Changes in hematological, physiological and menstrual status with training in elite female middle distance runners.

MICHAEL J. KING
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to the required standard

[redacted]
Dr. Howard A. Wenger, Ph.D.

[redacted]
Dr. David Docherty, Ph.D.

[redacted]
Dr. Michael D.D. McNeely, M.D.

[redacted]
Dr. Richard D.H. Backus, M.D.

[redacted]
Dr. John E. Peterson, M.D.

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Supervisor: Dr. Howard A. Wenger, Ph.D.

ABSTRACT

In order to describe the relationships between maximal aerobic power($\dot{V}O_2max$), performance tests(PT), training volume(TRIMPS), and menstrual function, nine elite female middle distance runners($\dot{V}O_2max$=58.2±5.7 ml.kg-1.min-1)from the University of Victoria volunteered to participate in a (9) week training study. Mean absolute $\dot{V}O_2max$ increased 5.4% from 3.2 l.min-1 to 3.5 l.min-1 ($p<0.01$) as did mean relative $\dot{V}O_2max$ which increased 7.0% from 58.2 ml.kg-1.min-1 to 63.6 ml.kg-1.min-1 ($p<0.01$). Post hoc Cluster Analysis identified (3) menstrual function groups, the amenorrheic (AM n=2), the oligomenorrheic (OM n=2) and the eumenorrheic (EU n=5). A fourth group, menstrual dysfunction (MD n=4) was formed for statistical purposes by collapsing the AM and OM groups together.

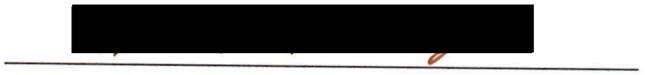
No relationship occurred between the hematological variables, hemoglobin (HG), hematocrit (HCT), serum iron (SI), iron binding capacity (IBC), percent saturation of transferrin (%SAT), serum ferritin (FER) and the training volume performed(TRIMPS), or between PT and TRIMPS.

HG and HCT and PT scores were significantly correlated ($p<0.05$) with $\dot{V}O_2max$ and significant correlations ($p<0.05$) did occur between HG and SI and the number of kilocalories (KCALS) consumed on the nutritional survey (NUTR).

Menstrual dysfunction(MD) was associated with the hematological variables. Mean levels for HG, HCT, SI, IBC, %SAT and FER were higher in the OM and AM groups when compared to the EU group. And the MD group demonstrated significantly higher ($p<0.05$) mean FER levels than the EU group as well as higher VO2max scores, faster PT times and completed more TRIMPS than the EU athletes.

Mean FER levels significantly deteriorated ($p<0.05$) over the (3) sample times (77.0±27.7, 65.3±31.0, 62.3±24.6 ug.litre-1). It was also demonstrated that AM athletes consumed fewer mean KCALS, while female middle distance runners in this study ingested more iron from supplements than through natural food sources.

EXAMINERS:



Dr. Howard A. Wenger, Ph.D.

Dr. David Docherty, Ph.D.

Dr. Michael D.D. McNeely, M.D.

Dr. Richard Backus, M.D.

Dr. John E. Peterson, M.D.

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DEDICATION

To my Grandmother, Mother and late Father who impressed upon me the value of higher education.

Chapter I

INTRODUCTION

Increased frequency of participation in vigorous exercise by females has led to an increase in both training related iron deficiency and exercise induced amenorrhea (Newhouse and Clement, 1988; Prior, 1988; Diehl et al., 1986; Williams, 1984; Dufaux et al., 1981; Ehn et al., 1980). Iron deficiency with concomitant low hematological indices may impact on performance by reducing the amount of oxygen arriving at the exercising tissues. The absence of adequate oxygen forces obligatory anaerobic glycolysis resulting in lactic acid which may produce muscular fatigue. Is this the case in elite female athletes experiencing menstrual dysfunction? Without regular menstruation, the major avenue of blood loss, are amenorrheic or oligomenorrheic athletes associated with higher hematological indices?

Several studies have indicated training related iron deficiency in females participating in endurance running (Newhouse and Clement, 1988; Pate et al., 1986; Sullivan, 1986; Schoene et al., 1983). However, only a few authors (Banister and Hamilton, 1985) have attempted to quantify the training volumes. The lack of quantification of training volumes makes the association of training data with the hematological parameters difficult. Therefore, this study quantified volumes of training

with Training Impulse Units(TRIMPS) which are the product of training duration and heart rate data (Banister, 1988; Banister and Hamilton, 1985).

It has been demonstrated that female runners are prone to a negative iron balance due to several factors: increased sweating; hemoglobinuria; gastrointestinal distress; hemolysis; a decreased rate of iron absorption and menstrual blood losses (Newhouse and Clement, 1988; Sullivan, 1986; Pate, 1986; Nickerson and Tripp, 1983; Valberg et al., 1976; Beaton et al., 1970). Iron balance is derived from several clinical hematological assays. This research focused on the levels of hemoglobin (HG), hematocrit (HCT), serum iron (SI), iron binding capacity (IBC), percent saturation of transferrin (%SAT) and serum ferritin (FER).

Iron is stored in two compounds: ferritin and hemosiderin (Bauer, 1974). Plasma ferritin has clinically demonstrated a high correlation with total body iron storage (Byryne et al., 1981; Tilkian and Conover, 1975; Bauer, 1974).

The value of ferritin as an indicator of iron storage is well established (Finch and Huebers, 1986; Clement and Sawchuck, 1984; Valberg et al., 1976) and FER levels have been shown to accurately reflect the size of total body iron reserves in female athletes (Newhouse and Clement, 1988; Newman, 1986; Schwarzkopf et al., 1986; Brown et al., 1985; Clement and Sawchuck, 1984; Schoene et al., 1983; Clement and Asmundson, 1982; Valberg et al., 1976). Asymptomatic ferritin levels determined by IRMA, are proposed to be 20-160 ug.litre-1 (Clement and Sawchuck, 1984).

Sixty to seventy percent of total body iron is found in HG and/or myoglobin. The remaining 30-40% forms the labile pool of iron storage located in the reticuloendothelial cells of the bone marrow, liver and spleen (Guyton, 1981). Mean HG levels are 110-160 g.litre-1 in females and 130-180 g.litre-1 in males (Bryne et al., 1981).

Other valuable laboratory tests, for the determination of an athletes iron profile include; HCT, SI, IBC and %SAT.

The HCT is expressed as the percentage of packed erythrocytes and formed elements of a given volume of blood (Byrne et al., 1981). Normal values are 0.40-0.54 for males and 0.37-0.47 for females. SI measures the plasma iron bound to the protein transferrin. SI is noted to have a diurnal variation which warrants consistent testing protocols and sampling times in order to assure meaningful results (Bauer, 1974). Normal physiologic ranges are 15-42 umoles.litre-1 in males and 12-31 umoles.litre-1 in females (Bryne et al., 1981). IBC measures the maximal saturation of free transferrin with iron (Bauer, 1974). An elevation in IBC is often associated with iron deficiency anemia. Normal physiologic ranges are 48-90 umoles.litre-1 for males and 45-73 for females (Bryne et al., 1981). %SAT describes the proportion of the iron binding protein which is saturated with iron. When the degree of saturation is low, iron is mainly utilized for hemoglobinization, while a high %SAT reading indicates a shunting toward storage (Bauer, 1974). Normal range is reported to be 0.20-0.55 (Bryne et al., 1981).

This study attempted to demonstrate the relationship between select hematological variables and training volume in TRIMPS. Other relationships tested were: and the correlation between select hematological parameters and the physiological variables $\dot{V}O_2max$ and the 1200 metre performance test(PT); the correlation between hematological variables and dietary intake. Many researchers find detrimental effects on $\dot{V}O_2max$ and performance with reduced hematological variables (Newhouse and Clement, 1988; Dufaux et al., 1981; Ehn et al., 1980; DeWijn et al., 1971). Yet some authors have challenged the effect of iron deficiency without anemia on endurance performance (Risser et al., 1988; Celsing et al., 1986; Celsing and Ekblom, 1986). Inadequate nutritional intakes have been blamed for contributing to poor iron storage and iron deficiency anemia in female athletes (Kaiseraurer et al., 1989; Snyder et al., 1989; Clark et al., 1988; Clement and Sawchuck, 1984; Clement and Asmundson, 1982).

High training volumes and inadequate nutritional practices are also cited as precursors to athletic amenorrhea (Cumming, 1988; Shangold, 1985; Williams, 1984)

Amenorrhea, defined as the cessation of menses in a female who was previously menstruating, appears to be a common syndrome in female endurance athletes (Bitner, 1985; Shangold, 1985; Baker et al., 1981; Dale et al., 1979). Several characteristics have been cited which define those who may be predisposed to amenorrhea with exercise: prior menstrual irregularity; age of onset of menarche; a history of weight loss; rapid weight loss; low body fat percentage; low energy intake; and volume, intensity

and mode of training (Cumming, 1988; Fisher et al., 1986; Nielson and Fleck, 1985; Williams, 1984; Wakat et al., 1982). The term eumenorrhea is used to describe regular menstruation in females, while oligomenorrhea is used clinically to describe infrequent and light mentrual cycles (Williams, 1984). Although the exact etiology of athletic amenorrhea is not known. It could be speculated by the elimination of menstruation, the amenorrheic athlete may support a greater iron storage.

There is little research into the cause and effect of iron deficiency and menstrual dysfunction. However, females have been shown to have an increased iron requirement directly related to the extent of menstruation (Clement and Sawchuck, 1984; Beaton et al., 1970; Shaw, 1970), and a high prevalence of iron deficiency has been observed in exercising female populations (Diehl et al., 1986; Dufaux et al., 1981; Ehn et al., 1980), with particular reference being made to female endurance runners (Newhouse and Clement, 1988; Pate, 1986; Sullivan, 1986; Banister and Hamilton, 1985; Schoene et al., 1983).

Several studies cite an iron cost to training, showing a marked depletion of iron stores, measured by serum ferritin, over the course of a competitive season (Risser et al., 1988; Diehl et al., 1986; Nickerson and Tripp, 1983). Diehl et al. (1986) and Risser et al. (1988) quantified menstrual flow in various collegiate female athletes with no significant correlation to iron storage. However, these studies did not describe the menstrual status (ie. AM, OM or EU) nor the quantity of training.

Therefore, this research was conducted to describe: the changes pre to post study in the physiological variables ($\dot{V}O_2max$ and PT); the volume of training performed (TRIMPS); and menstrual function(AM, OM, EU). This research also set out to identify the relationships between: the hematological variables and training volume (TRIMPS); the hematological variables and the physiological variables($\dot{V}O_2max$ and PT); the physiological variable (PT) and training volume (TRIMPS); and between the hematological variables and dietary intake. The final purpose of this research was to determine if menstrual status was associated with the hematological and the physiological variables ($\dot{V}O_2max$ and PT). The specific purposes of this study are described in nine (9) research questions.

1.1 Were there changes pre to post study in $\dot{V}O_2max$, PT, body weight(WT), HRmax and percent body fat(BF)?

1.2 Were there any variations in the training volumes (TRIMPS) monitored pre to post-study?

1.3 Which set of menstrual quantification data best describes the conditions of amenorrhea(AM), oligomenorrhea(OM) and eumenorrhea(EU)?

1.4 What is the relationship between training volume and the hematological parameters measured every three weeks?

1.5 What is the relationship between $\dot{V}O_2max$, the three performance tests(PT1, PT2, PT3) and the corresponding hematological parameters?

1.6 What is the relationship between performance test scores (PT2 and PT3) and the training volume as determined by the TRIMP scores?

1.7 Does a relationship exist between dietary intake and the hematological profile of an elite female runner in high volume training?

1.8 Was menstrual status associated with the hematological profile of the athletes?

1.9 Was menstrual status associated with the fitness variables ($\dot{V}O_2max$ and PT)?

Chapter II

METHODS

Nine healthy, elite female middle distance runners ($\dot{V}O_2max$=58.2+5.7 ml.kg-1.min-1) from the University of Victoria Vikes Racing Team volunteered and signed informed consent (Appendix A). Prior to experimentation, all subjects participated in a familiarization lecture, which outlined experimental design, the physical demands of testing, test protocols and inventories required. The physical characteristics of the subjects are shown in Table 1.

The subject pool consisted of 9 elite female middle distance runners, 2 which were Nationally Carded for Athletics and were of International calibre, 1 whom had just returned from a scholarship in the U.S., 2 who were national class competitors and 4 who competed at a minimum of varsity level athletics.

2.1 Physiological Testing

The illustration of the experimental protocol (Figure 1), summarizes the times of data collection.

Aerobic power ($\dot{V}O_2max$) was assessed before and after study by open circuit spirometry during a single, continuous multistage treadmill run. A modified Canadian Association of Sport Sciences (C.A.S.S.) protocol was used (Appendix E), incorporating increasing speed until ventilatory threshold was reached, and

subsequent increased grade to $\dot{V}O_2max$ (MacDougall et al., 1982). Respiratory gases were collected and analyzed every 30 seconds by a Beckman Metabolic Measurement Cart (MMC), and heart rates were recorded every minute by telemetry utilizing the PE3000 Sport Tester system. The MMC was calibrated prior to and after each VO_2max assessment with primary standard gases of known concentrations. Subjects were considered to have attained $\dot{V}O_2max$ if one or more of the following criteria were met; a plateau or decline in VO_2 with an increased power output(0.5mph or 2%grade), or a respiratory exchange ratio(R) greater than 1.15.

Anthropometric measurements of height(HT) in centimetres, body weight(WT) in kilograms and the skin fold thickness in millimetres of 4 sites (triceps, biceps, subscapular, illiac crest) were measured prior to each $\dot{V}O_2max$ assessment. Percent body fat (BF) was determined by the methods described in Durnin and Wormersley (1974).

2.2 Performance Testing

Each subject was required to perform (3) 1200 metre time trials on a 400 metre synthetic track, at the beginning of each training phase (Figure 1). Each performance test(PT) was conducted individually with no lap times given. The time for completion was recorded in seconds(s) and exercise heart rate was monitored every 5 s by telemetry using the PE3000 system.

2.3 Blood Testing

Venous blood samples were collected from the antecubital vein at a consistent weekday and hour during weeks 0, 2, 5 and 8. Two Becton-Dickinson vacutainers, one red top(chemistry) and one lavender top(hematology), were analyzed by Island Medical Laboratories(IML) Victoria, B.C. for HG, HCT, SI, IBC, %SAT and FER.

HG (g.litre-1) and HCT, were analyzed by a Coulter S Autoanalyzer. SI and IBC (umoles.litre-1) were determined by a Cobasbio Random Access Analyzer. FER concentration (ug.litre-1) was determined by Two-Site Immunoradiometric Assay(IRMA), as described by Bio-Rad Laboratories (1981).

All samples were drawn, handled, prepared and analyzed by either a medical pathologist, nurse or qualified laboratory technician of Island Medical Laboratories.

2.4 Inventories

Each subject was required to fill out a weekly training volume(TV) survey (Appendix C), which gave details regarding exercise duration and intensity. This raw data was used to produce training impulse scores(TRIMPS). Quantification of training volume by TRIMP units requires the product of the

duration of exercise in minutes by the factor created when dividing heart rate exercise minus heart rate rest over heart rate max minus heart rate rest(TRIMP= d(min)xHRex-HRr/HRmax-HRr). In accordance with Banister and Hamilton (1985), the heart rate rest was taken before getting out of bed, and HRmax was the highest heart rate achieved during the $\dot{V}O_2$max test.

Subjects were requested to record characteristics of all menstrual cycles occurring within the 9 week study. Information was to be transcribed onto the Estimated Menstrual Blood Loss Questionnaire (MQ) designed specifically for this study (Appendix B).

Each subject was required to participate in 3 dietary surveys, which encompassed one initial, middle and final week throughout the 9 week study (Figure 1). The 3 day nutritional surveys (NUTR) were provided and analyzed by the Sport Medicine Council of B.C.(High Performance Sport Science Unit) at the University of British Columbia. The comprehensive computerized analysis utilized the Canadian Nutrient File database (1986), which is based on the U.S. Department of Agriculture Nutrient Database release 4, (1984). A sample copy of the dietary survey is in Appendix D.

2.5 Statistical Analyses

All statistical analyses were completed utilizing the SPSSX package. Means(X), standard deviations(SD) and standard errors(SE) were calculated for all dependent variables. The

Pearson product moment coefficient of correlation (r) was used to describe relationships between dependent variables (Ary, 1985). To determine significant differences pre to post-test paired or dependent t-tests were utilized. Independent t-tests were run on data separated by group membership.

A Cluster Analysis was performed (Norusis, 1985), to determine the group membership of the 9 subjects with respect to the 3 catagories of menstrual function; AM, OM and EU. Athletes were provided with several copies of the MQ survey. Each subject was instructed to complete the questionnaire as accurately as possible at the onset of menses. If no menses occurred, the athlete checked off the box indicating no cycle and entered the date.

Menstrual activity recorded by the subjects produced raw data which described the number of sanitary devices required each day of the cycle and the estimated flow rate. The raw data were then used to generate numerous menstrual cycle variables (Figure 2), including; prior group assignment, number of complete cycles per study(cycles/study), date of cycles, length of cycles in days, total sanitary devices required per cycle(sum/p(1)), average sanitary devices required per day in each cycle, average subjective flow rate, and total number of sanitary devices required for the entire 9 week study period(total/p). The generated menstrual function variables were used in the Cluster Analysis to delineate group membership based on similar menstrual cycle characteristics.

The alpha level for significance was set prior to statistical interpretation as follows; $p>0.1$ no significance, $p<0.1$ questionable significance, $p<0.05$ a phenomenon and $p<0.01$ significant.

Figure 1. Experimental Protocol

TRAINING WEEK

0	1 (PHASE	2	3 I)	4 (PHASE	5	6 II)	7 (PHASE	8	9 III)
AP1	NUTR1	BL2	TV	PT2	BL3	NUTR2	PT3	BL4	AP2
HT1	TV	TV	MQ	TV	TV	TV	TV	NUTR3	HT2
WT1	MQ	MQ		MQ	MQ	MQ	MQ	TV	WT2
BF1								MQ	BF2
PT1									BL5
BL1									

VARIABLE LIST

AP-Aerobic Power($\dot{V}$02max)
HT-Height
WT-Weight
BF-Percent Body Fat
PT-Performance Test
BL-Blood Sample
NUTR-Nutrition Survey
TV-Training Volume Survey
MQ-Menstrual Quantity Survey

Table 1. Physical characteristics of the subjects on the pre-test.

Subject	Age (yrs)	Height (cm)	Weight (kg)	BF (%)	$\dot{V}O_2max$ (l.min-1)	$\dot{V}O_2max$ (ml.kg-1.min-1)
TJ	19	173.0	58.5	16.8	3.5	59.5
CM	29	167.0	56.5	14.0	3.2	56.8
PK	21	171.6	55.8	17.1	3.7	66.4
SS	20	160.7	50.2	21.5	2.5	48.8
CL	24	165.3	53.0	19.8	2.7	50.6
UM	30	165.0	52.0	23.6	3.4	64.5
TF	24	174.6	62.7	18.8	3.8	60.9
SB	25	165.8	57.3	22.0	3.2	56.6
LJ	23	167.6	53.1	21.1	3.2	59.4
Mean	23	167.8	55.4	19.4	3.2	58.2
SD	3	4.4	3.8	3.0	0.4	5.7

Chapter III

RESULTS

The results of the 9 week training program regarding $\dot{V}O_2max$, PT, dietary intake, hematological profile and menstrual status of the 9 elite female runners is described by research question. Reference is made to the sub-section assigned to each research question in the Introduction.

Physiological changes pre to post study see 1.1.

The physical characteristics of the subjects on the pre test are outlined in Table 1. Mean anthropometric measurements of HT, WT and BF did not differ significantly pre to post study.

Mean absolute $\dot{V}O_2max$ increased 5.4% from test1 to test2, ($p<0.01$) as did mean relative $\dot{V}O_2max$ which improved 7.0%($p<0.005$) see Table 2.

Mean PT times were not significantly faster pre to post study (Figure 4).

Mean changes in TRIMP scores over the study, see 1.2.

The average number of TRIMP units performed by the subjects over the entire study was 2279±590, while the highest average week was week 4 which scored 277±85 TRIMPS. The lowest average week was week 9 which scored 224+80 TRIMPS. There was

considerable variation between subjects, however no significant differences were noted between TRIMP units performed on a weekly basis.

Description of the three (3) catagories of menstrual function, see 1.3.

The Cluster Analysis identified 3 main groups based on the data obtained regarding: number of cycles per study (cycles/study); total number of sanitary devices required in each cycle(sum/p(1)); the average number of sanitary devices required in each cycle(X/p(1)); and the total number of sanitary devices needed for the entire duration of the study(total/p). The analysis isolated an amenorrheic group(AM, n=2), cases PK and SB; an oligomenorrheic group(OM, n=2), cases TJ and UM; and a eumenorrheic group(EU, n=5), cases CM, SS, CL, TF and LJ. Figure 2 illustrates the menstrual quantity data, and the physical characteristics of the subjects by group membership are shown in Table 5.

The relationship between training volume (TRIMPS) and the athlete's hematological profile, see 1.4.

No discernible pattern developed and no significant relationships occurred between the hematological parameters of HG, HCT, SI, IBC, %SAT or FER with the number of TRIMP units performed on a daily or weekly basis.

It was noted that the mean FER level dropped pre to post-study (Figure 3). FER1 was significantly different from both FER2 ($p<0.01$) and FER3 ($p<0.05$).

The relationship between the fitness variables ($\dot{V}O_2max$ and PT) and the hematological profile, see 1.5.

There were relationships between $\dot{V}O_2max$ on test1 (VO_2ABS1 and $\dot{V}O_2REL1$), $\dot{V}O_2max$ on test2 ($\dot{V}O_2ABS2$ and $\dot{V}O_2REL2$) and the HG and HCT levels determined by blood samples drawn in closest time proximity to those $\dot{V}O_2max$ assessments (Table 4). Correlations ($r=.64$, $p<0.05$ and $r=.69$, $p<0.05$) existed between $\dot{V}O_2max$ on test1 and HG and HCT respectively; while strong relationships ($r=.92$, $p<0.01$ and $r=.90$, $p<0.01$) existed between $\dot{V}O_2max$ on test2 with respect to HG and HCT levels. It is apparent from Table 4, that relative $\dot{V}O_2max$ correlated higher in both situations than did absolute $\dot{V}O_2max$.

No relationships existed between any of the 3 PT and the hematological variables HG, HCT, SI, IBC %SAT or FER.

Both absolute $\dot{V}O_2max$(l.min-1) and relative $\dot{V}O_2max$(ml.kg-1.min-1) were negatively correlated to PT time in s. A correlation matrix (Table 3) demonstrates the $\dot{V}O_2max$ and its ability to predict the outcome of the PT.

The relationship between PT2 and PT3 and training volume (TRIMPS), see 1.6.

Negative correlations ($r=-.45$, $p<0.11$ and $r=-.58$, $p<0.07$) existed between the number of TRIMP units performed prior to PT2 and PT3 (ie. PTRIMPa and PTRIMPb), and the actual finishing time of the 1200 metre performance test.

The relationship between the hematological profile and dietary intake, see 1.7.

The only consistent positive correlations occurred between KCAL consumed and the HG and SI values which were obtained in closest time proximity to the dietary survey (Figure 1).

KCAL2 correlated with HG3 ($r=.81$, $p<0.01$) and SI3 ($r=.49$, $p<0.09$); while KCAL3 correlated with HG4 ($r=.59$, $p<0.05$) and SI4 ($r=.76$, $p<0.01$).

The athletes obtained more iron from supplements than they did from natural food sources: NUTR1 ($p<0.06$); NUTR2 ($p<0.06$) and NUTR3 ($p<0.03$).

When dietary variables were split by group membership, the AM group was found to have consumed fewer mean KCALS as compared to the OM or EU groups (Table 9). Mean nutritional analyses of the subjects by group membership is presented in Table 9.

The association between menstrual status and the hematological profile, see 1.8.

The small size of the 3 sub-groups would not allow for independent t-tests to be run successfully, however means and standard errors (SE) were calculated.

The OM group recorded the highest mean FER levels over the 3 suitable BL sessions (Table 8), and the AM group recorded higher mean values for HG, HCT, SI, IBC, %SAT and FER over the 4 serial BL sessions, when compared to the EU group.

Independent t-tests were performed on a collapsed data set, which incorporated a menstrual dysfunction(MD, n=4) group cases TJ, UM, SB and PK and the EU group. The combined group with menstrual irregularities had significantly higher FER levels over the 3 BL sessions: FER1($p<0.03$); FER2($p<0.07$) and FER3($p<0.01$).

The association between menstrual status and the fitness variables ($\dot{V}O_2max$ and PT), see 1.9.

The physical characteristics of the subjects by group membership are shown in Table 5. Again independent t-tests could not be performed on the small sub-groups, however means and SE were calculated.

The OM group ranked higher than either the AM or EU groups with respect to mean $\dot{V}O_2max$ scores on pre and post study assessment, while the AM group was higher on pre and post test $\dot{V}O_2max$ scores when compared to the EU group (Table 6).

The OM group registered faster mean PT times than the AM or the EU groups.

The AM group performed more TRIMP units prior to each BL session (ie BTRIMPa, BTRIMPb and BTRIMPc) as compared to the OM or EU groups (Table 7).

Table 2. Mean change in $\dot{V}O_2max$ from pre to post test.

Subject	$\dot{V}O_2max$			
	Pre test (l.min-1)	Post test	Pre test (ml. kg-1. min-1)	Post test
TJ	3.5	3.7	59.5	64.8
CM	3.2	3.3	56.8	57.8
PK	3.7	3.8	66.4	67.8
SS	2.5	2.8	48.8	57.5
CL	2.7		50.6	
UM	3.4	3.7	64.5	72.8
TF	3.8	4.2	60.9	66.0
SB	3.2	3.3	56.6	58.8
LJ	3.2	3.2	59.4	63.2
Mean	3.24a	3.5a	58.2b	63.6b
SD	0.43	0.43	5.7	5.4

Note: Paired letters denote significance at $p<0.01$ level.

Table 3. Correlation matrix of $\dot{V}O_2max$ and the three 1200m performance tests.

	PT1	PT2	PT3
$\dot{V}O_2ABS1$	-.63*	-.53+	-.81**
$\dot{V}O_2REL1$	-.69*	-.72*	-.85**
$\dot{V}O_2ABS2$	-.62*	-.48+	-.69*
$\dot{V}O_2REL2$	-.78*	-.92**	-.76*

Note: + significant at $p<0.1$ level.
* significant at $p<0.05$ level.
** significant at $p<0.01$ level.

Table 4. Correlation matrix of $\dot{V}O_2max$ and the hematological variables hemoglobin (HG) and hematocrit (HCT).

	HG1	HG4	HCT1	HCT4
$\dot{V}O_2ABS$	.53+	.89**	.60*	.84**
$\dot{V}O_2REL$	.64*	.69*	.92**	.90**

Note: + significant at $p<0.1$ level.
* significant at $p<0.05$ level.
** significant at $p<0.01$ level.

Table 5. Physical characteristics of the subjects by group membership.

	GROUP MEMBERSHIP		
	Amenorrheic (n=2)	Oligomenorrheic (n=2)	Eumenorrheic (n=5)
AGE(yr)	23.0 2.8	24.5 7.7	24.0 3.2
WT1(kg)	56.5 1.0	55.2 4.5	55.1 4.7
BF1(%)	19.5 3.4	20.2 4.8	19.0 3.0
WT2(kg)	56.7 .8	54.6 4.5	55.3 5.9
BF2(%)	18.7 2.4	21.4 1.6	19.6 2.5

Values are means +-SE.

Table 6. $\dot{V}O_2$max and performance test results by group membership.

	GROUP MEMBERSHIP		
	Amenorrheic (n=2)	Oligomenorrheic (n=2)	Eumenorrheic (n=5)
$\dot{V}O_2$ABS1	3.45	3.45	3.08
	.35	.07	.50
$\dot{V}O_2$REL1	61.5	62.0	55.3
	6.9	3.5	5.3
$\dot{V}O_2$ABS2	3.55	3.70	3.37
	.35	.01	.29
$\dot{V}O_2$REL2	63.3	68.8	61.1
	6.3	5.6	4.1
PT1(sec)	247.9	227.8	247.4
	0.8	29.8	17.9
PT2(sec)	240.8	222.2	249.1
	7.1	13.7	9.8
PT3(sec)	240.0	230.6	243.9
	10.2	0.9	12.6

Values are means +-SE.

Table 7. Mean trimp scores generated by subjects before blood test 1 through blood test 3, separated by group membership.

	GROUP MEMBERSHIP		
	Amenorrheic (n=2)	Oligomenorrheic (n=2)	Eumenorrheic (n=5)
BTRIMPa	591.8	568.1	441.9
	36.6	294.3	173.0
BTRIMPb	895.3	813.7	785.4
	48.3	378.3	174.0
BTRIMPc	862.3	859.1	635.3
	88.8	376.0	150.0

Values are means +-SE.

Table 8. Mean serum ferritin values on blood tests one through three, separated by group membership.

	GROUP MEMBERSHIP			
	AM (n=2)	OM (n=2)	MD (n=4)	EU (n=5)
FER1	91.0	108.0	99.5*	59.0
	11.0	23.0	11.5	6.7
FER2	86.5	90.5	88.5+	46.8
	18.5	33.5	15.6	6.7
FER3	76.5	93.0	84.8**	44.4
	2.5	3.0	5.0	6.8

Serum Ferritin values are in ug.litre-1.
Values are means +-SE.

Note: + significant at $p<0.1$ level.
* significant at $p<0.05$ level.
** significant at $p<0.01$ level.

Table 9. Mean nutritional analyses of the subjects by group membership for the three dietary surveys.

	GROUP MEMBERSHIP		
	Amenorrheic (n=2)	Oligomenorrheic (n=2)	Eumenorrheic (n=5)
KCAL1	1695.0	2000.0	2212.0
	63.6	183.0	800.1
Fe/food(mg)	14.8	14.8	16.4
	1.7	1.2	6.7
%RNI	105.0	105.0	116.0
	12.0	9.1	48.0
KCAL2	1535.0	2465.0	2042.0
	43.2	368.6	587.3
Fe/food(mg)	19.3	15.1	14.2
	1.9	0.9	4.3
%RNI	138.0	108.0	101.0
	10.2	9.7	36.7
KCAL3	1790.0	1805.0	1980.0
	169.7	403.0	537.2
Fe/food(mg)	23.1	14.7	15.2
	9.9	1.0	3.4
%RNI	164.0	104.0	108.0
	70.7	8.5	24.6

Values are means+-SE.

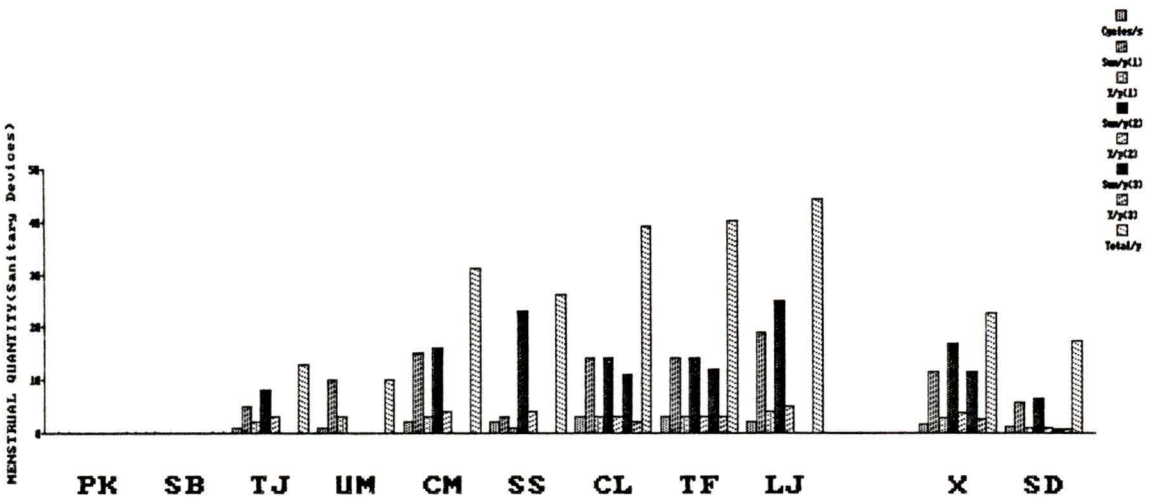

Figure 2. Menstrual Quantity

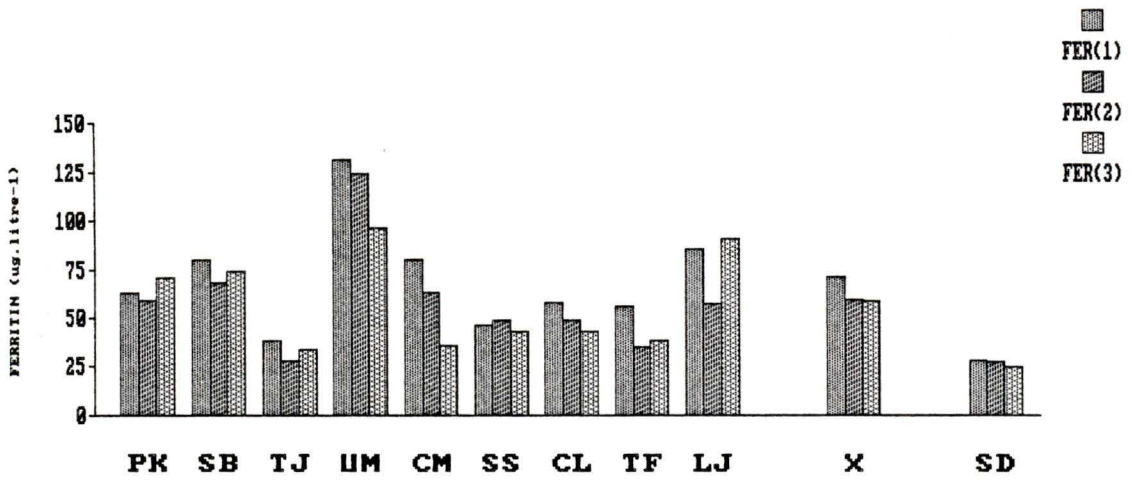

Figure 3. Mean Ferritin Levels

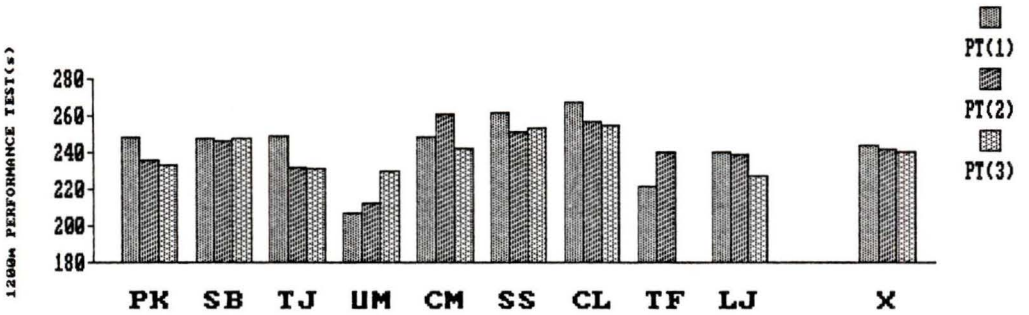

Figure 4. Performance Test Times

Chapter IV

DISCUSSION

Subjects completed all self-report inventories(TV, MQ and NUTR) on time. One injured subject could not complete final $\dot{V}O_2max$ testing, while another missed a PT with tendonitis; all other subjects completed the entire testing package.

Discussion of 1.1.

Athletes began the 9 week study after a rest period, which followed the outdoor track and field season. All athletes showed significant individual improvement in cardiovascular fitness (Table 2). Mean absolute $\dot{V}O_2max$ increased 5.4% ($p<0.01$) while mean relative $\dot{V}O_2max$ increased 7.0% ($p<0.01$) pre to post study. The percentage improvement in $\dot{V}O_2max$ is directly related to the subjects initial level of fitness and the distance from a possible end point of improvement(Pollock, 1973). Moderately trained individuals have been shown to improve 4-14% in $\dot{V}O_2max$ over 10 weeks of training, and the total improvement in an active athlete is rarely over 20% (MacDougall et al., 1982; Shephard, 1978; Pollock, 1973).

Mean PT times did not decrease over the (3) 1200 metre runs, the mean decrease in time as a percent (0.76%) was not significant (Figure 4). Unlike the homogeneous trend of improvement in $\dot{V}O_2max$, there was great individual variation in the PT times.

Familiarization with the $\dot{V}O_2max$ and PT protocol may have accounted for some of the improvement pre to post study, while a slight decrease in body weight in some individuals contributed to the larger improvement in relative $\dot{V}O_2max$. Although Banister and Hamilton (1985) did not monitor physiological changes in $\dot{V}O_2max$ they did report the female subjects completed on average an excess of 200 TRIMP units per week; the athletes in this study averaged a maximum of 277±85 and a minimum of 224±80 TRIMPS per week with a 9 week study average of 2279±590 TRIMP units. It may be speculated that the residual training level of these athletes at the pre test was very close to the plateau of achievable improvement. Therefore, even with the introduction of a substantial training volume only small gains in $\dot{V}O_2max$ are possible as the athlete reaches the top of their genetic limit (Hughson, 1986; Brooks and Fahey, 1984; MacDougall et al., 1982).

The subjects did not differ significantly in HT, WT, or BF pre to post study. With elite, physically mature athletes, large fluctuations in these anthropometric data are rare (Brooks and Fahey, 1984; MacDougall et al., 1982).

Discussion of 1.2.

No significant differences were noted between TRIMP units performed on a weekly basis, suggesting that each training week was reasonably consistent with respect to all other weeks throughout the study. These results were consistent with an aerobic build-up phase which entails completing high training volumes.

Individual raw scores were heterogeneous, with some athletes performing close to 400 TRIMP units per week while others with injuries struggled to complete 150 TRIMPS. The variation in these results can be explained by the variation in the subject pool which was outlined in the Methods section. In general the International calibre athletes preferred to complete higher training volumes.

Discussion of 1.3.

Researchers debate the best way to quantify menstrual blood losses (Marion et al., 1986; Levin and Wagner, 1986; Shaw, 1973). Hallberg and Nillson's Alkaline Hematin biochemical technique is the clinical standard, but the system is costly and labor intensive (Marion et al., 1986). This study utilized a menstrual survey (Appendix B), which asked subjects to report the number of sanitary devices required for each day of their menstrual cycle, along with a corresponding subjective flow rating. This format was undertaken with full knowledge of the previously described

problems with self-reporting, but due to the delicate nature of the other methods the subjects opted for a questionnaire (Marion et al., 1986; Shaw, 1973).

One indication for the quantification of menstrual blood flow is iron deficiency anemia (Shaw, 1973; Beaton et al., 1970). Excessive menstruation may be a factor contributing to the iron deficiency syndrome in female athletes (Newhouse and Clement, 1988; Diehl et al., 1986; Valberg et al., 1976; Beaton et al., 1970). Combined with occult gastrointestinal bleeding, hemolysis, poor iron absorption and an inadequate diet, the female athlete is at high risk for iron deficiency (Kaiseraurer et al., 1989; Synder et al., 1989; Clark et al., 1988; Sullivan, 1986).

In the present study a Cluster Analysis was performed on the data obtained from the MQ, and 3 distinct sub-groups were formed. The groups were AM (n=2), OM (n=2) and the EU (n=5). Figure 2 illustrates the variables which were used to separate the subjects into the 3 groups of menstrual function. The high amplitude of menstrual activity is quite apparent in subjects CM, SS, CL, TF and LJ, while the lower amplitude of TJ and UM beside the absence of activity in PK and SB help to delineate the groups. For statistical comparison, a Menstrual Dysfunction (MD n=4) group was formed by collapsing the AM and OM groups. This MD group was used to test the hypotheses, separating normal cyclic (ie. EU) versus the athletes with some form of menstrual irregularity.

The physical characteristics of the subjects by group membership are outlined in Table 5. Groups were similar in WT and BF, but differed in the fitness variables of $\dot{V}O_2max$ and PT (Table 6). The AM and OM groups performed more TRIMP units prior to each BL session (Table 7), indicating the completion of more training volume than the EU group. While WT, BF and training volume have been suspected as precursors to athletic amenorrhea (Baker et al., 1981; Frische and Revelle, 1970), recent research still fails to describe the exact etiology of athletic amenorrhea but less credence is given to theories regarding BF (Prior, 1988; Cumming, 1988; Sanborn et al., 1987).

The sub-groups did differ in mean FER levels (Table 8), with the OM group having a higher storage level of iron over the AM and finally the EU group. The mean nutritional analyses of the groups is presented in Table 9. The AM group consumed fewer KCALS than the EU and OM groups. These results are in agreement with others reported in the literature (Kaiseraurer et al., 1989; Clark et al., 1988; Deuster et al., 1986; Clement and Asmundson, 1982). While the athletes in this study consumed more iron from supplements, they did achieve in excess of 100% of the RNI for iron on the 3 NUTR surveys.

Discussion of 1.4.

Few authors have attempted to predict the effects of distance running training on select hematological variables. Banister and Hamilton (1985) recently quantified training volume

into TRIMPS and showed it affected SI and %SAT during training, while FER varied out of phase with training. The present study could not demonstrate any consistent correlations between mean weekly training in TRIMPS and any of the hematological variables. A small sample size (n=9) may have accounted for the multiple sporadic correlations which developed on these analyses. Other investigators have shown changes in the hematological variables with training, competition and/or point in the competitive season; however, most investigators have failed to specify the training volume making comparison to other literature difficult (Newhouse and Clement, 1988; Risser et al., 1988; Diehl et al., 1986; Pate et al., 1986; Brown et al., 1985; Dufaux et al., 1981; DeWijn et al., 1971). Mean FER levels did significantly deteriorate ($p<0.05$) over the 9 week training study (Figure 3) in agreement with Risser et al. (1988) and Diehl et al. (1986). This phenomenon is thought to be the iron cost associated with training and/or competition. However, neither mean FER level nor any other hematological variable correlated with the number of TRIMPS performed. Subjects in this study trained at a minimum 224 ± 80 TRIMPS per week, reaching a maximum 277 ± 85 TRIMPS in week four. No significant differences were noted between TRIMP1 through TRIMP9. These results suggest the training volume each week centred around the same approximate TRIMP score, thus indicating a consistent, cumulative training stress which helped produce the changes in $\dot{V}O_2max$.

Discussion of 1.5.

Iron deficiency has been implicated in lower $\dot{V}O_2max$ values, reduced endurance capacity, and higher exercising levels of lactic acid (Newhouse and Clement, 1988; Schwarzkopf et al., 1986; Schoene et al., 1983; Finch et al., 1979).

HG and HCT levels were significant predictors of $\dot{V}O_2max$ on both the pre and post study assessments (Table 4). The strong positive correlations indicate an increased level of HG or HCT may be related to performance on the $\dot{V}O_2max$ test. These findings are supported by animal research (Davies et al., 1982) which demonstrated iron deficient rats with sub-optimal HG had a 48% lower $\dot{V}O_2max$. Iron deficiency anemia, which is characterized by a marked drop in HG, will have an adverse effect on $\dot{V}O_2max$ and performance (Newhouse and Clement, 1988; Eichner, 1988; Clement and Asmundson, 1982; DeWijn et al., 1971). There is speculation if iron deficiency without anemia will cause a decrease in $\dot{V}O_2max$ and endurance performance. Celsing et al. (1986) used RBC transfusion to exclude the influence of low HG and HCT levels and showed no deleterious effect on $\dot{V}O_2max$ or performance. Our results could not identify any relationships between the select hematological variables and the PT, while $\dot{V}O_2max$ only correlated significantly with HG and HCT. However, these results are in agreement with current literature, which states iron deficiency anemia with a marked reduction in HG may affect $\dot{V}O_2max$ and

performance (Eichner, 1988; Celsing et al., 1986). Our subjects demonstrated a mean drop in FER (Figure 3) pre to post study, but this did not invoke a marked drop in any of the other hematological variables.

$\dot{V}O_2max$ was a reliable predictor of performance on a 1200 metre run (Table 3). Significant negative correlations occurred between absolute and relative $\dot{V}O_2max$ and the PT. These results are supported by animal research by Davies et al. (1984), Davies et al. (1982) and Henderson and co-workers (1986); and by Banister and Hamilton (1985) and Shephard (1978) in the human model. Therefore, maximal aerobic power may be used as a predictor of expected performance on an event related criterion test.

Discussion 1.6.

Negative correlations ($p<0.1$) occurred between the training volume (TRIMPS) performed by a subject and their corresponding PT. Increased levels of fitness are related to the duration, intensity, volume and mode of training performed (Hughson, 1986; MacDougall et al., 1982; Shephard, 1978; Pollock, 1973), however the present study could only demonstrate a weak relationship between performance and the TRIMPS completed. Perhaps the PT was not of the correct duration for the training phase, which was predominantly aerobic. The criterion test might have been better if set at 2000 metres.

Discussion of 1.7.

Suboptimal nutritional practices may limit performance through compromised iron storage. The physiologic parameters influencing iron uptake are dietary exposure, the quantity of storage iron present within the body and erythropoiesis (Kaiseraurer et al., 1989; Synder et al., 1989; Newhouse and Clement, 1988; Finch and Huebers, 1985; Clement and Sawchuck, 1984).

Subjects in this study completed 3 NUTR surveys (Appendix D) at planned intervals throughout the 3 training phases (Figure 1). Consistent positive correlations occurred between the total KCALS consumed and HG and SI values obtained in closest proximity to the NUTR. Many authors report inadequate energy consumption with concomitant low iron content (Synder et al., 1989; Risser et al., 1988; Clement and Asmundson, 1982). The athletes always consumed (Table 9) over 100% of the RNI for iron from natural food sources, excluding iron supplements. It was determined the athletes obtained significantly more iron from supplements.

Poor dietary and nutritional habits are indicated in iron deficiency anemia of elite female runners (Synder et al., 1989; Clark et al., 1988; Zierath, 1986; Clement and Asmundson, 1982). In female athletes too much iron may be utilized for red blood cell(RBC) production, enzymes and cytochromes; with a concomitant inadequate nutritional replacement allowing a negative iron balance to develop (Newhouse and Clement, 1988; Eichner, 1986; Clement and Sawchuck, 1984; Clement and Asmundson, 1982).

A positive HG response to iron supplementation is indicative of iron deficiency anemia (Newhouse and Clement, 1988); and the defects in VO2max are said to be primarily the result of diminished O2 delivery to the working tissues (Pate, 1986; Clement and Sawchuck, 1984; Guyton, 1981). Not all researchers are convinced that iron supplementation creates a HG response. Several studies have had little success demonstrating a hematological response to oral iron therapy (Synder et al., 1989; Newhouse, 1987; Schwarzkopf et al., 1986; Haymes et al., 1986; Banister and Hamilton, 1985; Brotherhood et al., 1975). Banister and Hamilton (1985) attribute the lack of response to oral iron therapy as a training increase in transferrin saturation.

Synder and co-workers (1989) express concern about oral iron supplementation, due to its possible effect on other trace mineral absorption rates. The contention is habitual or unguided self-prescription of iron supplements, during pre-latent or latent iron deficiency in a training athlete is unwarranted due to the possible elevated %SAT and that HG and HCT levels are unaffected until storage iron is so depleted that hemoglobinization and erythropoiesis can not occur (King, 1989; Banister and Hamilton, 1985; Clement and Sawchuck, 1984). At this point, when erythropoiesis is affected, oral iron therapy can be implemented. If a HG response is evoked, it may translate into an increased $\dot{V}$O2max.

Discussion 1.8.

Female endurance runners are at risk of a negative iron balance (Kaiseraurer et al., 1989; Newhouse and Clement, 1988; Ehn et al., 1980) and menstruation is the major avenue of iron loss (Sullivan, 1986; Marion et al., 1986; Diehl et al., 1986; Valberg et al., 1976; Beaton et al., 1970). Therefore, eliminating a major loss of iron (ie. menstruation) could have a positive effect on iron storage.

Unfortunately, independent t-tests could not be performed on the 3 sub-groups due to the small size. However, the AM and OM groups did consistently maintain higher levels on the hematological variables than did the EU group. The OM group demonstrated the highest (Table 8) mean FER levels. The MD group demonstrated significantly higher mean FER levels, which may suggest athletes with menstrual irregularities maintain higher iron storage. This data is contrary to Diehl et al.(1986) and Risser et al.(1989) who found no significant relationship between mentrual flow and serum ferritin level. However, these studies did not quantify menstrual flow or group subjects by menstrual function (ie. amenorrheic vs. eumenorrheic).

Athletic amenorrhea is prevalent in elite exercising females (Kaiseraurer et al., 1989; Prior, 1988; Williams, 1984; Baker, 1981). Cumming (1988) reports that 4% and 15% of exercising women are either amenorrheic and oligomenorrheic respectively. In this investigation 22% of the subjects experienced AM, 22% experienced OM and 56% were EU. Sanborn et al. (1982) found a sport specific trend to athletic amenorrhea. There was a 12% incidence in college swimmers and cyclists but a 43% incidence in females

running over 70 miles per week. While these training volumes were quantified only in miles, the athletes in this study also completed on average 50-60 miles of running per week. No clinical assessment was performed to determine whether the athletes in the menstrual dysfunction group had athletic amenorrhea as or a hormonal amenorrhea, however it may be surmised that some of the athletes were experiencing amenorrhea related to training stress.

Discussion of 1.9.

Results from this investigation showed the OM group ranked higher in both pre and post $\dot{V}O_2max$ scores (Table 6), and in PT than the AM and the EU groups. Accordingly, the MD group demonstrated higher mean $\dot{V}O_2max$ scores and faster PT times as compared to the regularly menstruating group.

The MD group performed more TRIMPS per week, and more TRIMPS prior to each BL session (Table 7). It should be noted that no one group, MD or EU, contained the more talented, intensely training athletes. For example each group contained 1 athlete of International calibre.

The higher iron storage in the MD group may have accounted for the higher $\dot{V}O_2max$. Suboptimal HG and HCT levels limit the ability of the central system to transport adequate O2 to the tissues. Increased iron demand coupled with inadequate iron replacement may induce movement from the pre-latent to the latent stage of iron deficiency. Further iron demand with concomitant

depleted iron storage forces available iron to be used solely in erythropoiesis. This leaves little iron for myoglobin, enzymes and/or mitochondrial cytochromes.

When erythropoiesis becomes affected by inadequate iron supply, full "blown" iron deficiency anemia can result. The combined effects are diminished $\dot{V}O_2max$ with accompanying anaerobic metabolism at the periphery. The resulting increased lactic acid followed by a drop in pH can explain local muscular fatigue (Hughson, 1986; Brooks and Fahey, 1984; Shephard, 1978; Wenger and Reed, 1976).

Athletic amenorrhea has been related to insufficient dietary intake (Kaiseraurer et al., 1989; Synder et al., 1989; Prior, 1988; Clark et al., 1988; Sanborn et al., 1987; Zierath, 1986). However, a poor diet and high energy drain are also contributing factors to pre-latent, and full blown iron deficiency anemia (Newhouse and Clement, 1988; Sullivan, 1986; Clement and Sawchuck, 1984; Clement and Asmundson, 1982; Valberg et al., 1976). Contradictory evidence does exist, but in the present study the OM and AM groups had higher hematological variables and higher physiological variables than the EU group.

This study may guide Physicians debating oral iron therapy for elite endurance athletes. The present research supports the view that marked changes in HG and HCT, as seen in iron deficiency anemia, may be associated with the performance of a VO2max test. No evidence was obtained to suggest that iron deficiency without anemia caused decreases in $\dot{V}O_2max$ or PT.

There remains considerable debate whether iron deficiency without anemia effects endurance performance (Newhouse and Clement, 1988; Celsing et al., 1986; Schoene et al., 1983). A HG response of 10.0 g.litre-1 with oral iron therapy in an athlete complaining of impaired endurance performance is indicative of an iron deficiency (Risser et al., 1988; Newhouse and Clement, 1988). In clinical practice an oral iron regimen would range from 100 to 300 mg of elemental iron daily for 12 to 24 weeks (Newhouse and Clement, 1988; Risser et al., 1988; Schwarzkopf et al., 1986; Clement and Sawchuck, 1984). The therapeutic value of oral iron therapy during the pre-latent and latent stages of iron deficiency, in order to "top-up" iron reserves, could be questioned. With high volumes of training there appears to be a marked decrease in iron absorption due to increased transferrin saturation and malabsorption at the gut (Newhouse and Clement, 1988; Banister and Hamilton, 1985). Indiscriminate large doses of iron supplements may result in inhibition and/or suppression of other trace minerals also important for performance. Since erythropoiesis remains unaffected until iron deficiency anemia occurs, the Physician may watch for a HG response to further justify oral iron therapy.

Unguided self-prescription of oral iron supplements by athletes is not recommended due to the remote potential for iron overload (hemochromatosis).

In summary, this investigation demonstrated $\dot{V}O_2max$ was related to HG and HCT levels and was negatively correlated with PT. Mean FER levels were higher in MD group as compared to EU

counterparts. Athletes within the collapsed MD group out performed their regularly menstruating EU colleagues with respect to $\dot{V}O_2max$ and PT. Perhaps there is a relationship between menstrual dysfunction and the hematological indices of the elite female runner.

REVIEW OF LITERATURE

Energy Systems

The ability of the energy production systems to meet the demands of energy consumption determine the outcome of endurance, power, speed and strength. Energy production combines the effects of the cardiovascular(CV), endocrine, respiratory, and nervous systems (Brooks and Fahey, 1984; MacDougall et al., 1982).

Shephard (1978) describes brief events requiring 10-60 seconds of effort, as predominantly fueled by phosphogens (alactic), and the anaerobic breakdown of glycogen to lactic acid (lactic). An accompanying increased acidity inhibits the enzymes phosphorylase and phosphofructokinase (PFK), which are required to metabolize glycogen, and fructose 6-phosphate respectively (Brooks and Fahey, 1984; MacDougall et al., 1982; Shephard, 1978). Brief bouts of exercise are limited by the capacity of the alactic supply of phosphogens (ie. creatine phosphate), and by the bicarbonate buffering capacity of the lactic system, which must contend with an exercise induced acidosis (Shephard, 1978).

Sustained events 60 seconds or longer are characterized by aerobic energy production (Brooks and Fahey, 1984; MacDougall et al., 1982; Shephard, 1978). Aerobic power ($\dot{V}O_2max$) is the ability to transport oxygen (O2) from the atmosphere to the working tissues (Hughson, 1986; Brooks and Fahey, 1984; MacDougall et al.,1982; Shephard, 1978).

$\dot{V}O_2max$ appears to be limited by cardiac output (Q), but the ability to sustain high levels of energy expenditure during competitive sport may be dependent on muscle metabolism (Hughson, 1986). Hughson (1986) reports the mechanism for the increase in endurance performance is the increase in aerobic metabolic pathways, specifically higher concentrations of succinate dehydrogenase (SDH). Increased enzymes of the oxidative metabolic pathways allow free fatty acid utilization and tend to shuttle glycogen to the end point of oxidation, diverting lactic acid production, thereby delaying the onset of acidosis which is suspected in fatigue (Hughson, 1986; Brooks and Fahey, 1984; Shephard, 1978; Wenger and Reed, 1976).

Normal physiology is governed by the principles described previously, however pathologic physiology may appear quite different, as in the case of an iron deficient athlete.

$\dot{V}O_2max$ is dependent upon the CV system, in that $\dot{V}O_2$ equals cardiac output multiplied by the arteriovenous oxygen difference, given by the equation $\dot{V}O_2 = Q \times (a\text{-}v)O_2$ difference. Cardiac output is responsible for providing movement of the labile pool of blood, which permeates working tissues with erythrocytes (RBC) containing a normal amount of hemoglobin (HG) in order that O_2 be delivered and carbon dioxide (CO_2) be removed (Hughson, 1986; Brooks and Fahey, 1984; Guyton, 1981). In the iron deficient athlete this hierarchical pattern is not maintained as depleted or reducing iron storage is believed to compromise $\dot{V}O_2max$ and

endurance performance (Newhouse and Clement, 1988; Diehl et al., 1986; Celsing et al., 1986; Clement and Sawchuck, 1984; Clement and Asmundson, 1982; Ehn et al., 1980; DeWijn et al., 1971).

Iron is stored as ferritin (FER) and hemosiderin in the liver, spleen and bone marrow (Guyton, 1981). Its main biological function is the formation of HG, myoglobin, iron containing enzymes (catalase, peroxidase), cytochromes a, b and c of the electron transport chain, nicotinamide adenine dinucleotide (NAD) and SDH (Finch and Huebers, 1986; Creager, 1983; Guyton, 1981; Brunner and Suddarth, 1975).

Poor iron storage is a precursor to exercise induced lactic acidosis as pyruvic acid is preferentially metabolized by lactate dehydrogenase (LDH) in the absence of adequate O2 (Newhouse and Clement, 1988; Finch and Huebers, 1986; Clement and Sawchuck, 1984; Pate, 1986). Anaerobic metabolism is limited as discussed previously, therefore, endurance capacity in the iron deficient athlete may be reduced due to the preferential anaerobiosis brought about by low iron storage (Newhouse and Clement, 1988; Hughson, 1986; Clement and Sawchuck, 1984; Pate, 1983).

Muscle Metabolism

Peripheral muscle fatigue can be due specifically to depletion of key metabolites or to the accumulation of other metabolites, which may effect the intracellular environment (Brooks and Fahey, 1984; Guyton, 1981; Shephard, 1978).

Peripheral muscle fatigue may be associated with high levels of lactic acid (Hughson, 1986; Brooks and Fahey, 1984; Shoene et al., 1983; Finch et al., 1979; Wenger and Reed, 1976).

Lactic acid dissociates to lactate and H+ ion. It is the H+ ion which causes pH to decrease in the muscle (Brooks and Fahey, 1984). A decrease in muscle pH impairs both energy production and force generation resulting in fatigue (Brooks and Fahey, 1984; Creager, 1983; Guyton, 1981). An increased concentration of H+ ion reduces energy production by the inhibition of phosphorylase and PFK activity in glycolysis and by decreasing the mobilization of free fatty acids (Wenger and Reed, 1976). Muscle force generation is affected when H+ ions compete with calcium for binding sites on troponin (Brooks and Fahey, 1984; Creager, 1983; Guyton, 1981). H+ ion will inhibit the combination of O2 with HG in the lungs, decreasing the amount of O2 available at the tissues (Brooks and Fahey, 1984; Creager, 1983).

When O2 delivery to a muscle is insufficient to produce the required energy for contraction, the muscle is forced to anaerobically metabolize the available substrates. Pyruvic acid is driven away from the tricarboxylic acid cycle (TCA) and catalyzed by LDH. The end point of this reaction is lactate and H+ ion (Brooks and Fahey, 1984; Creager, 1983; Guyton, 1981; Wenger and Reed, 1976).

Therefore, limited energy production, impaired muscle force generation, increased H+ ion concentration and decreased O2 delivery will negatively effect muscle performance.

Oxygen Transport

O2 is essential for aerobic energy production (Hughson, 1986; Shephard, 1978; Wenger and Reed; 1976). Maximum aerobic power is the highest rate at which energy can be released from the oxidative process exclusively (MacDougall et al., 1982). This rate is dependent upon two components, the peripheral and the central.

The peripheral component is the chemical ability of the tissues to utilize O2 in metabolizing fuels for energy (MacDougall et al., 1982). The central component requires the pulmonary, cardiac, blood, vascular and cellular mechanisms to transport O2 to the aerobic machinery of the muscle (MacDougall et al., 1982).

In the absence of O2, cellular respiration must be anaerobic. Anaerobic energy capacity is limited (Shephard, 1978). Anaerobic energy pathways generate lactic acid which negatively affects muscle performance (MacDougall et al., 1982; Wenger and Reed, 1976). Lactic acid has been shown to accumulate in the blood of exercising athletes at work intensities eliciting less than maximal O2 uptake (MacDougall et al., 1982). This is the result of an imbalance between lactic acid produced by muscle fibres contracting anaerobically, and the ability of the liver and kidney tissue to perform gluconeogenesis, converting excess lactate and pyruvate into glucose via the Corri Cycle (Brooks and Fahey, 1984; Guyton, 1981).

The aerobic system generates over 90% of the energy needed for endurance performance (Brooks and Fahey, 1984; MacDougall et al., 1982). The availability of O2 to exercising tissues is paramount to endurance performance. Transport of O2 is the responsibility of the CV system, which incorporates cardiac output($\dot{Q}$=SVxHR), peripheral resistance, (a-v)O2 difference, bicarbonate(HCO3-) buffering capacity, and HG concentration (Brooks and Fahey, 1984; Creager, 1983; Guyton, 1981).

Blood

Human blood is classified as a tissue composed of fluid(plasma), in which are suspended red (RBC) and white blood cells, platelets, fat globules and a great variety of chemical substances including carbohydrates, proteins, hormones and gases such as O2, CO2 and nitrogen (Brunner and Suddarth, 1975). The function of blood is the nutrition and respiration of tissues that are located far from food and air supplies; transportation of waste to excretory organs; chemical and thermal regulation and co-ordination of the body (Guyton, 1981; Brunner and Suddarth, 1975).

The free flowing erythrocytes (RBC) are the vehicles which transport O2 to the working tissues in combination with the iron containing pigment HG (Guyton, 1981). The RBC is both targeted for O2 delivery and CO2 removal, which ultimately takes place in the lungs. Normal HG can bind 1.34 ml of oxygen per gram of hemoglobin (Brooks and Fahey, 1984). HG has a great affinity for

O2 and CO2. Once in combination with these gases they form an oxyhemoglobin or carbaminohemoglobin complex (Brooks and Fahey, 1984; Creager, 1983; Guyton, 1981).

The Oxygen-Dissociation curve demonstrates the relationship between the partial pressure of O2(pO2) and the percentage saturation of HG, whereby increased temperature and decreased pH favor a shift to the right, liberating more O2 at the exercising tissues at the same given pO2 (Brooks and Fahey, 1984; Creager, 1983; Guyton, 1981; Brunner and Suddarth, 1975). In the exercising tissues where O2 tension is low and CO2 tension is high , oxyhemoglobin liberates its O2 in exchange for CO2. An intregal part in these reactions is 2, 3-diphosphoglycerate(2, 3-DPG), which in high concentrations also favors a shift to the right of the Oxygen-Dissociation curve (Brooks and Fahey, 1984; Guyton, 1981).

Physiology of the RBC

The RBC has a mean lifespan of 90 to 120 days (Guyton, 1981). After this time the constituent parts of the erythrocyte are reclaimed for use in new erythrocytes. A pathologic condition, hemolysis, can destroy a RBC prematurely. The RBC stroma is ruptured or dissolved due to heat, friction, physiologic stress or toxins, and the HG is liberated into the plasma. As a result, the body is unable to retain the HG, which is lost through the kidneys (hemoglobinuria) or feces (Eichner, 1986; Sullivan, 1986; Clement and Sawchuck; 1984).

New RBCs require differentiation of multipotential stem cells of the bone marrow, which is regulated by the hormone erythropoietin (Brunner and Suddarth, 1975). Erythropoiesis, the formation of new RBCs, requires several nutrients, including vitamin B12, folic acid, pyridoxine and iron, which is required for hemoglobin formation (Eichner, 1986; Valberg et al., 1976).

Low iron storage, coupled with inadequate iron absorption produces microcytic, faulty, immature RBCs which subsequently contain less than the normal complement of HG (Eichner, 1986; Finch and Huebers, 1986; Pate, 1983; Clement and Asmundson, 1982). If HG concentration is low, there are marginal effects on sedentary activity, but quite profound effects on certain types of athletic performance (Clement and Sawchuck, 1984).

Iron

Iron is stored in two compounds: ferritin and hemosiderin (Bauer, 1974). Iron is absorbed in the upper duodenum, where it is complexed with mucosal transferrin in the lumen. Finch and Huebers (1986) postulate that membrane uptake is the most potent regulator of iron absorption. Due to the tendency for iron to precipitate in solution, it must be held in a 24 polypeptide chain storage molecule called ferritin (Finch and Huebers, 1986). The smooth endoplasmic reticulum continually produces ferritin molecules to keep pace with loss. It should be noted that plasma ferritin differs from intracellular ferritin, as plasma ferritin contains virtually no iron (Finch and Huebers, 1986). Yet it is

the plasma ferritin which has clinically demonstrated a high correlation with total body iron storage (Byryne et al., 1981; Tilkian and Conover, 1975; Bauer, 1974).

The value of ferritin as an indicator of iron storage is well established from numerous studies (Finch and Huebers, 1986; Clement and Sawchuck, 1984; Valberg et al., 1976).

Sixty to seventy percent of total body iron is found in HG and/or myoglobin, the remaining 30-40% forms the labile pool of iron storage located in the reticuloendothelial cells of the bone marrow, liver and spleen (Guyton, 1981). Mean HG levels are 110-160 g.litre-1 in females and 130-180 g.litre-1 in males (Bryne et al., 1981). Asymptomatic ferritin levels determined by IRMA, are proposed to be 20-160 ug.litre-1 (Clement and Sawchuck, 1984).

FER levels have been shown to accurately reflect the size of total body iron reserves in female athletes (Newhouse and Clement, 1988; Newman, 1986; Schwarzkopf et al., 1986; Brown et al., 1985; Clement and Sawchuck, 1984; Schoene et al., 1983; Clement and Asmundson, 1982; Valberg et al., 1976).

Other valuable laboratory tests, for the determination of an athletes iron profile include; HCT, SI, IBC and %SAT.

The HCT is expressed as the percentage of packed erythrocytes and formed elements of a given volume of blood (Byrne et al., 1981). Normal values are 0.40-0.54 for males and 0.37-0.47 for females. SI measures the plasma iron bound to the protein transferrin. SI is noted to have a diurnal variation which warrants consistent testing protocols and sampling times in

order to assure meaningful results (Bauer, 1974). Normal physiologic ranges are 15-42 umoles.litre-1 in males and 12-31 umoles.litre-1 in females (Bryne et al., 1981). IBC measures the maximal saturation of free transferrin with iron (Bauer, 1974). An elevation in IBC is often associated with iron deficiency anemia. Normal physiologic ranges are 48-90 umoles.litre-1 for males and 45-73 for females (Bryne et al., 1981). %SAT describes the proportion of the iron binding protein which is saturated with iron. When the degree of saturation is low, iron is mainly utilized for hemoglobinization, while a high %SAT reading indicates a shunting toward storage (Bauer, 1974). Normal range is reported to be 0.20-0.55 (Bryne et al., 1981).

Iron Deficiency Syndrome in the Animal Model

Experimental animal research indicates that iron deficient rats differ as compared to iron sufficient rats in HG content, HCT, $\dot{V}O_2max$, workload achieved at $\dot{V}O_2max$, endurance capacity and preferential use of glucose as a metabolic fuel (Henderson et al., 1986; Davies et al., 1984; Davies et al., 1982).

Davies and co-workers (1984) surmise that the defects in $\dot{V}O_2max$ during iron deficiency are primarily the result of diminished O2 delivery; and decreased endurance capacity reflects impaired muscle mitochondrial function. In Davies et al. (1982) iron deficient rats had a 48% lower $\dot{V}O_2max$, a 93% lower endurance capacity and a significantly higher respiratory quotient(RQ) at any given workload. Blood lactate levels were higher both at rest and during exercise in the same iron deficient animals.

Henderson et al. (1986) note that when peripheral mitochondrial O2 utilization is limited, as in anemia, iron deficient animals will preferentially metabolize glucose and glycogen in an attempt to maximize ATP yield per mole of O2. The biochemical consequence of this pathway is excess pyruvic acid being shuttled to lactic acid.

Iron Deficiency in the Human Model

Healthy hematological adaptations to training are noted as expanded plasma volume, decreased HG and HCT, increased RBC mass and deformability and finally, decreased blood viscosity (Eichner, 1988; Newhouse and Clement, 1988; Pate, 1983). This transient dilutional anemia is considered to be a pseduoanemia which enhances circulation of a less viscous blood to working muscles. The hemodilution effect appears to be an early adaptation to endurance exercise, and is not considered a true anemia because a shortage of iron is not limiting RBC production (Eichner, 1988; Newhouse and Clement, 1988).

True iron deficiency and frank anemia to the point of faulty erythropoiesis will hinder some types of athletic performance (Newhouse and Clement, 1988; Risser et al., 1988; Diehl et al., 1986; Clement and Sawchuck, 1984; Shoene et al., 1983; Clement and Asmundson, 1982; Horwill, 1981; Ehn et al., 1980; Finch et al., 1979; Edgerton et al., 1979).

Iron deficiency is more prevalent in certain athletic populations, such as endurance runners (Newhouse and Clement, 1988; Schwarzkopf et al., 1986; Sullivan, 1986; Pate et al.,

1986; Brown et al., 1985; Clement and Asmundson, 1982; Dufaux et al., 1981). Contrary evidence to this case is provided by Risser et al. (1988) and Brotherhood et al. (1975), who determined little difference between athletes and non-athletes with respect to iron storage.

Iron deficiency is observed most frequently in exercising females as compared to exercising males (Newhouse and Clement, 1988; Pate et al., 1986; Schwarzkopf et al., 1986; Brown et al., 1985; Clement and Sawchuck, 1984; Valberg et al., 1976; Beaton et al., 1970). This difference may be attributed to the increased obligatory loss of iron in females through menstruation (Sullivan, 1986; Shaw, 1973; Beaton et al., 1970).

Females have an increased iron requirement directly related to menses (Marion et al., 1986; Levin and Wagner, 1986; Valberg et al., 1976; Shaw, 1973; Beaton et al., 1970).

Diehl and co-workers (1986) produced the only research to date which has attempted to correlate numerous menstrual cycle variables with FER levels. In their experiment women field hockey players failed to show any relationship between FER and menstrual cycle aberrations.

In female athletes poor diet, increased iron loss and reduced iron absorption can directly effect iron reserves (Synder et al., 1989; Newhouse and Clement, 1988; Clark et al., 1988; Clement and Asmundson, 1982; Horwill, 1981). The bioavailability of iron is greatest in the heme form which is present in red animal meats (Finch and Huebers, 1986). Heme iron is required to synthesize HG, myoglobin, catalase, peroxidase and mitochondrial

cytochromes (Finch and Huebers, 1986). Non-heme iron compounds are required in the formation NADH, SDH and the cytochromes of the electron transport chain (Finch and Huebers, 1986).

As a group, female athletes tend to consume fewer average kilocalories, and generally consume a diet low in red meats (Synder et al., 1989; Clark et al., 1988; Clement and Sawchuck, 1984; Clement and Asmundson, 1982). It appears only 10% of ingested dietary iron actually gets absorbed across the lumen, and athletes lose more and absorb less iron, than non-exercising controls (Newhouse and Clement, 1988; Banister and Hamilton, 1985; Clement and Sawchuck, 1984; Ehn et al., 1980). An unpublished work by Clement et al. (1983), found a decreased rate of iron absorption in iron deficient exercising females, who absorbed only 29% as compared to iron deficient non-exercising females who absorbed 70% of the iron ingested. This 2:1 ratio of iron absorption, favoring non-active controls was demonstrated in an earlier work by Ehn et al. (1980), who utilized radioactive tagged iron. Banister and Hamilton (1985) speculated high fatigue levels elevated transferrin saturation, inhibiting iron uptake. Sullivan (1986) feels gastrointestinal distress and trauma associated with the pounding of training, are responsible for defects in the absorptive sites in the duodenum. As yet no clear definition exists to describe this problem, which appears to occur only in intensely training individuals.

Therefore, female athletes can expect to battle a negative iron balance due to training stress, sweat losses, urinary excretion (hemoglobinuria), gastrointestinal stress, menstrual

losses, hemolysis, a decreased iron absorption rate, poor dietary iron consumption and a training related elevation in transferrin saturation (Synder et al., 1989; Clark et al., 1988; Sullivan, 1986; Diehl et al., 1986; Valberg et al., 1976).

Pate et al. (1986) found female runners were significantly lower than a control group in HG, HCT, IBC and FER. Brown and co-workers (1985) determined a relationship which indicated that habitual female athletes had lower FER levels than did non-athletes, even though HG levels did not differ significantly.

Iron deficiency without anemia can reduce physical work capacity and lead to excess lactate production (Clement and Sawchuck, 1984; Finch et al., 1979; Edgerton et al., 1979). Decreased concentrations of myoglobin and iron containing enzymes, important for aerobic energy production, may account for reduced endurance capacity observed in iron deficient subjects with normal HG (Clement and Sawchuck, 1984). Shoene et al. (1983) examined 9 iron deficient female athletes after 2 weeks of iron repletion therapy, blood lactate levels at maximum exercise decreased significantly from 10+-0.6 mmol before therapy to 8.42+-0.7 after therapy.

Iron repletion appears to be highly individualized. Valberg et al. (1976) noted oral iron therapy needs to be continued for two months in order to replenish iron stores, even after HG levels have reached normal concentrations. Supplementation of ferrous salts lowers heart rate, blood lactate and increases oxidative capacity in the previously iron deficient female athlete (Schwarzkopf et al., 1986; Haymes et al., 1986; Clement

and Sawchuck, 1984). Unguided, self-prescription of supplemental iron is not recommended by physicians, as iron may irritate the gastrointestinal tract, overload iron storage (hemochromatosis) and/or may initiate other trace mineral deficiencies (Synder et al., 1989; Newhouse and Clement, 1988; Risser et al., 1988).

Menstruation doubles the average daily loss of iron (Newhouse and Clement, 1988; Diehl et al., 1986; Sullivan, 1986; Clement and Sawchuck, 1984; Valberg et al., 1976; Shaw, 1973; Beaton et al.,1970). Menstrual blood losses are most commonly cited as the reason female athletes encounter iron deficiency anemia. Therefore, of the factors mentioned previously, menstrual blood losses figure prominently in the equation which places female athletes in a negative iron balance.

Athletic Amenorrhea

The exact etiology of amenorrhea is suspect, however, the most noted symptom is the absence of the menstrual cycle. Amenorrhea would appear to limit the physiologic loss of iron by the cessation of menstrual blood loss. As yet no data exists to support or denounce the theory that amenorrhea is a conservation mechanism which limits iron losses by selectively stopping reproductive functioning during intense training, thereby preventing depletion of iron storage.

Athletic amenorrhea or secondary amenorrhea, the absence of menses in a women who had been previously menstruating, appears to be a common syndrome in todays competitive female athlete (Kaiseraurer et al., 1989; Prior, 1988; Cumming, 1988; Deuster et

al., 1986; Sanborn et al., 1987; Fisher et al., 1986; Nelson et al., 1986; Nielson and Fleck, 1985; Calberg, 1983; Baker, 1981; Dale et al., 1979).

Cumming (1988) reports 2% of normal reproducing females are amenorrheic, and 5% are oligomenorrheic; those figures jump to 4% and 15% respectively in athletic women. Amenorrhea appears to be most prevalent in women with; a young gynecological age, small stature, low body fat, high stress and abnormal dietary practices (Kaiseraurer et al., 1989; Cumming, 1988; Zierath, 1986; Deuster et al., 1986).

Frische and Revelle (1970) proposed a critical mass hypothesis, to explain athletic amenorrhea, whereby a certain amount of body fat was required for estrone conversion. Speroff (1981), Cann (1982) and Longscope (1978), dispute this claim, suggesting aromatization of androstenedione to estrone occurs by definition in adipose tissue, but not exclusively, since conversion can take place at an equal rate in muscle. Sanborn et al. (1987) determined no causal link between body fat percentage and the incidence of amenorrhea.

Instead evidence points to a multi focal cause, where several factors may combine to elicit the amenorrheic response. Baker (1981), found runners experiencing secondary amenorrhea had lower body weight for height prior to onset of training, greater weight loss with the onset of training and a lower percent body fat when compared to regularly menstruating runners. In her review Williams (1984) agrees that runners with amenorrhea have a significant and rapid weight loss at the onset of training.

Prolonged amenorrhea is linked to several hormonal aberrations which may produce deleterious side effects. Data suggests that exercise induced amenorrhea correlates with a central suppression, meaning low serum leutinizing hormone (LH), low follicle stimulating hormone (FSH) and low estrogen levels. These low levels reflect an alteration in the hypothalamic control of gonadotropin release (Fisher et al., 1986; Loucks and Horvath, 1985; Bitner, 1985). Low circulating levels of estrogens are implicated in accelerated osteoporosis associated with athletic amenorrhea (Nielson et al., 1986; Fisher et al., 1986; Jones et al., 1985; Rebar, 1982; Gonzlez, 1982; Speroff, 1981).

Fisher et al. (1986) determined bone mineral content of the lumbar spine was lower in amenorrheic women, and speculation has developed over the increased occurrence of stress fractures in amenorrheic athletes. Prior (1988) disputes these claims, stating the same compulsive behavior causes overtraining and that, coupled with inadequate dietary practices, are to blame for stress fractures.

Multiple related factors associated with high training loads appear to elicit athletic amenorrhea, however, a clearly defined etiology is not demonstrated at this time. As yet, no body of literature has examined in detail the effect of amenorrhea on iron deficiency in the female endurance athlete.

Several descriptive characteristics which predispose female athletes to exercise induced amenorrhea are: prior menstrual irregularity, sudden weight loss, low percent body fat, low energy intake, stress, volume, intensity and mode of training (Prior, 1988; Cumming, 1988; Fisher et al., 1986; Deuster et al., 1986; Bitner, 1985; Nielson and Fleck, 1985; Calberg, 1983; Wakat et al., 1982; Baker et al., 1981; Schwartz, 1981; Dale et al., 1979; Feicht et al., 1978).

Athletic amenorrhea appears to be more prevalent in certain athletic populations. Sanborn et al. (1982) found a 12% incidence of amenorrhea in college swimmers and cyclists but a 43% incidence in women running over 70 miles per week. In runners, training mileage, pace per mile and $\dot{V}O_2max$ are good predictors of the number of cycles an athlete will have (Gray and Dayle, 1983; Baker et al., 1981; Feicht et al., 1978).

Athletic amenorrhea is reversible with decreased activity, producing no long-term reproductive side effects (Cumming, 1988; Loucks and Horvath, 1985; Shangold, 1985; Bitner, 1985; Williams, 1984; Stager, 1984; Cumming and Rebar, 1983). Therefore exercise induced amenorrhea appears to be a transient syndrome which may be reversed with cessation of intense physical training.

Recent research by Kaiseraurer and associates (1989), found nutritional inadequacy, coupled with daily intense exercise and a potential energy deficit, could elicit athletic amenorrhea. Prior (1988) agrees, stating the compulsive behavior of amenorrheic athletes to pound out miles and consume fewer kilocalories, may be the underlying factor.

Summary

In conclusion, energy must be provided for muscular contraction by one or by a synergistic combination of the three energy systems (alactic, lactic or aerobic). Iron deficiency appears to limit oxidative energy production, forcing obligatory anaerobic energy production. This in turn, produces excess lactic acid which dissociates to lactate and H+ ion. The resulting decrease in pH may cause muscle dysfunction in the form of fatigue. Fatigue can limit endurance performance. These events could be different in a female athlete with exercise induced amenorrhea, as the cessation of menses may slow iron depletion during heavy training by the elimination of menstrual blood losses.

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APPENDIX A: INFORMED CONSENT

INFORMED CONSENT

CHANGES IN HEMATOLOGICAL, PHYSIOLOGICAL AND MENSTRUAL STATUS WITH TRAINING IN ELITE FEMALE MIDDLE DISTANCE RUNNERS

PROCEDURE

SUBJECTS WILL BE ASKED TO PERFORM (2) VO2MAX TESTS 9 WEEKS APART, ALSO (3) PERFORMANCE TEST RUNS OF 1200M WILL BE REQUIRED. ANTHROPOMETRIC MEASUREMENTS CONSISTING OF HEIGHT, WEIGHT AND BODY FAT PERCENTAGE WILL COINCIDE WITH THE VO2MAX TESTS.

BIOCHEMICAL ANALYSIS

BLOOD SAMPLES WILL BE OBTAINED BY VENUPUNCTURE UTILIZING A QUALIFIED PHYSICIAN OR NURSE FROM ISLAND MEDICAL LABORATORIES. THE PROCEDURE IS CONSIDERED SAFE WITH LITTLE RISK OF COMPLICATION ALTHOUGH WITH ANY LACERATION THERE IS MINIMAL RISK OF INFECTION. SOME MINOR BRUISING OR DISCOLOURATION MAY OCCUR AT THE SITE OF THE VENUPUNCTURE.

CONSENT

I HAVE READ THE ABOVE AND AGREE TO PARTICIPATE IN THIS RESEARCH PROJECT AT MY OWN RISK. I AM 19 YEARS OR OLDER AND REGULARY TAKE PART IN STRENUOUS ACTIVITY AT LEAST AS INTENSE AS THESE TESTS. I REALIZE I MAY EXPECT A THOROUGH EXPLANATION/DEMONSTRATION OF ANY PROCEDURES AND THAT I MAY TERMINATE PARTICIPATION AT ANY TIME IN ANY ORALL PROCEDURES OF MY OWN VOLITION. HAVING VOLUNTARILY ASSUMED PARTICIPATION AND THE RISKS THEROF IN THE PROJECT. I HEARBY DISCLAIM AND RELEASE THE UNIVERSITY OF VICTORIA, ITS AGENTS, SERVANTS OR EMPLOYEES, INCLUDING ALL PERSONNEL INVOLVED IN THE RESEARCH PROJECT, FROM ANY AND ALL LIABILITY THAT MIGHT OTHERWISE ARISE AS A RESULT OF MY PARTICIPATION AS A RESEARCH SUBJECT IN THIS STUDY.

NAME:
SIGNATURE:
ADDRESS:
PHONE:
DATE:

THIS RESEARCH SPONSORED BY THE UNIVERSITY OF VICTORIA
RESEARCH IDENTIFICATION NO. 92-88
ANY QUESTIONS OR PROBLEMS PLEASE CONTACT MIKE KING 479-0859

APPENDIX B: ESTIMATED MENSTRUAL BLOOD LOSS QUESTIONNAIRE

This research supported by the University of Victoria , Research Identification # 92-88

University of Victoria
Sport and Fitness Centre Elite Athlete Research

ESTIMATED MENSTRUAL BLOOD LOSS

NAME _____ **DATE** _____

WITH THE ONSET OF MENSES, PLEASE ADVISE AS ACCURATELY AS POSSIBLE:

A. Day 1 — no's required= _____ — ☐ Tampons ☐ Sanitary Napkins
blood loss estimated as — ☐ Heavy ☐ Moderate ☐ Low

B. Day 2 — no's required= _____ — ☐ Tampons ☐ Sanitary Napkins
blood loss estimated as — ☐ Heavy ☐ Moderate ☐ Low

C. Day 3 — no's required= _____ — ☐ Tampons ☐ Sanitary Napkins
blood loss estimated as — ☐ Heavy ☐ Moderate ☐ Low

D. Day 4 — no's required= _____ — ☐ Tampons ☐ Sanitary Napkins
blood loss estimated as — ☐ Heavy ☐ Moderate ☐ Low

E. Day 5 — no's required= _____ — ☐ Tampons ☐ Sanitary Napkins
blood loss estimated as — ☐ Heavy ☐ Moderate ☐ Low

IF NO MENSES OCCURRED IN _____ **PLEASE INDICATE BY CHECKING THIS BOX:**
Date (dd/mm/yr)

☐ Amenorrheic(no cycle)

This research supported by the University of Victoria, Research ID# 92-88.

APPENDIX C: TRAINING VOLUME SURVEY

UNIVERSITY OF VICTORIA
SPORT AND FITNESS CENTRE ELITE ATHLETE RESEARCH

WEEKLY TRAINING LOG SUMMARY

ATHLETE'S NAME: _____ WEEK # ____

KNOWN VARIABLES

1. Heart Rate Max =

2. Heart Rate Rest=

1. Sunday _____

Training run= _____ = _____ + _____ X HRmax = _____
miles/km duration/min

Other Session=

2. Monday _____

Training run= _____ = _____ + _____ X HRmax= _____
miles/km duration/min

Other Session=

3. Tuesday _____

Training run= _____ = _____ + _____ X HRmax= _____
miles/km duration/min

Other Session=

4. Wednesday _____

Training run= _____ = _____ + _____ X HRmax= _____
miles/km duration/min

Other Session=

5. Thursday _____

Training run= _____ = _____ + _____ X HRmax= _____
miles/km duration/min

Other Session=

6. Friday _____

Training run= _____ = _____ + _____ X HRmax= _____
miles/km duration/min

Other Session=

7. Saturday _____

Training run= _____ = _____ + _____ X HRmax= _____
miles/km duration/min

Other Session=

Please fill in the above information as accurately as possible, including the heart rates. Heart rates can also be taken by corotid pulse (ie. feel for 10 sec -1x6.)

APPENDIX D: 3 DAY DIETARY SURVEY

Sport Medicine Council of B.C.

High Performance Sport Science Unit

COMPUTERIZED NUTRITION ANALYSIS

Diet Information Form

Name _____

Address _____

_____

Telephone _____

Sport & Event(s) _____

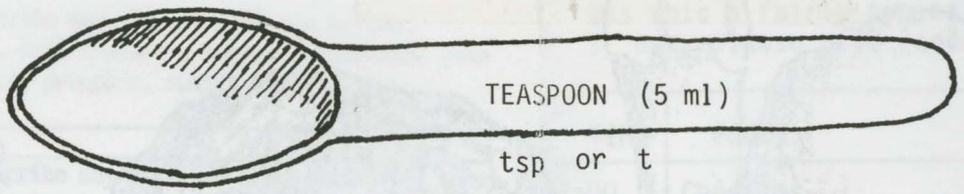
_____

Club _____

* Results of this analysis may be released to your coach and team physician. Please indicate if you do not approve.

APPENDIX D: 3 DAY DIETARY SURVEY

I. VOLUME



TEASPOON (5 ml)

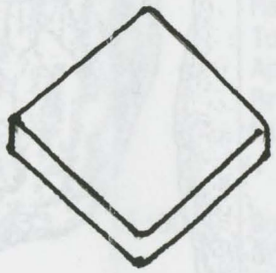
tsp or t

HEAPING TEASPOON -----> LEVEL -----> TEASPOON

---- 8 oz. (250 ml)

---- 4 oz. (125 ml)

1 CUP (250 ml)

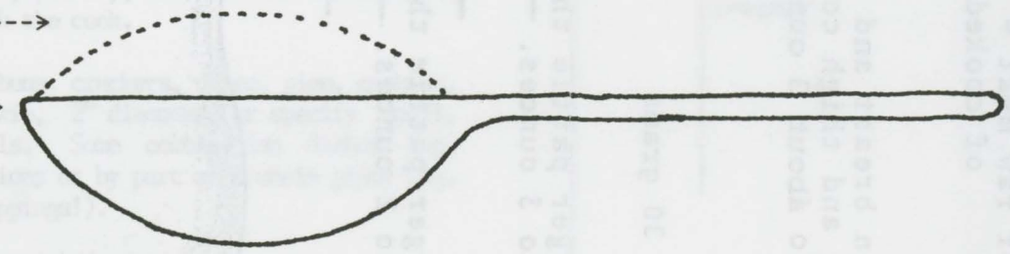


1 TEASPOON BUTTER (5 ml)

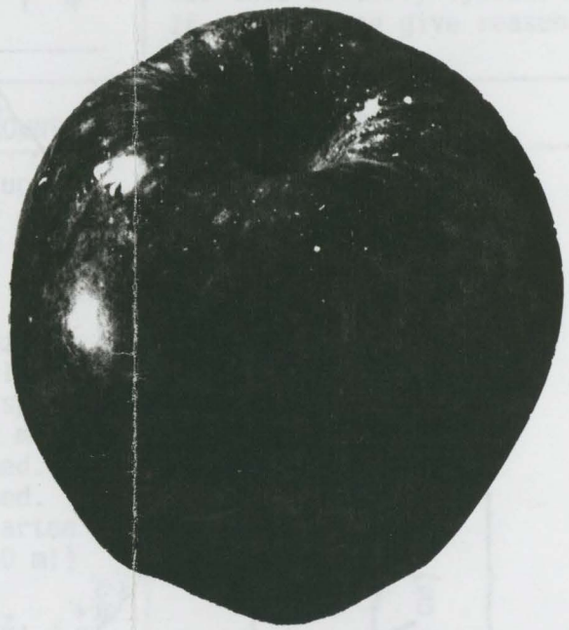
TABLESPOON (15 ml)

Tbsp or T

HEAPING TABLESPOON -----> LEVEL -----> TABLESPOON



II. SIZE



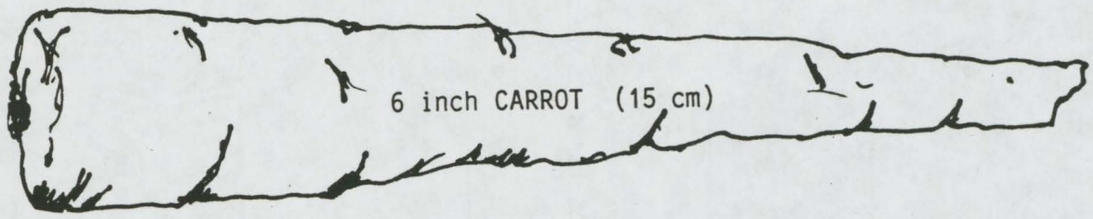
MEDIUM APPLE

* Use this model to descibe peaches, oranges, potatoes, tomatoes, etc.

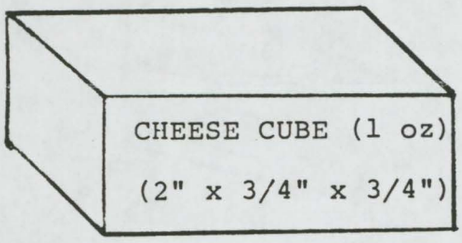
MEDIUM MUFFIN (100 grams)

SCALE (cm) - TO HELP IN DETERMINING DIMENSIONS

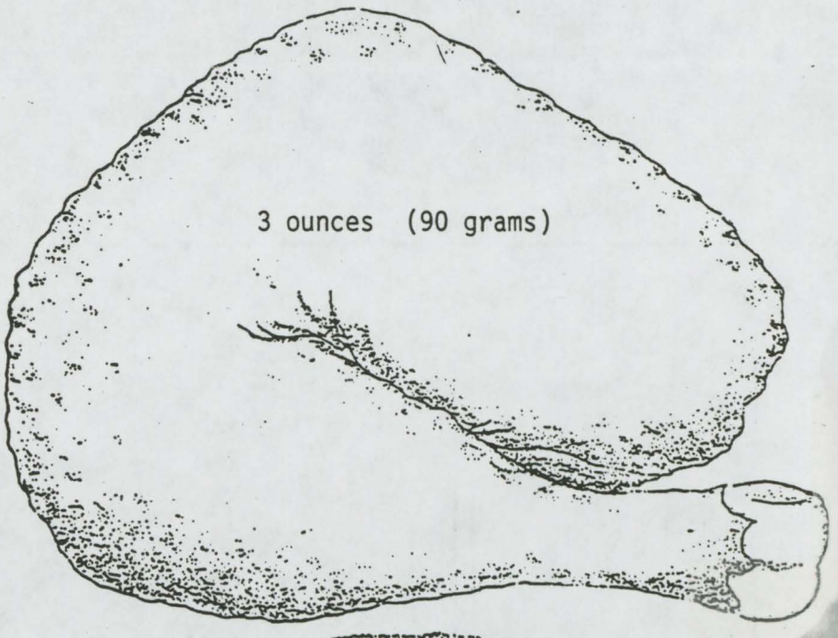
II. SIZE



III. WEIGHTS



4 ounces of raw meat = 3 ounces of cooked meat

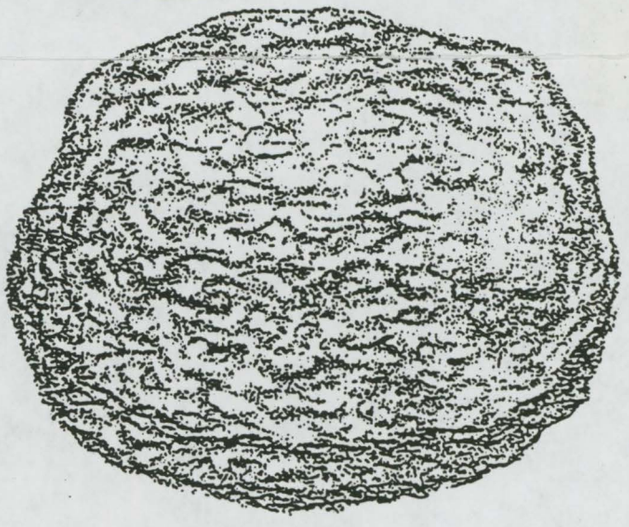


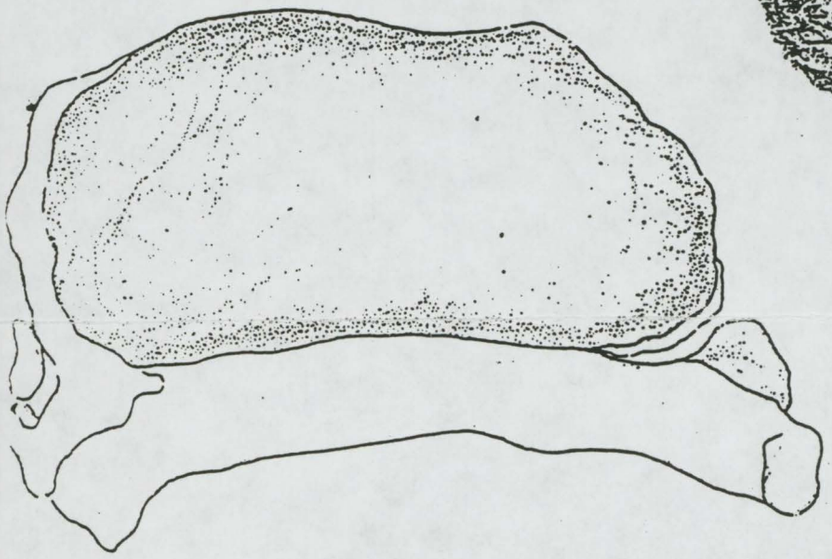
Half a chicken breast and wing or one leg and thigh cooked is equal to about 3 ounces of meat.

1 ounce= 30 grams

One hamburger pattie this thick is equal to 3 ounces.

One hamburger pattie this thick is equal to 2 ounces.



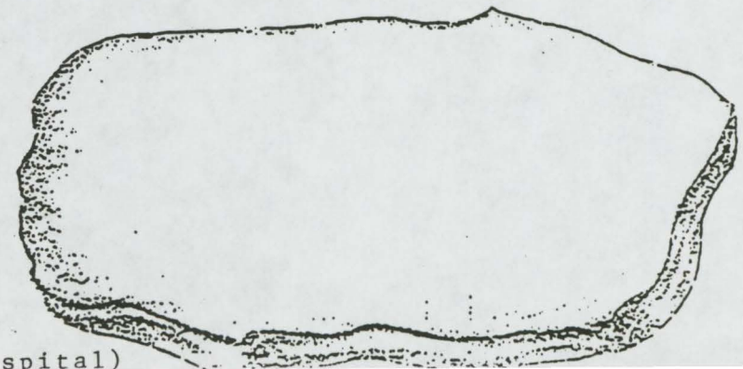


A pork chop this thick is equal to 3 ounces.

A pork chop this thick is equal to 2 ounces.

One slice of meat this thick is equal to 3 ounces.

Two slices of meat this thick are equal to 3 ounces.



(Adapted from Vancouver General Hospital)

APPENDIX D: 3 DAY DIETARY SURVEY

The following information is used to assess the nutritional adequacy of your diet. Please answer all questions and record food intake as accurately as possible.

I. PERSONAL DATA

Sex: Female___ Male___
Birthdate: _____
Age: _____ Years
Height: ___cm OR ___ft ___inches
Weight: ___kg OR ___lbs.
Desired Weight (if applicable) :
___kg OR ___lbs.

II. DIET INFORMATION

1. Describe any special diet you follow. (eg. diabetic diet, weight loss/weight gain diets, pregnant, nursing, etc.)

_____

_____

2. Describe any nutrient supplements you take. (eg. vitamin/mineral supplements) Include brand names. _____

_____

Dosage per day: _____

3. Describe any medications you take.

_____

_____

III. FOOD RECORD

List everything you eat or drink for 3 days (ideally 2 weekdays + 1 weekend day). Record immediately after each meal and snack to ensure accuracy. Be sure to include:

1. ALL FOOD AND DRINKS, including all snacks, soft drinks, wine, alcohol, cream and sugar in coffee and tea, butter or sauces on vegetables, jams, relishes, candies, butter, margarine or mayonnaise in sandwiches, salad dressings, etc. Combination foods must be broken down. (eg. omelette = 2 eggs + 1 oz. cheddar cheese + 1/2 Tbsp. vegetable oil in pan).

2. THE AMOUNT OF FOOD that was consumed. This is extremely important to ensure that an accurate analysis can be performed. Be as specific as possible. Use the attached diagrams and the following instructions to record precise portion sizes.

   a) Use VOLUME measures (cups, Tbsp, tsp or mls) for cereals, rice, pasta, vegetables, cut fruit, beverages, peanut butter, mayonnaise, butter, margarine, sauces, gravies, soups, honey, sugar, jams, jellies, etc.

   b) Use WEIGHTS (ounces or grams) for meat, fish, poultry, cheese. Use the attached diagrams , read food labels for weight or ask the cook.

   c) Use SIZE for fruits, vegetables, muffins, buns, crackers, cakes, pies, cookies, desserts, etc. Give dimensions (eg. 1 cookie, 2" diameter) or specify small, medium or large based on the attached models. Some combination dishes (eg. pizza, lasagna) may be described by dimensions or by part of a whole piece (eg. 1/4 of 12" pizza and remember to list the toppings!).

3. THE TYPE OF FOOD & BRAND NAMES, if applicable. Be specific.
   eg. whole wheat bread 2 slices mozzarella cheese 30 grams
   2% milk 1 cup Ritz crackers 4 crackers

4. THE TIME OF DAY these foods and drinks were consumed.

** THE MORE ACCURATELY YOU RECORD, THE MORE MEANINGFUL IS THE ANALYSIS. **

SAMPLE DAY
Was this a fairly typical day? Y N
If not, please give reason: _____

TIME	FOODS	AMOUNT
7:00	Cheerios	1 cup
	Milk, 2%	1/2 cup
	Orange juice, canned	280 ml
noon	Ham Sandwich:	
	Cooked ham	2 oz.
	Whole wheat bread	2 slices
	Margarine	1 tsp.
	Tomato	1/2 med.
	Apple	1 med.
	Bran muffin	1 med.
	Milk, 2%	1 carton (250 ml)
6:00	Roast beef	3 oz.
	Gravy	2 Tbsp.
	Carrots, cooked	1/2 cup
	Salad:	
	Romaine lettuce	1 cup
	Tomato	1/2 med.
	Radishes	2 med.
	Mushrooms	1/4 cup
	Regular Italian Dressing	2 Tbsp.
	Watermelon	1 cup
	Coffee	1 cup
	Creamer	1 Tbsp.

* Please print clearly.
Add extra sheets if necessary.

DAY 1 DATE _____
Was this a fairly typical day? Y N
If not, please give reason: _____

TIME	FOODS	AMOUNT

DAY 2 DATE _____

Was this a fairly typical day? Y N

If not, please give reason: _____

TIME	FOODS	AMOUNT

DAY 3 DATE _____

Was this a fairly typical day? Y N

If not, please give reason: _____

TIME	FOODS	AMOUNT

Sport Medicine Council of B.C.

High Performance Sport Science Unit

COMPUTERIZED NUTRITION ANALYSIS

Diet Information Form

Name _____

Address _____

_____

Telephone _____

Sport & Event(s) _____

_____

Club _____

* Results of this analysis may be released to your coach and team physician. Please indicate if you do not approve.

APPENDIX E: $\dot{V}O_2$ MAX ASSESSMENT DATA SHEET

Research I.D. # 92-88

UNIVERSITY OF VICTORIA ELITE ATHLETE RESEARCH
RUNNERS HEMATOLOGY STUDY

DATE _____

NAME _____ AGE _____ WT _____ HT _____

BODY COMPOSITION

TRICEPS	_____	_____	_____	=	_____
BICEPS	_____	_____	_____	=	_____
SUBSCAP	_____	_____	_____	=	_____
ILLIAC	_____	_____	_____	=	_____

_____ mm = _____ %B

ENDURANCE PERFORMANCE

WARM-UP SPEED @ _____ mph

		heart rate (bpm)
_____ MINUTES @ 5 mph	_____	_____
_____ MINUTES @ 6 mph	_____	_____
_____ MINUTES @ 7 mph	_____	_____
_____ MINUTES @ 8 mph	_____	_____
_____ MINUTES @ 3.5 mph	_____	_____
_____ MINUTES @ 9 mph	_____	_____
_____ MINUTES @ 9.5 mph	_____	_____
_____ MINUTES @ 10 mph	_____	_____
_____ MINUTES @ 10.5 mph	_____	_____
_____ MINUTES @ 11.0 mph	_____	_____
_____ MINUTES @ 11.5 mph	_____	_____
_____ MINUTES @ 12 mph	_____	_____
_____ MINUTES @ 2% GRADE	_____	_____
_____ MINUTES @ 4% GRADE	_____	_____
_____ MINUTES @ 6% GRADE	_____	_____
_____ MINUTES @ 8% GRADE	_____	_____
_____ MINUTES @ 10% GRADE	_____	_____

V02 MAX _____ L/MIN (absolute) MAX HEART RATE ___

_____ ML/kg/MIN (relative)

VITA

SURNAME: King **GIVEN NAMES:** Michael John

PLACE OF BIRTH: London, Ontario

DATE OF BIRTH: 9th October, 1963

EDUCATIONAL INSTITUTIONS ATTENDED, WITH DATES OF ENTERING AND LEAVING:

The University of Western Ontario 1982 to 1986

The University of Victoria 1986 to 1989

DEGREES AWARDED, WITH DATES AND NAMES OF INSTITUTIONS:

Honours Bachelor of Arts in Physical Education 1986
The University of Western Ontario

Masters in Exercise Physiology 1989
The University of Victoria

AWARDS:

Ontario Scholar 1982

The Sandy Hunter Memorial Award 1982

University of Western Ontario Physical Education Grant 1986

University of Victoria Graduate Supplement Award 1987

PUBLICATIONS:

Iron deficiency in endurance athletes, British Columbia Athletics Record, 1988.

Bad blood: the facts about iron deficiency in athletes, Island Runner, 9, 1989.

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Author [redacted]

Michael John King

December 7th 1989