred, they suggested increasing dietary carbohydrate levels past 648 $g \cdot d^{-1}$ might maximise muscle glycogen repletion past normal levels. Blom, Vaage, Randel, and Hermansen (1980), recommend intakes of as much as 588 - 840 g carbohydrate per day (or 8.4 - 12.0 $g \cdot kg^{-1} \cdot d^{-1}$) for a 70 kg subject over 12 hours may be required for full inter-workout repletion.

Though a great deal of the endogenous carbohydrate was ingested during exercise, a mock Tour de France study by Brouns et al., (1989) required 17.5 (1.0) $g \cdot kg^{-1} \cdot d^{-1}$ to remain in positive carbohydrate balance. Consequently, it is suggested that the linear relationship between carbohydrate intake and training volume may not be limited by any set amount but by the maintenance of a stable balance of glycogen usage and carbohydrate need (i.e. as long as training volume increases, more carbohydrate is required).

Therefore, the strong correlation with intakes exceeding 10 $g \cdot kg^{-1} \cdot d^{-1}$ obtained for A.H. may have been attributed to his metabolic requirements. Incidentally, the two highest intake values for P.N. and A.H. fell to the left of their respective correlation lines indicating that the relationship at the top end of the intake range may have been levelling off. Further research is required to quantitatively determine the point at which increasing dietary carbohydrate intake no longer aids in muscle glycogen repletion.

Given that the carbohydrate in question has a high glycemic index, (maltodextrin has the same glycemic response as glucose according to Guezennec, Satabin, Duforez, Merino, Peronnet & Koziet, 1989) the correlations observed between intake and training performance are due to a combination of many factors but primarily due to the immediate availability after exercise (and therefore the time available to replete muscle glycogen) and the amount of dietary intake (Coyle, 1991; Keizer, Kuipers, van Kranenburg & Geurten, 1987).

There is evidence to suggest that changes in training volume were influenced by changes in total dietary carbohydrate intake due to post exercise supplementation. The literature recommends spacing post exercise supplementation of 1.0 to 2.0 $g \cdot kg^{-1}$ every 2 hours for up to six hours after exercise (Ivy et al., 1988a; 1988b; Blom et al., 1987a; 1987b; 1989; Reed et al., 1989; Keizer et al., 1987). Are there possible drawbacks associated with ingesting all 4 $g \cdot kg^{-1}$ immediately after exercise? For these three athletes the question was not important because the 4 $g \cdot kg^{-1}$ supplementation was only required to cover their dietary need for between 2 and 3 hours..

One of the more subsiduary findings of this study was the consistency in meal time after exercise. Timing of the first self selected dietary intake after exercise during supplemented and unsupplemented conditions were similar, suggesting that neither treatment nor placebo drinks decreased intake due to a satiation effect. The concern that the ingestion of a placebo would discourage food ingestion longer than during the baseline conditions due to a satiating effect was not realised. Athletes always ingested a late meal before retiring for the night and this meal occurred within 2 hours after exercising in 48 of 51 meals and always within 3 hours. Subjects characteristically utilized this time to clean up, ride home, unpack and prepare dinner.

Initial studies by Ivy et al., (1988a; 1988b), Blom and colleagues (1987a, 1987b; 1989), Reed et al., (1989) and Keizer et al., (1987) established the importance of ingesting carbohydrate immediately after exercise to maximise the glycogen repletion in the first 4 to 6 hours after exercise. They demonstrated that immediate ingestion of between 1 and 3 $g \cdot kg^{-1}$ resulted in muscle glycogen repletion rates of between 5 and 10 $\mu mol \cdot g^{-1}$ wet $wt^{-1} \cdot h^{-1}$ in the first 2 hours after ingestion compared to 4.1 $\mu mol \cdot g^{-1}$ wet $wt^{-1} \cdot h^{-1}$ when ingestion was delayed 2 hours. Based on these studies, the 4 $g \cdot kg^{-1}$ ingested immediately after exercise in this current study should have optimised muscle glycogen resynthesis in the first 2 hours after exercise.

The results of Ivy et al., (1988a) demonstrated that glycogen storage rates decreased approximate 44% in the second two hours after immediate ingestion of a 2 $g \cdot kg^{-1}$ bw solution. In a second study, involving similar depletion strategies and demonstrating similar storage rates for placebo, Ivy et al., (1988) demonstrated that when either 1.5 or 3.0 $g \cdot kg^{-1}$ bw carbohydrate were ingested every 2 hours muscle glycogen storage decreased approximately 24% in the second 2 hour segment. The net cost to glycogen storage of the single dose in the second 2 hours post exercise can be estimated to be the difference in storage rates between the two studies (or 20%). This may be partially due to the decreasing blood glucose and insulin levels in the second two hours of the unrepeated trial since both increase dramatically after the ingestion of the second load. The difference in glycogen repletion rates in the first and second two hour time periods may represent the cost incurred by an athlete who delays his first meal greater than 2 hours after the supplement.

Costill (1981) reported similar muscle glycogen synthesis over 24 hours between runners who ate 525g of carbohydrate either in two large meals or seven smaller meals (70% carbohydrate). These results suggested ingestion timing was not a limiting factor in full repletion within 24 hours after exercise. Differences in muscle glycogen levels were 125.6 +/- 10.9 and 101 +/- 20.9 mmol·kg-1 wet tissue, respectively. Coyle and Coyle (1993) have suggested that the reduced gastric emptying rate following a large meal may have resulted in extended glucose entry into the blood stream, similar to the effect of ingesting a series of small meals, as a reason for the lack of difference reported by Costill. It is possible the same result occurred with the single dose employed in this current study thereby extending the glycogenic drive provided by the digesting substrate into the second two hours after exercise. If so, then the cost to net glycogen repletion within 24 hours may be negligable.

Hunt, Smith, and Jiang (1985) reported mean emptying rates of 1.3 g·min^{-1}, 0.75 g·min^{-1} and 0.63 g·min^{-1} in the first 30, second 30 and subsequent 60 min after ingestion of 600 ml of a 25% maltodextrin solution. They demonstrated that increasing either fluid volume or energy density increased mean emptying rates and emptying time. Therefore, it is reasonable to suggest that the 1.09 to 1.31 litres of 25% maltodextrin used in the current study produced greater gastric delivery rates and emptying time than solutions of lower caloric density and volume reported in the literature for smaller volumes.

During supplemented conditions, athletes ingested all the carbohydrate at one time. The slower gastric emptying rate due to the high supplement concentration and high solution volume should not have limited glycogen storage (Reed, Brozinick, Lee, & Ivy, 1989). Instead it may have provided a repletion stimulus for as long as the insulin levels remained high and carbohydrates and sugars continued emptying from the athlete's gut. Calculating the actual amount ingested, and based on quoted gastric emptying rates (Foster, Costill & Fink, 1980; Hunt et al., 1985), total gastric emptying time can be estimated at between 3.0 and 6.0 hours. This provided the athletes an adequate window of time in which it was possible to warm-down, pack-up and return home without compromising glycogen storage between the cessation of exercise and eating dinner. This time acted as a repletion buffer between exercise termination and the first meal and may illustrate the most clinically significant result of post exercise supplementation on glycogen repletion. Subjects in this study consistently ate solid meals within three hours after exercise. Therefore, post exercise supplementation effectively functioned to bridge the gap between the training cessation and the first large meal after exercise.

Excluding the first 2 to 6 hours after exercise an athlete's eating pattern should effect the net amount of glycogen reabsorbed over the 22 hour period. Ivy et al., (1988a, 1988b) and Blom et al., (1987) suggest that maximum muscle glycogen repletion occurs if 1.5 - 2.0 g of carbohydrate are ingested every 2 hours after exercise. While timing may be critical during the first repletion phase (0 - 6 hours after exercise) it may not be true after 6 hours. Considering the outcome of Costill's work on meal frequency (1981) and all other work correlating ingestion volume with glycogen storage, it is suggested that while ingesting adequate carbohydrate volume immediately after exercise is important, total ingestion volume over the 24 hours after exercise may be a significant factor in maximizing glycogen repletion between workouts.

Recommending that athletes ingest a large quantity of liquid carbohydrate immediately after exercise has been done to ensure compliance and for ease of administration. To ensure a more uniform delivery from the gut over the 2 to 3 hours after exercise, fat and protein may be added in the drink or meal to retard gastric emptying rates and to increase storage rates. In an unsubstantiated report, Zawazki, Yaspelkis and Ivy (1992) have observed a 38% faster rate of storage with the addition of protein to a post exercise carbohydrate drink. The addition of protein increased plasma insulin and decreased plasma glucose compared with the carbohydrate treatment alone.

Although the supplement drink induced a satiation effect on the subjects, it effectively lasted no more than 2 hours. The drink did not inhibit the athletes from ingesting food within 3 hours after the study. Because *ad libitum* dietary carbohydrate intake during supplemented conditions was similar to intake during unsupplemented conditions, the supplement did not appear to interfere with dietary intake of other foods. Therefore concerns over its use as a meal replacement were not realised.

Recommendations from several studies include various repletion strategies that space ingestion of 3 - $6 g \cdot kg^{-1} \cdot d^{-1}$ evenly in amounts of 0.7 to 2.0 $g \cdot kg^{-1}$ for as long as 6 hours after exercise (Blom et al., 1987; Coyle & Coyle, 1993; Ivy & Katz, 1988). While these procedures approach the ideal of continuous intravenous glucose intake they may prove to be challenging, time consuming and impractical. It is suggested that expecting complete compliance among elite athletes, particularly post adolescent athletes, is unrealistic and difficult to moniter.

Meeting the dietary carbohydrate need of training can be difficult due to a variety of reasons. Research has demonstrated that when athletes are allowed to eat *ad libitum*, significantly lower glycogen synthesis rates occur due to lower carbohydrate intakes (Costill, 1981; Keizer, 1987; Piehl, 1975). In some cases, a normal carbohydrate rich diet alone cannot meet the energy and carbohydrate requirement of exercise (Brouns et al., 1989). Coyle (1991) suggested one of the reasons athletes do not consume enough carbohydrates is because they become satiated with the fat in their diet and go through periods of their day when recovery of glycogen stores is suboptimal. Apart from increasing total dietary carbohydrate intake, ingesting the drink immediately after training may increase inter workout glycogen repletion due to a variety of factors. It is unclear whether the benefits of ingesting the entire amount of supplement immediately instead of steadily over four hours, outweigh other factors. While the procedure is time saving, simple and compliance easily monitored, possible limitations include gastric retention, temporary satiation and impalitability.

Keizer et al., (1987) compared the differences between liquid or solid high carbohydrate meals and found similar glycogen resynthesis rates even though plasma glucose and insulin were significantly higher for the solid meal. Liquid meals were considered more advantageous as post-exercise supplements since they could function to also rehydrate the athlete and offered a more pure source of carbohydrate to maximise glucose delivery in the first hours after exercise. Solid meals may include fat constituents which may interfere with maximal carbohydrate absorption or decrease absorption time immediately after exercise.

Training at intensities of between 60 and 80% of $\dot{V}O_2$max at the durations used in this study has been demonstrated to decrease submaximal training heart rates (Fox, Bowers & Foss, 1989). One of the anticipated advantages of using training heart rate as an indicator of training intensity was to benefit from a resultant, indirect application of a consistent aerobic load based on increasing submaximal $\dot{V}O_2$. However, none of the six training summary graphs in the study displayed any increasing trends in training results. This is perhaps due to the high level of aerobic fitness of the subjects prior to the study. Changes in heart rate in response to exercise occurs within one week and are not as significant in individuals who are already aerobically fit (Winder et al., 1976). Neither training nor maturation effects were apparent in the results. The increasing performance plot in the case of J.T. was considered more a function of his improving health.

A decrease in performance was observed during the last cycle of training for all athletes. Fatigue or overtraining was not suspected since there were no changes in resting heart rates and results from two subjects, not included in this report, who trained under supplementation during the last phase increased their training volume. Both P.N. and A.H. ingested less carbohydrate during that phase than in any previous phase. This may have been an indication of decreasing motivation.

Ecological validity of the study was determined by considering the post study debriefing notes, post study performance results and subject feedback. Five of the six subjects involved in the study graduated their competitive status within two months after completion of the study and two obtained national ranking and professional sponsorship with their subsequent racing results. All subjects continued to use the product mixture until the end of that season (four months) and reported changes in their eating habits due to participation in the study.

The long term effects of supplementation on dietary carbohydrate intake should be investigated. Dietary carbohydrate intake may have diminished as the study progressed, although it was difficult to deduce a common trend over the six ingestion phases. The concern is that over the long term, athletes may become overly dependent upon a supplement drink to provide the adequate intake levels for maximum glycogen repletion. Doing so could diminish the dietary intake of the complementary nutrients, minerals and vitamins present in whole foods, necessary in a complete diet. This current study has demonstrated the supplement to be of value to augment and not replace dietary carbohydrate intake during periods of high volume, exhaustive training.

For the three athletes studied, there is weak evidence to support the claim that dietary intake 24 hours prior to each training session effected training performance in a subsequent session. There is, however, evidence to support the claim of an effect of mean dietary intake on total training volume in two and three subsequent sessions. The results suggest that consistent post exercise supplementation at a level of $4g{\cdot}kg^{-1}{\cdot}d^{-1}$ b.w. over 3 days of training increased the training volume over those three days, increased carbohydrate volume to levels of $10\ g{\cdot}kg^{-1}{\cdot}d^{-1}$, and increased relative carbohydrate intake an average of 13%. It is suggested that the supplement helped to maximise glycogen repletion during the first rapid synthesis phase occurring in the hours after exercise and before the first major meal. In addition the supplement served to increase total dietary carbohydrate intake, thereby maximising total muscle glycogen resynthesis in the period of time between training sessions. It is questionable whether the same results could have been obtained had the supplement amount been less. While a smaller dose (1 to 2 $g \cdot kg^{-1}$) of carbohydrate would be as effective in the span of time after exercise and before eating, it is doubtful if athletes would have met the ingestion criterion suggested for maximal inter-workout muscle glycogen repletion.

REFERENCES

Argentieri, M.P., Ennis, P. and Piper, L. (1988). Deflection velocities in heart rate and end-tidal oxygen graphs during incremental exercise on a windload trainer. In Burke, E.R. and Newson, M.M. (Eds.), Medical and Scientific aspects of cycling. Champaign, Ill., Human Kinetics, 101-107.

Ahlquist, L.E., Bassett, D.R., Sufit, R., Nagle, F.J., and Thomas, D.P. (1992). The effect of pedaling frequency on glycogen depletion rates in type I and type II quadriceps muscle fibers during submaximal cycling exercise. European Journal of Applied Physiology., 65, 360 - 364.

Astrand, P.O. (1981). Aerobic and anaerobic energy sources in exercise. In P.E. diPrampero and J.R. Poorman (Eds.), Medicine sport : Vol. 13. Physiological Chemistry of Exercise and Training, Karger, Basel, 22 -37.

Barr, S.I. and Costill, D.L. (1992). Effect of increased training volume on nutrient intake of male collegiate swimmers. International Journal of Sports Medicine, 13(1), 47 - 51.

Barr, S. I. (1989). Energy and nutrient Intakes of elite adolescent swimmers Journal of the Canadian Dietetic Association, 50(1), 20-24.

Bergstrom, J., Hermansen,L., Hultman, E., & Saltin, B. (1967). Diet, muscle glycogen and physical performance. Acta Physiologica Scandinavica, 71, 140-150.

Bergstrom, J., and Hultman, E. (1967). A study of the glycogen metabolism during exercise in man. Scandanavian Journal of Clinical Laboratory Investigations, 19, 218-228.

Bergstrom, J., Hultman, E., and Roch-Norland. (1972). Muscle glycogen synthase in normal subjects: Basal values, effect of glycogen depletion by exercise and of a carbohydrate rich diet following exercise. Scandanavian Journal of Clinical Laboratory Investigations, 29, 231-236.

Blom, P.C.S., Hostmark, A.T., Vaage, O., Kardel, K.R. and Maehlum, S. (1987). Effect of different post-exercise sugar diets on the rate of muscle glycogen synthesis. Medicine and Science in Sports and Exercise, 19, 491-496.

Blom, P., Vaage, O., Kardel, K. and Hermansen, L. (1982). Effect of different carbohydrates on rate of muscle glycogen resynthesis after prolonged exercise. Medicine and Science in Sports and Exercise, 14, 136 -144.

Borg, G.A.V. (1982). Psychophysical bases of perceived exertion. Medicine and Science in Sports and Exercise, 14(5), 377 - 381.

Bourey, R.E., Coggan, A.R., Kohrt, W.M., Kirwan, J.P., King, D.S. and Holloszy, J.O. (1990). Effect of exercise on glucose disposal: response to a maximal insulin stimulus. Journal of Applied Physiology, 69:1689 - 1694.

Brener, W., Hendrix, T.R., McHugh, P.R. (1983). Regulation of gastric emptying of glucose. Gastroenterology, 85, 76-82.

Brouns F., Saris, W.H.M., Beckers, E., Adlercreutz, H., van der Vusse, G.J., Keizer,H.A., Kuipers, H., Menheere, P., Wagemakers, A.J.M., and ten Hoor, F. (1989a). Metabolic changes induced by sustained exhaustive cycling and diet manipulation. International Journal of Sports Medicine, Vol. 10, supplemental 1, S49-S62.

Brouns, F., Saris, W.H.M., Stroecken, J., Beckers, E., Thijssen, R., Rehrer, N.J., and Hoorten, F. (1989b). Eating, drinking, and cycling, a controlled Tour de France simulation study. Part 1. International Journal of Sports Medicine, 10, S32-S40.

Brouns, F., Saris, W.H.M., Stroecken, J., Beckers, E., Thijssen, R., Rehrer, N.J., and Hoorten, F. (1989c). Eating, drinking, and cycling, a controlled Tour de France simulation study. Part 2. International Journal of Sports Medicine, 10, S41-S48.

Burke, L.M. and Read, R.S.D. (1988). A study of dietary paterns of elite Australian football players. Canadian Journal of Sport Sciences, 13(1), 15 - 19.

Burke, L.M. and Read, R.S.D. (1987). Diet patterns of elite Australian male triathletes. Physician and Sport Medicine, 15(2), 140 -142; 151 -155.

Clark,N., Nelson, M. and Evans, W. (1988). Nutrition education for elite female runners. Physician and Sport Medicine, 16(2), 124 -128;130;1133-134 ;136.

Coggan, A. R. and Coyle, E. F. (1989). Metabolism and performance following carbohydrate ingestion late in exercise. Medicine and Science in Sports and Exercise, 21(1), 59 - 65.

Conlee, R.K., Lawler, R. and Ross, P. (1983). Effect of fructose or glucose ingestion on glycogen repletion in muscle and liver after exercise of fasting. Medicine and Science in Sports and Exercise, 14, 137-144.

Costill, D.L., Bowers, R., Branam, G. and Sparks, K. (1971). Muscle glycogen utilization during prolonged exercise on successive days. Journal of Applied Physiology, 31, 834 - 838.

Costill, D.L., Draig, F., Fink,W.J. and Katz, A. (1983). Muscle and liver glycogen resynthesis following oral glucose and fructose feedings in rats. In H.G. Knuttgen, J.A. Vogel, and J. Poortmans (Eds), Biochemistry of Exercise: International Series on Sports Sciences, Vol. 3,). Chicago: Human Kinetics Publishers, Inc., 281 -285.

Costill, D.L., Flynn, M.G., Kirwan, J.P.,Houmard, J.A.,Mitchell, J.B., Thomas, R. and Park, S.H. (1988). Effects of repeated days of intensified training on muscle glycogen and swimming performance. Medicine and Science in Sports and Exercise, 20(3), 249 - 254.

Costill D.L. and Miller, J.M. (1980). Nutrition for endurance sport: carbohydrate and fluid balance. International Journal of Sports Medicine, 1, 2-14.

Costill, D.L. Pascoe, D.D., Fink, W.J., Robergs, R.A., Barr, S.I. et al. (1990). Impaired muscle glycogen resynthesis after eccentric exercise. Journal of Applied Physiology, 69, 46-50.

Costill, D.L. and Saltin, B. (1974). Factors limiting gastric emptying during rest and exercise. Journal of Applied Physiology, 37(5), 679-683.

Costill, D.L.,Sherman,W.M., Fink,W.J., Maresh,C., Witten,M. and 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. (1991). Timing and method of increased carbohydrate intake to cope with heavy training, competition and recovery. Journal of Sports Science, 9, 29-51, 51-52.

Coyle, E.F. and Coyle, E. (1993). Carbohydrates that Speed Recovery from Training The Physician and Sports Medicine, 21, 111-123.

Durnin. J.V. and Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thicknesses: measurements on 481 men and women aged from 16 - 72 years. British Journal of Nutrition, 32, 77 - 97.

Ekblom, B., and Hermansen, L. (1968). Cardiac output in athletes. Journal of Applied Physiology,. 25(5): 619-625.

Ekblom, B., Astrand, P., Saltin, B., Stenberg, J., and Wallstrom, B. (1968). Effect of training on circulatory response to exercise. Journal of Applied Physiology, 24(4), 518-528.

Faria, I., Dix, C. and Frazer, C. (1978). Effect of body position during cycling on heart rate, pulmonary ventilation, oxygen uptake and work output. Journal of Sports Medicine and Physical Fitness, 18,49 - 56.

Fell, R.K. Terblanche, S.E., Ivy, J.L., Young, J.C. and Hollooszy, J.O. (1982). Effect of muscle glycogen content on glucose uptake following exercise. Journal of Applied Physiology, 52, 434-437.

Foster, C., Costill, D.L. and Fink, W.J. (1979). Effects of preexercise feedings on endurance performance. Medicine and Science in Sports and Exercise, 11, 1-5.

Friedman, J.E., Neufer,P.D. and Dohm, G.L. (1991). Regulation of Glycogen Resysnthesis Following Exercise. Sports Medicine. 11 (4), 232-243.

Grandjean, A.C. (1986). Nutrition for swimmers. Clinics in Sport Medicine, 5, 65-76.

Guezennec, C.Y., Satabin, P., Duforez,F., Merino, D., Peronnet, F. and Koziet, J (1989). Oxidation of corn starch, glucose, and fructose ingested before exercise. Medicine and Science in Sports and Exercise, 21(1),45-50.

Hamilton, M.T., Gonzalez-Alonso, J., Montain, S.J. and Coyle, E.F. (1991). Fluid replacement and glucose infusion during exercise prevent cardiovascular drift. Journal of Applied Physiology 71(3): 871-877.

Heigenhauser, G. J. F., Sutton, J. R. and Jones, N.L. (1983). The effect of glycogen depletion on the ventilatory response to exercise. Journal of Applied Physiology, 54(2), 470 - 474.

Hermansen, L., Hultman, E. and Saltin, B. (1967). Muscle glycogen during prolonged severe exercise. Acta Physiology of Scandanivia, 71, 129-139.

Hermansen, L., and Odd, V. (1977). Lactate disappearance and glycogen synthesis in human muscle after maximal exercise. American Journal of Physiology, 233(5), E422-E429.

Hermansen, L., and Osnes, J. (1972). Blood and muscle pH after maximal exercise in man. Journal of Applied Physiology, 32, 304-308.

Hodges, R.E., Krehl, W.A. (1965). The role of carbohydrates in lipid metabolism. American Journal of Clinical Nutrition 17, 334-346.

Houston, M. E. (1980). Diet, training and sleep: A survey study of elite Canadian swimmers. Canadian Journal of Applied Sport Sciences, 5:3 161 -163.

Hultman, E. (1967). Physiological role of muscle glycogen in man, with special reference to exercise. Circulation Research, 20, 21, Supplement 1, I99-I114.

Hultman, E. (1967). Studies on muscle metabolism of glycogen and active phosphate in man with special reference to exercise and diet. Scandanavian Journal of Clinical Laboratory Investigations, supplement 94 (19), 1-64

Hultman, E., Bergstrom, J. and Roch-Norland, A.E. (1971). Glycogen storage in human skeletal muscle. In B. Pernow & B. Seltin (Eds.) Muscle Metabolism During Exercise, Plenum Press, New York 143 - 151.

Hunt, J.N., Smith, J.L., and Jiang, C.L. (1985). Effect of meal volume and energy density on the gastric emptying rate of carbohydrates. Gastroenterology, 89:1326-1330.

Ivy, J., Costill,W., Fink,W. and Lower,R.W. (1979). Influence of caffeine and carbohydrate feedings on endurance performance. Medicine and Science in Sports, 11, 6 - 11.

Ivy, J.L., Katz, A.L., Cutler, C.L., Sherman, W.M. and Coyle, E.F. (1988a). Muscle glycogen synthesis after exercise: effect of time of cargohydrate ingestion. Journal of Applied Physiology, 64(4), 1480 - 1485.

Ivy, J.L., Lee, M.C., Broznick, J.T. and Reed, M.J. (1988b). Muscle glycogen storage after different amounts of carbohydrate ingestion. Journal of Applied Physiology, 65, 2018-2023.

Jacobs, I. (1989). Effect of glucose polymer ingestion on glycogen depletion during a soccer match. Canadian Journal of Sport Sciences, 14:2, 112-116.

Jang, K.T., Flynn, M.G., Costill, D.L., Kirwan, J.P., Houmard, J.A. Mitchell, J. B. and D'Acquisto, L.J. (1987). Energy balance in competitive swimmers and runners. Journal of Swim Research, 3, 19.

Jansson, E. (1980). Diet and muscle metabolism in man. Acta Physiologica Scandinavica. Supplemental 487, 1-24.

Jansson, P. (1989). Training Lactate Pulse Rate. Oy Liitto, Oulu: Polar Electro Oy Finland.

Keizer, H.A. Kuipers, H., van Kranenburg G., Guerten P. (1987). Influence of lipid and solid meals on muscle glycogen resynthesis, plasma fuel hormone response, and maximal physical work capacity. International Journal of Sports Medicine 8:99-104.

Kiens, B., Raben, A.B., Valeus, A-K. and Richter, E.A. (1990). Benefit of dietary simple carbohydrates on the early poistexercise muscle glycogen repletion in male athletes. Medicine and Science in Sports and Exercise 22: S88, abstract.

Kochan, R.G., Lamb, D.R., Lutz, S.A., Perrill, C.V., Reimann, E.M. and Schlender, K.K. (1977). Glycogen synthase activation in human skeletal muscle: Effects of diet and exercise. American Journal of Physiology. 236:E660-E666.

Kuipers, H., Keizer, H.A., Brouns, F. and Saris, W.H. (1987). Carbohydrate feeding and glycogen synthesis during exercise in man. Pfluners Archives, 410 (60), 652 -6.

Kujala, U.M., Heinonen, O.J., Kuist, M., Karkkainen, O.P., Marniemi, J., Nittymaki, K. and Havar, E. (1989). Orienteering performance and ingestion of glucose and glucose polymers. British Journal of Sports Medicine, 23(2), 105 - 108.

Lohmann, K., Liebold, F. and Heilmann, W. (1978). Diminished insulin response in highly trained athletes. Metabolism, 27, 521 - 528.

MacDougall, J.D., Ward, G.R., Sale, D.G. and Sutton, J.R. (1977). Muscle glycogen repletion after high intensity intermittent exercise. Journal of Applied Physiology, 42,129 - 137.

Maehlum, S., Felig, P. and Wahren, J. (1978). Splanchnic glucose and muscle glycogen metabolism after glucose feeding during postexercise recovery. American Journal of Physiology: Endocrinology, 4(3), E255 - E260.

McCafferty, W.B., Gliner, J.A., Horvath, S.M. (1978). The stress of coaching. Physician and Sportsmedicine, 6(2): 66-71.

Millard - Stafford, M.L., Cureton, K.J., and Ray, C.A. (1988). Effect of glucose polymer diet supplement on responses to prolonged successive swimming, cycling and running. European Journal of Apllied Physiology, 58, 327 - 333.

Nilsson, L.H. and Hultman,E. (1973). Liver glycogen in man: the effect of total starvation or a carbohydrate - poor diet followed by carbohydrate refeeding. Scandanavian Journal of Clinical Laboratory Investigations, 33, 5-10.

Nieman, D.C. Butler, J.V., Pollet, L.M., Dietrich, S.J. and Lutz, R.D. (1989). Nutrient intake of marathon runners. Journal of the American Dietetics Association, (9), 1273-8.

Minami, H., McCallum, R.W. (1984). The physiology and pathophysiology of gastric emptying in humans. Gastroenterology 86:1592-1610.

Noakes, T.D. (1988). Carbohydrate ingestion and muscle glycogen depletion during marathon and ultramarathon racing. European Journal of Applied Physiology, 57 (4), 482- 489.

Noakes, T.D., Rehrer, N.J. and Maughan, R.J. (1991). The importance of volume in regulating gastric emptying. Medicine and Science in Sports and Exercise, 23(3), 307-313.

Montain, S.J., and E.F. Coyle (1992). The influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. Journal of Applied Physiology. 73:1340-1350.

Mourot, J., Thouvenot, P., Couet, C., Antoine, J.M., Krobicka, A. and Debry, G. (1988). Relationship between the rate of gastric emptying and glucose and insulin responses to starchy foods in young healthy adults. American Journal of Clinical Nutrition. 48(4), 1035-1040.

Noakes, T.D., Rehrer, N.J. and Maughan, R.J. (1991). The importance of volume in regulating gastric emptying. Medicine and Science in Sports and Exercise, 23, 307-313.

Noble, B.J., Borg, G.A.V., Jacobs, I., Ceci, R. and Kaiser, P. (1983). A category - ratio perceived exertion scale: relationship to blood and muscle lactates and heart rate. Medicine and Science in Sports and Exercise. 15(6), 523-528.

Parsonson, B.S. and Baer, D.M. (1978). The analysis and presentation of graphic data. In Kratochwill, T.L. (Ed.), *Single subject research: Strategies for evaluating c change*. New York: Academic Press, 1978.

Piel, K. (1973). Time course for refilling of glycogen stores in human muscle fibres following exercise-induced glycogen depletion. Acta Physiologica Scandinavica 90, 297-302.

Reed, M.J., Bozinick,Jr., J.T., Lee, M.C., Ivy, J.L. (1989). Muscle glycogen storage postexercise: effect of mode of carbohydrate administration. Journal of Applied Physiology, 66, 720-726.

Saltin, B. and Karlsson, J. (1971). Muscle glycogen utilization during work of different intensities. In Pernow, B., and Saltin, B. (eds.): *Muscle Metabolism during exercise*. New York, Plenum Press, 289-299.

Saltin, B. and Karlsson, J. (1971). Muscle glycogen utilization during work of different intensities. In: pernow, B. and Saltin, B. (eds), Muscle Metabolism during Exercise. New York, Plenum Press, 289-299.

Saris, W.J.M., van Erp-Vaart, J.A., Brouns, F., Westerterp, K.R., and ten Hoor, F. (1989). Study on food intake and energy expenditure during extreme sustained exercise: the Tour de France. International Journal of Sports Medicine 10:S26 - S31.

Sahlin, K., Edstrom, L., and Sjoholm, H. (1983). Fatigue and phosphocreatine depletion during carbon dioxide-induced acidosis in rat muscle. American Journal of Physiology, 245:C15-C20

Schwellnus, M.P., Gordon, N.F., van Zyl, G.G., Cilliers, J.F., Grobler, H.C., Kuyl, J., and Kohl, H.W. (1990). Effect of a high carbohydrate diet on core temperature during prolonged exercise. British Journal of Sports Medicine 24(2), 99 - 102.

Seiple R.S., Vivian, V.M., Fox, E.L. and Bartels, R.L. (1983). Gastric emptying characteristics of two glucose poylmer-electrolyte solutions. Medicine and Science in Sport and Exercise, 155, 366 -369.

Sherman, W.M. (1992). Recovery from endurance exercise. Medicine and Science in Sports and Exercise, 24, S336 - S339.

Sherman, W.M., Costill, D.L, Fink, W.J., Miller, J.M. (1981). Effect of exercise - diet manipulation on muscle glycogen and its subsequent utilization during performance. International Journal of Sports Medicine, 2, 114-118.

Sherman W.M. and Wimer, G.S. (1991). Insufficient dietary carbohydrate during training: does it impair athletic performance? International Journal of Sport Nutrition, 1, 28-44.

Short, S.H. and Short, W.R. (1983). Four - year study of university athletes' dietary intake. Journal of the American Dietatic Association, 82(6), 632 -645.

Simonsen, J.C., Sherman, W.M., Lamb, D.R., Dernbach, A.R., Doyle, A.J. and Strauss, R. (1991). Dietary carbohydrate, muscle glycogen and power output during rowing training. Journal of Applied Physiology, 70(4), 1500 - 1505

Sjogaard, G. Nielsen, B., Mikkelsen, F., Saltin, B. and Burke, E. (1986). Physiology in bicycling. Movement Publications, Ithica, N.Y.

Wasserman, K. (1986). The anaerobic threshold: definition, physiological significance and identification. Advances in Cardiology, 35, 1 - 23.

Wenger, H.A. & Bell, G.J. (1986). The interactions of intensity, frequency and duration of exercise training in altering cardiorespiratory fitness. Sports Medicine, 3, 346 - 356.

Wheeler, K. B. (1989). Sports nutrition for the primary care physician: The importance of carbohydrate. The Physician and Sports Medicine, 17(5), 106-117.

Williams, C. (1985). Nutritional aspects of exercise - induced fatigue. Proceedings from the Nutrition Society 44, 245 - 256.

Winder, W.W., Hagberg, J.M., Hickson, R.C., Ehsani, A.A. and McLane, J.A. (1978). Time course of sympathoadrenal adaptation to endurance exercise training in man. Journal of Applied Physiology, 45(3), 370-374.

Wolf, M.M. (1978). Social validity: The case for subjective measurement or how applied behaviour analysis is finding its heart. Journal of Applied Behaviour Analysis, 11, 203 -214.

Wright, D.A., Sherman, W. M. and Dernbach, A.R. (1991). Carbohydrate feedings before, during or in combination improve cycling endurance performance. Journal of Applied Physiology, 71(3), 1082 - 1088.

Young, K. and Davies, C.T.M. (1984). Effect of diet on human muscle weakness following prolonged exercise. European Journal of Applied Physiology, 53, 81-85.

Zawadzki, K.M., V.V. Yaspelkis, and Ivy J.L. (1992). Carbohydrate - protein complex increases the rate of muscle glycogen storage after exercise. Journal of Applied Physiology, 72,1854 - 1859.

APPENDIX A

LITERATURE REVIEW

The purpose of this study was to determine the ecological validity of using a carbohydrate supplement drink to increase training volume by meeting the dietary requirements necessary to increase inter workout glycogen storage on a daily basis. It also demonstrated the use of single subject methodology as an appropriate experimental tool to validate results obtained from between group research to the individual case. This chapter summarises the effect of exercise intensities and durations on glycogen depletion, the determinants of muscle glycogen repletion from metabolic and dietary perspectives and then reviews the effect of dietary carbohydrate intake on exercise performance. Evidence will be presented to support the use of heart rate as an indicator of training intensity in the dependent variable, followed by a brief review of single - subject methodology.

The relationship between intramuscular glycogen stores and endurance during prolonged sub maximal exercise is well established. Glycogen depletion rates are primarily determined by exercise intensity (Saltin & Karlsson, 1971; Foster, Costill, & Fink, 1979). Submaximal exercise is limited by intramuscular glycogen and time to exhaustion decreases linearly over time with pre exercise muscle glycogen (Bergstrom, Hermansen, Hultman, & Saltin, 1967). Bergstrom et al. investigated the effect that manipulation of pre-exercise glycogen levels had on aerobic work capacity. Subjects with initial muscle glycogen levels of 35, 100, and 200 $\mathrm{mmol \cdot kg^{-1}}$ exercised until exhaustion at 70% of $\dot{V}O_2$ max for 60, 115 and 170 min, respectively. The higher glycogen levels did not increase initial exercise speed but allowed athletes to maintain race pace longer. They hypothesised that as glycogen depleted fibres failed to contract, total force output decreased. Therefore, a direct correlation of muscle glycogen stores with the capacity to sustain prolonged exercise was established.

At low intensities of work both carbohydrates and fats are used as energy substrates (Hultman, Bergstrom & Roch-Morland,1971). As training intensity increases, muscle cells become more dependent upon glycogen. Hermansen Hermansen, Hultman, and Saltin (1967) demonstrated that two hours of cycling at 30% $\dot{V}O_2$ max reduced muscle glycogen by 20% whereas performing at 75% $\dot{V}O_2$ max for two hours resulted in almost total depletion. Exercise at between 70 - 80% of $\dot{V}O_2$ max could continue until muscle glycogen levels approached zero whereas at lower intensities a much slower rate of glycogen utilisation occurred. This was because at work rates lower than 70% of maximal oxygen uptake fatty acid utilisation augmented glycogen usage to a greater degree. At intensities lower than 60% of $\dot{V}O_2$ max, exercise continued for hours without significant muscular depletion due to a much greater reliance on fatty acids metabolism (Saltin et al., 1971). In general, at exercise intensities of 65 - 85 % of $\dot{V}O_2$ max , muscle glycogen decreases exponentially over time with exhaustion correlating with muscle glycogen depletion. Exercise of approximately 60 - 80% of $\dot{V}O_2$ max can be continued for 2 - 3 hours before exhaustion.

Above 90 % of maximum aerobic output glycogen is the predominant catabolic substrate but lactate and subsequent proton generation impede sustained work at these intensities (Saltin & Karlsson, 1971). Exhaustion occurs before nearing full tissue glycogen depletion. Since the limiting factors are residuals of anaerobic exercise the highest depletion rate attainable for sustained submaximal work occurs under the anaerobic threshold.

When depletion occurs as a result of repeated bouts of work above the level of maximum oxygen uptake, replacement of the catabolized glycogen can occur from lactate and glycogen precursors (Hermansen & Vaage, 1977). However, after prolonged exercise these metabolises are not available so muscle glycogen repletion depends exclusively on blood glucose. During periods of fasting, or exercise of long duration, liver glycogen stores are mobilised to maintain blood glucose. During and immediately post-exercise, net glucose uptake increases 10 -20 fold above the resting value to meet 75 -90% of the carbohydrate metabolism of muscle (Wahren, Felig, Ahlborg, & Jorfeldt, 1971). During exercise this increased intercellular blood glucose transport is met by a concomitant increase in hepatic glucose output. Hepatic gluconeogenesis increases in an attempt to replace the diminishing supply of liver glycogen but cannot meet the immediate demand through gluconeogenic pathways (from lactate, pyruvate, glycerol and gluconeogenic amino acids). After 40 - 60 min of exercise, peripheral utilisation of blood glucose begins to reduce hepatic glycogen stores (Wahren, 1966). Blood glucose levels are maintained at a constant level during continuous muscle and hepatic glycogen depletion through a combination of decreased insulin secretion and an increase in both insulin and glucose sensitivity by the muscle cell. Blood glucose levels do not drop until hepatic levels near a depleted state.

Unlike some animals, gluconeogenesis from lipid and protein stores does not offer a significant amount of glucose in humans. Glycogen storage is limited by dietary carbohydrate intake so athletes depend on a daily dietary consumption of carbohydrates to provide the necessary substrate. Studies have shown that full inter-workout glycogen repletion can occur in as little as 24 hours provided sufficient dietary carbohydrate is consumed (Bergstrom et al., 1967; MacDougall, Ward, Sale, & Sutton, 1977) . Costill, Sherman, Fink, Maresh, Witten, and Miller, (1981) had trained male runners perform a 16.1 km run at 80% to exhaustion then fed them increasing amounts of carbohydrate (188 - 648 g). Increasing amounts of dietary carbohydrate correlated the amount of glycogen stored within 24 hours after depletive training ($r = 0.84$, $p < 0.05$) and displayed the direct relationship between dietary intake volume with muscle glycogen storage.

The quality of the ingested fuel from simple sugars to glycogen may also affect the speed of transformation . Over an eight hour period both glucose and sucrose transform almost 1.5 - 2.0 times faster than fructose (Bloom, Vaage, Kardel, & Hermansen, 1982). Fructose must be metabolised in the liver in order to produce glucose whereas when sucrose is ingested it is hydrolysed to equal amounts of glucose and fructose. Costill and

Miller (1980) found that while there was no significant difference between glycogen resynthesis through simple or complex carbohydrate ingestion over 24 hours, after 48 hours the complex carbohydrate diet produced a significantly higher level of glycogen than the simple sugar. Although it was suggested that the difference in glycogen storage was due to the influence starches have in maintaining plasma insulin levels longer than glucose (Hodges & Krehl, 1965) another study indicated that there was no difference in glycogen synthesis between a simple or complex carbohydrate diet 20, 32 or 44 hr after exhaustive exercise (Kiens, Raben, Valeus & Richter, 1990).

As well as requiring a diet high in carbohydrate, athletes must also maintain an adequate intake of minerals, vitamins, proteins and fats. If athletes are chronically hypocaloric they can compromise glycogen synthesis by depriving the body of adequate anabolic substrates, cofactors and salts (Wheeler, 1989). Dietary surveys of elite athletes show a prevalence of inadequate dietary carbohydrate, mineral and caloric intakes (Sherman & Wimer, 1991; Short S.H. & Short, 1983; Clark, Nelson & Evans, 1988; Burke & Read, 1988). The highest observed relative mean carbohydrate intake found in the literature was 59.5% (2435 kcal$\cdot$ d^{-1}) in Australian elite male triathletes (Burke & Read). Grandjean (1986) reported that the diet of both male and female endurance athletes was characteristically comprised of 49 % carbohydrate.

Instances of inadequate dietary intake for athletes are frequently reported. Clark, Nelson, and Evans (1988) discovered that a significant proportion of elite female runners believed they ate wisely but actually had sub-optimal intakes. Costill (1988) described four swimmers who failed to increase dietary intake in response to an increase in their training volume as "insensitive to changes in their daily energy expenditure and having difficulty in maintaining a caloric and carbohydrate balance."

Full repletion between workouts is dependent upon the time available, the type of carbohydrate, glycogen resynthesis rate determinants (such as adequate substrate availability), insulin response characteristics, blood flow, arteriovenous glucose difference, and aerobic fitness (Lohmann,Liebold, and Heilmann, 1978). Relative strength of the repletive stimulus is also influenced by the extent of depletion (Costill et al., 1988). The activity of glycogen synthase I, the rate limiting enzyme in the glycogen resynthesis pathways, is inversely related with glycogen content with reported correlations as high as -0.88 (Costill). The highest enzyme activities (0.4 - 0.5 mmol glycosyl units $\cdot$ kg $^{-1}$ $\cdot$ min^{-1}) occurs immediately post exercise in glycogen depleted muscle and decreases exponentially after that.

The post workout physiological environment is also suited for glycogen repletion because the post depletive muscle cell exists in an optimal state for glucose absorption and glycogen synthesis. Blood pressure and heart rate remain high and muscle cells display an increased affinity for blood borne glucose due to increased membrane sensitivity and local dilatory effect around active muscle (Ivy, Katz, Cutler, Sherman & Coyle, 1988; Piehl, 1974). Insulin response characteristics and arteriovenous glucose also optimise cellular glucose absorption in the post depletive state (Lohmann et al., 1978). Splanchnic glucose output (SGO) increases and remains elevated to two times resting levels in the recovery period after exercise (Maehlum, Felig, & Wahren, 1978). Since the body regains homeostasis relatively soon after the cessation of exercise, carbohydrate ingestion has been recommended immediately after a glycogen depletive workout for optimal gluconeogenesis (Friedman, Neufer, & Dohm, 1991).

Studies by Ivy et al., (1988a; 1988b), Blom et al., (1987a, 1987b; 1989), Reed et al., (1989) and Keizer et al., (1987) established the importance of ingesting carbohydrate immediately after exercise to maximise the glycogen repletion in the first 4 to 6 hours after exercise. Ivy and coworkers found that the rate of glycogen storage was significantly higher (300 % increase above basal levels during the first two hours) when a carbohydrate supplement (2 g$\cdot$ kg $^{-1}$ of body weight in a 25 % solution) was administered immediately after exercise. Biopsy samples taken at the vastus lateralis of trained cyclists showed that delaying carbohydrate ingestion by two hours led to a 45% slower ($p < 0.05$) activity compared with immediate ingestion. The rate increase occurred despite elevated plasma glucose and insulin levels and comparable glycogen synthase activity. It was suggested that the difference in rate between the two treatments was due to the increased insulin action, increased blood flow, greater activation of muscle glucose transport, and a greater arteriovenous glucose difference, occurring immediately after exercise. Delaying the ingestion of the carbohydrate by two hours actually resulted in a rate of glycogen synthesis 47 % slower than immediate ingestion. Immediate ingestion resulted in an average muscle glycogen repletion rate of 7.7 μmol·g wet wt^{-1}·h^{-1} in the first 2 hours after ingestion compared to 4.1 μmol·g wet wt-1·h^{-1} when ingestion was delayed 2 hours. After 4 hours, significantly more muscle glycogen was stored in the immediate treatment compared to the delayed treatment. They suggested that the reduced rate of muscle glycogen storage was due to a lower rate of blood glucose uptake.

To provide for maximum glycogen storage a number of studies have investigated ingesting various amounts of carbohydrate every 2 hours after exercise. Blom et al. (1987) fed subjects 0.35, 0.7 or 1.4 g· kg $^{-1}$ immediately after depletive exercise and at two hour intervals after for six hours. They found no significant difference in storage rates at ingestion amounts of 0.7 and 1.4 g· kg $^{-1}$ averaging 5.2 mmol·kg^{-1}·h^{-1}. However, Ivy et al. (1988b) observed higher glycogen synthesis rates (5.2 compared to 4.6 and 0.5 mmol·kg^{-1}·h^{-1}) when 3.0 rather than 1.5 g· kg $^{-1}$ or placebo was ingested immediately after and at 2 hour intervals for 4 hours. Blom (1989) fed or infused subjects with approximately 3.0 g· kg $^{-1}$ carbohydrate evenly within 3 hours of exercise and found similar glycogen storage rates of 8.8 mmol·kg^{-1}·h^{-1}.

These studies suggest that when carbohydrate beverages are consumed at a rate of 0.7 - 3.0 g· kg $^{-1}$ immediately and every two hours for the next 3 - 6 hours after exercise mean muscle glycogen storage will be between 5 and 10 mmol·kg^{-1}·h^{-1}. Higher repletion rates have been reported (12 - 25 mmol·kg^{-1}·h^{-1}) through glucose infusion and hyperinsulinemic clamp procedures (Bergstrom & Hultman, 1972; Bourey, Coggan, Kohrt, Kirwan, King, & Holloszy, 1990) but as such forms of methodology are considered neither ethical nor practical.

A few authors have suggested that two synthesis phases exist and that the rapid resynthesis observed in the first 4 to 6 hours is primarily provoked by low muscle glycogen levels (Blom et al., 1987; Fell, Terblanche, Ivy, Young, & Hollooszy, 1982; Keizer, Kuipers, van Kranenburg & Guerten, 1987). After that, synthesis is controlled by the insulin-induced activation of glycogen synthase and only capable of moderate storage rates (approximately 3 mmol·kg^{-1}·h^{-1}). Minimal levels of insulin and blood glucose are necessary within the first phase to develop the appropriate cellular conditions and offer enough substrate for glycogen repletion (Ivy et al., 1988a). Blom et al., suggest that insulin activation of glycogen synthesis occurs at a threshold of 21μU·ml^{-1}. Ivy et al., (1988a) found that blood insulin decreased to 20 - 25μU·ml^{-1} and blood glucose dropped to near pre-exercise levels 2 hours after immediate ingestion. This would explain the decrease in the glycogen storage rate from 7.7 to 4.3 μmol·g wet wt^{-1}·h^{-1}.

Since intravenous glucose infusion alone has not demonstrated any greater synthesis rates (Reed, Bozinick, Lee, & Ivy, 1989) glucose entry into the blood does not appear to be a limiting factor. The degree to which blood glucose, insulin and other factors such as blood FFA.'s and blood flow effect muscle glycogen storage in the first hours after exercise is unclear.

Excluding the first 2 to 6 hours after exercise, an athlete's eating pattern may effect the net amount of glycogen reabsorbed over a 22 hour period. Ivy et al., (1988a; 1988b) and Blom et al., (1987) suggest that maximum muscle glycogen repletion occurs if 1.5 -

2.0 g of carbohydrate are ingested every 2 hours after exercise. While consistent delivery of carbohydrate may be important in the first repletion phase (0 - 6 hours after exercise) it may not be as important after 6 hours.

Costill et al., (1981) reported similar muscle glycogen synthesis over 24 hours between runners who ate 525g of carbohydrate either in two large meals or seven smaller meals (70% carbohydrate). The differences in muscle glycogen levels were determined to be insignificant at 125.6 (10.9) and 101 (20.9) mmol·kg^{-1} wet tissue, respectively. Coyle and Coyle (1993) have suggested that the reduced gastric emptying rate following a large meal may result in extended glucose entry into the blood stream similar to the effect of ingesting a series of small meals, as a reason for the lack of difference reported by Costill. It is suggested that while it is important to ingest adequate carbohydrate volume immediately after exercise, total ingestion volume, irrespective of ingestion frequency, may play a significant role in maximising glycogen repletion between workouts.

Costill et al., (1981) investigated the effect of dietary carbohydrate volume on muscle glycogen storage by progressively increasing dietary carbohydrate intake. They determined that consumption of 525 to 648 g (for a 72 kg man) carbohydrate during the 24 hours after strenuous running resulted in normal muscle glycogen levels. Because no plotting of intake volume occurred, they suggested increasing dietary carbohydrate levels past 648 g·d^{-1} might maximise muscle glycogen repletion past normal levels.

The relationship between muscle glycogen storage and dietary carbohydrate intake volume becomes even more pronounced with successive days of training. Costill, Bowers, Branam and Sparks (1971) demonstrated that daily bouts of exhaustive exercise produced a progressive decrease in pre exercise muscle glycogen levels as much as 50%. Costill's (1980) research on the effect increased training has on the self-selected dietary intake of swimmers reported that over ten days 8 out of 12 athletes automatically increased carbohydrate intake (8.2 g·kg^{-1}·d^{-1}) to meet the increased caloric demands of training while four did not (5.3 $g \cdot kg^{-1} \cdot d^{-1}$). Over the ten days of the study the first group experienced a 15% decrease in pre-exercise muscle glycogen content whereas the other group dropped 20%. Simonsen, Sherman, Lamb, Cernbach, Doyle and Strauss (1990) compared changes in power output for three 2,500m time trials of rowers training 4 weeks under diets of either 5 or 10 $g \cdot kg^{-1} \cdot d^{-1}$. After training, pre-exercise muscle glycogen increased from 94 to 155 $mmol \cdot kg^{-1}$ with the high carbohydrate diet yet the moderate carbohydrate diet only maintained muscle glycogen at 119 $mmol \cdot kg^{-1}$. Keizer (1987) and Sherman, Doyle and Lamb (1991) also support the contention that ingestion of 5 to 8 $g \cdot kg^{-1} \cdot d^{-1}$ carbohydrate over the long term will either maintain or allow pre-exercise muscle to gradually decline whereas ingesting 10 $g \cdot kg^{-1} \cdot d^{-1}$ or more may increase pre-exercise muscle glycogen levels. Recommendations for maximal muscle glycogen storage reported by Blom (1980) suggest ingesting between 1.4 and 2.0 g $glucose \cdot kg^{-1}$ bw which would equate to 588 - 840 g carbohydrate per day (or 8.4 - 12.0 $g \cdot kg^{-1} \cdot d^{--1}$) for a 70 kg subject over 12 hours. The relationship between dietary CHO intake and maximal glycogen storage has been shown to exceed 10 $g \cdot kg^{-1} \cdot d^{-1}$ and be as high as 16 $g \cdot kg^{-1} \cdot d^{-1}$(Brouns, Saris, Stroecken, Beckers, Thijssen, Rehrer, and Hoorten, 1989c). Recommendations for maximum inter workout repletion range from 8.4 to 12.0 $g \cdot kg^{-1} \cdot d^{-1}$ reflecting the metabolic needs of the particular athletes.

The effect of diet on performance is often difficult to measure. However, in their research on swimmers (Costill et al., 1980) reported that the 4 out of 12 that didn't increase their dietary carbohydrate intake were unable to tolerate the heavier demands of their training program. Simonsen, Sherman, Lamb, Dernbach, Doyle, and Strauss (1991) also noticed that after training, power output increased 10.7% when following a high carbohydrate diet (10 $g \cdot kg^{-1} \cdot d^{-1}$) but only 1.6% with the moderate diet (5 $g \cdot kg^{-1} \cdot d^{-1}$).

If athletes do not consume the daily required dietary volume of carbohydrate, a deficit begins to occur. Performance and training begin to plateau and the symptoms of fatigue (such as weight loss, loss of energy and predisposition to illness) can develop (Williams, 1985). Chronic glycogen depletion can also interfere with anaerobic work as creatine phosphate and adenosine triphosphate regeneration is partially dependent upon adequate glycogen stores (Costill, 1988).

Acute fatigue during prolonged exercise is most directly effected by decreases in blood glucose. The hypoglycemic condition develops as hepatic and muscle glycogen levels become depleted. This is commonly experienced by marathon runners when they "hit the wall". Experiments using percutaneous needle biopsy techniques to obtain samples of human muscle support this observation (Bergstrom & Hultman, 1966; Gollnick, 1969). Glycogen depletion has also been correlated to decrements in skill and coordination highlighting the dependence of the central nervous system upon an adequate blood glucose supply (Kujala, Heinonen, Kuist, Karkkainen, Marniemi, Nittymaki & Havar, 1989).

If sufficient carbohydrate is not ingested between daily workouts consistent daily declines in glycogen stores and subsequent decrements in training can develop (Costill et al.,1988; Costill, Bowers, Branam & Sparks,1971). Sufficient levels of carbohydrate repletion has been expressed in terms of absolute mass, relative caloric value and as absolute mass per body weight.

Keizer, Kuipers, van Kranenburg and Geurten (1987) compared the effects of ingesting either a liquid and or solid supplement (192 - 422 g based on body weight and energy expenditure during exercise) after exercise. They noted that while athletes are not often hungry immediately after exercise liquid food was tolerated better than solid food in the first 1 to 2 hours. Liquid food did not cause any of the feelings of a distended stomach and slight nausea that accompanied solid food. Coyle (1991) has recommended athletes avoid eating meals with a high protein and fat content, especially during the first 6 hours after exercise because this often suppresses hunger and limits carbohydrate intake. However, when eating the last meal before an extended period without food he suggests that a limited amount of fat and protein be added to the meal to develop a more sustained gastric emptying rate.

Adequate substrate availability is influenced by a number of gastric emptying variables including the osmolality, volume, caloric density, electrolyte content, temperature and pH of ingested solutions, the level of hydration and individual variability (Noakes, Rehrer, & Maughan, 1991). Gastric emptying rates for fluid decreases logarithmically with time, and the emptying rates change with increasing volume and energy density (Blom, Hostjmar, Odd Vaage Kristin & Maehlum, 1987; Hunt, Smith, & Jiang, 1985). Hunt et al. reported mean emptying rates of 1.3 $g \cdot min^{-1}$, 0.75 $g \cdot min^{-1}$ and 0.63 $g \cdot min^{-1}$ in the first 30, second 30 and subsequent 60 min after ingestion of 600 ml of a 25% maltodextrin solution. They demonstrated that increasing either fluid volume or energy density increased mean emptying rates and emptying time.

Many factors such as emotions, diet, muscle temperature, environmental temperature, air humidity and sickness can influence heart rate (HR) (Bergstrom et al., 1967; Janssen, 1989; McCafferty, 1978,). Bergstrom et al. determined a correlation of 0.91 between pulse rate and glycogen utilisation. They concluded that the rate of glycogen depletion is related to the relative work load, rather than to the absolute, therefore, exercise HR was a good measure of the relative work load. A more recent study by Hamilton, Gonzalez-Alonso, Montain and Coyle (1991) supported Hultman's work in an investigation of the decrease in training heart rate that follows decreasing hydration levels in what is known as the "cardiovascular drift". Hamilton et al. showed that ingesting enough water to maintain training body weight minimised the drift to a 5 - 7% increase within a 120 min exercise bout at 70% of $\dot{V}O_2max$. Without active fluid replacement, HR increased another 10% due to decreasing stroke volume. The 5 - 7% increase in HR and cardiac output (CO) during the fluid replacement trials was in response to increased whole body oxidative metabolism in tissues that react directly or indirectly to blood glucose concentration because the increase in oxidative metabolism was suppressed by hyperglycemia. With enough fluid intake to maintain hydration, HR was shown to increase concomitantly with VO_2 due to increased caloric expenditure. These studies indicate that heart rate is a good indicator of training intensity and therefore glycogen depletion rates.

Various research (Heigenhauser, Sutton & Jones, 1983; Sahlin & Katz, 1990 and Schwellnus, Gordon, van Zyl, Cilliers, Grobler, Kuyl, & Kohl, 1990) determined that training heart rate increases with decreasing muscle glycogen content Heigenhauser et al. depleted muscle glycogen from 59.1 to 17.1 $\mu mol \cdot g^{-1}$ and showed that at any given power output, depleted state O_2 intake, heart rate, blood pH and ventilation were significantly higher than the repleted condition. Skill and coordination also appear to be negatively influenced by muscle glycogen depletion (Kujala et al., 1989). Therefore, if heart rate is maintained, decreasing muscle glycogen levels should decrease power output. At a lower power output less work will be achieved than with higher muscle glycogen levels in an equal amount of time.

The foundation of coaching technology depends on both single-subject and case models. Even when working in a group setting, such as a team swim workout or football scrimmage, the coach is concerned with the training impact on the individual athlete. Single subject studies are most concerned with the practical or ecological benefit of the observed results. Interventions can be evaluated under the circumstances in which they usually occur rather than in academic or laboratory settings. Conclusions obtained primarily through visual or subjective analysis may be of more benefit to the individual athlete or sport specific foci. The dependence on gross change, reproducibility, reversal and stability of results acts as a filter or qualifier to determine validity of the effect.

While addressing the same topic, single subject and between-group research designs take different methodological approaches. Any discussion about the strengths and weaknesses inherent within their structures must be done with respect to an experimental context. Each design is better suited to answer a different question. This review argues the validity of utilising single-subject design when determining the clinical significance of data determined from between-group research. This is done with a brief survey of literature supporting the use of single-subject design in studies of clinical and behavioural psychology, an argument to support using similar techniques in a study of applied exercise physiology, an explanation of basic single-subject designs, and a description of the methods used to evaluate the data obtained in single-subject experiments.

With the advent of statistical analysis techniques by Gosset (Studentized *t* test in 1908), R. A. Fisher (introduced key arguments supporting the analysis of variance), and others, psychological journal publications began to place a greater emphasis on results obtained in large sample studies utilising statistical analyses. In this model the effect of an intervention was determined by statistical significance. Until then, published investigations into human behaviour would draw general conclusions from a collection of research in samples of five or less subjects (Robinson & Foster, 1979) and focus on the clinical significance of the intervention.

Information obtained from group research, while important, represents a conglomerate result that reflects the effects for most subjects who are representative of a specific population. Conclusions derived from group research may not apply to an individual because of unique differences in their (physiology, psychology, technique, and genetic endowment). In addition, results can be affected by the influences of group dynamics present or absent in the study or the actual environment. An in depth study of the individual response to an intervention is not possible within the parameters of a between-group study. Single-subject designs permit the in-depth study of the individual response to an intervention and may provide more insight into the clinical significance of the intervention.

Research on exercise physiology has relied on the methodology of between group designs. Such a methodological approach may not be applicable in a practical setting. In comparison, other disciplines, such as psychology, psychiatry, education, rehabilitation, social work and counselling have employed single-subject methodology. The individual nature of the research questions in these disciplines is often best suited to single-subject designs.

"Single subject design" has been most prevalent in the field of clinical psychology and in the development of psychotherapy techniques (Kazdin,1982). Compilation of comprehensive case studies form the basis for establishing new clinical methods. Single subject designs have been instrumental in discovering, documenting and determining treatments for relatively rare clinical disorders.

Kazdin (1982) presents many of the problems facing clinical psychology regarding the development and application of experimental data. Researchers are very rarely involved as practitioners and vice-versa. Therefore they have little appreciation for the issues pertinent in the field of practice. The carefully controlled experimental environment does not include the social variables normally present for the subject. The personal characteristics of the patient, their education and background, may not be representative of the college students typically used in experimental research and, therefore, the results in the studies may not be relevant to an individual case. In group designs the administrators may be preselected on the basis of availability and knowledge and thus may not reflect the ordinary range of capabilities in practical situations. Experimental results may not address the questions and concerns that arise in a clinical setting. Kazdin (1982, p.14) compares the validity of single subject versus between-group design models clearly by stating:

> The clinician is not concerned with presenting a standardised technique but with providing a treatment that is individualised to meet the patient's needs in an optimal fashion. The results of research that focuses on *statistically significant* changes may not be important if the clinician is interested in producing a *clinically significant* effect, i.e., a change that is clearly evident in the patient's everyday life. The results of the average amount of change that serves as the basis for drawing conclusions in

> between-group research does not address the clinician's need to make decisions about treatments that will alter the individual client.

He further suggests that many experiments conducted in groups are designed to meet criteria for statistical evaluation and may in fact distort the more germane relationship - the effects of treatments on individuals. Uncontrolled case studies can only produce anecdotal and not causal evidence. However, carefully controlled and designed single subject studies producing a continuous series of discrete data points can be used to determine cause and effect relationships for an individual.

Between-group designs are often selected because of the inherent advantages of statistical evaluation and the controlled environment. Between-group designs operate effectively to answer a broad range of research questions and are accepted methods to make general observations and statements about a general population from the data obtained from a population sample. However, the process of evaluating treatment and intervention in an individual case is arguably more suited to single-subject methodology. Conditions of homogeneous groups, random assignment of subjects to groups, standardisation of treatments among subjects, and large enough sample sizes may not be possible when the focus of the study is on individual responses in a practical setting. Single subject design can more specifically examine the individual effect of the intervention without losing results or data to result groupings.

Compared to descriptive, case-studies, the focus of single-subject methodology is to provide a systematic, rigorous means of evaluating the effects of an intervention in an individual case. Single-subject design establishes causality by demonstrating consistency of response to the alternating application and withdrawal of the intervention in varying environments over many cycles.

While there is little literature to support the use of single-subject design methodology in the clinical sport science environment, much of the same concerns discussed in the field of psychology can be applied to coaches and athletes in application of information derived through between-group research.

As opposed to uncontrolled case studies, contemporary single-subject methodology follows stringent rules for application. Developed from psychological research into the experimental analysis of behaviour and from applied behaviour analysis, these guidelines serve to provide a more direct causal link between intervention and result. What follows is a brief survey of the general requirements of single-subject designs and are taken primarily from Kazdin (1982).

Continuous assessment of performance over time is the principal requirement of single-case studies. Observations occur as often as practical to determine both pattern and stability of performance under any condition. Contrasted against between group research where one observation is made amongst a large group, many observations over time provides a valid indication of performance in single-case studies.

Prior to introducing the intervention, information about the subjects condition is typically collected during the baseline phase of the study. Baseline assessment serves both descriptive and predictive functions. The descriptive function provides information about the subject and the level of performance whereas the predictive function suggests the future level of performance if the intervention is not provided and conditions continue as they exist. The quality of performance recorded during the baseline phase serves as a gauge to determine changes in performance with time. Observations are made until predictions can be made within a reasonable level of certainty. This is typically established by demonstrating consistency in trend (or slope) and variability in data.

Stable baseline trends and trends going in the opposite direction to the expected treatment pose no problem for analysis. However, baseline trends moving in the same direction as the predicted treatment trend must be of considerably less magnitude to detect a change in performance due to change in trend upon intervention.

Variability of data refers to the fluctuation of data over time under a specific condition. As a general rule, the greater the variability in data the more difficult it is to draw conclusions with intervention. Change due to intervention becomes undefinable when the boundaries of variability exceed or overlap the magnitude of performance due to the intervention. Acceptable options for modifying excessive performance variability include the blocking of consecutive data and the plotting of blocked averages.

The most basic research design involving single subjects is termed the ABAB design and refers to performance data obtained over time through a series of alternating conditions. Typically performance is continuously recorded during an initial baseline period, during treatment and during subsequent baseline and treatment sessions. A causal relationship is suggested if performance changes with intervention, reverts to baseline levels when the treatment is withdrawn and demonstrates another reversal when the treatment is reapplied.

While the first and subsequent intervention and subsequent baseline conditions perform both predictive and descriptive functions they also test the accuracy of the prediction based on performance in the previous condition. Change after the first intervention phase may be due to other factors such as history and maturation and cannot be ruled out until observing the effect of subsequent treatment reversals. Each change in condition offers another opportunity for validation of the study hypotheses and strengthens the argument for a causal relationship between the intervention and performance.

A return to baseline conditions confirms two predictions, those from the previous treatment condition and from the first baseline phase (ie. if the intervention had not been implemented, would performance have changed?). Failure to return to baseline performance levels may not disprove the intervention effect. Multiple baseline designs have been created to answer this question by introducing different baselines at different points in time to investigate and rule out possible extraneous factors associated with the intervention such as a change in home or work situations, illness or weather. To accomplish this effect, different baselines are chosen to change subject, setting, time or condition variables.

A causal relationship between the intervention and performance is most often established through the analysis and visual inspection of graphically represented data. Occasionally statistical methods are employed to augment and further define the results. This process of validating an intervention effect on performance is referred to as the *experimental criterion.*

In general, clinical studies are most concerned with the applied significance of the intervention. The *therapeutic criterion* questions whether the intervention makes a significant difference in the client's life? Results need to be obvious and significant to meet this criteria. Consequently Kazdin (1982) and others support the suitability of visual inspection for determining significance in single-subject design experiments. The relatively unrefined and insensitive nature of visual inspection is regarded as a strength since only those changes which appear reliable and strong are accepted as supportive evidence. Weak results do not pass the vigorous criteria of visual inspection. This is contrasted with the sensitivity of statistical analysis which can determine significance even if the data are very weak. Thus the insensitivity of visual analysis is considered an advantage because it encourages investigators to carefully construct study designs to produce large effects.

The experimental criterion is met by observing the effect of the intervention on performance over time. Studies are carefully designed in phase pattern, order and number so that extraneous variables plausibly effecting results are discounted. Effect is determined through analysis of many factors including some of the following identified by Parsonson and Baer (1978): stability of baselines, variability within and between phases, overlap between scores of adjacent phases, the number of data points in each phase, changes in trend within and between phases, changes in level (means) between phases, the analysis of data across similar phases, and evaluation of the overall pattern of the data. Kazdin considered the factors related to the magnitude and rate of change as most prominent. Magnitude refers to changes in mean and level while rate refers to changes in trend and latency.

Mean changes refer to a shift in the average rates of performance across phases. Changes in level refers to the shift or discontinuity of performance from the end of one phase to the beginning of the next phase and is independent of a change in mean. A change in level is determined by a change in performance immediately after a shift in phase. Changes in mean and level are mutually exclusive. A trend or slope is established by assessing the direction of performance data with time either within a phase or amongst phases. The period between the onset of a condition and changes in performance is referred to as the latency of change. An immediate change in performance with a shift in phase more strongly indicates causality than a latent change. Changes in performance long after a phase shift introduce suspicion that an extraneous source may have influenced the change.

Changes in mean, level, trend and latency exist as separate characteristics but may occur either alone or in combination. The experimental criterion is met by summarising these and more indicators. Data that demonstrates consistency with replication and display minimum variability provide validation for the effect

In clinical studies the therapeutic criterion is evoked to evaluate the applied significance of the intervention. While a causal relationship may be clearly established through experimental criteria, the clinical import may or may not exist and deserves discussion. Wolf (1978) introduced the concept of social validation to evaluate the clinical or applied importance of the intervention. Social validation addresses the social implications of treatment focus, procedure and effect on performance. The two proposed methods to determine social validation are social comparison and subject evaluation. The social comparison model compares the performance of the behaviour of the client to the mean functioning level of his/her peers. The subjective method asks the peers of the client to determine any clinical or significant change in the performance of the client.

APPENDIX B

P.N. Cycle means (SD) of calorie, carbohydrate, protein and fat content.

Cycle: Condition:	1 B	2 S	3 B	4 P	5 S	6 P	Rest Days
Excluding Supplement:							
Calories (kcal)	2698	2430	2555	3419	2998	2352	2874
SD	389	276	352	884	272	675	868
CHO (g)	411	371	363	396	432	324	411
SD	35	74	95	88	40	86	98
Protein (g)	94	72	99	135	130	122	118
SD	14	1	33	77	74	12	76
Fat (g)	89	82	92	154	95	94	95
SD	23	13	10	77	9	32	37
Including Supplement:							
Calories (kcal)	3099	3785	2891	3698	4370	2602	-
CHO (g)	482	691	416	443	749	366	-

P.N. Day means (SD) of dietary content

Variable		Condition				
		B	P	S	Combined B & P	Rest Days
n = days		n = 2	n = 2	n = 2	n = 4	n = 10
Excluding supplementation:						
Calories (kcal)	Mean	2627	2886	2714		2874
	SD	101	754	402		868
Carbohydrate (g)	Mean	387	360	402		411
	SD	34.0	51.0	43.0		97.8
Protein (g)	Mean	97	104	101		118
	SD	4.0	44.0	41.0		76.1
Fat (g)	Mean	91	124	89		95
	SD	2.0	42.0	9.0		36.9
Including supplementation:						
Calories (kcal)	Mean	2995	3150	4078	3073	
	SD	147	775	414	464	
Carbohydrate (g)	Mean	449	405	720	463	
	SD	47	54	41	49	

P.N. Mean (SD) of two day distance totals (km)

Condition	n	Mean (SD)
Baseline	2	113.60 (1.34)
Placebo	1	111.21 (4.91)
Supplement	2	119.5 (2.48)
Baseline and Placebo	3	112.41 (3.25)

A.H. Cycle means (SD) of calorie, carbohydrate, protein and fat content.

Cycle:	1	2	3	4*	5	6	
Condition:	B	S	B	S	S	P	Rest Days
	n = 3	n = 3	n = 3	n = 2	n = 3	n = 3	n = 10
Excluding supplement:							
Calories (kcal)	3594	4613	3935	3635	3725	4468	3309
SD	1625	963	613	531	1505	2727	860
CHO (g)	526	637	716	520	471	532	514
SD	195	206	115	97	147	46	126
Protein (g)	108	139	96	138	128	119	93
SD	72	50	22	45	68	50	28
Fat (g)	141	179	85	118	151	165	111
SD	76	94	35	60	72	165	51
Including supplement:							Mean
Calories (kcal)	4149	6214	4310	5280	5262	4328	4924
CHO (g)	612	1013	789	916	848	592	795

4* two day average due to an injury and a subsequent single session withdrawl.

A.H. Day means (SD) of dietary content

Variable		Condition				
n = days		B n = 2	P n = 1	S n = 3	Combined B & P n = 3	Rest days n = 10
Excluding supplementation:						
Calories (kcal)	Mean	3765	4468	3991		3310
	SD	241	n.a.	541		860
Carbohydrate (g)	Mean	621	532	543		514
	SD	134	n.a.	85		126
Protein (g)	Mean	102	119	135		93
	SD	8.5	n.a.	6.1		28.1
Fat (g)	Mean	113	165	149		111
	SD	39.6	n.a.	30.5		50.7
Including supplementation:						
Calories (kcal)	Mean	4230	4708	5585	4389	
	SD	114	n.a.	545	287	
Carbohydrate (g)	Mean	700	592	926	664	
	SD	125	n.a.	83	108	

J.T. Mean (SD) of two day distance totals (km)

Condition	n	Mean (SD)
Baseline	2	120.84 (6.15)
Placebo	2	118.95 (9.86)
Supplement	2	127.24 (5.83)
Baseline and Placebo	4	119.89 (6.80)

J.T. Cycle means (SD) of calorie, carbohydrate, protein and fat content.

Cycle:	1	2	3	4	5	6	
Condition:	B	S	B	P	S	P	Rest Days
Excluding Supplement:							
Calories (kcal)	3907	2509	3003	3306	3773	-	3186
SD	845	326	512	689	963		568
CHO (g)	602	382	419	468	677	-	446
SD	102	59	98	81	86		89
Protein (g)	125	63	103	102	82	-	94
SD	43	11	26	67	6	-	32
Fat (g)	118	91	105	120	96	-	119
SD	36	27	52	34	28		33
Including Supplement:							
Calories (kcal)	4260	4190	3203	3626	5494		4155
CHO (g)	675	781	462	515	1077		702

J.T. Day means (SD) of dietary content

Variable n = days		B n = 2	P n = 1	S n = 2	Combined B & P n = 3	Rest Days n = 9
Excluding supplementation:						
Calories (kcal)	Mean	3455	3306	3141		3186
	SD	639	n.a.	894		651
Carbohydrate (g)	Mean	511	468	530		446
	SD	129	n.a.	209		93
Protein (g)	Mean	114	102	73		94
	SD	16.0	n.a.	13.0		33.0
Fat (g)	Mean	112	120	94		119
	SD	9.0	n.a.	4.0		46.0
Including supplementation:						
Calories (kcal)	Mean	3732	3626	4842	3696	
	SD	747	n.a.	922	532	
Carbohydrate (g)	Mean	569	515	929	551	
	SD	151	n.a.	209	111	

A.H. Mean (SD) of two day distance totals (km)

Condition	n	Mean (SD)
Baseline	2	129.7 (1.49)
Placebo	1	122.2 (n.a.)
Supplement	3	136.48 (3.86)
Baseline and Placebo	3	127.18 (4.43)

Individual and group means (SD) of dietary content during day 2, 3 and 4. Grouped by Condition.

	Baseline	Placebo	Supplement
P.N.			
Not Including Supplement	n=2	n=2	n=2
Cal (kcal)	2627 (101)	2886 (754)	2714 (402)
Protein (g)	97 (4)	104 (44)	101 (41)
Fat (g)	91 (2)	124 (42)	89 (9)
CHO (g)	387 (37)	360 (51)	402 (43)
Including Supplement			
Cal (kcal)	2995 (147)	3150 (775)	4078 (414)
CHO (g)	449 (47)	405 (54)	720 (41)
A.H.			
Not Including Supplement	n=2	n=1	n=3
Cal (kcal)	3765 (241)	4468 (n.a.)	3991 (541)
Protein (g)	102 (8)	119 (n.a.)	135 (6)
Fat (g)	113 (40)	165 (n.a.)	149 (31)
CHO (g)	621 (134)	532 (n.a.)	543 (85)
Including Supplement			
Cal (kcal)	4230 (114)	4708 (n.a.)	5585 (545)
CHO (g)	700 (125)	592 (n.a.)	926 (83)
J.T.			
Not Including Supplement	n=2	n=1	n=2
Cal (kcal)	3455 (639)	3306 (n.a.)	3141 (894)
Protein (g)	114 (16)	102 (n.a.)	73 (13)
Fat (g)	112 (9)	120 (n.a.)	94 (4)
CHO (g)	511 (129)	468 (n.a.)	530 (209)
Including Supplement			
Cal (kcal)	3732 (747)	3626 (n.a.)	4842 (922)
CHO (g)	569 (151)	515 (n.a.)	929 (209)

APPENDIX C

Recommended Nutrient Intakes

Subject	Cal. kcal	CHO g	Prot. g	Fat g	Chol. mg	Ca mg	Fe mg	K mg	Na mg	A IU	D IU	E mg	C mg	B-1 mg	B-2 mg	Niacin NE
P.N.	3276	450	60.2	109	300	800	9.0	4900	2450	5000	100	9.0	40.0	1.31	1.64	23.59
A.H.	4221	580	66.5	141	300	800	9.0	5411	2706	5000	100	10.0	40.0	1.69	2.11	30.39
J.T.	4494	618	70.8	150	300	800	9.0	5761	2881	5000	100	10.0	40.0	1.80	2.25	32.35

Nutrition Recommendations, Health & Welfare Canada 1990.

Vitamin B-1 (thiamine)
Vitamin B-2 (riboflavin)

Individual and group means (SD) n=days
Percent recommended energy intake (% REI) during days 2, 3 and 4.

	n (days)	Not Including Supplement Cal (%)	Protein (%)	Fat (%)	CHO (%)	Including Supplement Cal (%)	CHO (%)
P.N.							
Baseline	2	104 (3.1)	161 (6.4)	108 (2.7)	112 (9.2)	114 (0.9)	126 (6.4)
Placebo	2	114 (30)	174 (23)	148 (51)	104 (14)	125 (30)	117 (16)
Supplement	2	108 (17)	169 (69)	105 (11.1)	116 (13)	**176 (3.6)**	**191 (35)**
A.H.							
Baseline	2	108 (2.7)	146 (22)	91 (22)	129 (20)	130 (3.6)	157 (27)
Placebo	1	137 (n.a.)	179 (n.a.)	152 (n.a.)	120 (n.a.)	146 (n.a.)	133 (n.a.)
Supplement	3	122 (17)	203 (9.0)	138 (29)	122 (20)	**172 (17)**	**208 (15)**
J.T.							
Baseline	2	100 (18)	161 (21)	96 (8.3)	108 (27)	107 (21)	120 (31)
Placebo	1	96 (n.a.)	144 (n.a.)	104 (n.a.)	99 (n.a.)	105 (n.a.)	116 (n.a.)
Supplement	2	91 (26)	102 (19)	82 (2.7)	112 (44)	**138 (21)**	**195 (44)**
Group Means:							
Baseline	3	104 (3.9)	156 (8.7)	99 (8.6)	116 (11)	117 (12)	134 (21)
Placebo	3	116 (21)	166 (19)	1135 (27)	108 (11)	125 (21)	122 (9.4)
Supplement	3	107 (16)	158 (51)	108 (29)	117 (5.2)	**161 (21)**	**198 (8.8)**

Nutrition Recommendations, Health and Welfare Canada 1990

P.N. Cycle means and Recommended Nutrient Intake (%RNI) for calorie, carbohydrate, protein and fat content

Cycle:	1	2	3	4	5	6	
Condition:	B	S	B	P	S	P	Rest Days
	n = 3	n = 3	n = 3	n = 3	n = 3	n = 3	n = 10
Excluding Supplement:							
Calories (kcal)	2698	2430	2555	3419	2998	2352	2874
% RNI	82	74	78	104	92	72	101
CHO (g)	411	371	363	396	432	324	436
% RNI	91	82	81	88	96	72	96
Relative (g/kg)	5.83	5.26	5.15	5.62	6.13	4.60	6.18
Protein (g)	94	72	99	135	130	73	118
% RNI	156	120	165	225	217	122	197
Fat (g)	89	82	92	154	95	94	113
% RNI	81	75	84	104	87	86	103
Including Supplement:							Means
Calories (kcal)	3099	3785	2891	3698	4370	2602	3408
% RNI	94	115	88	112	134	77	103
CHO (g)	482	691	416	443	749	366	524
% RNI	107	153	93	98	166	81	116
Relative (g/kg)	6.84	9.80	5.90	6.28	10.62	5.19	7.44

A.H. Cycle means (SD) and Recommended Nutrient Intake (%RNI) for calorie, carbohydrate, protein and fat content

Cycle:	1	2	3	4*	5	6	
Condition:	B	S	B	S	S	P	Rest Days
	n = 3	n = 3	n = 3	n = 2	n = 3	n = 3	n = 10
Excluding Supplement:							
Calories (kcal)	3594	4613	3935	3635	3725	4468	3162
% RNI	85	109	93	86	88	106	75
CHO (g)	526	637	716	520	471	532	506
% RNI	91	110	123	90	81	92	88
Relative (g/kg)	7.46	9.04	10.16	7.38	6.68	7.55	7.18
Protein (g)	108	139	96	138	128	119	83
% RNI	87	209	145	208	193	179	67
Fat (g)	141	179	85	118	151	165	103
% RNI	163	127	61	84	107	117	120
Including Supplement:							Means
Calories (kcal)	4149	6214	4310	5280	5262	4328	4924
% RNI	98	147	102	125	124	103	116
CHO (g)	612	1013	789	916	848	592	795
% RNI	106	175	136	159	146	102	137
Relative (g/kg)	8.68	14.37	11.19	12.99	12.03	8.40	11

4* two day average due to injury and a subsequent single session withdrawl.

J.T. Cycle means and Recommended Nutrient Intakes (%RNI) for calorie, carbohydrate, protein and fat content

Cycle:	1	2	3	4	5	6	
Condition:	B	S	B	P	S	P	Rest Days
	n = 3	n = 3	n = 3	n = 3	n = 3		n = 9
Excluding Supplement:							
Calories (kcal)	3907	2509	3003	3306	3773	-	3186
% RNI	87	56	67	74	84	-	71
CHO (g)	602	382	419	468	677	-	446
% RNI	97	62	68	76	110	-	
Relative (g/kg)	8.54	5.42	5.94	6.64	9.60	-	6.33
Protein (g)	125	63	103	102	82	-	94
% RNI	176	88	146	144	115	-	131
Fat (g)	118	91	105	120	96	-	119
% RNI	79	61	70	80	64	-	79
Including Supplement:							Means
Calories (kcal)	4260	4190	3203	3626	5494	-	4155
% RNI	93	91	71	81	120	-	91
CHO (g)	675	781	462	515	1077	-	702
% RNI	109	126	75	89	174	-	115
Relative (g/kg)	9.57	11.08	6.55	7.30	15.28	-	9.96

Dietary Composition. Mean (SD)

	Condition											
Subject	Baseline			Placebo			Sup.			Sup. (inc. CHO)		
	CHO	Fat	Prot	CHO	Fat	Prot	CHO	Fat	Prot	CHO	Fat	Prot
	%cal SD	%cal SD	%cal SD	%cal SD	%cal SD	%cal SD	%cal SD	%cal SD	%cal SD	%cal SD	%cal SD	%cal SD
P.N.	59.9	27.2	12.9	52.1	35.0	13.0	57.5	28.4	14.2	70.8	19.5	9.8
n=2,2,2	3.25	2.12	1.13	5.94	3.60	2.33	2.55	1.27	3.75	3.11	0.07	3.04
A.H.	66.1	24.2	9.7	54.8	34.3	11.0	53.8	33.2	13.5	66.4	24.0	9.7
n=2,1,3	10.0	8.98	1.06				3.51	3.49	1.70	2.65	3.26	0.80
J.T.	61.3	26.7	12.1	60.4	28.7	10.9	64.2	26.8	9.1	78.2	16.5	5.4
n=2,1,2	4.31	3.39	0.92				7.57	6.65	0.92	2.90	3.11	0.21
Means	62.4	26.0	11.6	55.8	32.7	11.6	58.5	29.5	12.3	71.8	20.0	8.3
SD	3.25	1.61	1.67	4.23	3.45	1.18	5.27	3.33	2.76	5.96	3.77	2.50
				Placebo & Baseline			Unsupplemented					
Means				59.1	29.4	11.6	58.9	29.4	11.8			
SD				4.97	4.36	1.29	4.74	3.83	1.75			

Individual and group means (SD) of dietary composition during day 2, 3 and 4 (% caloric intake)

	Before Supplementation			After Supplementation
	Baseline	Placebo	Supplement	Supplement
P.N.				
	n=2	n=2	n=2	n=2
CHO	60 (3.3)	52 (5.9)	58 (2.6)	71 (3.1)
Fat	27 (2.1)	35 (3.6)	28 (1.3)	20 (0.1)
Protein	13 (1.1)	13 (2.3)	14 (3.8)	10 (3.0)
A.H.				
	n=2	n=1	n=3	n=3
CHO	66 (10)	55 (n.a.)	54 (3.5)	66 (2.5)
Fat	24 (9.0)	34 (n.a.)	33 (3.5)	24 (3.3)
Protein	10 (1.1)	11 (n.a.)	14 (1.7)	9.7 (0.8)
J.T.				
	n=2	n=1	n=2	n=2
CHO	61(4.3)	60 (n.a.)	64 (7.6)	78 (2.9)
Fat	27 (3.4)	29 (n.a.)	27 (6.7)	17 (3.1)
Protein	12 (0.9)	11 (n.a.)	9.1 (0.9)	5.4 (0.2)
Group Means	n=3	n=3	n=3	n=3
CHO	62 (3.3)	56 (4.2)	59 (5.3)	72 (6.0)
Fat	26 (1.6)	32 (3.9)	30 (3.3)	20 (3.7)
Protein	11.7 (1.5)	12 (1.2)	12 (2.8)	8.3 (2.5)

Means (SD) of percent recommended nutrient intake (% RNI) during days 2, 3 and 4

Variable		Condition			
		Baseline n = 6	Placebo n = 4	Supplement n = 7	Rest Days n = 3
Excluding supplementation:					
Calories	Mean	82.17	89.00	84.14	82.30
	SD	8.57	18.51	16.29	16.29
Carbohydrates	Mean	91.83	82.00	90.14	85.00
	SD	18.36	9.52	17.15	12.77
Proteins	Mean	145.83	167.50	164.29	131.67
	SD	31.12	44.95	54.35	65.00
Fat	Mean	89.67	96.75	86.43	100.67
	SD	36.90	16.92	23.65	20.60
Including supplementation:					
Calories	Mean	91.00	93.25	122.29	
	SD	10.88	16.94	17.30	
Carbohydrates	Mean	104.33	82.50	157.00	
	SD	20.12	9.40	17.30	

APPENDIX D

Perceived exertion summary for the six cycles of three training days - P.N.

Day 2 - (Front Push)

Sub.	Cycle	Cond.	VT1			VT2			VT3			VT - 20(2)		
			Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot
P.N.	1	B	16	12	28	17	14	31	17	17	34			
P.N.	**2**	**S**	**17**	**15**	**32**	**17**	**14**	**31**	**18**	**17**	**35**	**15**	**13**	**28**
P.N.	3	B	15	12	27	15	13	*28	16	14	*30	15	9	*24
P.N.	4	P	12	8	20	12	8	20	13	8	21	11	8	19
P.N.	**5**	**S**	**12**	**9**	**21**	**12**	**10**	**22**	**13**	**10**	**23**	**12**	**8**	**20**
P.N.	6	P	13	10	23	12	8	*20	11	9	*20	8	8	*16

Day 3 (Specific Endurance)

Sub.	Cycle	Cond.	VT - 20(1)			VT - 20(2a)			VT - 20(2b)			
			Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot	
P.N.	1	B	15	13	28	14	13	*27	16	16	*23	
P.N.	**2**	**S**	**14**	**11**	**25**	**13**	**10**	**23**	**13**	**10**	**23**	
P.N.	3	B	16	10	26	14	8	*22	13	10	*23	
P.N.	4	P	12	11	23	13	9	*22	15	9	24	
P.N.	**5**	**S**	**12**	**11**	**23**	**13**	**11**	***24**	**14**	**10**	***24**	sore stomach
P.N.	6	P	12	8	20	11	9	20	10	8	18	

Day 4 (End Push)

Sub.	Cycle	Cond.	VT - 20(1)			VT1			VT3			VT - 20(2)		
			Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot
P.N.	1	B	11	8	19	14	12	26	17	16	33	15	12	27
P.N.	**2**	**S**	**13**	**10**	**23**	**14**	**12**	**26**	**17**	**14**	**31**	**14**	**11**	**25**
P.N.	3	B	11	8	19	15	11	26	16	12	28	12	8	20
P.N.	4	P												

			VT - 20(2)			TT(1)			TT(2)			TT(end)		
P.N.	**5**	**S**	**13**	**9**	**21**	**12**	**9**	**21**	**15**	**12**	**27**	**17**	**14**	**31**
P.N.	6	P	11	8	19	11	9	19	13	10	23	15	13	28

* did not achieve target hr.

Perceived exertion summary for the six cycles of three training days - A.H.

Day 2 - (Front Push)

Sub.	Cycle	Cond.	VT1			VT2			VT3			VT - 20(2)		
			Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot
A.H.	1	B	15	10	25	16	11	27	17	11	*28	16	10	26
A.H	**2**	**S**	**17**	**10**	**27**	**17**	**11**	**28**	**18**	**10**	**28**	**15**	**10**	**25**
A.H.	3	B	17	10	27	16	11	27	17	11	28	16	10	26
A.H	**4**	**S**	**16**	**10**	**26**	**15**	**10**	**25**	**16**	**11**	***27**	**15**	**9**	**24**
A.H	**5**	**S**	**14**	**10**	**24**	**14**	**10**	**24**	**15**	**11**	**26**	**15**	**10**	**25**
A.H.	6	P	14	11	25	15	11	26	17	11	*28	14	11	25

Day 3 (Specific Endurance)

Sub.	Cycle	Cond.	VT - 20(1)			VT - 20(2a)			VT - 20(2b)		
			Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot
A.H.	1	B	17	10	27	17	10	27	16	12	28
A.H	**2**	**S**	**15**	**10**	**25**	**14**	**10**	**24**	**14**	**10**	**24**
A.H.	3	B	15	9	24	15	8	23	14	9	23
A.H	**4**	**S**	**14**	**10**	**24**	**14**	**10**	**24**	**15**	**11**	**26**
A.H	**5**	**S**	**15**	**10**	**25**	**16**	**11**	**27**	**15**	**11**	**26**
A.H.	6	P	12	10	22	16	10	26	14	11	25

Day 4 (End Push)

Sub.	Cycle	Cond.	VT - 20(1)			VT1			VT3			VT - 20(2)		
			Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot	Lgs	Lng	Tot
A.H.	1	B	15	10	25	18	12	30	19	11	30	16	10	26
A.H	**2**	**S**	**14**	**9**	**23**	**17**	**12**	**29**	**17**	**11**	**28**	**14**	**9**	**23**
A.H.	3	B	14	9	25	15	10	25	17	11	28	16	10	26
A.H.	4	did not participate												

			VT - 20(2)			TT(1)			TT(2)			TT(end)		
A.H	**5**	**S**	**13**	**9**	**22**	**14**	**10**	**24**	**15**	**11**	**26**	**16**	**11**	**27**
A.H.	6	P	14	10	24	16	11	*27	17	12	*29	-	-	-

did not achieve target hr.

APPENDIX E

Informed Consent Form

This study is the basis of a thesis to be presented to the Faculty of Education, University of Victoria, Victoria, British Columbia, by Philip J. Skinder in partial fulfilment of the requirements for the Degree of Master of Arts in the field of Physical Education. The observation period will be from February 9, 1992 until March 15, 1992. The scope of the study will be delimited to tests of $\dot{V}O_2$ max, time trials and the following of daily prescribed workout protocols. Dietary and training record will be maintained starting three days prior to the study and for the next 34 days. Anthropometric measurements including body weight and skin folds will be recorded before and after the 35 day trial. I will refrain from any other vigorous physical activity during the scope of the experiment.

The data gathered throughout the study period will be available to me and my coach at the end of the testing period. However, this information will not be available to anyone else without my written permission. The anonymity and confidentiality of all data will be protected. I am free to withdraw from the study at any time without fear of reprisals.

Data collected during the study will be used in the thesis and will become the property of Philip J. Skinder. It cannot be published or reproduced in any form without his permission in writing.

I have read the preceding statement, understand it, and give my informed consent to take part in the study.

I confirm that I have been given a satisfactory and complete explanation of the procedures in which I am to participate. I further confirm that I have been advised that I may ask for further explanation and or demonstration of such procedures at any time. I also acknowledge that I have been advised that I may terminate participation in any or all of the procedures at any time as a matter of my own personal discretion or volition. I hereby waive and disclaim any entitlement against the University of Victoria, the personnel involved in this research study, the research investigators, or any other person in respect of liability that may arise from my participation as a research subject in this study.

Printed Name: ______________________

Signed : ______________________

Date: ______________________

UNIVERSITY OF VICTORIA SPORTS AND FITNESS CENTRE

INFORMED CONSENT OF PHYSIOLOGICAL ASSESSMENTS

In order to assess physiological function(s) the following laboratory tests will be performed:

Lab Initial   Subject Initial

_______ _______ Submaximal Cardio-Respiratory Function

You will exercise on an ergometer up to 75% of predicted maximum heart rate. The following indicated variables will be measured:

a) ventilatory responses ___   c) thermoregulatory responses ___

b) heart rate responses ___   d) other ___

_______ _______ Maximal Cardio-Respiratory Function

You will exercise on an ergometer with progressively increasing loads to elicit **maximal** responses in the following indicated variables:

a) oxygen consumption ___   c) ventilation ___

b) heart rate ___   d) other ___

_______ _______ Submaximal and/or Maximal Muscular Contractions

You will perform submaximal or maximal muscular contractions in the following modes:

isometric ___ isotonic ___ isokinetic ___ eccentric ___

_______ _______ Maximal Anaerobic Function

You will exercise maximally on an ergometer for a period of ____ seconds to elicit responses in the following indicated variables:

a) heart rate ___ b) blood lactate ___ c) other ____

_______ _______ Blood Chemistry

Blood samples will be taken prior to, during, or post-exercise by:

a) venipuncture ___ b) finger tip prick ___

_______ _______ Body Composition

Lean body mass and percent body fat may be assessed by:

a) anthropometric measures ___ b) body densiometry ___

Consent Form

Lab Initial	Subject Initial	
_______	_______	Tests will be administered by qualified personnel under the direct supervision of the investigator(s).
_______	_______	Blood samples will be taken by a qualified laboratory technician or registered nurse.
_______	_______	Training will be monitored by the investigator(s) or trained assistants.
_______	_______	Test and/or training data/results will be treated in a confidential manner and used only to describe group responses unless specific approval has been given to other use of the material by each subject, or guardian where necessary.
_______	_______	While it is highly unlikely that a subject should be injured or taken ill during a test or training session, lab personnel are trained in emergency procedures and emergency equipment is on-site at all times.
_______	_______	All laboratory activity will be completed proximal to medical and/or paramedical assistance.
_______	_______	The maximal exercise loads imposed will not exceed those which might be expected of an athlete during sports performance.
_______	_______	If a record is achieved; athletes name, sport, record value and date of testing may be posted on the U.Vic Sport and Fitness Centre Records Board.

I have read the above and agree to participate in this research project/ fitness appraisal at my own risk. I regularly take part in strenuous physical activity at least as intense as these tests. I realize that I may expect a thorough explanation and/or demonstration of any procedures and that I may terminate participation at any time in any or all procedures of my own volition.

Having voluntarily assumed participation and the risks thereof, in the project, I hereby disclaim and release the University of Victoria, its agents, servants or employees, including all personnel involved in the research project/ fitness appraisal from any and all liability that might otherwise arise as a result of my participation as a research subject in this study/or fitness appraisal.

NAME: ______________________________ DATE: ______________
(please print)

SIGNATURE: ______________________

Physical Activity Readiness
Questionnaire - PAR-Q
(revised 1994)

PAR - Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO		
☐	☐	1.	Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
☐	☐	2.	Do you feel pain in your chest when you do physical activity?
☐	☐	3.	In the past month, have you had chest pain when you were not doing physical activity?
☐	☐	4.	Do you lose your balance because of dizziness or do you ever lose consciousness?
☐	☐	5.	Do you have a bone or joint problem that could be made worse by a change in your physical activity?
☐	☐	6.	Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
☐	☐	7.	Do you know of any other reason why you should not do physical activity?

If you answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- **You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.**
- **Find out which community programs are safe and helpful for you.**

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal — this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively.

DELAY BECOMING MUCH MORE ACTIVE:

- If you are not feeling well because of a temporary illness such as a cold or a fever — wait until you feel better; or
- If you are or may be pregnant — talk to your doctor before you start becoming more active.

Please note: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: **The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.**

You are encouraged to copy the PAR-Q but only if you use the entire form

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction

NAME ____

SIGNATURE ____ DATE ____________

SIGNATURE OF PARENT ________________________________ WITNESS ____________
or GUARDIAN (for participants under the age of majority)

continued on other side

Supported by Health Canada Santé Canada

APPENDIX F

Name
Date:
Type of ride: Day 2
Windtrainer: stopping time from 30 km/hr. =

Ambient Temp.:
Wind:
Humidity:
Tire Pressure: previous (p.s.i.)
today : (p.s.i.)

Weight: Before: After:

							Resistance			
				R.P.E.	Heart Rate		Wind t.	Cog Wheel		Comments:
Dist.	Spd.	Time	Focus	Lg/Ht	Range	Actual		Big	Sml	
km		min.			b.p.m.	b.p.m.				
0		0								
		:05:00	spin							
		:10:00	build VT-20							
		:15:00	VT - 20%							
		:20:00		*						
*		:25:00	Build VT-10	*						
		:30:00	VT - 20%							
		:35:00								
		:40:00								
*		:45:00		*						
		:50:00	VT - 10%							
*		:55:00		**						
		1:00.00	VT							
		1:05.00	VT							
*		1:10:00	VT	*						
		1:15:00	spin							
*		1:20:00	Build VT-10							
		1:25:00	VT							
		1:30:00	VT							
*		1:35:00	VT	*						
		1:40:00	spin							
*		1:45:00	Build VT-10							
		1:50:00	VT							
		1:55:00	VT							
*		2:00:00	VT	*						
*		2:05:00	spin							
		2:10:00	VT - 20%	*						
		2:15:00								
		2:20:00								
*		2:25:00		*						
		2:30:00	Spin							
		2:35:00	(& drink)							

										Type of Liquid: Volume of Liquid:

General Comments About the Ride:

Name:

Date:

Type of ride: Day 3

Windtrainer: stopping time from 30 km/hr. =

Ambient Temp.:

Wind:

Humidity:

Tire Pressure: previous (p.s.i.)

today : (p.s.i.)

Weight: Before After

							Resistance			
				R.P.E.	Heart Rate		Wind t.	Cog Wheel		Comments:
Dist.	Spd.	Time	Focus	Lg/Ht	Range	Actual		Big	Sml	
km		min.			b.p.m.	b.p.m.				
0		0								
		:05:00	spin							
		:10:00								
*		:15:00	Build VT-20							
		:20:00	VT - 20%	*						
		:25:00								
		:30:00								
		:35:00		*						
		:40:00								
*		:45:00								
		:50:00								
		:55:00								
		1:00.00		*						
		1:05.00								
		1:10:00								
*		1:15:00		*						
		1:20:00	spin							
*		1:25:00		*						
		1:30:00	VT - 20%							
		1:35:00								
		1:40:00								
		1:45:00								
		1:50:00		*						
		1:55:00								
*		2:00:00								
		2:05:00								
		2:10:00								
		2:15:00		*						
		2:20:00								
*		2:25:00		*						
		2:30:00	spin							
		2:35:00	(& drink)							
		2:40:00								
		2:45:00								
										Type of liquid
										Volume

General Comments About the Ride:

Name:
Date:
Type of ride: DAY 4 supplemental
Windtrainer: stopping time from 30 km/hr. =

Ambient Temp.:
Wind:
Humidity:
Tire Pressure: previous (p.s.i.)
today : (p.s.i.)

Weight: Before:			After:				Resistance			
				R.P.E.	Heart Rate		Wind t.	Cog Wheel		Comments:
Dist.	Spd.	Time	Focus	Lg/Ht	Range	Actual		Big	Sml	
km		min.			b.p.m.	b.p.m.				
0		0								
		:05:00	spin							
		:10:00	Build VT-20							
		:15:00	VT - 20%							
*	*	:20:00		*						
*		:25:00	Build VT-10	*						
		:30:00	VT - 20%							
		:35:00								
		:40:00								
*		:45:00		*						
		:50:00	spin							
*		:55:00	choice							
*		1:00.00	choice	*						
*		1:05.00	TT start	*						
*		1:10:00	TT							
*		1:15:00								
*		1:20:00		*						
*		1:25:00		*						
*		1:30:00								
*		1:35:00								
*		1:40:00		*						
*		1:45:00		*						
*		1:50:00								
		1:55:00		*						
		2:00:00	TT end							

Type of Liquid:
Volume of Liquid:

General Comments About the Ride:

Name:
Date:
Type of ride: DAY 4
Windtrainer: stopping time from 30 km/hr. =

Ambient Temp.:
Wind:
Humidity:
Tire Pressure: previous (p.s.i.)
today : (p.s.i.)

Weight: Before: After:

							Resistance			
				R.P.E.	Heart Rate		Wind t.	Cog Wheel		Comments:
Dist.	Spd.	Time	Focus	Lg/Ht	Range	Actual		Big	Sml	
km		min.			b.p.m.	b.p.m.				
0		0								
		:05:00	spin							
		:10:00	Build VT-20							
		:15:00	VT - 20%							
*		:20:00		*						
*		:25:00	Build VT-10	*						
		:30:00	VT - 20%							
		:35:00								
		:40:00								
*		:45:00		*						
		:50:00	spin							
*		:55:00	Build VT-10							
*		1:00.00	VT	*						
*		1:05.00		*						
		1:10:00	spin							
*		1:15:00	Build VT-10							
*		1:20:00	VT	*						
*		1:25:00		*						
		1:30:00	spin							
*		1:35:00	Build VT-10							
*		1:40:00	VT	*						
*		1:45:00		*						
		1:50:00	spin							
*		1:55:00	VT-20	*						
*		2:00:00		*						
*		2:05:00		*						
*		2:10:00		*						
*		2:15:00	spin	*						
*		2:20:00	& drink	*						

Type of Liquid:
Volume of Liquid:

General Comments About the Ride:

Name:

Date:

Time	Foods:	Cooking Procedure	Amount

Sleep (approx. hours):

Waking heart rate (b.p.m.):

General Comments:

Name:

Date:

Time	Foods:	Cooking Procedure	Amount

Sleep (approx. hours):

Waking heart rate (b.p.m.):

General Comments:

VITA

Surname: Skinder Given Names: Philip Joseph

Place of Birth: __________________ Date of Birth: ________________

Educational Institutions Attended:

University of Victoria	1984 to 1995

Degrees Awarded:

B.Sc. (Biochemistry)	University of Victoria	1989

Honours and Awards:

None

Publications:

PARTIAL COPYRIGHT LICENCE

Title of Thesis: THE EFFECT OF A GLUCOSE POLYMER SUPPLEMENT DRINK INGESTED IMMEDIATELY POST EXERCISE ON TRAINING VOLUME

Autho [redacted]
PHILIP JOSEPH SKINDER
AUGUST 31, 1995