CAUSES OF HYPONATREMIA IN NEW ZEALAND FEMALE ULTRADISTANCE TRIATHLETES

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A thesis submitted in partial fulfilment of the requirements for the degree of

Master of Science

In

Nutritional Science

Massey University, Albany, Auckland, New Zealand

2001

Abstract

The Ironman Triathlon is an ultradistance event typically dominated by male competitors. However, the event has become increasingly popular and is now attracting greater female participation [1, 2]. While hyponatremia (plasma sodium concentration < 135mmol/L) has been recognised as a serious complication of prolonged exercise, the aeitology remains unclear and controversial. The postulated causes of hyponatremia include the consumption and retention of excessive volumes of fluid or large unreplaced sodium losses in the sweat.

This study was undertaken to investigate the nutritional, biochemical, hormonal and physical status of New Zealand female ultradistance triathletes, specifically, those competing in the New Zealand Ironman Triathlon, on 3 March 2001. The study was to determine the causes of hyponatremia in these athletes.

Twenty-seven (n=27) ultradistance female triathletes training for the New Zealand Ironman Triathlon were recruited for the study. Participants completed: (a) a brief demographic questionnaire; (b) two 7-day food diaries to evaluate dietary intake during the peak of training (6-7 weeks prior to the event) and during the taper (1 week before the event); and (c) a menstrual status questionnaire. Body composition was assessed by calibrated digital scales and bioelectrical impedance analysis **(BIA)** 19 hours before the race and within 15 minutes of each individual completing the race. Blood and urine samples were

collected and analysed 19 hours before the race and within 15 minutes of each individual completing the race.

Complete medical information was available for 19 of the 27 recruited female triathletes (70%). Post-race plasma sodium concentrations were inversely related to body weight changes. A mean weight loss of 1.6 ± 1.1kg ($p = < 0.001$) equated to a percentage dehydration of 2.4 ± 1.8%. One athlete from the study group had asymptomatic hyponatremic (post-race plasma sodium concentration 134mmol/L). The athlete was the smallest subject in the study (53.4kg), finished the race 1kg heavier and was moderately overhydrated by 1.9%. A lowered post-race plasma sodium concentration was also related to lowered haematocrit (Hct). The lowered Hct indicated that the fluid was retained in the extracellular space, which caused dilutional hyponatremia.

The athlete with the asymptomatic hyponatremia was the only athlete taking a progesterone only, oral contraceptive pill. Progesterone is believed to contribute to postovulatory fluid retention.

The mean daily energy intake **(MDEI)** results for the study group from the first and second 7-day food diaries were 10811 ± 211 kJ/day (2672 ± 511 kcal/day) and 10155 ± 1820 kJ/day (2487 ± 410 kcal/day) respectively. This was between 22-35% lower than the expected daily energy expenditure **(EDEE)** 13874 – 15610 kJ (3319 – 3734 kcal). It has been suggested that the difference may be

due to inaccurate reporting of intake. The lower MDEI resulted in carbohydrate **(CHO)** intake expressed as grams of CHO per kilogram (kg) body weight **(BW)** per day appearing below the recommended 7-10g CHO/kg BW/day. All subjects had a fat intake below the 30-33%, and a protein intake above the 12-15% recommended for the general New Zealand population. Most of the athletes met or exceeded the Recommended Dietary Intake (RDI) for most micronutrients.

The study concluded that the likely cause of exercise associated hyponatremia was probably dilutional hyponatremia due to the consumption and retention of large volumes of low sodium or sodium free fluids before and during the race. Many subjects would benefit from individualised dietary advice to balance the increased energy expenditure of heavy training and to determine the volume of fluids needed for ultradistance events.

Acknowledgments

I would like to thank the following people for the valuable assistance they have provided during the completion of this research project:

Dr Clare Wall	Lecturer, Institute of Food, Nutrition and Human Health, Massey University
Dr Dale Speedy	Sports Physician, Sports Care, Manurewa, Auckland
Keith Thorpe	Ironman New Zealand Race Director
Jane Patterson	Ironman New Zealand Event Manager
Medlab Taupo	Nurse/phlebotomists Beverley Garmey & Nancy Stratford
Medlab Hamilton	Biochemistry Manager Graeme Broad

I would also like to thank the New Zealand Dietetic Association and the late Neige Todhunter, and Susie Cropper Heinz/Watties Australasia for providing funding for this research project.

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1 Introduction

Since its creation in the late 1970s, the Ironman Triathlon event has been dominated by male competitors. Over the last few years the event has become increasingly popular and is now attracting greater female participation [1, 2]. The Ironman Triathlon by definition is an aerobic, ultradistance race involving three consecutive disciplines: swimming 3.8km (2.4 miles), cycling 179.2km (112 miles) and running 42.2km (26 miles, 385 yards).

Maintenance of fluid balance, muscle fuel stores (glycogen) and blood glucose by the athlete is challenged because of the duration of the event. Although races usually start early morning to avoid the heat, an Ironman Triathlon takes considerably longer than eight hours to complete. As the temperature increases over the course of the race sweat losses can be substantial so that fluid intake becomes more critical.

Hyponatremia (plasma sodium concentration < 135mmol/L) has been recognised as a serious complication of prolonged exercise. However, no reference to 'exercise associated' hyponatremia can be found in the literature prior to the 1980s. The aeitology of hyponatremia remains unclear and controversial; the postulated causes include the consumption and retention of excessive volumes of fluid or large unreplaced sodium losses in the sweat.

Male and female hormones influence anatomy and physiology after puberty. Collected data on the male endurance athlete therefore can not be applied to the female endurance athlete because potential exists for these different hormonal and physiological processes to influence athletic performance. Consequently, it is important to be aware of the physical, biochemical, hormonal, and nutritional status of the female endurance triathlete competing in ultradistance events in order to study and determine the cause of hyponatremia in these athletes.

Assessment of body compositional changes and biochemical alterations that occur during prolonged exercise (and the relationship to each other) could help identify risks associated with the development of hyponatremia and provide a protocol for the reduction of incidence.

The nutritional requirements of female ultradistance triathletes are different when compared to sedentary females due to the effect of exercise on the physiology. The estimation of daily energy requirements, the carbohydrate **(CHO)** and protein intakes based on kilogram **(kg)** body weight **(BW)**, and fluid and micronutrient (especially sodium) intakes will be greater due to the increased energy expenditure.

Recommendations for food and fluid intake during heavy training, the taper leading into an event, as well as the day of the competition should be designed for each individual based on assessment of body composition, biochemical and hormonal status, and energy expenditure.

2 Literature review

2.1 Ironman Triathlon

Triathlon races typically involve three consecutive disciplines (swim, bike, run) and were developed as endurance events for individual competitors. The triathlon season extends over the summer months and races usually start early morning to avoid the heat. The Ironman Triathlon is an aerobic event that is raced over long distances. Navy Commander John Collins developed the ultra distance event in Hawaii in the late 1970s. Collins proposed the combination of the annual Waikiki Roughwater Swim (2.4 miles), the Around Oahu bike race (112 miles, then a two day event) and the Honolulu marathon (26.2 miles). He said that, *"Whoever finishes first we'll call the Ironman"*. Fifteen men participated in the first event February 18, 1978; 12 finished. The Ironman event went on to become an annual race in Hawaii and eventually became so popular it required a number of qualifying races around the world. This included Ironman New Zealand, which first took place March 24, 1985 [1].

2.1.1 New Zealand Ironman Triathlon

Statistical information from Ironman New Zealand shows that 214 athletes competed in 1985 compared with 779 athletes in 2000. The number of female athletes competing has also increased from 20 females in 1985 to 106 females in 2000 [2]. Female athletes race the same course as male athletes.

The New Zealand Ironman Triathlon was raced for 14 years in Auckland but was relocated in 1999, to Taupo, a premier holiday destination in the middle of the North Island of New Zealand. The swim course is 3.8km (2.4 miles) in a fresh water lake, the bike course is 179.2km (112 miles) and the run course is 42.2km (26 miles, 385 yards).

At best, ultra distance events like Ironman Triathlon take approximately eight hours and 30 minutes to complete but most competitors take considerably longer. The duration of the event poses a challenge for the athlete with regard to maintenance of fluid balance, muscle fuel stores (glycogen) and blood glucose levels. As the temperature increases over the course of the race, sweat losses can be considerable so that fluid intake becomes critical.

2.2 Water and Sodium Regulation

2.2.1 Water and sodium regulation under normal conditions

The body water of healthy individuals is conserved on a daily basis by factors that control input and output of both water and electrolytes [3]. There are wide variations in the amount of water and sodium consumed daily yet the composition, volume and distribution of body fluids in normal individuals is maintained within a narrow range. Water balance is regulated by hypothalamic control over anti-diuretic hormone **(ADH)** release from the posterior pituitary as presented in figure 2.1 [4]. ADH is released in response to increased osmolality of the blood, a decrease in plasma volume or left atrial pressure, and angiotensin II. In the kidney, ADH increases the permeability of the distal tubules and collecting ducts, thus increasing water re-absorption [4].

Sodium is the principal electrolyte in the extra-cellular fluids and is the major contributor to serum osmolality. Serum osmolality regulates ADH and therefore water balance. If sodium concentration decreases in the blood, angiotensin II stimulates the release of ADH, which regulates the secretion of aldosterone. Aldosterone acts to increase sodium re-absorption in the distal tubules of the kidney as well as in the intestine [4, 5].

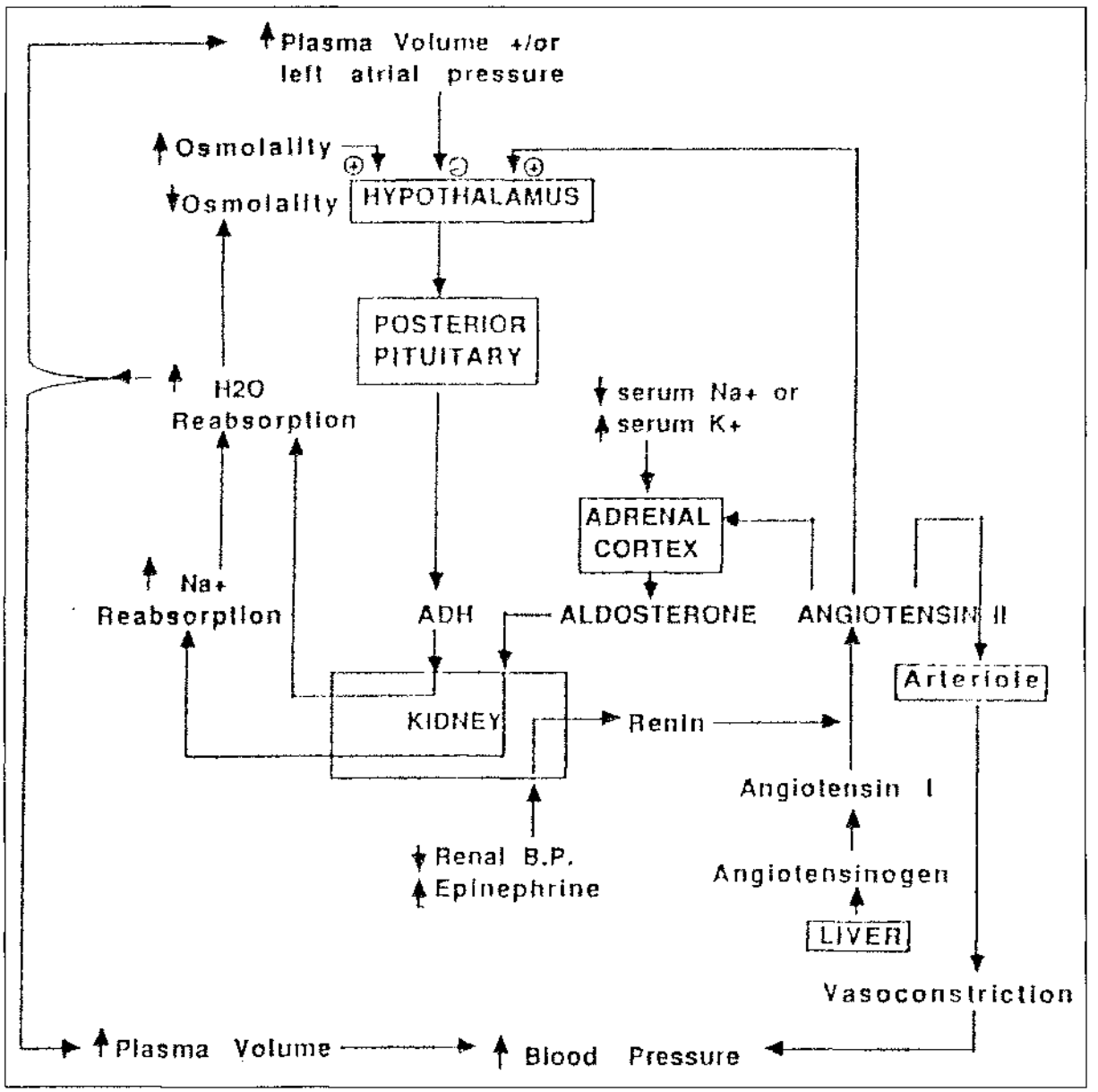


Figure 2.1 Fluid and Electrolyte Regulation. [4]

2.2.2 Water and sodium regulation in endurance exercise

Even before significant sweat losses have occurred, mechanisms for the conservation of water and sodium have been activated in the body [4]:

(a) There is a marked drop in plasma volume at the onset of exercise because water from the plasma moves into the exercising muscle,

(b) The decrease in plasma volume decreases renal blood pressure, and stimulates ADH release and water re-absorption,

(c) The decrease in renal blood pressure stimulates renin release from the kidney, which results in aldosterone secretion and sodium re-absorption [4, 6],

(d) As exercise continues, the production and evaporation of sweat is the major means for dissipating body heat [6].

2.2.3 Sweating – body heat dissipation during exercise

During exercise in a cold or cool environment, body heat is lost mainly through radiation and convection via the air movement around the body. Some evaporation of sweat and evaporative heat loss from the lungs may also contribute to maintenance of the heat balance [5]. However, when the ambient temperature is higher than skin temperature, the only mechanism to control the excessive rise in the core temperature is the evaporation of water from the skin and respiratory tract [7].

The transfer of heat to the skin is achieved by vasodilation of cutaneous circulation, thereby displacing blood to the periphery [8]. As much as 75% of heat loss is achieved by the evaporation of sweat, with approximately 2424 kJ (580kcal) of heat dissipated for each litre of sweat evaporated [7]. In hot, humid environments, where sweat drops from the body without evaporating, actual sweat rates will be higher than predicted. In windy, mild conditions, in which sweat can be dissipated by convection, actual rates may be somewhat lower [4].

2.2.4 Sweat Rate

Sweat is mostly water, about 99 percent, but it also contains a number of major electrolytes and other nutrients [5, 6, 9]. The major electrolytes found in sweat are sodium and chloride. These electrolytes are present because sweat is derived from extra-cellular fluid, such as plasma, and intercellular fluid [5]. Sweat is hypotonic in comparison to the fluids in the body and the net effect of sweat loss is an increase in plasma osmolality [7].

The degree to which an athlete sweats depends on temperature, humidity, solar radiation, exercise intensity, heat acclimatisation and cardio-respiratory fitness [9]. In an observational case study by Armstrong et al [10] ten subjects performed eight hours of moderate, intermittent exercise (40-45% VO2max) in hot conditions (41°C, 21% Relative Humidity (RH)) daily for 10 days to measure the effects of dietary sodium consumption on heat acclimatisation and physical performance.

Volunteers had to live continuously for 17.5 days in a research facility which contained sleeping and dining quarters, and an environmentally controlled chamber. The daily diet (3600 kilocalories) had a specified sodium composition of 137 mEq (8g NaCl/day). The prescribed salt consumption fell within the normal range of the adult daily intake in the United States of (137-223 mEq sodium daily). At the conclusion of each day's exercise regimen, the volunteers returned to the 21°C environment. On the initial day of exercise-heat exposure, the body mass was measured (+/- 50g) immediately prior to exercise and at the end of each hour of exercise to allow the calculation of sweat rate (corrected for water intake, urine output, faecal excretion, and food consumption). To maintain body weight, volunteers were encouraged to drink pure water and flavoured water (< 1 mmol sodium/L) *ad libitum* from canteens, during both exercise and rest. An average sweat rate of 0.7 L per hour was reported [10].

One volunteer experienced symptomatic hyponatremia during the research investigation. It was suggested that the volunteer was moderately hyper-hydrated at the beginning of the research – plasma indices (plasma sodium 134mmol/L, osmolality 282 mmol/kg, haemoglobin 15.6g/dL and haematocrit 0.45) were more than 2 Standard Deviations (2SD) from the means of the control subjects [10]. The 'low normal' initial plasma sodium has been suggested to have predisposed the volunteer to hyponatremia. The total sodium loss (urine and sweat) and sweat volume of the hyponatremic volunteer were within ± 1 SD of the mean of the control subjects during hours 0-4. The authors have reported that sodium loss was not critical to the development of hyponatremia but that excessive fluid retention was [10].

The 1985 retrospective evaluation of three athletes who completed the 'Comrades ultra marathon' were reported to have similar sweat rates [11]. This was considerably lower than when Alberto Salazar (a well-conditioned, world-class athlete), competing in the 1984 Olympic marathon, in Los Angeles, USA, was reported to have had a sweat rate of 3.7 L per hour [12].

It has been reported that women have more subcutaneous fat and have lower sweat rates than men and should monitor their body weight loss as a result of endurance exercise in order to calculate the required volume of fluid replacement [13, 14].

2.2.5 Heat acclimatisation

The composition of sweat may vary somewhat from individual to individual and will even be different in the same individual when acclimatised to the heat, as contrasted to the non-acclimatised state [5]. In trained athletes many of the body's adaptations to cope with heat can happen quite quickly i.e. within 5 days. The majority of adaptations take place within 10 days but complete acclimatisation may take up to three weeks [14]. Wenger reported that well-conditioned, heat acclimatised athletes, have sweat sodium concentrations in the

range of 5-30 mmol/L sweat (115-690 mg sodium/L sweat); typically non-acclimatised athletes lose much more sodium and have sweat sodium concentrations in the range of 40-100 mmol/L sweat (920-2300mg sodium/L sweat) [15].

2.3 Fluid Balance during Exercise

Fluid balance during exercise is dependent upon the rates of fluid ingestion, gastric emptying, and intestinal absorption [16].

2.3.1 Fluid ingestion before and during exercise

It is difficult to make recommendations for fluid ingestion during endurance and ultra endurance exercise that encompass the specific physiological issues and practical considerations of various sports events. Previous attempts have met with criticism for their failure to suit extremes of exercise or to understand practical issues in sport [17]. For example, the American College of Sports Medicine **(ACSM)** (1987) position paper [18] entitled *'The Prevention of Thermal Injuries During Distance Running'* emphasised the need for regular fluid intake during races of 10km and longer, and encouraged runners to ingest 100-200ml at every aid station (every 2-3km). Coyle and Montain reported that if the recommendation were taken literally fluid intake could range from 300ml/hour for a slower runner (10km/hour; 100ml/3km) to 2000ml/hour for a faster runner (20km/hour; 200ml/2km) [19]. Thus dehydration could occur at one extreme and gastrointestinal discomfort at the other. The uncertainty from the 1987 position paper is said to have been addressed in the most recent ACSM (1996) position paper, *'Exercise and Fluid Replacement'* [20].

The first ACSM recommendation from the 1996 paper stated that individuals should consume a nutritionally balanced diet and drink adequate fluids during the 24-hour period before an event, especially during the period that includes the meal prior to exercise, to promote proper hydration before exercise or competition [20]. The New Zealand Food and Nutrition Guidelines, though not

specific for athletes, are in agreement with the ACSM recommendation. The guidelines include the recommendations *"Prepare meals with minimal added fat and salt"* and *"Choose pre-prepared foods, drinks and snacks that are low in fat, and salt"* [21]. The recommended daily dietary allowances for sodium are approximately 1100mg to 3100mg.

In a small (eight subjects – five male and three female – all well trained cyclists), observational, crossover designed study, Barr et al. [22] compared the responses during 6-hours of exercise in the heat (30°C, 50% RH) with three fluid replacement conditions. The three interventions included were no fluid, plain water to balance sweat and urinary losses and similar amounts of a saline solution containing sodium in amounts greater than is found in commercially available beverages (25 mmol/L). The no fluid condition resulted in decreased plasma volume (plasma sodium rose steadily and was significantly higher than both plain water and saline trials at 2-hour, 4-hour and post-exercise). In addition the study group experienced other deleterious effects of dehydration and the trial was terminated 1.5 hours prior to its scheduled completion. Conversely, plasma volume was maintained during the plain water and saline trials. Saline ingestion was not associated with significantly higher plasma sodium levels. Sodium losses were substantial in both the plain water and saline trials but were not large enough to present a risk of hyponatremia (low blood salt/sodium) (plasma sodium concentration <135 mmol/L) during 6-hours of exercise. The observed post-exercise plasma sodium level for the water trial was 135 mmol/L (+/- 0.5), and for the saline trial 137.3 mmol/L (+/- 0.7). However, the authors reported that had the subjects continued to exercise the risk of hyponatremia would not be inconceivable. The authors concluded that those athletes who restricted their sodium intake in accordance with nutrition recommendations aimed at the general public and who also engaged in prolonged exercise over several days with resultant excessive sodium loss through sweat were at greater risk [22]. The observational study by Armstrong et al. [10], reported that a "low normal" initial

plasma sodium and subsequent sodium losses contributed to the rapid onset of hyponatremia in the athlete.

A recent report stated that simply adding salt to the diet (or eating high-salt foods) and consuming a selected volume of a salt-containing beverage (sports drink) would ensure better fluid retention and could prevent a sodium deficit [9]. However, in the Barr et al. [22] study, saline ingestion (sodium concentration 25 mmol/L) did not prevent a decrease in plasma sodium. The saline solution contained sodium in amounts higher than generally found in commercial sports drinks. The higher sodium concentration needed (2-4 times greater than that of commercial beverages) to support pre-exercise sodium levels would reduce palatability of the solution and could lead to the risk of dehydration as a result of inadequate fluid ingestion [22].

It has been reported that athletes may benefit from consuming a large volume/bolus of fluid as they can comfortably tolerate (e.g. 300-500ml) just prior to exercise (e.g. 15 minute before starting exercise). In addition to providing fluid, advocates suggest that this strategy helps "prime the stomach" to stimulate a more rapid gastric emptying of subsequent fluid intake during activity [23].

The second and perhaps most significant ACSM recommendation from 1996, stated that: during exercise, athletes should start drinking early and at regular intervals in an attempt to consume fluids at a rate sufficient to replace all the water lost through sweating, or consume the maximal amount that can be tolerated [20]. Ideally, a fluid intake plan should replace about 80% of your sweat losses to ensure greater cardiac output, lower core temperature and reduce the rate of perceived exertion [24, 25]. The recommendation recognises that an optimal intake may be difficult to achieve and that some degree of dehydration may be inevitable when sweat rates are sustained in excess of 1 L/hour [20, 26]. However, athletes should be warned that very high rates of fluid ingestion (> 1.5 L/hour) or sodium-free fluid ingestion to match sweat loss sustained for many

hours, can lower plasma sodium concentration and precipitate the development of hyponatremia [27, 28].

In a randomised crossover designed trial, Vrijens and Rehrer [28] assessed whether replacing sweat losses with sodium-free fluid would lower the plasma sodium concentration and thereby precipitate the development of hyponatremia. Ten male endurance athletes cycled for 1-hour pre-trial (to estimate fluid needs) and two 3-hour experimental trials at 55% VO2 max at 34°C, 65% RH. Water or Gatorade (63g/L carbohydrate, 18-mmol/L sodium) was given every 15 minutes at a rate equal to the estimated fluid loss. Plasma sodium concentrations decreased to a greater extent with water ingestion than with the ingestion of the Gatorade. One subject developed hyponatremia (plasma sodium 128 mmol/L) at 2-hour 30-minutes in the water trial.

The study by Barr et al. [22] is the most comparable to this study, however there was no significant difference between the two fluid replacement conditions in the Barr et al study. The difference in the results compared to the Barr et al. study may be explained by environmental differences. In the Vrijens et al. study [28] the environmental chamber was set at 34°C, 65% RH, whereas the chamber in the Barr et al. study was set at 30°C, 50% RH.

In the Vrijens et al. study [28], more fluid was lost and replaced with ingested fluids. The sweat rate was 1.36 +/- 0.20 L/hour with water and 1.38 +/- 0.21 L/hour with Gatorade. This can be compared with the lower sweat rate in the Barr et al. study [22] of 0.79 L/hour with water and 0.81 L/hour with saline solution. Although the exercise intensity was the same in both studies 55% VO2 max, the temperature and humidity was greater in the Vrijens et al. study [22, 28]. The sodium content of sweat increases with an increased sweat rate [6]. The greater temperature and humidity of the Vrijens et al. study was, therefore, likely to have caused greater sodium loss through increased sweat rate [22,28].

2.3.2 Gastric Emptying

Gastric emptying is considered to be the first limiting step in making fluid available to the circulation [29]. Several descriptive studies involving running and cycling, at the same relative intensities, have shown that there is no difference in the regulation of gastric emptying rate **(GER)** between rest and exercise up to intensity levels of 70-80% VO_2 max [30, 31].

The GER is determined by the volume and the composition/energy content of the fluid consumed [16]. The common assumption that frequent small sips is the best pattern of fluid ingestion is not supported by the fact that ingesting a large fluid volume results in greater gastric emptying [32]. After the ingestion of a fluid bolus there is a rapid emptying phase followed by a phase of reduced emptying once the volume in the stomach has been reduced to about 30% of the initial content [29, 31, 33].

The chemical composition/energy density of the fluid is undoubtedly the most significant factor influencing GER [34]. In a small (six subjects – male) observational, crossover designed study Vist and Maughan [34] compared the half-emptying time of four different solutions on four separate occasions. A double sampling gastric aspiration method (which makes it possible to follow the time course of gastric emptying) was used to investigate the relative importance of osmolality and carbohydrate content. The method compared the rate of gastric emptying of isoenergetic solutions of glucose and glucose polymer at high and low concentrations [34]. The four solutions studied were:

(a) A dilute (40 g/L) monomeric glucose solution (LG, 230 mosmol/kg),

(b) A dilute (40 g/L) glucose polymer solution (LP, 42 mosmol/kg),

(c) A 188 g/L glucose solution (HG, 1300 mosmol/kg), and

(d) A 188 g/L glucose polymer solution (HP, 237 mosmol/kg).

Tests were conducted 3-5 days apart and subjects were seated throughout the study.

The dilute solutions (40 g/L) of glucose and glucose polymer were both rapidly emptied from the stomach. The glucose polymer solution (LP) emptied faster (14 minutes) than the free glucose solution (LG) (17 minutes) [34]. Increasing the carbohydrate content of the solution decreased the rate of gastric emptying [34]. Both of the concentrated solutions (HP and HG) had a slower rate of gastric emptying than the dilute solutions. Although HP and HG had the same carbohydrate content, the osmolality was different. The greater osmolality of HG may have contributed to its slower rate of gastric emptying when compared to HP [34]. The dilute glucose solution (LG) emptied faster than the concentrated glucose polymer solution (HP) with the same osmolality (LG, 230 mosmol/kg; HP, 237 mosmol/kg) [34]. The results indicate that carbohydrate content appears to have greater influence than osmolality in gastric emptying of liquids and that the addition of carbohydrate in quantities up to 4% did not inhibit GER [34].

A sports drink is defined as a solution with 4-8% (4-8g/100ml) carbohydrate and 500-700mg/L (20-30mmol) sodium [21]. A high content of electrolytes may reduce GER but the sodium content observed in most sports drinks mimics the sweat content of athletes and will not reduce GER [35]. Fructose, the sweetest monosaccharide, is often added to sports drinks to increase the sweetness without the addition of more glucose or polymers [36] but has been reported to cause gastrointestinal distress [7].

GER has been measured in 50 young (18-31 years) healthy subjects (32 males, 18 females) by means of sequential scitigraphy with two radioactive markers [37]. Significant differences were found in gastric emptying of both solids and liquids among sexes; women emptied the stomach more slowly than men regardless of age, weight, height, or body surface. A relationship was found to exist between gastric emptying and the phase of the menstrual cycle. The authors observed that women have slower GER when compared to men, but during ovulation this is reversed [37]. A progesterone effect was suggested to be responsible for the faster emptying during ovulation reported in this study [37].

2.3.3 Intestinal absorption

Fluid loss during exercise can impair thermoregulatory function. Fluids ingested during activity must be absorbed to promote rehydration. As there is little net absorption of water and solutes in the stomach, the rate at which a drink is delivered to the absorptive surface of the proximal small intestine (duodenum and jejunum) will influence how quickly absorption occurs [38]. At rest the maximal fluid absorptive capacity of the proximal small intestine is reported to be about 1.9 to 2.3 L/hour (this is similar to the highest values ever recorded for gastric emptying) [39]. Currently the maximum rates of fluid absorption by the small proximal bowel during exercise are not known. However, Noakes has estimated that intestinal water absorption during exercise is limited to between 750-1000ml/hour [26].

The proximal small intestine absorbs about 50-60% of any given fluid load, the ileum absorbs 20% and the colon 15% [39]. Absorption of water in the intestine is a passive process caused by the creation of local osmotic gradients. Introduction of water into the intestine places water in contact with the brush border membrane of the enterocytes covering the intestinal villi i.e. the presence of an osmotic gradient across a semi-permeable membrane. Water (0 mosmol. /kg) moves into the plasma (280 mosmol /kg) to dissipate the osmotic gradient, plasma is diluted by the entering water [16].

Plain drinking water is hypotonic, with osmolalities generally in the order of between 5-15 mosmol/ kg; they would be expected to promote the most effective rates of water absorption [38]. However, water absorption from an isotonic or hypotonic solution containing a transported monosaccharide such, as D-glucose, is more rapid than from water itself [40]. The relatively poor rates of water absorption from water is thought to be due to the efflux of electrolytes down concentration gradients pulling water across the mucosa into the intestinal lumen [38].

The brush border membrane is the major barrier for absorption of carbohydrate. Within the membrane there are energy dependent active transporters. These transporters usually require the presence of the cation sodium (Na+) which are co-transported with the carbohydrate [38]. Other electrolytes do not appear to affect water and solute absorption to the same extent as sodium [16, 38].

Drinks with an osmolality greater than 290-mosmol/ kg are effectively hypertonic and when ingested they cause an efflux of water into the proximal small intestine, which reduces the rate of net water absorption. This effect renders the solution ineffective as a rehydration beverage [38].

The most effective osmolality range for solutions containing carbohydrate and sodium is between 200-260 mosmol/kg [38]. Increasing the concentration of glucose in the intestinal lumen to 10% (550 mosmol/kg) can cause fluid secretion and gastrointestinal distress [41]. Glucose polymers taste less sweet than mono- or disaccharides and offer the potential advantage of increasing the quantity of glucose delivered to the intestine at decreased osmolality [36, 39, 42].

Endogenous sodium is secreted into the intestinal lumen rapidly when sodium-free or low sodium solutions are perfused [16, 38]. It has, therefore, been postulated that exogenous sodium added to rehydration fluids, intended for use during exercise, is not required to activate the energy dependent transporters in the brush border membrane of the proximal small intestine [43]. While no perfusion studies have shown that the exclusion of sodium from glucose solutions has a detrimental effect on net water absorption, substitution of mannitol or magnesium for sodium results in a 23% and 45% reduction respectively in glucose absorption [38].

The addition of sodium to drinks intended for consumption during prolonged exercise such as ultraendurance events may be necessary. Hyponatremia has

been reported in these events where large sweat losses can be expected and the exercise intensity is necessarily low, making it possible to consume and absorb large volumes of fluid [11].

2.4 Hyponatremia

The first hyponatremia study was published in 1916 by Haldane [44]. Compulsive water drinking was reported to have induced the complications of hyponatremia in psychiatric patients who subsequently died [44].

Hyponatremia was not a condition commonly associated with exercise until recently. Traditionally it was believed that all persons who collapsed during or after exercise were dehydrated and would require intravenous fluid therapy [26, 45].

The most commonly encountered medical condition at endurance events is exercise-associated collapse **(EAC)** [45, 46, 47, 48]. EAC is not in itself a diagnosis, as it gives no indication of the cause of the condition [47]. Roberts describes the main complaint of EAC as the inability to stand or walk unaided as a result of dizziness, light-headedness or faintness [48]. Most collapsed runners were believed to suffer from exercise-induced dehydration with or without hyperthermia [46]. Because serum sodium concentration is not frequently measured in collapsed athletes, the diagnosis of hyponatremia is often missed or mistaken for exercise-induced hypoglycaemia or exertional 'heat-stroke' [48,49].

The development of hyponatremia requires that fluid must be ingested at high rates for many hours, probably for at least four to six hours (see section 2.4.2 for aeitology – postulated causes of hyponatremia). Hence the condition will be extremely uncommon in any race that finishes in less than four hours and becomes more evident in races lasting longer than eight hours [49]. The condition could develop after a race, however, if the athlete continues to ingest fluid at high rates [49].

Hyponatremia usually occurs in athletes of average ability (recreational, as opposed to highly trained – competitive athletes), and is reported predominantly in those athletes who finish the race with the last 25-50% of entrants [27]. Female athletes have been reported to be at significantly increased risk of the development of hyponatremia [27, 51].

2.4.1 Clinical features and symptoms

No reference to 'exercise associated hyponatremia' can be found in the literature prior to the 1980's. Symptomatic hyponatremia during prolonged exercise was first reported in 1981 by Noakes, in two athletes competing in the 1981, 90km Comrades Marathon footrace in South Africa [50]. In 1985 Noakes et al, provided the first clinical histories of these two cases plus reported another two cases in the medical literature [11].

Symptoms of hyponatremia may be mild and non-specific. They include: fatigue, nausea and malaise. The athlete may only become aware of the symptoms upon completion of the race [49, 51]. Symptoms of moderate severity include headache, confusion, restlessness, and disorientation [49]. More specific and severe symptoms include raised intracranial pressure, grand mal seizures, pulmonary edema, respiratory arrest, coma and death [11, 49, 52, 53]. It has been recognised from the aforementioned symptoms that exercise associated hyponatremia is a serious complication of prolonged exercise that has the potential to be life-threatening if misdiagnosed [11, 49, 51, 52, 53, 54, 55].

2.4.2 Aetiology – Postulated causes of hyponatremia

The aetiology remains unclear and controversial.

(a) Excessive losses of sodium through urine and sweat (either chronically before, or acutely during exercise); or

(b) The consumption and retention of large volumes of low-sodium or sodium-free fluids; or

(c) A combination of these two factors,
are the factors that are most often debated in the literature [4].

Hiller and other authors have postulated that 'massive', unreplaced sodium losses, in the presence of dehydration is important in the development of hyponatremia [46, 56, 57, 58]. Hiller reported that the combination of dehydration and hyponatremia was easily understandable if one considered that sweat rates of 1.5 litres per hour with 0.25 normal saline solution (NSS) sweat concentration were usual [46,56]. Hiller stated that with an average Ironman finish time of 13 hours, an athlete would lose about 19.5 litres of water and about 39g of sodium chloride [46]. In a survey of 39 Ironman finishers in 1985, the average fluid intake was much less than 19.5 litres and water (sodium-free fluid) was the commonly chosen drink. It is therefore possible that the athletes can finish the race both dehydrated and hyponatremic [46]. Hiller et al. reported that this is salt depletion heat exhaustion and the appropriate treatment for this condition is volume replacement with a solution such as 5% dextrose in normal saline solution (D5NSS) [46,56]. Hiller et al. also reported that 3 to 4 pints of fluid (571ml = 1 pint) per hour is required to maintain fluid balance under typical race conditions [46].

Sixty-four athletes (53 male and 11 female) from the 1984 Hawaiian Ironman Triathlon participated in the first observational study of ultraendurance triathletes [57]. The study by Hiller et al. [57] assessed plasma electrolyte and glucose changes during the 1984 Hawaiian Ironman Triathlon. Pre-race (21 to 24 hours before the race) and post-race (within 5 minutes of the subject completing the race) blood samples were taken from each subject. Blood was drawn from the brachial vein while the subject was supine. During the 1984 race, environmental conditions were particularly severe: land air temperatures exceeded 34°C (100°F), Relative Humidity (RH) was high coupled with bright sunlight and high wind [46]. All 64 athletes had normal pre-event sodium levels that ranged from plasma sodium 141.8 mmol/L to 137.8 mmol/L. The post-race sodium levels

ranged from plasma sodium 114 mmol/L to 145 mmol/L. Seventeen of the 64 athletes (27%) were hyponatremic. One of the top ten finishers had a post-race plasma sodium level of 125 mmol/L – indicating that this is not a problem of under-training or inexperience [46]. Hyponatremia was defined as a blood sodium concentration of <135 mmol/L. The pre- or post-race weight of the subjects was not recorded in this study.

In a recently published article, Speedy et al. [59] recommended that weight measurements for all athletes before the race be a compulsory requirement of race registration, and subsequent inclusion of a post-race weight in the athlete's triage assessment if they present for medical care. Speedy et al. [59] also reported that knowing the weight change an athlete has sustained during a race is particularly helpful in deciding whether an athlete's symptoms are due to dehydration or over-hydration.

The interpretation of Hiller has met with opposition from Noakes and others who documented weight gain or diuresis during recovery in hyponatremic athletes and therefore reported that hyponatremia developed from fluid retention or fluid overload [10, 11, 27, 51, 53, 59, 60, 61, 62, 63]. Detailed studies by Nose and colleagues concluded that it is the sodium content of the extracellular space that regulates the extracellular volume [64, 65]. The normal response to uncorrected sodium chloride losses during exercise would be a reduction in the extracellular volume in proportion to the sodium chloride deficit [64, 65]. Noakes has reported that the proponents of the theory that large, unreplaced, sodium chloride losses induce acute hyponatremia of exercise have ignored the fundamental physiological findings of Nose et al. [27].

Alternatively, Noakes and others postulated that hyponatremia developed from fluid retention or fluid overload. What they did not resolve is the issue of the location of the retained fluid. There are two possible mechanisms:

(a) The first possible mechanism is that the "missing" sodium chloride is not irreversibly lost in sweat or urine but is translocated into a 'third space' [27]. Unabsorbed fluid may pool in the gastrointestinal tract (the third space) with movement of sodium ions from the extracellular fluid into the gastrointestinal tract [26, 27]; or
(b) The expansion of the extracellular volume, causing hyponatremia by dilution of a normal or slightly reduced total extracellular sodium chloride content (because of sweat or urine sodium losses or both) [27].

Until recently reports of changes in plasma sodium concentration and the relationship of hyponatremia to changes in body weight had included case studies [10, 11, 60] and smaller studies [58]. The theory of fluid retention was also supported by the findings of Vrijens and Rehrer [28]. The authors showed an inverse correlation between the rate of urine production and the rate of plasma sodium change – urinary losses were less than the mean in the hyponatremic subject [28].

Fluid overload – as a result of an expansion of the extracellular volume was confirmed in the largest observational, cohort study of ultraendurance triathletes [51]. Six hundred and five athletes from the 1997 New Zealand Ironman Triathlon participated in the study by Speedy et al. [51]. Participating subjects were weighed 2-days before the race (because of time constraints) wearing minimal clothing, without shoes (in addition 323 athletes were weighed again immediately prior to the race to establish the extent of any weight change). Subjects were re-weighed post-race at the finish line or at the medical tent wearing race clothing including shoes. The mean weight of a pair of running shoes was made as an adjustment to the post-race weight. Blood was collected by routine venipuncture within 15 minutes of subjects finishing the race. Blood was not collected before the race. Urgent assays for plasma sodium concentrations were carried out on 89 athletes presenting for medical care. Samples collected from 284 well athletes were analysed the following morning. The race conditions were 21°C, 91% RH.

Water, Powerade, and Coca-Cola were the fluids available at the support stations (every 12km on the cycle course and every 1.8km on the run course) [51].

For the purposes of the study, mild hyponatremia was defined as a plasma sodium concentration of 130-134 mmol/L. Severe hyponatremia was defined as a plasma sodium concentration below 130 mmol/L. Athletes who presented to the medical tent with symptoms of hyponatremia and a plasma sodium concentration below 135mmol/L were categorised as "symptomatic" for hyponatremia. Hyponatremic athletes who did not present for medical care after the race were defined as "asymptomatic" for hyponatremia [51].

Full data on pre- and post-race weights, and post-race plasma sodium concentration were available on 330 race finishers (55%) (292 male, 38 female); 58 (18%) of these finishers were hyponatremic. Fluid overload was reported in eight of the 11 athletes (73%) diagnosed with severe hyponatremia (plasma sodium concentration <130 mmol/L). The subjects either gained weight or maintained their pre-race weight. Relative weight change in those athletes with severe hyponatremia ranged from –2.4% to +5%. Lower post-race plasma sodium concentrations were also related to the lower haematocrit levels. This suggested that exercise associated hyponatremia was a result of the expansion of the extracellular space [51]. It was reported in the study by Speedy et al. [51] however, that those athletes defined as 'mild hyponatremic' (plasma sodium concentration 130-134 mmol/L) had a wide range of weight changes; some were overhydrated and some were dehydrated.

In the small 1991 Hawaiian Ironman Triathlon study (30 subject – 26 male, 4 female) O'Toole et al. [58] observed that although the hyponatremic athletes were slightly dehydrated, they lost significantly less weight (fluid) than did the normonatremic athletes. Nine of the 30 athletes (30%) were diagnosed with hyponatremia. Hyponatremia in this study was defined as <130 mmol/L [58]. The

authors suggest that inappropriate retention or compartmentalisation of fluid may occur in combination with mild dehydration and without gross overload [58].

This suggestion may explain the 'mild hyponatremia' seen in the athletes with a wide range of weight changes in the Speedy et al. [51] study. However, hyponatremia defined as <130mmol/L in the O'Toole et al. [58] study, would have classified those athletes with 'severe hyponatremia' in the Speedy et al. [51] study. The majority of the athletes (73%) with severe hyponatremia in the Speedy et al. [51] study were overhydrated not dehydrated as seen in the O'Toole et al. [58] study.

2.4.3 Incidence

The incidence of hyponatremia is unclear, and varies considerably among studies. Part of the problem is definition. Some studies include mild cases (<135 mmol/L) while others only report severe cases (<130 mmol/L) [49].

Environmental conditions also impact on the prevalence of hyponatremia. Hiller et al. [57] reported the incidence of hyponatremia at the 1984 Hawaiian Ironman Triathlon as high as 27%. The land course temperature at the event exceeded 34°C (100°F). Speedy et al. [61] reported hyponatremia to be present in 9% of athletes requiring medical care after the 1996 New Zealand Ironman Triathlon. The land course temperature at the event was between 17-24°C (68-80°F). Both studies defined hyponatremia as plasma sodium concentration <135 mmol/L.

Since the 1980's the drinking practices of competitors in ultraendurance events contrast to those ultraendurance athletes, who, up until 1969 were actively discouraged from drinking during exercise [26]. Indeed, the International Amateur Athletic Federation (IAAF) rules in force in 1953 stated that "refreshments (water was the only drink available) shall only be provided by the organisers of the race after 15 kilometres (km) or 10 miles, and thereafter every 5 km or 3 miles. No other refreshment may be carried or taken by the competitor other than that

provided by the organisers" [66]. Noakes reported the 1991 personal communication between himself and J. Mekler, world record holder at the ultramarathon distance [66]. J Mekler said, *"In those days, it was quite fashionable not to drink, until one absolutely had to. After a race, runners would recount with pride 'I only had a drink after 30 or 40 kilometres'. To run a complete marathon without any fluid replacement was regarded as the ultimate aim of most runners, and a test of their fitness"* [67].

The 1969 Wyndham and Strydom study [68] provided the stimulus to change the IAAF rules governing the conduct of international distance races [26]. The authors measured the rate of fluid ingestion and the levels of dehydration that developed in runners in 32-km footraces and reported that athletes drank less than they sweated [68]. Wyndham and Strydom stated that there was a linear relationship between levels of dehydration of >3% and the athletes' post-exercise rectal temperature [68]. The authors concluded that to prevent heat injury during exercise weight loss, dehydration must be avoided [68]. However, the most dehydrated runners of the 32-km footraces won the races, and despite inadequate fluid intake, showed no evidence of having a greater health risk or being predisposed to heat-stroke [26]. Noakes reported that no study had yet shown that dehydration to the levels present in endurance athletes (1-4% of body weight), exercising under more moderate environmental conditions poses any major health risks [67].

Several authors have reported that it is metabolic rate that determines sweat rate and body temperature during exercise [4, 11]. Barr and Costill stated that fluid loss at a given body weight is greater as running speed increases and that fluid loss at a given running speed increases with body weight [4]. Thus heavier runners will sweat heavily only if they also run fast and maintain high metabolic rates during exercise; in reality, heavier runners tend to run slower than lighter runners [27].

The IAAF rule changes, stimulated by the Wyndham and Strydom study [68], allowed for fluid consumption every 2.5 km after the first 5 km of long distance races [66]. The American College of Sports Medicine (ACSM) supported the rule change and produced a position statement in 1975 that emphasised a high fluid intake during exercise to prevent dehydration [69]. In support of this paper sports medicine practitioners have counselled athletes to avoid dehydration during exercise by drinking more than their thirst dictates, as thirst may be an unreliable index of fluid needs during exercise [61]. The IAAF rules were changed again in 1990 to allow carbohydrate drinks and water to be provided at aid stations, every 3 km, for long distance races [70].

Hyponatremia was not a condition commonly associated with exercise until it was first recognised at the 1981 88-km Comrades Marathon and the 1982 through 1985 Hawaiian Ironman Triathlon [27]. At this time ultradistance events became extremely popular with *Time Magazine* (September 2, 1985) calling triathlon the "sport of the eighties". Participation was reported as 3665 finishers (1981 Comrades Ultra-endurance footrace) and, 4583 starters (1982-1985 Hawaiian Ironman Triathlon) [11, 46]. The popularity attracted an increasingly large number of less competitive participants (the athlete competing in the Ironman Triathlon for example would cycle 180 km in approximately 7 hours (26 km/hour) and run 42 km in 4 hours (10.5 km/hour)) [27]. Noakes has stated that these less competitive athletes were exercising at slower paces so their sweat losses were low (0.7 L/hour) and they were making ample use of the multiple aid stations (provided by the IAAF rule changes), taking plenty of time to stop and drink [11, 27, 70]. The rule changes have increased the average fluid intake during competition from 100ml/hour in the late 60's and early 70's [68] to between 500 – 2000ml/hour from the mid 1980's to the present time [53, 60]. Noakes has postulated that multiple aid stations, combined with the inclusion of less competitive athletes has a close temporal relationship to the exercise associated hyponatremia cases reported in the mid-1980's [27].

2.4.4 Incidence of hyponatremia at New Zealand Ironman Triathlon over 3 consecutive years (1996-1998)

Hyponatremia was defined as (plasma sodium <135 mmol/L) for all three races.

Environmental conditions for the three races were comparable. At the 1996 race, ambient air temperature was between 17-24°C with a relative humidity (RH) 97.6%. Ambient air temperature was 21°C with RH 91% for the 1997 race and 19.4°C with RH 87% at the 1998 race (Table 2.1) [51, 59, 61].

Table 2.1 Race conditions for three consecutive New Zealand Ironman Triathlons (1996 – 1998)

					Sports Drink			
NZ Ironman	Temperature	Relative Humidity	Aid Station Frequency		Brand	Sodium		CHO
	°C	%	Cycle (km)	Run (km)		(mg/L)	(mmol/L)	(g/L)
1996	17-24.0	97.5	12	1.8	Sports Schweppes Plus	391	17	7
1997	21.0	91	12	1.8	Powerade	250	10.9	8
1998	19.4	87	20	2.5	Powerade	250	10.9	8

The frequency of the aid stations was the same in 1996 as in 1997, every 12 km on the cycle course and every 1.8 km on the run course [51, 61]. The proprietary sports drink in 1996 was Schweppes Sportsplus containing 391mg/L of sodium (17 mmol/L) and 7% carbohydrate) [61]. The sports drink available in 1997 was Powerade (containing 250mg/L of sodium (10.9 mmol/L) and 8% carbohydrate) (Table 2.1) [51].

One hundred and nineteen of the 689 race starters in the 1996 race sought medical care (17%). Consent was obtained from 95 of those athletes to use their medical information for the study. A diagnosis of hyponatremia was made in eight

of the 95 athletes presenting for medical care (9%). Hyponatremia was calculated in 1.2% of race starters (Table 2.2) [61].

Table 2.2 Incidence of hyponatremia (HN) over three consecutive New Zealand Ironman Triathlons (1996 – 1998)

NZ Ironman	Number of race starters	Number of athletes seeking medical care	Number of consenting athletes or (complete data on race finishers)	Number of athletes with HN	Percentage of HN found in consenting athletes	Percentage of HN found in race starters
1996	689	119	95	8	9.0	1.2
1997	660	115	330	58	18.0	8.8
1998	650	134	117	4	3.4	0.6

Six hundred and five of the 660 race starters in the 1997 race consented to participate in a hyponatremia study. Complete data was available for 330 consenting participants (292 men and 58 women). One hundred and fifteen of the 660 race starters in the 1997 race sought medical care (17%), twenty-six of these athletes (23%) were hyponatremic. Fifty-eight of the 330 athletes with complete data available were hyponatremic (18%). Hyponatremia was calculated in 8.8% of race starters (Table 2.2) [51].

One hundred and thirty four of the 650 race starters at the 1998 race sought medical care (20%). One hundred and seventeen of the 134 consented to having their medical information used for the study. Four of the 117 athletes presenting for medical care (3.4%) were hyponatremic. Hyponatremia was calculated in 0.6% of race starters (Table 2.2) [59].

There was a significant reduction in the number of athletes receiving medical care for hyponatremia from 1997 to 1998. The drinking practices of the athletes

were altered from 1997 to 1998. The frequency of aid stations was decreased on the cycle course from every 12km to every 20km, and the frequency of the run course aid stations decreased from every 1.8km to every 2.5km (Table 2.1) [59]. Speedy et al. [59] conclude that although it is difficult to prove a causal relationship, the interventions (reduced access to fluid and warnings of the dangers of drinking too much) were associated with a significant reduction in the number of athletes diagnosed with hyponatremia. The authors also acknowledged that confounding factors, including the awareness of the research project on the incidence of hyponatremia, may have accounted for the greater percentage of athletes who had plasma sodium concentrations measured in 1997 than in 1998 [59].

The incidence of hyponatremia in female ultradistance triathletes competing in the 1998 New Zealand Ironman was not specifically documented in the published material. However, in the 1996 New Zealand Ironman there were 83 female race starters. Nineteen of the 95 athletes who consented to have their medical information studied were female. Three of the 19 female athletes were diagnosed with hyponatremia (16%). Three of the 83 female race starters were hyponatremic (3.6%) (Table 2.3) [61]. Permission was granted from the Auckland Ethics Committee and the author, Dr Dale Speedy, to review collected data from the 1997 New Zealand Ironman. There were 79 female race starters in the 1997 event. Complete medical information was available for 38 female participants (48%). Seventeen of the 38 female athletes were hyponatremic (45%). Seventeen of the 79 female race starters were diagnosed with hyponatremia (22%). There was complete medical information gathered on 22 New Zealand female ultradistance triathletes competing in the 1997 New Zealand Ironman. Ten New Zealand female athletes of the 38 female participants with complete medical information were hyponatremic (26%). Ten of the 22 New Zealand female athletes were diagnosed with hyponatremia (45%). Ten New Zealand female athletes of the 79 female race starters were hyponatremic (13%) (Table 2.3).

Table 2.3 Incidence of hyponatremia (HN) in female triathletes competing in the 1996 and 1997 New Zealand Ironman Triathlons

NZ Ironman	Number of female race starters	Number of consenting female athletes or (complete data on race finishers	Number of female athletes with HN	Percentage of HN found in consenting female athletes with complete medical data	Percentage of HN found in female race starters
1996	83	19	3	16	3.6
1997	79	38 22 consenting NZ female athletes with full data	17 10 NZ female athletes with HN	45 10/38 = 26 10/22 = 45	22.0 13.0

2.4.5 Hyponatremic encephalopathy

Women and men are equally likely to develop hyponatremia and hyponatremic encephalopathy post-operatively [71]. However, in a case-control study, Ayus et al. [71] observed those of the 34 case patients who developed permanent brain damage or died 33 (97%) were women. Among the women with brain damage, 25 (76%) were menstruant. Case patients included 40 women (62%) and 25 men (38%) – with postoperative hyponatremic encephalopathy; control patients included 367 women (54%) and 307 men (46%) - who had postoperative hyponatremia without encephalopathy. The much higher mortality from postoperative hyponatremia in menstruant women relative to that of postmenopausal women (or men) was thought to result from a diminished ability of the female brain to adapt to hyponatremia by limiting the amount of brain swelling [71]. As a person ages, brain volume declines progressively, whereas skull size remains constant in adult life. Thus, elderly people of either gender

have more room in the skull for the brain to expand than do younger people. The increased risk for menstruant women to develop hyponatremic encephalopathy did not depend on either the speed of development or the magnitude of the hyponatremia. The authors suggest that since elevated plasma levels of vasopressin are essentially a universal postoperative occurrence (2-4 days postoperative) it may be important to avoid the use of hypotonic intravenous solutions in the immediate postoperative period [71].

2.5 The Female Athlete

Very little work has focused on the influence of hormonal cycles on exercise performance. The female athlete is exposed to a rhythmic variation in either endogenous hormones (as during a regular ovulatory menstrual cycle), or exogenous hormones (administered as the oral contraceptive pill). Given that oestrogens and progestogens can have individual or interactive effects on a variety of metabolic processes, the potential exists for an influence on athletic performance [72].

2.5.1 Menstrual Phases

For women aged 20 to 40 years an average menstrual cycle lasts 28 days but may range from 20 to 45 days [73, 74]. The menstrual cycle is typically divided into three phases. The first, the period of menstrual bleeding *(menses)*, is called the *menstrual phase* and occurs from Days 1 to 4 (or 5). Menstrual bleeding, or *endometrial regression*, is usually referred to as the first phase of the cycle because of the simplicity of noting Day 1 (it actually marks the completion of the menstrual cycle). The second phase is called the *follicular, proliferative or regenerative phase*. This phase is characterised by the development of a mature follicle under the primary influence of the gonadotropins, *luteinising hormone* **(LH)** and *follicle-stimulating hormone* **(FSH)**, and by a thickening of the endometrial lining of the uterus under the influence of oestrogen. The third phase is dominated by the hormone progesterone and is called the *luteal,*

progestational, or secretory phase. This phase follows ovulation and continues until menstrual flow occurs again [75].

2.5.2 Neuroendocrinology of the menstrual cycle

The menstrual cycle is under the hormonal control of the endocrine system as well as the neurogenic stimulus of the nervous system [75]. This interaction occurs primarily between the hypothalamus and the pituitary gland [75]. The hypothalamus, located beneath the cerebral hemisphere, secretes *gonadotropin-releasing hormone* **(GnRH)** into the hypothalamic-pituitary portal vessels. The blood to the anterior pituitary gland transports GnRH, where it stimulates the release of the anterior pituitary hormones, LH and FSH [75]. Cyclic fluctuations of LH and FSH act on the ovary to release the steroid hormones, oestrogen and progesterone [75].

Estradiol (E2) is the major oestrogen, while estrone (E1) and estriol (E3) are less potent oestrogens [72]. The primary function of the oestrogens in the menstrual cycle is to stimulate the growth of the uterine endometrium. Oestrogens also exert major feedback effects on the secretion of GnRH, LH and FSH (complicated positive and negative feedback mechanisms) [75]. The facilitation of calcium uptake into bone, as well as the fat deposition around breasts, buttocks and thighs, is promoted by oestrogen [72]. Oestrogens contribute to a protective effect against atherosclerosis by decreasing the total and low-density lipoprotein **(LDL)** cholesterol levels and by increasing the high-density lipoprotein **(HDL)** cholesterol level [72].

The main function of progesterone is to prepare the endometrial lining of the uterus for the implantation of a fertilised ovum (egg) [75]. Like oestrogen, progesterone also exerts a feedback effect on the secretion of GnRH, LH and FSH [75]. The increase in progesterone levels during the luteal phase is responsible for:

(a) The biphasic basal body temperature curve and increased core body temperature (of 0.3°C – 0.5°C); and
(b) Greater basal metabolic rate during this time [72, 73].

Progesterone is believed to also contribute to postovulatory fluid retention through a complex feedback mechanism involving aldosterone and the renin and angiotensin system [73]. Under the influence of progesterone there is an increased excretion of water and sodium from the kidney as a consequence of antagonism of aldosterone. The resultant stimulation of the renin and angiotensin system then paradoxically increases aldosterone secretion and promotes an increase in ADH [72] and thus water re-absorption [6].

Twenty-eight studies were performed on thirteen normotensive male volunteers (22-44 years of age) to determine the effect of progesterone on renal sodium handling in man [76]. Male subjects were chosen to avoid cyclic changes in endogenous oestrogen and progesterone secretion. It was demonstrated that administration of progesterone consistently produced an increase in sodium excretion to normal subjects on either low (13-40 mmol of sodium/day) or high (240 mmol of sodium/day) sodium intakes and that progesterone inhibits sodium reabsorption in the proximal tubule in the nephron [76].

Water conservation via alteration in osmotic regulation of plasma ADH by endurance training was also suggested in twelve males (24-49 years of age) participating in the case-control study by Freund et al. [77]. The study addressed ADH levels in relation to hydration status at rest in six endurance-trained and six untrained subjects. The endurance trained subjects regularly ran 32miles/week (50km/week) and were capable of running 10km in under 40 minutes. No differences between groups for base-line hormonal, electrolyte or renal measurements were found. Following the ingestion of 1% of lean body weight as water, there were no significant differences in plasma aldosterone or renin activity between the groups. However, ADH levels were higher in trained than

untrained subjects at 30 minutes post-drink. The higher plasma ADH values in the trained subjects is probably responsible for the blunted diuresis and is in spite of similar plasma osmolality values between the groups.

The majority of previous studies examining hormonal and renal responses have used male subjects. Recent developments in our understanding of an athlete's physiology (mainly in relation to the male endurance runner) have revealed new areas of interest that need to be assessed with specific reference to the female athlete [74].

Sixteen females (18-37 years of age) who had been running a minimum of 35 miles/week (56km/week) for at least 1 year participated in the observational study by De Souza et al. [78]. The effects of menstrual cycle phase (early follicular vs. midluteal) and menstrual status (eumenorrhea vs. amenorrhea) on plasma arginine vasopressin **(AVP)**, renin activity, and aldosterone were studied before and after 40 minutes of submaximal running (80% maximal O2 uptake).

Eumenorrhea was defined as the consistent recurrence of menstruation at intervals of 23-33 days. Amenorrhea was defined as the absence of menstruation for 3 or more consecutive months. Menstrual phase was associated with no significant differences in pre-exercise plasma AVP or renin activity in the eumenorrheic runners. Plasma AVP and renin activity were significantly elevated at 4 minutes after exercise in the eumenorrheic runners during both menstrual phases and returned to pre-exercise levels by 40 minutes post-exercise.

Menstrual status was associated with no significant differences in pre-exercise AVP or renin activity, however amenorrheic runners had elevated resting levels of aldosterone. Post-exercise responses in the amenorrheic runners were comparable with the eumenorrheic runners during the early follicular phase. Pre-exercise plasma aldosterone was significantly elevated during the midluteal phase in the eumenorrheic runners. Plasma aldosterone, post-exercise resulted

in a greater response during the midluteal phase, and was greater when compared to the amenorrheic runners [78]. The authors suggested that progesterone was the factor responsible for the observed differences, because progesterone is highest at this time [78]. Other studies have also shown that plasma aldosterone peaks during the midluteal phase of the menstrual cycle due to elevated levels of progesterone [79, 80].

In a more recent observational study, twelve healthy adult, pre-menopausal, normal-cycling women, aged 20-23 years, were studied over the follicular (6-10 days) and luteal (19-22 days) phases of the menstrual cycle to determine AVP responses to exercise [81]. Peeke and colleagues demonstrated similar findings to that of De Souza et al. [78]: there was a significant increase in AVP concentrations after exercise, in both phases of the menstrual cycle, but the responses in the two phases were not significantly different [81]. Aldosterone was not measured in this study.

2.5.3 Heat tolerance and thermoregulation

Women generally have a large surface area to mass ratio, relatively great adiposity, and a menstrual cycle. All these properties may influence thermoregulation with regard to its effectiveness and therefore may affect heat tolerance [82].

The phase of the menstrual cycle has an impact on thermoregulation in women [83]. During the luteal phase of the menstrual cycle, core temperature is approximately 0.4°C higher than during the follicular phase, at least in non-heat acclimatised women [83, 84].

The adaptations that occur with heat acclimatisation include decreased heart rate, decreased rectal temperature, increased plasma volume, and increased sweating rate [83]. Heat tolerance depends on the cardiovascular fitness of an individual more than gender. Young girls tolerate exercise in hot climates less

effectively than adult women because of the young girls' larger ratio of surface area to body mass and slower onset of sweating and sweating rate [83].

Hessemer and Bruck documented differences between the follicular and luteal phases in thresholds for sweating [85]. Ten healthy adult, pre-menopausal, normal-cycling women, aged 20-29 years, were studied over the follicular (4-7 days after the onset of menstrual flow) and luteal (4-8 days after elevation of waking rectal temperature) phases of the menstrual cycle. Cycle phases were verified by progesterone determinations in venous blood. The ten women exercised for 15 minutes on a cycle ergometer in the mid-luteal phase and early follicular phase at the same constant work rates (mean 122 W) and an ambient temperature of 18°C [85]. Exercise was performed between 3:00am and 4:30am (after a 4-hour resting period), when the luteal-follicular difference in body temperature is at its maximum. Pre-exercise oesophageal, tympanic and rectal temperatures averaged 0.6°C higher in the luteal phase. During the exercise period these temperatures were significantly higher in the luteal than the follicular phase (average 0.5°C). The thresholds for chest sweating and cutaneous vasodilation at the thumb and forearm were elevated in the luteal phase by an average of 0.47°C. The heart rate level during the exercise was significantly increased in the luteal phase by an average of 6.2 beats per minute. The mean exercise VO2 in luteal was 5.2% higher than in follicular, the metabolic rate was increased in luteal by 5.6%, but the net efficiency was 5.3% lower [85].

The authors reported that the elevated vasodilation and sweating thresholds could account for the observed postovulatory increases in core temperature during rest, as well as for the sustained temperature elevation during exercise [85]. Hessemer and Bruck also stated that the higher heat production during exercise in the luteal phase might also have contributed to the temperature elevation [85]. The authors alternatively postulated that the 5.6% elevation in exercise metabolic rate may be secondary to the 0.5°C higher core temperature

which could explain the increase in heart rate by 6.2 beats per minute in the luteal phase [85].

It was concluded from the Hessemer and Bruck study that during the luteal phase, short-lasting heavy exercise (15 minutes) is performed with higher core temperature and higher thermoregulatory, circulatory and aerobic-metabolic strain than during the follicular phase [72, 85].

Others have noted that the tests by Hessemer and Bruck were conducted between 3:00am and 4:30am when the deep body temperature difference between menstrual phases is at its maximum, and thus has little consequence in the sporting world [74].

In a more recent study involving short-duration heavy exercise, Lynch and Nimmo studied 15 women (five women using low-dose, monophasic oral contraceptive (OC) agents and ten normally menstruating women (Non-OC)) to determine the effect of OC's and the menstrual cycle (MC) on intermittent exercise performance [86]. The intermittent exercise involved repeated 20-second sprints starting at 14.3km/h, increasing by 1.2km/h until exhaustion, on an incline of 10.5% [86]. Like the results found in the Hessemer and Bruck study [85] temperatures were higher in the luteal phase when compared to the follicular phase of the menstrual cycle. Unlike the increase in heart rate by 6.2 beats per minute in the luteal phase reported in the Hessemer and Bruck study [85], Lynch and Nimmo revealed no significant difference in heart rate between menstrual phases or between the groups [86]. The study by Lynch and Nimmo [86] is in accordance with the majority of reports published to date, that state that heart rate, both at rest and during exercise, is not affected by the menstrual phase [87, 88, 89].

These reports however have all been completed using short duration activity. The current literature may not be applicable to extreme conditions, where there

may be potential implications for women participating in prolonged endurance events (e.g. marathons, ultra-marathons, and triathlons) at high ambient temperature during the luteal phase of their cycle [73].

Pivarnik et al. [90] investigated whether menstrual cycle phase would affect temperature regulation during an endurance exercise bout at 22°C with 60% RH. Nine eumenorrheic, aerobically trained, heat acclimated women aged 27.2 ± 3.7 years performed 60 minutes of cycling exercise at 65% of peak VO2, and subjects were tested in both follicular and luteal phases [90]. Sweat loss, VO2 and skin temperatures were not affected by menstrual cycle phase. Pre-exercise rectal temperature was 0.3°C higher during the luteal than during the follicular conditions, and this difference increased to 0.6°C by the end of the exercise. Pivarnik and colleagues reported that heart rate during luteal conditions were 10 beats greater when compared to follicular conditions [90]. These results support the findings of Hessemer and Bruck [85]. De Souza et al [88], found no menstrual phase effect on heart rate response in a group of runners who performed a 40 minute treadmill exercise at 80% VO2 max. However, neither ambient conditions nor pre-exercise heart rate values were reported. Although Pivarnik et al. [90] did not document alterations in performance, higher ratings of perceived exertion at the same level of exercise during the luteal phase as compared with the follicular phase were reported.

Pivarnik et al. [90], concluded that temperature regulation, cardiovascular strain and perceptual responses to steady-state exercise were adversely affected during the luteal phase of the menstrual cycle. Because the subject's VO2 values during exercise were similar in follicular and luteal conditions, a difference in heart rate response suggests that the subjects were exercising at a higher percentage of their aerobic capacities in the luteal phase. Each subject's peak VO2 was measured only once, and tests were performed randomly with respect to the menstrual cycle – therefore it can not be said for sure that VO2max was not affected by the experimental conditions' [90]. Subjects were not able to

achieve thermal equilibrium during exercise performed after ovulation, despite similar sweat loss and skin temperatures in follicular and luteal conditions [90]. Pivarnik et al. [90] suggested that the effects were mediated by progesterone, because they were not seen in a subject whose P4 levels did not increase after her lutenizing hormone surge had occurred.

It has been postulated by Pivarnik et al. [90] there may be potential implications for prolonged athletic activity at high ambient temperature, as the woman is beginning to exercise at an already elevated core temperature.

2.6 Body Composition

The ability to accurately determine body composition would greatly enhance health-professionals understanding of the nutritional status of population groups. Methods for the *in vivo* assessment of body composition have progressed considerably in recent years. There are now many techniques available to estimate body composition in humans. There is anthropometry, bioelectrical impedance analysis **(BIA)**, hydrodensitometry, dual energy X-ray absorptiometry **(DEXA)** and total body potassium **(TBK)**. Many of these procedures have been limited to laboratory and clinical environments due to cumbersome, time consuming, expensive procedures. The development of newer procedures such as BIA, are more appropriate for field use and epidemiological studies [91].

2.6.1 Bioelectrical Impedance Analysis (BIA)

Bioelectrical impedance methods have the advantage of convenience, rapidity and non-invasiveness [92]. Bioelectrical impedance analysis is based on two theories:

1. The body consists of two compartments:

(a) A lean compartment which contains virtually all the water and conducting electrolytes of the body; and

(b) A fat compartment which contains little water and is hence non-conducting.

2. The Thomassett [93, 94] principles that state that the electrical impedance of a single geometric system is a function of conductor configuration, length, cross-sectional area and measurement signal frequency [91, 95].

The accuracy and precision of the BIA method are affected by instrumentation, subject factors, technician skill, environmental factors, and the prediction equation used to estimate fat-free mass **(FFM)** [96].

Multiple regression analysis has been used extensively to derive prediction equations to estimate body composition [97]. The publication of many regression equations for men [98, 99, 100, 101] and women [98, 99, 102, 103, 104] have resulted from the variance of body composition and anthropometric measures related to age and gender. Many of the regression equations however were derived on sample sizes of normally less than 75 [97]. Cooley and Lohnes have stated that equations derived on sample sizes of under 200 subjects should be viewed with caution [105].

If BIA is to be of use in estimating body composition, it must be accurate and valid. Lukaski et al. [106], and Segal et al. [107] have published conflicting data on the validity of the BIA method. Lukaski et al. [106] reported their standard errors for estimating **(SEE)** hydrostatically measured percent body fat **(% fat)** with BIA were low: 2.7% fat for males and 3.1% fat for females. The authors used gender-specific regression equations that estimated fat-free weight **(FFW)** from the ratio of height squared to bioelectrical resistance (ht2/*R*). In contrast, Segal et al. [107] reported SEE of 6.1% fat for a combined sample of men and women, and used empirically derived equations provided by the manufacturer of the BIA system (R. J. L. Systems, Detroit, MI).

Jackson et al. [108] designed a study to examine the reliability and validity of the BIA method and compare its accuracy with results obtained by standard anthropometric methods. BIA, skinfold fat, and hydrostatically measured % fat were obtained on 44 female and 24 male subjects, and each subject was tested four times by two testers on two different days [108]. An additional 26 men (n = 50) and 38 women (n = 82) were tested once and combined with the data used for the reliability analysis to cross-validate BIA estimates of % fat with hydrostatically determined % fat [108]. The BIA prediction accuracy reported by Lukaski et al. [106] was not confirmed by the Jackson et al. study [108]. The SEE for the BIA methods ranged from 4.6 to 6.4% fat compared with 2.6 and 3.6% fat for the sum of seven (Σ7) skinfolds equations [108]. The results of the Jackson et al. study [108] are in agreement with the findings reported by Segal et al. [107], who showed that the BIA method was less accurate than other anthropometric models. The authors stated that the results suggest that weight/height2 is the major source of variance in BIA prediction models [108].

2.6.2 *BIA and the female subject*

The study by Eaton et al. [109] compared the accuracy of four methods to assess body composition of seventy-seven caucasian women (age: 31.8 ± 8.6 years, weight: 59.5 ± 9.1kg, height: 162.4 ± 6.9 cm). Compared to % fat with hydrostatic weighing (24.9 ± 6.5%) an analysis of variance revealed no mean difference to BIA (25.7 ± 5.8%) and the SEE value for the BIA method was 4.2%. The authors concluded that the mean % fat differences between hydrostatic weighing and BIA were small and would allow for its use in assessing body composition even with a SEE value of 4.2% [109]. The authors also reported that % fat obtained from the BIA machine readings versus those computed from the manufacturer's equation indicated significant differences (machine 25.7%, equation 27.8%) [109].

In a more recent study Stout et al. [110] did not support the suggestion of Eaton et al. [109] that the equation supplied by the manufacturer is not the same as used by the BIA machine to calculate % fat. The estimated % fat values from the

BIA machine and BIA equation were highly correlated (r = 0.97) [110]. Stout et al. [110] investigated the validity of methods for estimating % fat in 41 non-athletic young women (age: 20.1 ± 2.3 years). The authors reported that the limited number of studies on the validity of BIA procedures [108, 109, 111] for estimating % fat in women have resulted in disparate findings. Results from the Stout et al. [110] study found Total Error **(TE)** values ranging from 4.3 to 7.2% fat, which indicated that the errors associated with all the BIA equations were substantially greater than those from (∑7: 2.3% fat) and (∑3: 2.4% fat). The authors reported that the accuracy with which BIA equations can estimate % fat may be partly a function of subject characteristics or the difference in BIA machines used for various investigations [110].

Adult women often report noticeable fluctuations in body weight **(BW)** related to the menstrual cycle. Temporary water retention has been assumed as the cause of the weight gain [111]. Because water content is the largest component of the fat-free body **(FFB)** (approximately 73%) it can have a significant influence on the FFB [112]. Thus, temporary increases in body water can affect a female's body density and lead to variable estimations of body fatness when using an indirect method such as hydrostatic weighing **(HW)** [112].

Bunt et al. [112] investigated the possibility that variability in body weight in females due to water retention causes differences in body density **(Db)** values determined by HW. Seven females were measured when they felt they were at their lowest (LO) and highest (HI) body weights (BW) during a menstrual cycle [112]. Significant mean differences were found in BW (kg) (LO = 58.9, HI = 61.1), Db (g. cc –1) (LO = 1.0430, HI = 1.037), and % fat as determined by HW alone (LO = 24.8%, HI = 27.6%) [112]. The authors concluded that changes in total body water (TBW) can in part result in significantly different Db values obtained from HW in females who did experience perceptible changes in BW during the menstrual cycle [112]. Lohman [113] has shown that variation in water content of

the FFB is the greatest contributor to variation in the density of the FFB among subjects.

In contrast to the findings by Bunt et al. [112], Lebrun et al. [89] found no significant differences in weight or % fat when 16 eumenorrheic women had their body composition measured by HW.

Eating, drinking, dehydration, exercise, and menstrual cycle stage may affect BIA measures [96]. Individual females who experience significant changes in BW during the menstrual cycle are believed to have varying states of hydration [114]. Fluctuating levels of estradiol and progesterone are believed to be the cause of this and are related to their influences on sodium and water retention [114]. Such fluid alterations may be expected to impact on BIA since the method relies on distinguishing hydrated lean body mass (LBM) or TBW from relatively anhydrous fat [106].

There have been conflicting results reported in the two studies that assessed the effects of the menstrual cycle on BIA. Chumlea et al. [115] investigated the effects of timing of the menstrual cycle and of oral contraceptive usage on BIA in 29 women aged 21 to 38 years (11 women were taking oral contraceptive). The type of oral contraceptive (combination or progestogen only) was not discussed in the paper. The ratio of height squared to bioelectrical resistance (ht2/*R*) did not vary significantly during the menstrual cycle. However, the detection of significant variations was reduced because parts of two successive menstrual cycles were used instead of one complete menstrual cycle.

In a more recent study Gleichauf and Roe [116] reported that BW and resistance changed significantly between the immediate post-menstruum and both the menses and the pre-menstruum in 25 women aged 20 to 41 years. The authors stated that the validity of the BIA technique for assessing body composition remains unconfirmed [116]. The study's results suggested that the effect on

resistance and fat-free mass estimates obtained during the menstrual cycle are related to changes in body weight, presumably caused by changes in water retention [116].

McKee and Cameron [117] included male control subjects in an attempt to discern variations in BIA associated with the menstrual cycle in women from those due to other physiological variables affecting both sexes. Body weight and BIA were measured two to five times per week for one menstrual cycle (21-34 days) using 42 women (6 taking an oral contraceptive, OC), aged 19.0-34.4 years, and for 22-32 days using 28 men aged 18.9-24.1 years [117]. Body weight in the non-OC women decreased significantly between menses and the late follicular phase, and increased significantly between the late follicular phase and the pre-menstrum. These changes were not correlated with matching BIA changes. BIA in the non-OC women, and body weight and BIA in the OC women and the men, did not differ significantly over the measurement period [117]. The authors concluded that body composition assessments based on BIA are not affected by the menstrual cycle [117].

Thorn et al. [111] have also reported that females who experience significant changes in BW may encounter fluctuation in appetite (due to changes in estrogen and progesterone levels) and this may lead to significant shifts in caloric intake.

2.7 Dietary Assessment

It is useful to evaluate the dietary intake of the athletic population as they may have different eating habits to the general population – in terms of food choices, serve sizes, frequency of consumption, food preparation and even language about food. Athletes, particularly adolescents and those involved in endurance training, have higher nutrient requirements and turnover of nutrients than the sedentary population [118].

Dietary evaluation involves collecting information on dietary intake and evaluating and interpreting dietary intake using the 'common' reference guidelines or standards available [118]. Dietary intake can be assessed by a number of data collection methods. Current food consumption methods include 1-7 day food record/diaries (using weighed – scales or estimated – household measures), or duplicate food collection [118]. Methods for collecting food consumption in the past (retrospectively) include 24-hour recall, food frequency questionnaires (FFQ) and diet histories (combination of 24-hour recall and FFQ) [118].

The diet record is considered the most 'accurate' and feasible method for research [119]. Although the weighed diet record is considered the 'gold' standard, a recent review of dietary intakes of athletes by Burke et al. [120], reported that 3 or 4 day diet records using household measures were predominantly the method of choice. Other authors have also reported that the estimated method using household measures was acceptable for research because of better compliance than the weighed method [121, 122]. It has been suggested that seven days of continuous recording was the most accurate method [123]. However, periods of recording for longer than 3 to 4 days have shown reduced accuracy, associated with memory interference, incomplete records and a high drop-out rate [124].

Collection of reliable and accurate dietary intakes of individuals and groups is difficult because of the influence of confounding effects and errors inherent in all dietary survey methods [118]. A number of errors are introduced at each stage of dietary assessment: from collection of food intake data to analysis and interpretation of these data [118].

There are several limitations when collecting nutritional data via the estimated method using household measures. Diet records are not representative of measuring usual diet unless repeated several times, two to three months apart [125]. Self-recording food intake changes an individual's eating behaviour by

discouraging snacking, inhibiting spontaneous food selection and consumption of mixed meals (because of difficulties estimating individual ingredients) [118]. Under-reporting of actual food intake either intentionally or unintentionally is higher in food records than other methods. Authors have reported that energy intake can be under-estimated by 20-50% [126, 127]. Under-reporting of food has been documented especially for women who are overweight [128], and endurance female athletes [129, 130].

The number of days needed to measure nutrient intakes reliably varies for different subjects and different nutrients. Basiotis et al. [131] have provided estimates for the number of days required to measure true average intake of a range of nutrients with given statistical confidence (95% confidence). Intakes of protein, fat and CHO required four, six and five to six days respectively to estimate true average intake in both males and females [131]. The accuracy of estimated dietary intake might also be affected by seasonal changes in food intake due to variation in food supply [132].

The number of days required to measure true average intake of micronutrients is much longer. Due to significant intraindividual variability, 5 to 10 days of urine or food collection, or more than 14 days of dietary recalls are necessary to characterise dietary sodium intake [133, 134]. Mean sodium intake obtained from food analysis and food composition tables were correlated to the measurement of urinary sodium excretion in a study by Sowers and Stumbo [135].

3 Aims of the Study

This study investigated the nutritional, biochemical, hormonal and physical status of New Zealand female ultradistance triathletes competing in the New Zealand Ironman Triathlon, 3 March 2001. The aim of the study was to determine the causes of hyponatremia in these athletes.

To ensure homogeneity, New Zealand female ultradistance triathletes competing in the New Zealand Ironman Triathlon were selected because gender, heat acclimation, type and intensity of training and level of fitness would be similar.

The first aim of the study was to calculate the nutritional intake of the athletes (via 7-day food diaries) eight weeks and one week prior to competition. The second aim was to assess body composition (using digital scales and bioelectrical impedance) and to determine water content shifts before and after competition. The third aim was to investigate biochemical status (via blood and urine tests) before and after the race. The fourth aim was to determine menstrual status (via blood test and questionnaire) and investigate whether there is a correlation between the incidence of hyponatremia and different stages of the menstrual cycle.

The overall aim of the study was to determine why female ultradistance triathletes are at increased risk from developing hyponatremia and to provide practical advice on how to lower the incidence.

4 Methodology

The subjects for this study were recruited from female athletes who competed in either of the two Half Ironman Triathlon competitions (Taupo Half Ironman Triathlon – Saturday 16 December 2000 and Mt Maunganui Half Ironman Triathlon – Saturday 6 January 2001). Those female athletes who then intended to go on and compete in the New Zealand Ironman Triathlon, 3 March 2001 were asked to participate in the study.

Subjects were invited to participate in the study by the provision of information sheets at the two venues and by contacting triathlon coaches who informed athletes in their regions about the study (appendix 1). The researcher was present at the two Half Ironman Triathlon venues to provide further information about the study. Athletes who agreed to participate gave their informed consent before the start of the study (appendix 2).

All New Zealand women who were participating in the New Zealand Ironman Triathlon, 3 March 2001 were eligible to be included. In total 27 female triathletes were recruited (n=27).

Permission was granted from Ironman New Zealand to use the event and ethical approval was given by the Auckland Ethics Committee (Ethics Committee of the Regional Health Authority) and by the Human Ethics Committee of Massey University, Albany, Auckland.

4.1 Dietary Assessment

Participants were asked to complete two, 7-day food diaries (appendix 3). The first diary was completed 6-7 weeks prior to the New Zealand Ironman Triathlon (approximately 13 January 2001) to evaluate dietary intake at the peak of training. The second diary was completed the week before the race (25 February 2001) to evaluate dietary intake in the taper before the event. Instructions on how

to measure food items and fluid consumed was given both verbally and in writing by the researcher. Metric spoon and cup measures were available for participants to borrow. However, all the participants had their own sets of standardised spoons and cups.

The food diaries were analysed using SERVE-NZ dietary analysis software (M. Williams Pty Ltd, Sydney, Australia) utilising the NZ food database version W3.0, supplied by the New Zealand Institute of Crop & Food Research Ltd, Palmerston North. Nutrient intake from dietary sources as well as from supplement products listed in the data base or for which nutrient content could be obtained (carbohydrate-electrolyte sports drinks, sports bars) were included in the analysis. Several participants were taking supplements/medications that were not listed in the database and these could not be included in the nutrient analysis but are reported separately (appendix 4).

The results from the nutrient analysis were compared with nutrition guidelines for physically active adults and athletes in New Zealand [136] and with the Recommended Dietary Intakes (RDI) for adult New Zealanders [137]. A personal communication with Professor Cliff Tasman-Jones revealed that the joint review of Nutrient Reference Values (NRVs) between New Zealand and Australia had yet to be submitted or adopted. Professor Cliff Tasman-Jones reported that the Nutrition Taskforce (1991) recommended that the revised Australian Dietary Intakes (1990) be adopted as an interim measure [138].

4.2 Body Composition/Blood Pressure

Athletes reported to the testing facility the day before the race, 19 hours before the start (appendix 5). All were in the pre-race taper period and had not exercised strenuously within the previous 12 hours. The pre-race procedure was designed to be similar to the expected post-race conditions. Each athlete walked to the testing site and was weighed in minimal clothing without shoes. All weights

were measured on calibrated digital scales (Tanita 1614, Wedderburn, Japan) on a hard level surface.

Bioelectrical impedance analysis (BIA) (see appendix 6 for technical details of the BIA technique) was used to determine fat mass (FM), fat free mass (FFM) and total body water (TBW) in the athlete pre-race. The BIA monitor was calibrated by an Auckland University of Technology lecturer before the measurements were taken. All participants were in the supine position on a wooden trestle table. Two electrodes were affixed to the right wrist and ankle. Weight and BIA results were recorded on the pre-event/post-event participant identification forms (appendix 7).

During the race, the athletes ate and drank *ad libitum*. No quantification of food or fluid intake was attempted. On completion of the race the study subjects were met at the finish line and escorted back to the testing facility (1-2 minutes). Shoes and socks were removed and the subject was weighed on the same pre-race calibrated scales. Athletes were then helped onto the wooden trestle table and into a supine position where BIA testing was repeated (within 15 minutes of each individual crossing the finishing line). Weight and BIA results were again recorded on the pre-event/post-event participant identification forms (appendix 7).

The athletes had their blood pressure measurement taken by a nurse/phlebotomist in either the sitting or supine postures approximately 19-hours before the race and within 15 minutes of finishing the race. Blood pressure readings were recorded on pre-event/post-event participant identification forms (appendix 7).

4.3 Medical/Hormonal Assessment

Blood samples were collected observing universal precautions for venipuncture by a nurse/phlebotomist from all consenting athletes approximately 19 hours

before the race and within 15 minutes of their finishing the race. Athletes were either in the sitting or supine postures for the venipuncture. Blood samples were immediately transferred into standard collection tubes that were supplied by Medlab. The purple collection tubes contained EDTA and the red collection tubes were devoid of added solution.

The bloods that were collected pre-race were refrigerated between 2 to 8°C and sent by courier to Medlab Hamilton for analysis after centrifugation. The bloods that were collected post-race were stored between 2 to 8°C after centrifugation at the Taupo Medlab centre and were sent to Medlab Hamilton for analysis by courier Monday morning.

A urine sample was also collected approximately 19 hours before the race. A post-race urine sample was collected from those athletes who were able to urinate and stored according to the protocol described at Taupo Medlab centre. Those athletes who were unable to urinate were given urine-collecting equipment and asked to collect their first urine and return to the researcher the following morning. Verbal instruction for the overnight storage of the sample was given to the athlete and/or caregiver. The urine samples collected by the researcher were added to the previously collected post-race urine samples and were sent to Medlab Hamilton for analysis by courier Monday morning.

The blood and urine samples were analysed by Medlab Hamilton for serum creatinine, serum protein, serum sodium, serum albumin, serum globulin, serum urea, urinary sodium and urinary creatinine (Roche/Hitachi Modular Analytics ISE 900/1800 electrolyte measuring unit and P800 photometric measuring unit); haemoglobin, haematocrit, mean cell haemoglobin (MCH), mean cell volume (MCV), neutrophils and monocytes (Coulter GEN•S System automated haematology analyser); lutenizing hormone and progesterone (ADIVA Centaur automated immunoassay analyser); and serum osmolality and urine osmolality (using Osmometer at Waikato hospital). Analysis of lutenizing hormone and

progesterone were completed on the pre-race blood sample only. The co-efficient of variation (CV) of the samples was determined by dividing the standard deviation by the mean and expressing as a percentage. The lower the CV the greater the likelihood that the result was meaningful.

In addition to the pre-race blood sample test for lutenizing hormone and progesterone the participants also completed a menstrual status questionnaire (appendix 8).

4.4 Temperature/Relative Humidity Assessment

Measurement of temperature (°C) and relative humidity (% RH) was recorded simultaneously at the beginning of the race (7am) and throughout the day until (5:30pm) using a HT-800 Thermo-Hygrometer.

4.5 Statistical Methods and delivery of data to participants

The results were calculated as the mean, standard deviation and range. Medical, anthropometrical and nutritional data was correlated and tabled to all the participants in the study for their information (appendix 9).

5 Results

Medical information was completed on 19 of the 27 recruited female athletes (70%).

5.1 Physical characteristics and Demographic Data

In table 5.1 information about age and the number of Ironman Triathlons entered is presented. The mean age of the subjects was 37.8 ± 8.9 years (range of 26-55 years). It was the first Ironman event for nine out of the nineteen participants (47%).

Table 5.1 Demographic data for each subject

Subject number	Age	Age group	Number of Ironman Triathlons
1	28	25-29	1
2	43	40-44	4
3	39	35-39	7
4	29	25-29	1
5	27	25-29	3
6	43	40-44	4
7	40	40-44	1
8	32	30-34	1
9	36	35-39	2
10	37	35-39	2
11	26	25-29	1
12	55	55-59	29
13	52	50-54	3
14	29	25-29	1
15	42	40-44	2
16	29	25-29	1
17	53	50-54	1
18	40	40-44	6
19	38	35-39	1
Mean	37.8		
SD	8.9		

5.2 Body Composition, Bioelectrical Impedance Assessment and Blood Pressure

Information about pre-race height, weight, and body mass index (BMI) is presented in table 5.2. The general pre-race descriptive characteristics of the study group were: height (mean 1.64 ± 0.07 metres, range 1.53-1.84 metres),

weight (mean 62.9 ± 7.1 kg, range 53.4-76.8 kg) and BMI (mean 23.3 ± 2.8, range 19.2-32.8).

Table 5.2 Pre-race anthropometric data for the study group

	Age (years)	Height (m)	Weight (kg)	BMI
Mean	37.8	1.64	62.9	23.3
SD	8.9	0.07	7.1	2.8
Range	26-55	1.53-1.84	53.4-76.8	19.2-32.8

Post-race weight (mean 61.7 ± 6.8kg, range 52.6-74.2kg), weight loss (mean 1.6 ± 1.1kg, range -3.4kg - +1.0kg) and percentage dehydration (mean 2.4 ± 1.8%, range 5.78 to -1.87%) is presented in table 5.3. The mean weight loss of 1.6kg between pre- and post-race was statistically significant ($p = < 0.001$).

Table 5.3 Post-race anthropometric data for the study group

	Weight (kg)	Weight loss (kg)	Percentage dehydration (%)
Mean	61.7	1.6	2.4
SD	6.8	1.1	1.8
Range	52.6 - 74.2	-3.4 – +1.0	5.78 – -1.87

Figure 5.1 shows the pre- and post race weight changes for the study group.

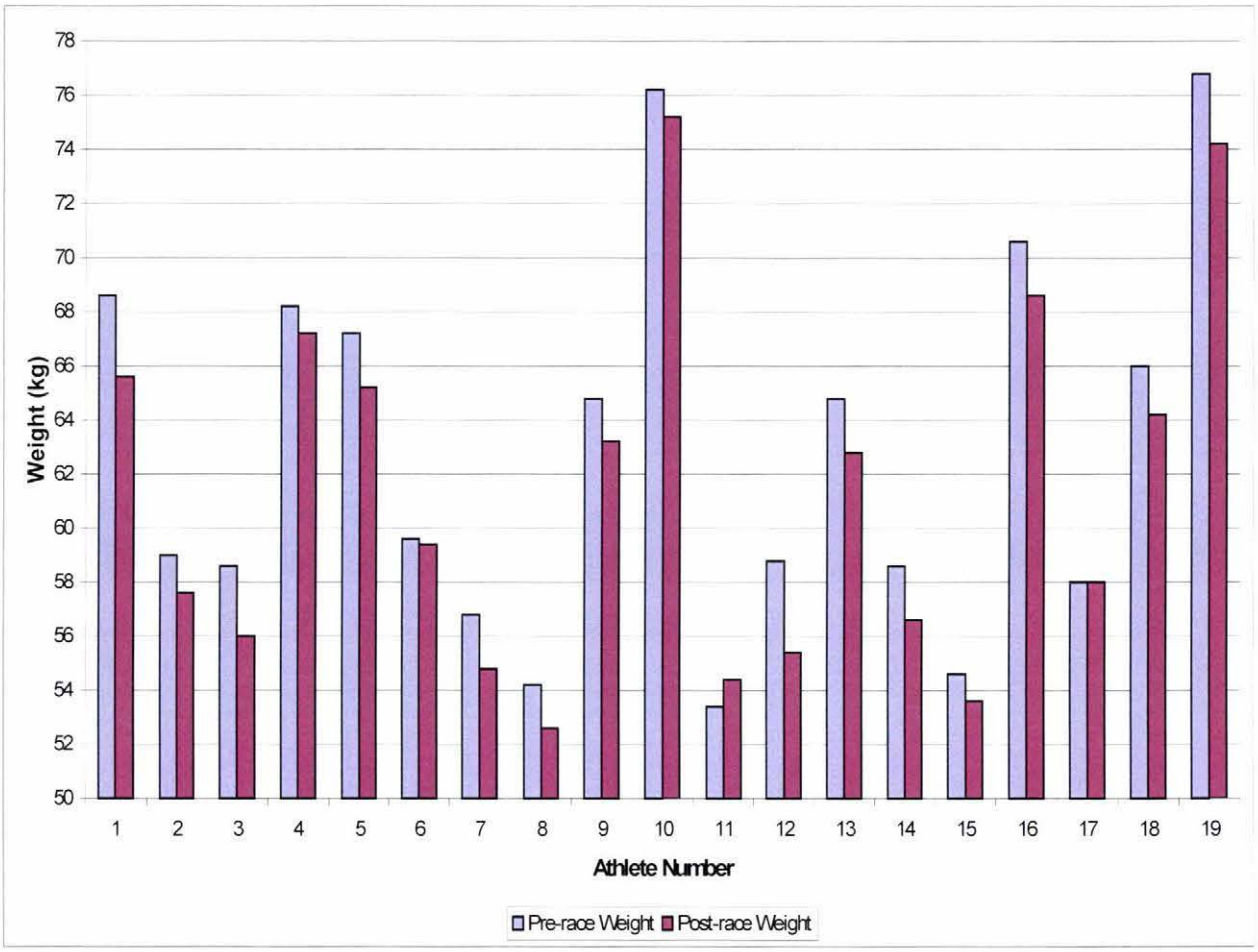


Figure 5.1 Pre- and post-race weight for the study group

In table 5.4 pre-race bioelectrical impedance assessment (BIA) of weight of fat mass (FM), percentage (%) body fat, weight of fat free mass (FFM), % FFM, weight of total body water (TBW) and % TBW is presented respectively.

Table 5.4 Pre-race BIA for the study group

	Fat Mass (FM) (kg)	%Fat	Fat Free Mass (FFM) (kg)	%FFM	Total Body Water (TBW) (kg)	%TBW
Mean	14.8	23.3	48.0	76.6	34.7	55.3
SD	4.5	5.1	5.3	5.1	3.8	3.7
Range	9.1-30.3	16.8-39.4	40.4-63.3	60.5-83.1	29.2-45.7	43.7-60.0

Although the same instrument was used to monitor the changes in the client body composition it was decided to omit the post-race BIA readings due to possible error. There would be an altered state of client hydration due to the fact that the athletes had ate and drank *ad libitum* during the race, had a mean percentage dehydration of 2.4% and had exercised continuously. The mean finishing time for all study participants was 755 minutes (12 hours 35 minutes), the range was 606 minutes (10 hours 5 minutes 36 seconds) to 974 minutes (16 hours 14 minutes 06 seconds).

Information about pre- and post-race systolic and diastolic readings is presented in table 5.5. The mean difference between pre- and post-race systolic was -2.65. The mean difference between pre- and post-race diastolic was +4.7.The mean pre-race blood pressure was 117/74 and the mean post-race blood pressure was 114/79.

Table 5.5 Pre-and post-race blood pressure readings for the study group

Subject	Systolic		Diastolic	
	Pre-race	Post-race	Pre-race	Post-race
Mean	116.59	113.94	74.18	78.88
SD	13.47	11.88	6.56	7.70
Range	100-142	94-138	65-86	70-100

5.3 Medical/Hormonal Assessment

Intake of selected blood tests is summarised in table 5.6.

Table 5.6 Results from pre- and post-race blood tests

TEST	Pre-race Mean ± SD	Post-race Mean ± SD	Difference	Pre-race Range	Post-race Range	Normal Range
Haemoglobin (Hb) (g/L)	132.9±5.6	137.7±7.2	4.8	122.0-143.0	122.0-149	115.0-155
Haematocrit (Hct)	0.40±0.20	0.40±0.20	0.00	0.36-0.44	0.35-0.44	0.33-0.48
Mean Cell Volume (MCV)	94.1±4.2	93.5±4.1	-0.6	88.6-100.9	85.7-100.2	80.0-96.0
Mean Cell Hb (MCH)	31.6±1.5	31.9±1.5	0.3	28.7-34.8	28.7-34.3	25.0-33.0
Total White Blood Cell Count (TWBC)	7.3±1.1	16.5±2.4	9.2	5.2-9.5	11.9-22.0	4-11
Neutrophils	4.5±1.0	13.9±2.0	9.4	2.9-6.2	9.6-17.8	2-7
Monocytes	0.5±0.2	1.0±0.4	0.5	0.2-0.7	0.5-1.6	0.1-1.2
S. Albumin (g/L)	42.9±2.0	45.9±2.2	3.0	40.0-47	43.0-49.0	40.0-50.0
S. Urea (mmol/L)	5.4±1.2	8.7±2.1	3.3	3.2-7.8	5.1-11.5	3.1-7.5
S. Creatinine (mmol/L)	0.09±0.01	0.11±0.01	0.02	0.08-0.1	0.10-0.14	0.08-0.13
S. Sodium (mmol/L)	141.4±1.3	140.8±3.5	-0.6	139.0-143.0	134.0-148	138-145
S. Protein (g/L)	70.5±2.9	75.7±3.5	5.2	63.0-76.0	70.0-82.0	65.0-85.0
S. Globulin (g/L)	27.6±2.2	29.7±2.5	2.1	23.0-31.0	25.0-34.0	25.0-35.0
S. Osmolality (mmol/kg)	292.6±4.7	299.1±8.9	6.5	286.0-306.0	281.0-312.0	280.0-300.0

Figure 5.2 shows the comparison between pre- and post-race plasma sodium concentrations. Athlete 11 recorded a post-race plasma sodium concentration of 134mmol/L (asymptomatic hyponatremia). Athlete 11 had the lowest recorded pre-race plasma sodium concentration of the group 139mmol/L.

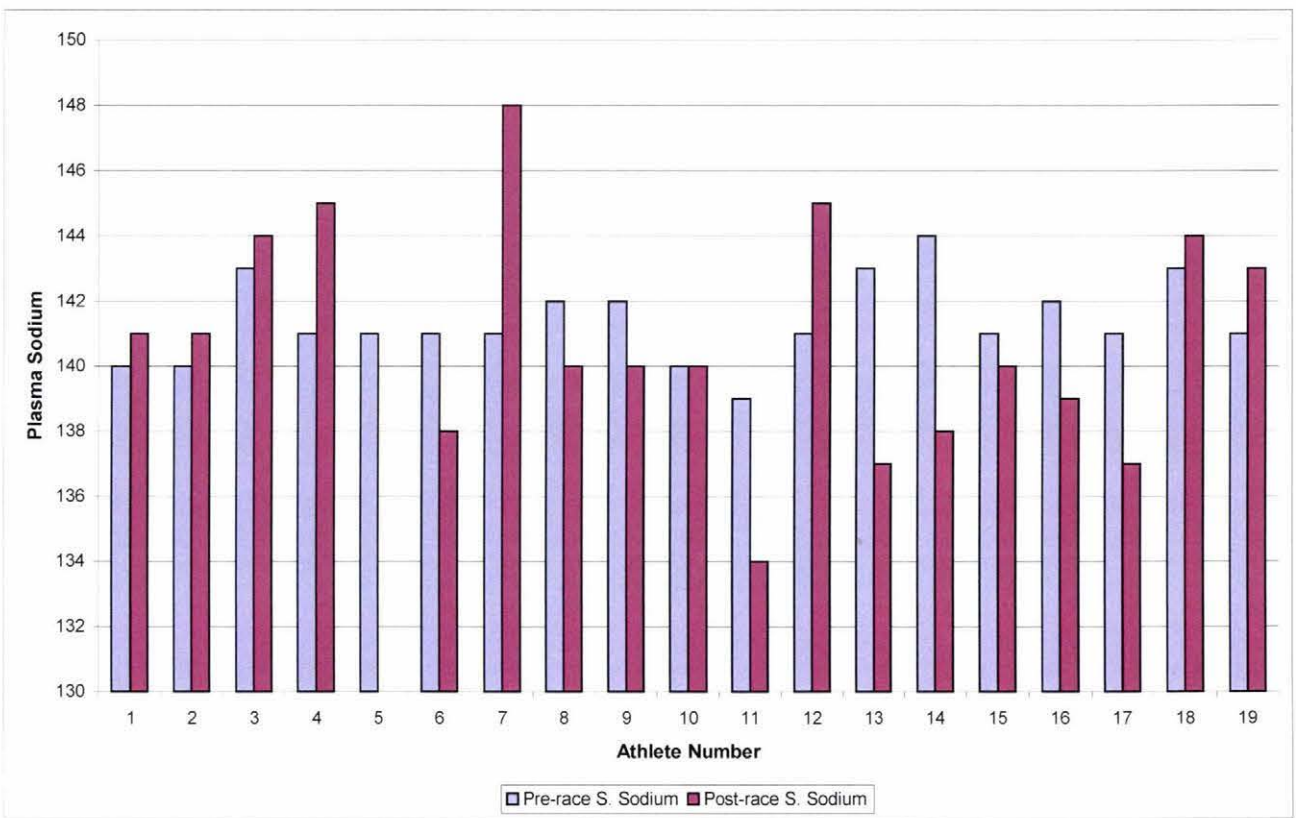


Figure 5.2 Pre- and post-race plasma sodium concentration comparisons

Figure 5.3 demonstrates that the post-race plasma sodium concentrations were related to percentage dehydration.

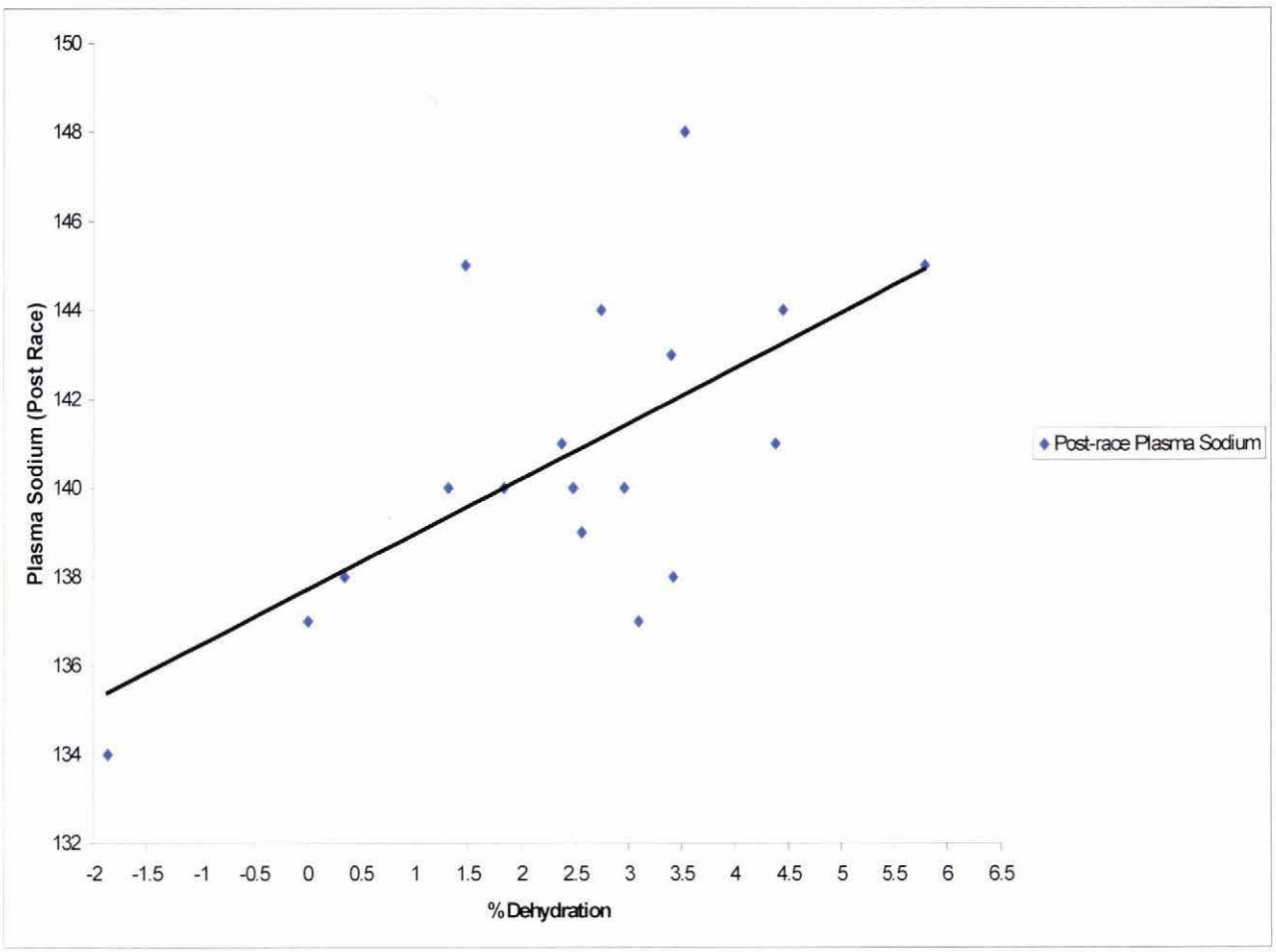


Figure 5.3 Post-race plasma sodium concentration vs percentage dehydration

The athlete with the post-race plasma sodium concentration of 134mmol/L (asymptomatic hyponatremia) had gained weight and was over-hydrated (percentage dehydration of -1.87%).

Figure 5.4 compares the difference in serum albumin of athlete 11 with asymptomatic hyponatremia to the normonatremic athletes (the 18 other athletes in the study). Athlete 11 had a serum albumin difference of -3, compared with the normonatremic athletes who recorded a difference in mean serum albumin of +3.

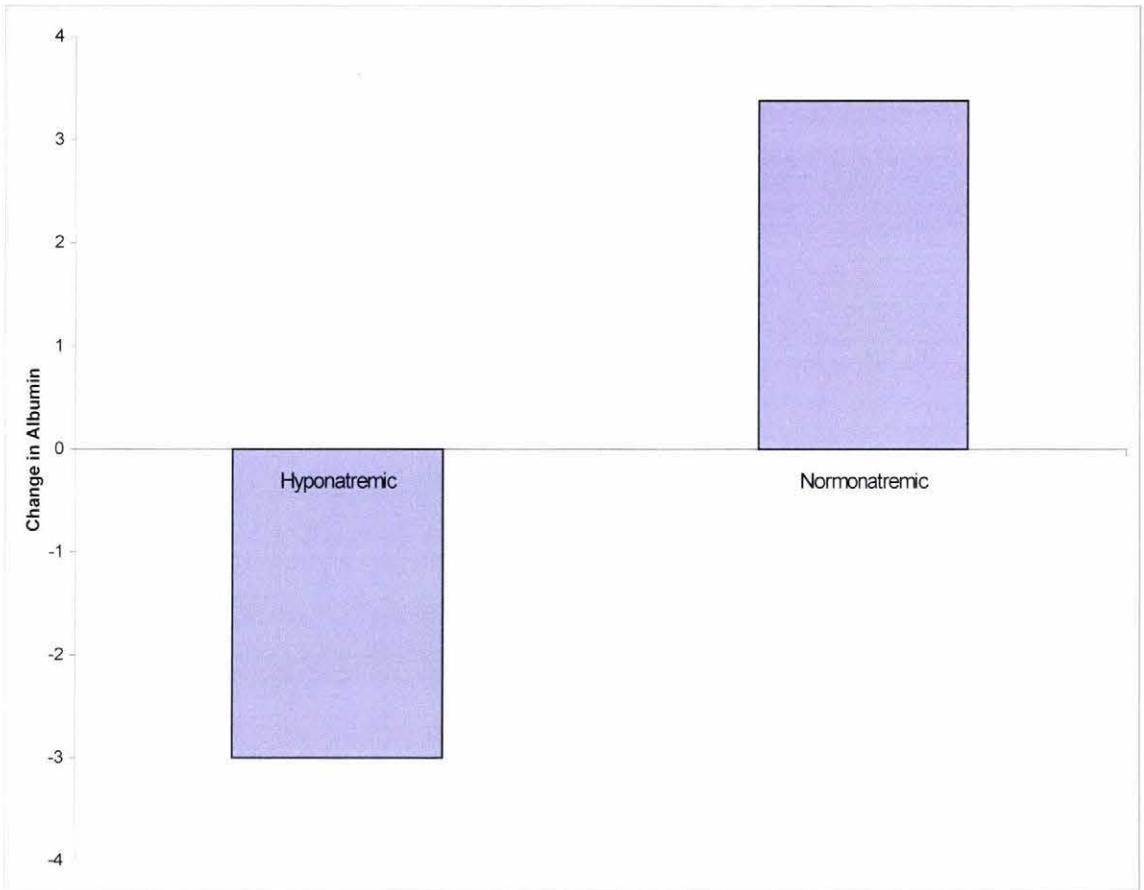


Figure 5.4 Difference in serum albumin for the hyponatremic athlete vs mean value for all normonatremic athletes

Figure 5.5 compares the difference in serum protein of athlete 11 with asymptomatic hyponatremia to the normonatremic athletes (18 other athletes in the study). Athlete 11 had a serum protein difference of -6.0 compared with the normonatremic athletes who recorded a difference in mean serum protein of +5.2.

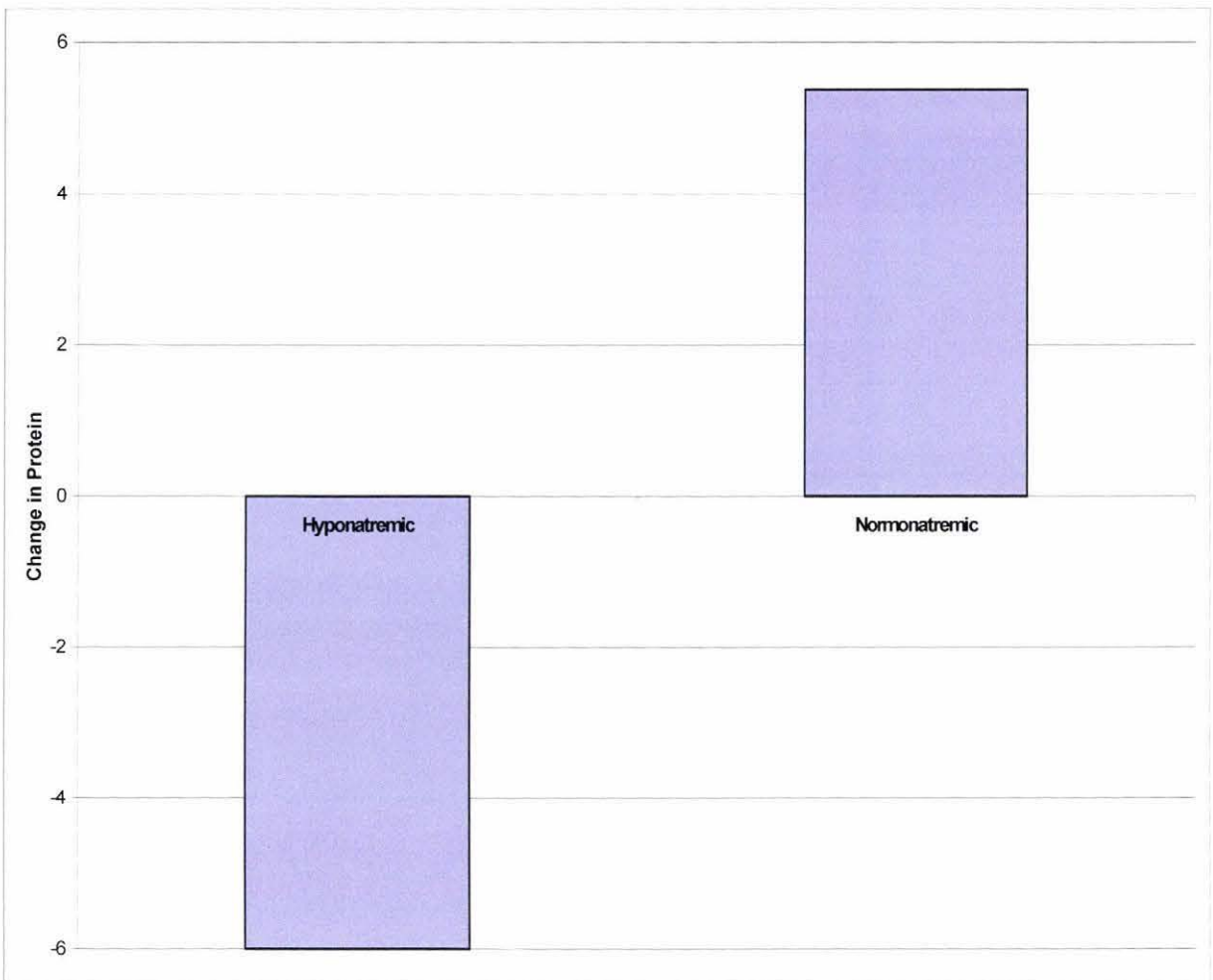
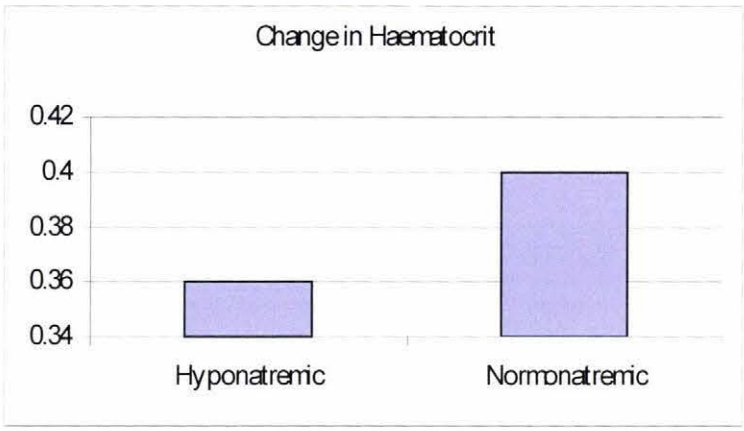


Figure 5.5 Difference in serum protein for the hyponatremic athlete vs mean value for all normonatremic athletes

There was an obvious difference when the mean post-race haematocrit (0.40) and haemoglobin (137.7) results for the study group were compared with athlete 11 post-race haematocrit (0.36) and haemoglobin (124g/L) results. When athlete 11 was also matched for age group there was continued variance. Figure 5.6 compares the post-race haematocrit and haemoglobin result of athlete 11 to the mean post-race results of the normonatremic athletes who were matched for age. Mean post-race haematocrit (0.40) and haemoglobin (135g/L) results in the normonatremic athletes who were matched for age.



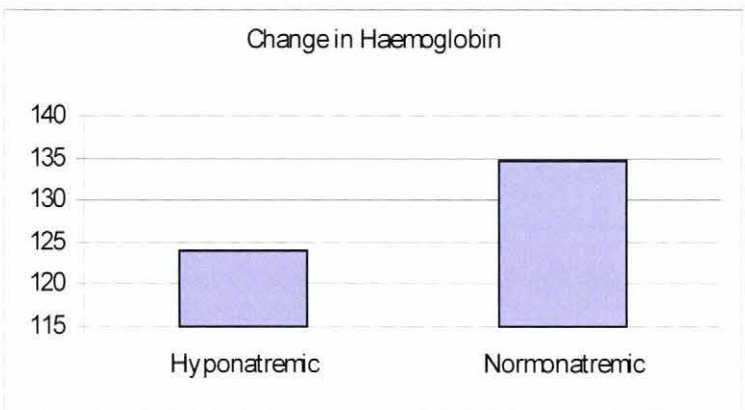


Figure 5.6 Comparison of post-race haematocrit and haemoglobin results

The mean difference for urinary sodium (-25.33mmol/L), urinary creatinine (8.43mmol/L), and urinary osmolality (255.93mmol/kg) is presented in table 5.7. Athlete 11 recorded no difference between pre- and post race urinary sodium, and recorded the lowest pre-race urinary sodium (8mmol/L) and the lowest post-race urinary sodium (8mmol/L). The first female finisher of the study group started the race with the greatest pre-race urinary sodium (206mmol/L) and also had the greatest post-race urinary sodium (111mmol/L). Athlete 11 achieved the lowest post-race urinary creatinine (-0.07mmol/L) and urinary osmolality (71mmol/kg).

Table 5.7 Results from pre- and post-race urine tests

	Pre-race Mean ± SD	Post-race Mean ± SD	Difference	Pre-race Range	Post-race Range
U. Sodium (mmol/L)	65.7 ± 54.7	37.5 ± 25.7	-25.33	8-206	8-111
U. Creatinine (mmol/L)	4.8 ± 3.5	12.8 ± 7.1	8.43	1.1-12.5	1.1-26.3
U. Osmolality (mmol/kg)	379.4 ± 260.5	568.8 ± 247.7	255.93	93-788	71-805

Hormonal assessment was made by measuring pre-race serum lutenizing hormone and progesterone, and by analysing answers given in the menstrual status questionnaire. This is summarised in table 5.8. Four of the 19 athletes were not taking an oral contraceptive pill and were diagnosed with amenorrhea. Seven of the athletes were taking an oral contraceptive pill.

Table 5.8 Individual Hormonal Assessment

Athlete	Lutenizing Hormone (IU/L)	Progesterone (nmol/L)	Comments
1	6.1	2	25-day cycle, no ovulation
2	2.1	< 1	OC – Loette, no ovulation
3	6.1	2	Amenorrhea, no ovulation
4	2.8	8	Possibly ovulating
5	5.6	< 1	Amenorrhea, no ovulation
6	2.7	< 1	25-day cycle, no ovulation
7	8.5	21	Possibly ovulating
8	5.9	< 1	OC – Mercilon, no ovulation
9	0.1	1	OC – Femodene, no ovulation
10	0.4	10	21-24-day cycle, possibly ovulating
11	9.4	4	OC – Femulin, no ovulation
12	32	< 1	OC – Kliogest, no ovulation
13	16.5	< 1	28-30-day cycle, no ovulation
14	9.7	9	21-day cycle, possibly ovulating
15	2.3	44	26-day cycle, ovulation
16	1.1	21	28-day cycle, possibly ovulating
17	26.7	2	OC – Trisequens, no ovulation
18	3.5	10	35-day cycle (period arrived race day)
19	8.0	3	24-day cycle, no ovulation

Lutenizing Hormone

0-6 Not indicative of ovulation

6-25 Possibly ovulating

25-80 ovulation

Progesterone

> 25 ovulation has occured

10-25 post ovulation

Information of oral contraceptive details is presented in table 5.9. Six of the seven athletes who were taking an oral contraceptive pill were using a combination pill and athlete 11 was the only athlete taking a progesterone only pill.

Table 5.9 Oral contraceptive (OC) details for individual oral contraceptive users

Subject number	Brand name	Oestrogen	Progestogen	Combination (C) or Progestogen only (P)
2	Loette	0.02mg ethinyloestradiol	0.1mg levonorgestrel	C
6	Diane-35	0.035mg ethinyloestradiol	2.0mg cyproterone acetate	C
8	Mercilon	0.02mg ethinyloestradiol	0.15mg destrogestrel	C
9	Femodene	0.03mg ethinyloestradiol	0.075mg gestrodene	C
11	Femulin		0.05mg ethynodiol diacetate	P
12	Kliogest	2.0mg oestradiol	1.0mg norethisterone	C
17	Trisequens	2.0mg oestradiol	1.0mg norethisterone	C

5.4 Dietary Assessment

5.4.1 Energy and Macronutrient Intake

All 19 subjects (100%) completed and returned the first of the two 7-day food diaries. The first testing period (approximately 6-7 weeks prior to the New Zealand Ironman Triathlon) was chosen to evaluate dietary intake in the peak of training.

Seventeen of the 19 subjects (89%) returned the second 7-day food diaries. The second testing period (the week before the event) was chosen to evaluate the dietary intake during the tapering of exercise before the event.

The average daily energy, macronutrient, fibre and sodium intake for the first 7-day period is presented in table 5.10, the second 7-day period is presented in

table 5.11. Carbohydrate (CHO) and protein intakes are presented as intake in grams as well as grams per kg body weight. The distribution of carbohydrate, protein, mono-unsaturated fat, poly-unsaturated fat, saturated fat and alcohol to total mean energy for the first 7-day period is presented in figure 5.7, the second 7-day period is presented in figure 5.8.

Table 5.10 Nutritional Data collected 6-7-weeks prior to the event

	Energy (kJ)	Energy (kcal)	CHO (g)	g/kg BW	Protein (g)	g/kg BW	Fat (g)	Fibre (g)	Sodium (mg)
Mean	10811	2672	393	6.3	125	2.0	76	14.9	3194
SD	2211	511	102	1.5	26	0.6	23	5.4	723
Range	6203 – 15944	1795 – 3852	221 – 564	3.8 – 8.7	85 – 175	1.1 – 3.2	29 – 124	7.9 – 25.9	1804 – 4748

* BW = Body Weight

Table 5.11 Nutritional Data collected 1-week prior to the event

	Energy (kJ)	Energy (kcal)	CHO (g)	g/kg BW	Protein (g)	g/kg BW	Fat (g)	Fibre (g)	Sodium (mg)
Mean	10155	2487	364	6.0	115	1.9	71	15.4	3244
SD	1820	410	84	1.5	24	0.4	19	5.1	789
Range	6321 – 13829	1508 – 3353	201 – 496	2.6 – 8.3	69 – 153	0.9 – 2.6	39 – 101	7.4 – 24.8	1944 – 5243

The mean daily energy intake for the first 7-day period was 10811 ± 2211 kiloJoules/day (kJ/day) or 2672 ± 511 kilocalories/day (kcal/day). A large variation in the range of mean daily intakes was seen (6203-15944 kJ/day, 1795-3852 kcal/day).

The mean daily energy intake for the second 7-day period was 10155 ±1820 kiloJoules/day (kJ/day) or 2487 ± 410 kilocalories/day (kcal/day). A large variation in the range of mean daily intakes was seen (6321-13829 kJ/day, 1508-3353 kcal/day).

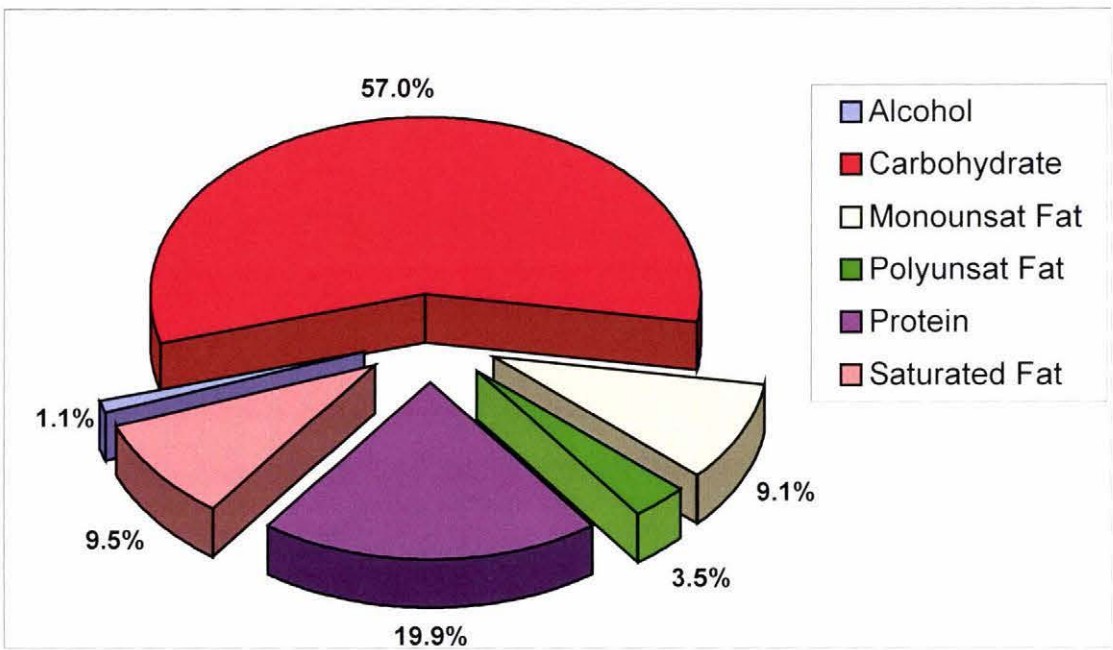


Figure 5.7 The first 7-day food diary mean energy contribution

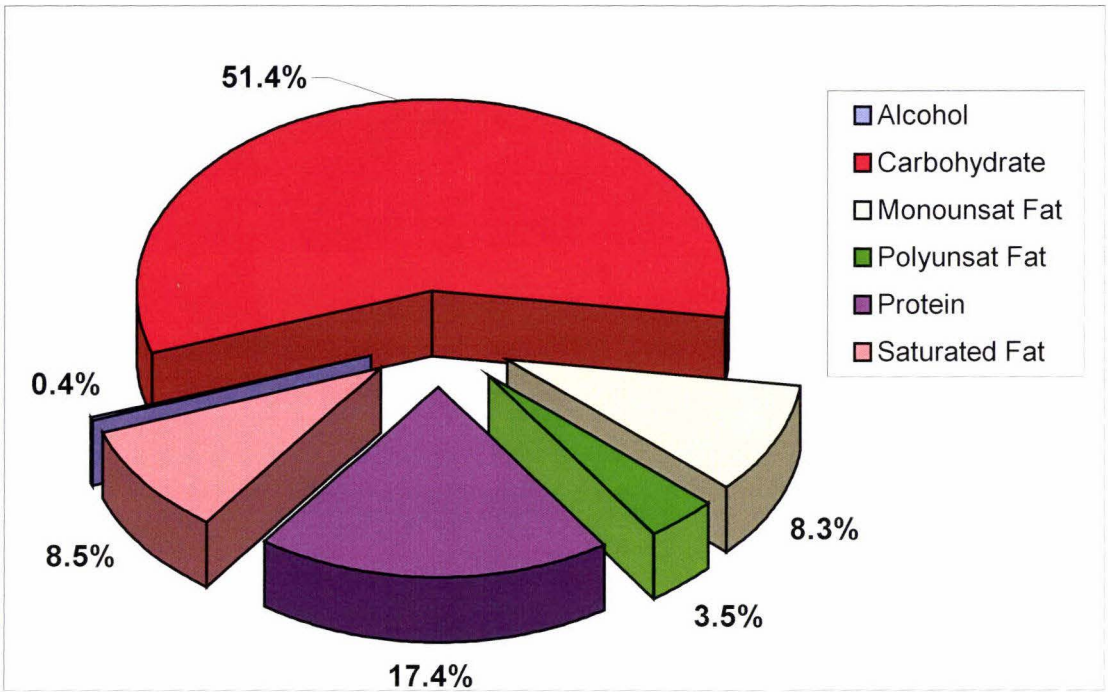


Figure 5.8 The second 7-day food diary mean energy contribution

The mean daily carbohydrate (CHO) intake for the first 7-day period was 393g and this represented 57.0% of the total daily energy intake. The mean CHO intake expressed as grams of CHO per kg body weight (BW) per day for the first 7-day period was 6.3 ± 1.5 g/kg BW/ day (range 3.8-8.7 g/kg BW/ day).

The mean daily carbohydrate (CHO) intake for the second 7-day period was 364g and this represented 51.4% of the total daily energy intake. The mean CHO intake expressed as grams of CHO per kg body weight (BW) per day for the second 7-day period was 6.0 ± 1.5 g/kg BW/ day (range 2.6-8.3 g/kg BW/ day).

Mean daily intake of dietary fibre for the first 7-day period was 14.9 ± 5.4 g/day (range 7.9-25.9 g/day). Mean daily intake of dietary fibre for the second 7-day period was 15.4 ± 5.1 g/day (range 7.9-24.8 g/day).

The mean daily protein intake for the first 7-day period was 125 ± 26 g/day (range 85-175 g/day) and this represented 19.9% of the total daily energy intake. The mean daily protein intake for the second 7-day period was 115 ± 24 g/day (range 69-153 g/day) and this represented 17.4% of the total daily energy intake.

The mean protein intake was expressed as grams of protein per kg body weight (BW) per day. The first 7-day period was 2.0 ± 0.6 g/kg BW/ day (range 1.1-3.2 g/kg BW/ day) and the second 7-day period was 1.9 ± 0.4 g/kg BW/ day (range 0.9-2.6 g/kg BW/ day).

The mean daily fat intake for the first 7-day period was 76 ± 23 g/day (range 29-124g) and this represented 22.1% of the total daily energy intake. Fat intake (22.1%) was below the guideline for percentage of energy from fat (30-33%) recommended for the general New Zealand population [131]. The mean daily fat intake for the second 7-day period was 71 ± 19 g/day (range 39-101g) which represented 20.3% of the total daily energy intake.

There would have been a discrepancy between % fatty acids (SFA, MUFA and PUFA) of total energy intake and % fat of total energy intake due to the figures for fatty acids not including the glycerol fraction. Total % fat is not shown.

The individual subjects' mean daily water intake, as well as the mean daily water intake for the group for the first and second 7-day periods are presented in table 5.12. The mean daily water intake for the study group was 3359.5 ± 1335.2ml and 3252.3 ± 1368.3ml for the first and second 7-day period respectively. The mean water intake was also expressed as milligrams of water per kg body weight (BW) per day. The first 7-day period was 53.6 ± 20.4 mg/kg BW/ day (range 20.6 – 100.0 g/kg BW/ day) and the second 7-day period was 52.7 ± 20.8 mg/kg BW/ day (range 23.3 - 97.0 g/kg BW/ day).

Table 5.12 Mean water intake for 1st and 2nd 7-day period

Athlete	Weight (kg)	1st Food Diary (ml)	ml/kg	2nd Food Diary (ml)	ml/kg
1	68.6	3314.1	48.3	3851.6	56.1
2	59.0	2842.3	48.2	3735.3	63.3
3	58.6	3384.2	57.8	3051.8	52.1
4	68.2	4270.4	62.6		
5	67.2	6721.8	100.0	5072.9	75.5
6	59.6	1228.5	20.6	1389.9	23.3
7	56.8	3196.6	56.3	3311.0	58.3
8	54.2	3014.8	55.6	2227.7	41.1
9	64.8	2013.7	31.1	3750.8	57.9
10	76.2	3659.0	48.0		
11	53.4	4702.6	88.1	4956.8	92.8
12	58.8	2690.4	45.8	2651.7	45.1
13	64.8	3216.2	49.6	2881.5	44.5
14	58.6	1673.1	28.6	2366.7	40.4
15	54.6	3476.3	63.7	2888.2	52.9
16	70.6	5948.0	84.2	6849.8	97.0
17	58.0	3312.4	57.1	2100.0	36.2
18	66.0	2756.8	41.8	2070.6	31.4
19	76.8	2409.9	31.4	2133.4	27.8
Mean	62.9	3359.5	53.6	3252.3	52.7
SD	7.1	1335.2	20.4	1368.3	20.8
Range	53.4 – 76.8		20.6 – 100.0		23.3 – 97.0

Mean alcohol intake represented 1.1% of total energy intake (range 0-5.61%) for the first 7-day period. The mean alcohol intake for the second 7-day period represented 0.4% of total energy intake (range 0-5.40%). Six of the 19 athletes (32%) consumed no alcohol in the first 7-day period compared with eight athletes

in the second 7-day period. Those who consumed alcohol restricted their intake to one to two days only.

5.4.2 Micronutrient Intake

Intake of selected micronutrients for first 7-day period is summarised in table 5.13, the second 7-day period is summarised in table 5.14. Recommended Dietary Intakes (RDI) value and the percentage of those athletes who did not meet the RDI are included to allow for comparison. The RDI values for adult females (19-54 years) were used although one subject was 55 years at the time of this study.

Table 5.13 Analysis of the first 7-day food diary micronutrient intake and comparison to the RDI for New Zealanders

NUTRIENTS	MEAN	RANGE	RDI	% OF ATHLETES BELOW RDI
VITAMINS				
Vitamin A Eq. (ug)	1618	561-3600	750	5%
Thiamin (mg)	4.1	0.8-9.6	0.8	0
Riboflavin (mg)	2.5	1.5-3.7	1.2	0
Niacin (mg)	22.1	14.8-29.4	13	0
Vitamin B6 (mg)	2.5	1.4-4.9	0.9-1.4	0
Folate (ug)	383	237-626	200	0
Vitamin B12 (ug)	5.7	2.3-25.2	2.0	0
Vitamin C (mg)	168	51-249	30	0
Vitamin E (mg)	12.6	6.0-22.2	7.0	16%
MINERALS				
Calcium (mg)	1636	871-2587	800	0
Iron (mg)	18.6	12.4-28.9	12-16	0
Magnesium (mg)	478	282-680	270	0
Phosphorous (mg)	2127	1235-3271	1000	0
Potassium (mg)	5282	3435-8687	1950-5460	0
Selenium (ug)	84.4	46.9-201	70	37%
Sodium (mg)	3194	1804-4748	920-2300	0
Zinc (mg)	14.5	10.2-22.7	12	37%

The group mean met or exceeded the RDI for all listed vitamins and minerals for the first 7-day period.

As individual athletes, all subjects met or exceeded the RDI for thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, vitamin C, calcium, iron, magnesium, phosphorous, potassium and sodium for the first 7-day period. One

athlete (5%) did not meet the RDI for vitamin A, three athletes did not meet the RDI for vitamin E. Seven athletes did not meet the RDI for selenium or zinc.

Table 5.14 Analysis of the second 7-day food diary micronutrient intake in comparison to the RDI for New Zealanders

NUTRIENTS	MEAN	RANGE	RDI	% OF ATHLETES BELOW RDI
VITAMINS				
Vitamin A Eq. (ug)	1320	814-2679	750	0
Thiamin (mg)	3.3	1.0-7.2	0.8	0
Riboflavin (mg)	2.3	1.3-3.1	1.2	0
Niacin (mg)	20.5	13.1-26.8	13	0
Vitamin B6 (mg)	2.8	1.1-5.9	0.9-1.4	0
Folate (ug)	420	196-779	200	6%
Vitamin B12 (ug)	4.1	1.9-7.0	2.0	6%
Vitamin C (mg)	196	54-320	30	0
Vitamin E (mg)	14.4	6.7-20.8	7.0	6%
MINERALS				
Calcium (mg)	1498	695-2598	800	12%
Iron (mg)	19.0	13.3-28.9	12-16	0
Magnesium (mg)	491	300-692	270	0
Phosphorous (mg)	2049	1222-2987	1000	0
Potassium (mg)	5294	2742-8690	1950-5460	0
Selenium (ug)	76.0	38.7-144.0	70	47%
Sodium (mg)	3244	1944-5243	920-2300	0
Zinc (mg)	13.9	7.4-21.9	12	29%

The group mean met or exceeded the RDI for all listed vitamins and minerals for the second 7-day period.

As individual athletes, all subjects met or exceeded the RDI for thiamin, riboflavin, niacin, vitamin B6, vitamin C, iron, magnesium, phosphorous,

potassium and sodium for the second 7-day period. One athlete (6%) did not meet the RDI for folate, vitamin B12 and vitamin E. Two athletes (12%) did not meet the RDI for calcium, eight athletes did not meet the RDI for selenium and five athletes did not meet the RDI for zinc.

Analysis of the 7-day food diaries did not include multi-vitamin preparations or any other types of supplements (except for sports drinks, bars, gels and liquid meal replacements). Athletes were asked to record all supplements/medications consumed throughout the 7-day period. See appendix 4 for full listing of supplements/medications consumed over the two 7-day food-recording periods.

During the first 7-day period the athlete who did not meet the RDI for vitamin A was not taking a supplement. One athlete who did not meet the RDI for vitamin E was not taking any supplements and the other two athletes who did not meet the RDI for vitamin E were taking multi-vitamin supplements. Four out of the seven athletes (57%) who did not meet the RDI for selenium were not taking a multi-vitamin. Three out of the seven athletes who did not meet the RDI for zinc were not taking a multi-vitamin supplement.

During the second 7-day period the athlete who did not meet the RDI for folate was not taking a supplement. The athlete who did not meet the RDI for vitamin B12 was taking a multi-vitamin. The athlete who did not meet the RDI for vitamin E, calcium, selenium and zinc was taking a multi-vitamin. The other athlete who did not meet the RDI for calcium was not taking a supplement. Three out of the eight subjects (38%) who did not meet the RDI for selenium were not taking a supplement. Three out of the five subjects (60%) who did not meet the RDI for zinc were not taking a supplement.

5.5 Temperature/Relative Humidity Assessment

Information about temperature and relative humidity, in relation to times during the day of the event, is presented in table 5.15.

Table 5.15 Temperature/Relative Humidity with relation to race times

TIME	TEMPERATURE (°C)	RELATIVE HUMIDITY (% RH)	COMMENTS
7am – Race start 0 hour 0 minutes	14.9	96.5	Rain
8am 1 hour 0 minutes	18.0	75.7	Rain stopped
12 noon 5 hours 0 minutes	19.1	66.8	Cloudy
12:30pm 5 hours 30 minutes	21.4	58.1	Sunshine
1pm 6 hours 0 minutes	24.7	55.9	Sunshine
3pm 8 hours 0 minutes	24.1	57.2	Sunshine
3:30pm 8 hours 30 minutes	27.6	45	Hot
4pm 9 hours 0 minutes	27.6	51	Hot
5:30pm 10 hours 30 minutes	21.0	69.5	Wind picking up
10pm 13 hours	16.0	62	Cooling down

6 Discussion

New Zealand female ultradistance triathletes competing in the New Zealand Ironman Triathlon, 3 March 2001, were selected for this study as this ensured that the group was as homogeneous as possible with regard to gender, heat acclimatisation, type and intensity of training and level of fitness. Other studies investigating causes of hyponatremia have included both male and female subjects [11, 22, 51, 53, 57, 59, 61] and ultra-marathon running [11, 53], cycling [22] and Ironman ultradistance triathlon [51, 57, 59, 61].

6.1 Body Composition and Medical Assessment

Triathletes aim to maintain a low body fat level to gain an increased power to mass ratio and to eliminate extra weight to carry across distance. In the present study, the mean pre-race weight was 62.9 ± 7.1 kg (range 53.4-76.8 kg) (table 5.2) and percentage pre-race body fat (measured by BIA) was 23.3 ± 5.1% (range 16.8 –39.4%) (table 5.4).

It has been observed by the researcher, that the weight and body fat of female triathletes is typically greater for ultra-endurance participants than shorter sprint and Olympic distance triathletes. It is suggested that ultra-endurance triathlon attracts an older aged athlete, and the physiological changes with increasing age give rise to greater body fat and weight. The mean age of the participants in this study was 37.8 ± 8.9 years.

It is interesting to note that the youngest athlete of the study (athlete 11, 26 years) was the only one of the group to record a post-race plasma sodium concentration < 135mmol/L (table 5.6). In table 6.1 the incidence of hyponatremia (HN) in female triathletes competing in the 1996 and 1997 New Zealand Ironman Triathlons is compared to the incidence of hyponatremia reported in the 2001 New Zealand Ironman Triathlon.

Table 6.1 Incidence of hyponatremia (HN) in female triathletes compared from 1996, 1997 and 2001.

NZ Ironman	Number of female race starters	Number of consenting female athletes	Number of female athletes with HN	Percentage of HN found in consenting female athletes	Percentage of HN found in female race starters
1996	83	19	3	16%	3.6
1997	79	70 35 consenting NZ female athletes	20 10 NZ female athletes with HN	29% 10/70 = 14% 10/35 = 29%	25.0 13.0
2001	133	43 seeking medical care + 19 NZ females as part of the HN study Total=62	4 3 = medical tent 1 = HN study 2 NZ female athletes with HN 1 = medical tent 1 = HN study	4/62 = 6% 2/62 = 3% 1/19 = 5%	4/133 = 3.0% 2/133 = 1.5%

The difference between mean pre- and post-race weight was 1.6 ± 1.1 kg (table 5.3), this was statistically significant (p= < 0.001). The weight loss equated to a mean percentage dehydration of 2.4 ± 1.8% (table 5.3). Dehydration was the most common diagnosis made for the athletes in this study.

An inverse relationship between post-race plasma sodium concentration and percentage dehydration was reported in this study (figure 5.3). The athletes with the higher plasma sodium concentrations had lost the greatest amounts of weight. Conversely, the athlete with the lowest post-race plasma sodium concentration (134 mmol/L) had gained weight. Athlete 11 recorded a pre-race weight of 53.4 kg (9.5 kg below the mean pre-race weight) (table 5.2), finished the race 1 kg heavier (54.4 kg) (figure 5.1) and was moderately over-hydrated by 1.9% (table 5.3).

The inverse relationship reported in the present study agrees with the findings of Speedy et al. [51]. Speedy et al. reported an inverse relationship between post-race plasma sodium concentrations and percentage change in body weight in 330 ultradistance triathletes [51]. Interestingly, athletes in the Speedy et al. [51] study were weighed before the race in minimal clothing and without shoes but, were re-weighed post-race wearing their running clothes and shoes. Although an adjustment for weight of running shoes was made, by weighing 18 athletes with and without running shoes, the researcher of the present study observed that weather conditions deteriorated during the 1997 race and ended with rain. If the subjects were not towelled down and their sodden, heavy shoes removed, the retained rainwater could have contributed to an increase in post-race weight. It has been reported that to measure percentage dehydration the athlete has to be weighed before and after the exercise session in minimal clothing, and to be towelled dry [24]. The calculated percentage dehydration would be less accurate if the subjects of the Speedy et al. [51] study had not observed this protocol. The subjects in the present study were weighed pre- and post-race with minimal clothing, no running shoes and were towelled off.

Athlete 11 was the smallest subject in the study and had the lowest recorded pre-race fat mass by BIA. Noakes [139] cited in [51] suggested that the smaller size of women may explain their greater risk for the development of hyponatremia because they are more likely to maintain body weight during the race.

Although the development of BIA methods are more appropriate for field use, a major source of error for this method is the intra-individual variability in whole body resistance due to factors that alter the clients' state of hydration. Factors such as eating, drinking, dehydrating and exercising alter the individual's hydration state. This affects total body resistance and the estimation of FFM [96]. It was decided to omit the post-race BIA readings due to this probable error.

Approximately 60% of the body's weight is water [5]. About 65% of the body's water is stored inside the cells as intracellular water and the remaining 35% is outside the cells as extracellular water [5]. The extracellular water is further divided into the intercellular (interstitial) water between or surrounding the cells and vascular water (within the blood vessels) [5]. The percentage of pre-race total body water (%TBW) of 55.3 was calculated by dividing the mean TBW in kilograms by the pre-race mean weight of 62.9 kg. The results indicated that all the athletes in the study were in a well-hydrated state before the race. Athlete 11 recorded one of the highest TBW of 59.9%.

The mean pre-race blood pressure of the study group was 117/74 this dropped to a mean post-race blood pressure of 114/78. It is suggested that the dehydration seen in the normonatremic athletes was the reason for the decrease. Hypervolaemia is the suggested cause for the increase from pre-race blood pressure 114/70 to post-race blood pressure 120/78 recorded by athlete 11 with asymptomatic hyponatremia.

It was interesting to note that although athlete 11 recorded a 'normal' initial plasma sodium level (139 mmol/L, table 5.6), it was the lowest pre-race plasma sodium recorded by the group. The findings in this study agree with Armstrong and colleagues [10] who have concluded that a large intake and retention of water and a 'low normal' initial plasma sodium were primary factors that contributed to the rapid onset of hyponatremia in their subject. However, Vrijens

and Rehrer [28] did not support this, their hyponatremic subject had an initial plasma sodium of 144 mmol/L and was not fluid overloaded.

Athlete 11 ingested a mean daily water intake of 4956.8ml (1704.5ml greater than the group mean) for the second 7-day period (the week before the race). The mean daily water intake for the study group for the same period was 3252.3 ± 1368.3ml and represented a mean daily water intake expressed as milligrams of water per kg body weight (BW) per day of 52.7 ± 20.8 mg/kg BW/ day. Athlete 11 ingested 92.8mg/kg BW/day (40.1mg/kg BW/day greater than the group mean).

The main feedback device for the control of body water is the osmolality of the various body fluids [5]. Athlete 11 recorded a 'normal' initial serum osmolality (286 mmol/kg, table 5.6), however it was the lowest pre-race serum osmolality recorded by the group. The lowest post-race serum osmolality (281 mmol/kg, table 5.6) of the group was also recorded by athlete 11. In the body a number of different substances affect osmolality, including glucose, protein and electrolytes, most notably sodium [5]. The low pre-race serum osmolality suggests that athlete 11 had ingested and retained a large amount of sodium-free beverage before the start of the race.

The further reduction of serum osmolality (post-race) also suggests that athlete 11 continued to ingest sodium-free or sodium-low fluids over the race without the excretion of the excess fluid during the race. In essence, athlete 11's body fluids became diluted. Extracellular sodium content is the most important variable (besides transcapillary colloid osmotic pressure) that influences plasma fluid shifts during exercise [5].

Athlete 11's post-race serum creatinine and urea values were not greater than the normonatremic athletes in this study. The inability of athlete 11 therefore, to excrete the excess water consumed during the race was not due to reduced

glomerular filtration rate (GFR). Nonsteroidal anti-inflammatory drugs (NSAIDs) used because of their analgesic (pain reduction) and anti-inflammatory benefits can cause a reduction in renal perfusion, leading to a depressed GFR and accentuated effects of ADH [140]. Athlete 11 was not consuming NSAIDs.

The dehydration observed in the normonatremic athletes explained the increased concentration of serum protein and serum albumin whereas, associated with the post-race plasma sodium concentration of 134 *mmol/L* (asymptomatic hyponatremia) of athlete 11, were marked reductions in serum albumin (figure 5.4) and serum protein (figure 5.5).

Noakes [27, 60] cited in [141] has speculated that the physiologic abnormality causing hyponatremia during exercise is an inappropriate renal response to high rates of fluid ingestion during very prolonged exercise, leading to fluid retention. Associated with athlete 11's lower plasma sodium concentration in this study was also a large reduction in hematocrit (Hct) (Figure 5.6). The lowered Hct indicated that the fluid was retained in the extracellular space (due to alterations in renal response) which caused dilutional hyponatremia. This finding agrees with the report by Speedy et al. [51] who demonstrated a relationship between post-race plasma sodium concentration and Hct.

In the 'Letters to the Editor' [141] Mink stated that certain susceptible individuals, when subjected to the stress of long distance events, may develop inappropriate anti-diuretic hormone (ADH) clinical syndrome stimulated by the haemeodynamics of endurance activities. Freund et al. [77] also reported that ADH levels were higher in trained than untrained male subjects following the ingestion of 1% of lean body weight as water. Freund and colleagues stated that, the higher plasma ADH values in the trained athletes were probably responsible for the blunted diuresis and is in spite of similar plasma osmolality values between the groups [77].

In contrast, Hiller et al. [57] reported that plasma ADH concentrations were not elevated in athletes with exercise associated hyponatremia.

6.2 Hormonal Assessment

The female athlete is exposed to a rhythmic variation in either endogenous hormones (i.e. during a regular ovulatory menstrual cycle), or exogenous hormones (administered as the oral contraceptive (OC) pill). Given that oestrogens and progestogens can have individual or interactive effects on a variety of metabolic processes, the potential exists for an influence on athletic performance [72].

The involvement of progesterone and its contribution to post-ovulatory fluid retention has been speculated by this author to have impacted on the occurrence of hyponatremia in Athlete 11. Athlete 11 was the only athlete in the study to be taking a progesterone-only oral contraceptive. Noakes [139] has proposed that another hormone or hormones (perhaps as yet unidentified or when acting in concert) cause the abnormal renal response that leads to the abnormal fluid retention that causes the hyponatremia during exercise.

Progesterone causes an increased excretion of water and sodium (natriuresis) from the kidney as a consequence of aldosterone antagonism. Paradoxically aldosterone secretion is then further increased via stimulation of the renin-angiotensin. Aldosterone promotes the increase of ADH secretion and therefore greater water re-absorption by the kidney [72, 73]. In another small case-controlled study by Oparil et al. [76] sodium excretion increased 3 hours after the administration of progesterone to normal subjects on either a high or low sodium intake. The authors of the study concluded that progesterone may inhibit sodium reabsorption at the proximal as well as distal sites in the nephron of the kidney [76]. Athlete 11 recorded the lowest pre- and post race urinary sodium (8mmol/L) and the lowest post-race urine osmolality (71mmol/kg).

Hyponatremia is more common in women and slower runners (those athletes who finish in the last 25-50% of entrants) [27,51]. The mean finishing time for all study participants was 755 minutes (12 hours 35 minutes). Athlete 11 finished in the last 50% of the study group with a time of 756 minutes (12 hours 35 minutes 52 seconds). Slower runners sweat less (sweat rate is determined by the metabolic rate, which is proportional to mass times running speed) but have greater opportunity to drink more as they walk and run at a lower exercise intensity [142].

Five of seven marathon runners in a recent study by Ayus et al. [143] were menstruant women who were treated for noncardiogenic pulmonary edema. Noncardiogenic pulmonary edema can be the initial manifestation of hyponatremic encephalopathy [143]. The authors suggested that gender may be a predisposing factor for hyponatremia [71, 143].

The mean age of the participants in the study was 37.8 ± 8.9 years. Most were of menstruant age. For women aged 20 to 40 years an average menstrual cycle lasts 28 days but may range from 20 to 45 days [73, 74]. Six of the seven female athletes who were taking an OC pill were using a combination pill. Athlete 11 was taking a progesterone only pill. Six of the female athletes who were not on an OC pill were possibly ovulating or at ovulation. Three of these athletes finished in the last 50% of the study subjects and had a serum osmolality value that had declined from the pre-race value (a trend seen in athlete 11). Serum osmolality increased in 82% of the subjects due to dehydration. Plasma sodium concentrations had also declined in these three subjects from the pre-race value (although remained within the normal range).

The majority of previous studies examining hormonal and renal responses have used male subjects. Recent developments in our understanding of an athlete's physiology have revealed new areas of interest that need to be assessed with specific reference to the female athlete [74].

It has been postulated by Pivarnik et al. [90] that there may be potential implications for prolonged athletic activity at high ambient temperature as the female athlete, in the luteal phase of her menstrual cycle, is beginning to exercise at an already elevated core temperature. Cyclic fluctuations of lutenizing hormone (LH) and follicle stimulating hormone (FSH) act on the ovary to release the steroid hormones, oestrogen and progesterone [75]. The increase in progesterone levels during the luteal phase (which follows ovulation and continues until menstrual flow occurs) is responsible for the biphasic basal body temperature curve and increased core body temperature (0.3°C – 0.5°C) and greater basal metabolic rate during this time [72, 73].

The author of this study has suggested that the three athletes who finished in the last 50% of this study, with the similar trend of declined serum osmolality as the athlete with hyponatremia, may have also been affected by progesterone. The increase in core body temperature experienced at ovulation due to increased levels of progesterone may have inhibited sodium reabsorption and prevented the excretion of excess ingested fluid. This phenomenon could have contributed to the decline in serum osmolality seen in these athletes.

6.3 Dietary Assessment

The determination of energy balance requires the estimation of energy consumed and expended each day. Basal metabolic rate (BMR) is the energy required to maintain the systems of the body and to regulate body temperature at rest [144]. Because assessment of BMR requires the individual to stay overnight in the laboratory, many researchers measure resting metabolic rate (RMR). In general BMR and RMR usually differ less than 10%. Factors that influence RMR are age, gender and body size (including the size of an individual's fat-free mass - FFM). These factors are included in prediction equations for RMR.

Thompson and Manore [145] found that for both male and female endurance athletes the Cunningham (1980) equation best predicted RMR, with the Harris-Benedict (1919) equation being the next best predictor. Because the Cunningham (1980) equation requires the measurement of FFM in kilograms, the Harris-Benedict (1919) equation is easier to use in settings where FFM can not be directly measured [144]. The two equations for estimating RMR are presented in table 6.2.

Table 6.2 Equations for estimating RMR

Cunningham (1980)
RMR = 500 + 22 (FFM)
Harris-Benedict (1919)
Females: RMR = 655.1 + 9.56 (weight-kg) + 1.85 (height-cm) – 4.68 (age-years)

In table 6.3 the mean FFM of the study group (calculated in the pre-race BIA assessment, refer table 5.4) is used in the Cunningham (1980) equation to calculate RMR. For comparison, the mean weight, height and age of the study group (refer table 5.2) is used in the Harris-Benedict (1919) equation to calculate RMR and is also presented in table 6.3.

Table 6.3 Estimation of RMR using mean body compositional data

Cunningham (1980)
RMR = 500 + 22 (48 kg)
1556 kilocalories (6504 kJ)
Harris-Benedict (1919)
Females: RMR = 655.1 + 9.56 (62.9 kg) + 1.85 (164 cm) – 4.68 (37.8 years)
1383 kilocalories (5781 kJ)

Once RMR has been estimated, total daily energy expenditure can then be estimated using factorial methods. The easiest method for assessing total energy

expenditure multiplies RMR by an appropriate activity factor (a multiple of RMR), with the resulting value representing total daily energy expenditure. The energy cost of activities normally includes the effects of thermogenesis (spontaneous physical activity – SPA and the thermic effect of food – TEF). Regardless of the method used to calculate energy expenditure, it should be noted that all values are estimates [144].

It has been reported that athletes, especially endurance athletes, require higher values for their 24-hour energy expenditure due to long workouts at high intensity [146]. In a study of elite female athletes using the double-labelled water technique for measuring energy expenditure, Haggarty and colleagues reported a ratio of 24-hour energy expenditure to BMR of 2.79 [129]. Warwick concluded that the mean 24-hour energy expenditure in very active female athletes appears to be about 2.0 x BMR [147]. The multiple 2.4 (the average of 2.0 and 2.79), was used to calculate the expected daily energy expenditure (EDEE) for the study group and is shown in table 6.4 using the calculated RMR from the two prediction equations in table 6.3. (See appendix 11A for a summary of individual subjects' RMR and EDEE values).

Table 6.4 Expected daily energy expenditure (EDEE)

Cunningham (1980)
RMR = 1556 kilocalories (6504 kJ) x 2.4
3734 kilocalories (15610 kJ)
Harris-Benedict (1919)
Females: RMR = 1383 kilocalories (5781 kJ) x 2.4
3319 kilocalories (13874 kJ)

The mean daily energy intake (MDEI) results for the study group from the first and second 7-day food diaries are, respectively, 10811 ± 2211 kJ/day (2672 ± 511 kcal/day) and 10155 ± 1820 kJ/day (2487 ± 410 kcal/day). When compared with the EDEE 13874 – 15610 kJ (3319 – 3734 kcal), which used the mean FFM,

age, weight and height of the study group, the MDEI results from the first and second 7-day food diaries were between 22-35% below what was expected. The difference between EDEE values and MDEI results from the two sets of food diaries is presented in table 6.5.

Table 6.5 Differences between expected daily energy expenditure (EDEE) and mean daily energy intake (MDEI) results

First Food Dairy	
EDEE Cunningham – MDEI	**EDEE Harris-Benedict – MDEI**
15610 kJ (3734 kcal) – 10811 kJ (2672 kcal)	13874 kJ (3319 kcal) – 10811 kJ (2672 kcal)
4799 kJ (1062 kcal)	3063 kJ (647 kcal)
4799/15610 = 31%	3063/13874 = 22%
Range (-7 – 59%)	Range (-26 – 53%)
Second Food Dairy	
EDEE Cunningham – MDEI	**EDEE Harris-Benedict – MDEI**
15610 kJ (3734 kcal) – 10155 kJ (2487 kcal)	13874 kJ (3319 kcal) – 10155 kJ (2487 kcal)
5455 kJ (1247 kcal)	3719 kJ (832 kcal)
5455/15610 = 35%	3719/13874 = 27%
Range (19 – 59%)	Range (6 – 58%)

The difference of 22-35% between EDEE and MDEI of the two diaries may be due to inaccurate reporting of intake. Several recent studies have found that the bias of reporting errors is towards under-reporting dietary intake [148, 149]. Edwards et al. [148] found that the mean reported energy intake of a group of female distance runners was 32% below the double-labelled water estimates of energy expenditure over the same period of energy balance monitoring.

Edwards et al. [148] also stated that the energy discrepancies in individual runners ranged from 4 to 58% and were greatest in the heavier runners and those who displayed greater dissatisfaction with their body image. The energy discrepancies in individual subjects in the first food diary ranged from –7 to 59% (Cunningham equation, table 6.5) and –26 to 53% (Harris-Benedict equation, table 6.5). There was one athlete who appeared to over-report or over-consume their usual intake. Although this athlete was in the peak of training for the

Ironman event, the athlete reported being on Christmas vacation and eating more food and drinking more alcohol at this time than was usual. The athlete who recorded the greatest under-reporting or under-consumption (53% - Cunningham) and (59% - Harris Benedict) has recently admitted to having an eating disorder, and has discussed poor self-image with the researcher.

The energy discrepancies in individual subjects' in the second food diary ranged from 19 to 59% (Cunningham equation, table 6.5), and 6 to 58% (Harris-Benedict equation, table 6.5). Two athletes recorded the lowest energy discrepancy for the Cunningham equation (19%). The first athlete in the group to finish the event had been receiving monthly nutritional advice from a sports dietitian. The second athlete had the lowest recorded pre-race weight 53.4kg – athlete 11. The athlete who recorded the greatest under-reporting or under-consumption (59% - Cunningham) and (58% - Harris-Benedict) was the heaviest subject of the group (76.8kg) and recorded the slowest race time. (See appendix 11B for a summary of individual subjects' EDEE values compared with MDEI results from the first and second 7-day food diaries).

Burke et al. [120] reported, that it seemed reasonable to expect that:

(a) Most athletes will under-report or under-consume their usual intakes when filling in dietary records, and
(b) That groups or individuals who are bodyweight/physique conscious or are dissatisfied with their body image are at the highest risk for significant under-estimation.

The authors also stated that the best accuracy with self-reported dietary assessment tools might be expected from athletes who are confident of their eating habits and body image, and who are highly motivated to receive valuable feedback [120].

The mean daily carbohydrate (CHO) intake for the first and second 7-day period was 393g and 364g respectively. This represented 57.0% and 51.4% of the total

daily energy intake for the first and second 7-day period respectively. These results were above the guideline for percent energy from carbohydrate (≥ 50%) recommended for the general New Zealand population [132].

However, the mean CHO intake expressed as grams of CHO per kilogram (kg) body weight (BW) per day for the first and second 7-day food diaries was 6.3 ± 1.5g and 6.0 ± 1.5g respectively. In the peak of training (6-7 weeks from the race) athletes normally train for greater than 2 hours a day. The recommendation for endurance training of 1-3 hours daily and for CHO loading (to super-compensate muscle glycogen stores) is a CHO requirement of 7-10g CHO/ kg BW/ day [150].

Athletes (especially female athletes) who eat lower energy intakes than might be expected may need to devote a greater proportion of their dietary intake (65 to 70% of total energy) to CHO intake and even then may fail to meet the absolute CHO intakes suggested for optimal daily glycogen recovery [120]. It is likely that female athletes become conscious of their dietary patterns or body compositional goals when taking part in dietary surveys, and consequently they under-eat or under-report their intake during the observation period [120]. If under-reporting is the major contributor to energy discrepancies, the true CHO intakes of female athletes will be higher than estimated. However, it is also likely that moderate energy restriction occurs either periodically or over the long term, which limits total CHO intake [120].

The recommended daily intake of dietary fibre for the New Zealand general population is 25-30 g/day [132]. The mean daily intake of dietary fibre for the first and second 7-day period was 14 ± 5.4g and 15.4 ± 5.1g respectively. Seventeen of the 19 athletes (89%) and all 17 athletes (100%) consumed less than the recommended level of 25-30 g/day in the first and second 7-day period respectively. A reduction in CHO foods with a high fibre content is recommended for athletes who suffer with gastro-intestinal problems [136].

The mean contribution of protein to total energy of 19.9% and 17.4% for the first and second 7-day period respectively was above the recommendation for percent energy from protein for the general New Zealand population of 12-15% [132]. The general recommended dietary intake (RDI) for protein for females in the general New Zealand population is 45g/day [137]. All subjects in both 7-day periods had a greater protein intake than recommended for the general population. The mean daily protein intake for the first and second 7-day period was 125 ± 26g and 115 ± 24g respectively.

The RDI for protein for the general population based on BW is 0.8 g/kg BW [137]. For athletes involved in endurance training the recommended intake of protein is 1.2-1.4 g/ kg BW/ day [136]. The mean protein intake expressed as grams of protein per kg BW per day for the first and second 7-day periods was 2.0 ± 0.6g and 1.9 ± 0.4g respectively. The energy discrepancy due to under-reporting or under-consumption of food, coupled with the low CHO intake expressed as grams per kg BW per day may have lead to an increase in protein consumption to cover energy costs over this time. Another factor that may have interfered with the achievement of adequate CHO intake and hence a higher protein intake could have been the promotion of fad diets encouraging lower CHO intakes (e.g. PR nutrition and the Zone diet).

It is recommended that athletes restrict their fat intake to a moderate level to allow for adequate CHO consumption [136]. The mean daily fat intake for the first and second 7-day periods was 76 ± 23g and 71 ± 19g respectively. This represented 22.1% and 20.3% for the first and second 7-day periods respectively and all subjects had a fat intake below the 30-33% recommended for the general New Zealand population [132].

The mean daily water intake for the study group was 3359.5 ± 1335.2ml and 3252.3 ± 1368.3ml for the first and second 7-day periods respectively. The mean

water intake was also expressed as milligrams of water per kg body weight (BW) per day. The first 7-day period was 53.6 ± 20.4 mg/kg BW/ day (range 20.6 – 100.0 g/kg BW/ day) and the second 7-day period was 52.7 ± 20.8 mg/kg BW/ day (range 23.3 - 97.0 g/kg BW/ day).

The Recommended Dietary Intakes (RDI) for adult females (19-54 years) were used in this study although one of the subjects was aged 55 at the time of this study. The RDI value, two standard deviations above the estimated average requirement (EAR) is intended to ensure that most of the population would avoid the consequences of deficiency if the RDI in question were ingested daily [151]. Some athletes do not fit within the range of 'average' population standards on which nutrient standards are based. Athletes involved in strenuous endurance programmes are likely to need higher intakes of micro-nutrients (due to losses in sweat, urine and perhaps faeces) than RDIs, although such recommendations have not been established and are likely to be highly variable [152].

The group mean met or exceeded the RDI for all listed vitamins and minerals for the first and second 7-day period. As individual athletes, most subjects met or exceeded the RDI for the majority of the listed vitamins and minerals for both recorded periods. Those athletes who appeared not to meet the RDI were generally consuming a multi-vitamin preparation. The analysis of the 7-day food diaries did not include the consumption of these preparations.

The RDI for sodium for females aged 19-54 years is between 920-2300mg (40-100mmol) a day. Every study participant met or exceeded the RDI for sodium. The mean daily intake of sodium for the first and second 7-day period was 3194 ± 723 mg/day and 3244 ± 789 mg/day respectively. The first study participant to finish the race (605 minutes) had the highest recorded mean sodium intake of 5243mg (1999mg above the mean for the second 7-day period – the week prior to the race). This intake was greater than double the upper limit recommended for females of the same age group. The accurate measurement of sodium by

dietary intake methods is difficult however because of the extensive sodium distribution in foods, variable use of sodium compounds added by food manufacturers and the use of sodium (salt) in cooking [135]. A recent report stated that simply adding salt to the diet (or eating high-salt foods) and consuming a selected volume of a salt-containing beverage (sports drink) would ensure better fluid retention and could prevent a sodium deficit [9]. However, saline ingestion (sodium concentration 25 mmol/L) did not prevent a decrease in plasma sodium in the Barr et al. study [22]. Oparil et al. [76] also demonstrated that sodium excretion increased after the administration of progesterone whether the subjects were on a low sodium (40mmol/day) or high sodium (240mmol/day) intake.

Present data suggests that the aeitology of hyponatremia relates to excessive replacement of fluid lost by sweating (a dilute sodium solution) by ingesting large volumes of even more dilute fluids (water or sports drinks), all of which have relatively low sodium content [142]. The low pre-race and post-race serum osmolality suggests that athlete 11 had ingested and retained a large amount of sodium-free beverage before the start of the race and had continued to ingest sodium-free or sodium-low fluids over the race without the excretion of the excess fluid during the race. Athlete 11's progesterone intake via the oral contraceptive may have also prevented sodium reabsorption and exacerbated the fluid retention situation.

The sponsored sports drink for New Zealand Ironman 2001 was PB. Details of its composition are presented in table 6.6. Temperature and relative humidity (RH) with relation to time are reported in table 5.14 and are expressed as a range in table 6.6.

Table 6.6 Race conditions for three consecutive New Zealand Ironman Triathlons (1996 – 1998) compared with the conditions for 2001.

					Sports Drink			
NZ Ironman	Temperature	Relative Humidity	Aid Station Frequency		Brand	Sodium		CHO
	°C	%	Cycle (km)	Run (km)		(mg/L)	(mmol/L)	(g/L)
1996	17-24.0	97.5	12	1.8	Sports Schweppes Plus	391	17.0	7
1997	21.0	91	12	1.8	Powerade	250	10.9	8
1998	19.4	87	20	2.5	Powerade	250	10.9	8
2001	14.9-27.6	45-96.5	15	1.8	PB	580	25.2	6.8

As reported in the literature review, the incidence of hyponatremia between 1997 and 1998 was significantly reduced (23% to 3.4%) when the frequency of aid stations was decreased and pre-race education (newsletters and briefing) of the dangers of over-drinking were implemented as a hyponatremia preventative strategy [59].

In 2001, 239 athletes presented for medical care. Ten of the 239 athletes (4%) were hyponatremic; of these, 7 were male and 3 were female [153]. Information relating to the female athletes is presented in table 6.1.

The environmental conditions between 2001 and previous races were comparable, although slightly warmer in 2001. The aid station frequency had been increased from the recommended 2.5 km to 1.8 km for the run course. The sodium content of the sports drink had more than doubled (from 10.9 mg/L to 25.2 mg/L).

It is interesting to note that potato chips were offered at aid stations on the run course for the first time in the history of the New Zealand event. One potato crisp (2g weight) will provide 13mg of sodium.

6.4 Recommendations

Some recommendations can be made based on the information from the literature review combined with the data from this study.

To protect the health, enhance the performance and prevent hyponatremia of the female triathlete while she competes in ultraendurance events it is important to educate the athlete about water and sodium balance and the problems associated with water overload. The athlete should monitor weight changes before and after lengthy, heavy training sessions to determine mean sweat rates and thus hourly fluid replacement needs. Dietary advice should be given promoting the use of higher-salt foods and appropriate fluid requirements (sodium containing beverages – sports drink). Pre-race plasma sodium concentration and serum osmolality may be prudent tests to conduct to assess individual risk.

Dietary recommendations should also include an individual assessment of energy and macro-nutrients (carbohydrate, protein and fat) necessary to balance the increased energy expenditure of heavy training.

Because gender has been suggested as a predisposing factor for hyponatremia further research into the effect of progesterone and its contribution to postovulatory fluid retention is required. This would allow the female triathlete to be aware of her menstrual cycle so that she races at the optimum time (in the follicular phase when core temperature is not elevated). Based on the findings in this study it appears that progesterone plays a role in sodium balance and may be a factor in the aetiology of exercise associated hyponatremia.

6.5 Limitations

To make the study group as homogenous as possible only New Zealand females competing in the New Zealand Ironman triathlon were selected. This made the sample size relatively small (n=19). However, to the researcher's knowledge there are currently no other studies that have solely studied female ultradistance triathletes and the causes of hyponatremia.

Although the development of BIA methods are more appropriate for field use a major source of error is the intra-individual variability in whole body resistance due to factors that alter the client's state of hydration. Factors such as eating, drinking, dehydrating, exercising, and menstrual cycle alter the individual's hydration state. The accuracy and precision of the BIA method are also affected by instrumentation, technician skill, environmental factors and the prediction equation used to estimate FFM [96]. To minimise the possible error only the pre-race BIA results were used. The athletes were instructed not to have:

(a) Eaten 4-5 hours prior to testing,

(b) Exercised for 12 hours prior to testing,

(c) Consumed alcohol 24 hours prior to testing; and

(d) Emptied their bladder immediately before testing.

The post-race BIA results were omitted due to the fact that the athletes had ate and drank *ad libitum* during the race, had a mean percentage dehydration of 2.4% and had exercise continuously for over 10 hours.

Although the 7-day diet record is considered the most 'accurate' and feasible method for research, the collection of reliable and legitimate dietary intake is difficult because of the influence of confounding effects, and errors inherent in all dietary survey methods [118, 119, 123]. To estimate the true average intake of energy and macro-nutrients, 7 days of recorded food is required [131]. Micro-nutrient intakes require much longer. To ensure that diet records were representative of usual diet the 7-day record was repeated 6-8 weeks apart. It

has been reported that the estimated method using household measures was acceptable for research because of better compliance than the weighed method [121,122]. However, under-reporting or under-consuming of food either intentionally or unintentionally is higher in food records than other methods.

The conversion of food into nutrients is a major source of error in dietary surveys and is a reflection of the skills and knowledge of the researcher, the method of data collection and the food composition database utilised. When the food database did not contain a consumed food, substitutions and omissions were made. Errors in entering food for nutrient analysis may have been introduced however frequent checking was implemented to avoid error.

Results from the blood and urine collection may have been affected by the procedures used for collection, storage, and testing of the samples. However, collection was made observing the universal precautions for venipuncture by the trained nurse/ phlebotomist. Both blood and urine samples were transferred into standard collection tubes supplied by a Medlab registered laboratory and stored as per the laboratory manual protocols. The biochemistry manager determined the co-efficient of variation (CV) of the samples by dividing the standard deviation by the mean and expressing as a percentage. The lower the CV the greater the likelihood that the result was meaningful. A result was regarded as meaningful when achieving a CV of lower than 5%. All but one of the blood and urine results scored a CV below 5%.

7 Conclusions

The incidence of hyponatremia in this study was 5%. One athlete (athlete 11) from the study group of 19 females recorded a post-race plasma sodium concentration of 134mmol/L (asymptomatic hyponatremia). In agreement with the findings of Speedy et al. [51] the post-race plasma sodium concentrations in this study were inversely related to percentage dehydration. The athletes with the higher plasma sodium concentrations had lost the greatest amount of weight, compared to athlete 11 who had the lowest plasma sodium concentration and had gained weight (table 5.3).

The higher TBW reading recorded by athlete 11 coupled with her 'low normal' initial plasma sodium was speculated by the author to have been a direct outcome of:

1) the consumption and retention of a large intake of low-sodium or sodium free beverages before and during the event, as well as
2) progesterone's suggested effect on inhibition of sodium reabsorption at the proximal site of the nephron and the consequential fluid retention that develops.

The estimated mean daily water intake for athlete 11 was 4956.8ml (1704.5ml greater than the group mean) for the second 7-day period (the week before the race). This represented a mean daily water intake expressed as milligrams of water per kg body weight per day of 92.8mg/kg BW/day (40.1mg greater than the group mean). The further reduction of serum osmolality (post-race) also suggests that the individual continued to ingest sodium-free or sodium-low fluids over the race and was unable to excrete the excess fluid.

Associated with athlete 11's lower plasma sodium concentration in this study was a large reduction in hematocrit (Hct) (Figure 5.6). The lowered Hct indicated that

the fluid was retained in the extracellular space (due to alterations in renal response) which caused dilutional hyponatremia. This finding is in agreement with the report by Speedy et al. [51] who demonstrated a relationship between post-race plasma sodium concentration and Hct.

Athletes involved in strenuous endurance programmes are likely to need higher intakes of micro-nutrients (due to losses in sweat, urine and perhaps faeces) than RDIs, although such recommendations have not been established and are likely to be highly variable [152]. The RDI for sodium for females aged 19-54 years is between 920-2300mg (40-100mmol) a day. Every study participant met or exceeded the RDI for sodium. However the first study participant to finish the race had the highest recorded mean sodium intake of 5243mg (1999mg above the mean for the second 7-day period – the week prior to the race). This intake was greater than double the upper limit recommended for females of the same age group. A recent report stated that simply adding salt to the diet (or eating high-salt foods) and consuming a selected volume of a salt-containing beverage (sports drink) would ensure better fluid retention and could prevent a sodium deficit [9]. In contrast to this report Oparil et al. [76] demonstrated that sodium excretion increased after the administration of progesterone whether the subjects were on a low sodium (40mmol/day) or high sodium (240mmol/day) intake. Increasing sodium intake may not have the desired effect if the female is on a progesterone only oral contraceptive or is in the luteal phase of her menstrual cycle when progesterone levels are raised.

The dietary analysis revealed that the study group had a significantly lower mean daily energy intake (MDEI) than the calculated expected daily energy expenditure (EDEE). The MDEI results from the first and second 7-day food diaries were between 22-35% below what was expected. The difference has been speculated to be due to inaccurate reporting of intake.

Burke et al. [120] reported that it seemed reasonable to expect that most athletes will under-report or under-consume their usual intakes when filling in dietary records, and that groups or individuals who are bodyweight/physique conscious or are dissatisfied with their body image are at the highest risk for significant under-estimation.

The lower MDEI resulted in CHO intake expressed as grams of CHO per kilogram of body weight per day appearing below the recommended 7-10g CHO/ kg BW/ day. If under-reporting is the major contributor to energy discrepancies, the true CHO intakes of female athletes will be higher than estimated. However, it is also likely that moderate energy restriction occurs either periodically or over the long term, which limits total CHO intake [120].

Dietitians and sports nutrition consultants need to foster confidence in the female triathletes' eating habits and body image so that the client is more likely to alter food intake positively and meet the absolute CHO intakes suggested for optimal daily glycogen recovery.

Limitations to this study include:

- the relatively low sample size,
- intra-individual variability in whole body resistance when using BIA,
- errors inherent in all dietary survey methods,
- limitation in the food database
- collection, storage and testing errors of blood and urine samples.

Future research is needed to:

- validate methods for estimating energy expenditure against the doubly-labelled water technique in female ultradistance triathletes

- determine the effect of dietary modification of the consumption and retention of large volumes of low-sodium or sodium-free fluids on hyponatremia incidence
- determine whether excessive losses of sodium through urine and sweat (either chronically before or acutely during exercise) predicts hyponatremia
- conclude whether the neuroendocrinology of the menstrual cycle (especially progesterone) is partially responsible for the significantly increased risk of the development of hyponatremia in female ultradistance triathletes

Ironman events are becoming increasingly popular and are attracting more participants each year. Further studies should be carried out in the area of hyponatremia and the female ultradistance triathlete and the information used to protect her health and improve her performance while she is racing.

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Appendix 1

Invitation to Participate in a Research Study (Information Sheets)

This study has received ethical approval from the Auckland Ethics Committee.

.....If you have any questions?

If you have any questions about the study, either now or in the future, please feel free to call. If you need an interpreter, one can be provided.

Nikki Hart – Ph (09) 366 0336 work
Ph (09) 302 3121 home
Fax (09) 302 3185

Or my research supervisor,
Dr Clare Wall - Ph (09) 443 9748
Fax (09) 443 9640

If you have any queries or concerns about your rights as a participant in this study you may wish to contact:
Northland to Franklin – Health Advocates Trust Ph 0800 205 555
Franklin to Wellington – Advocacy Network Services Trusts Ph 0800 423 638
South Island – Advocacy South Island Trust Ph 0800 377 766

Thank-you for taking the time to read this information sheet.

Massey University
Institute of Food, Nutrition and Human Health.
Private Bag 102-904
North Shore Mail Centre
Auckland

Causes of hyponatremia (low blood salt) in New Zealand female ultradistance triathletes

Information sheet for New Zealand female Ironman competitors (3 March 2001)

You are invited to participate in the study "Causes of hyponatremia (low blood salt) in NZ female ultradistance triathletes". The study is part fulfilment of the completion of a Master of Science (Nutritional Science) at Massey University.

WHY AM I DOING THIS STUDY?

Dr Dale Speedy reported that nearly half, (45%) of the female race finishers at New Zealand Ironman 1997 were hyponatremic (low blood salt) compared with 14% of male race finishers.

Exercise associated hyponatremia is a life-threatening condition that begins with non-specific symptoms. The cause of hyponatremia remains uncertain.

By participating in this study you will help determine why female ultradistance triathletes are at more risk for hyponatremia.

WHO IS IN THIS STUDY?

All New Zealand women who are participating in the New Zealand Ironman Triathlon, 3 March 2001 are eligible to be included.

WHAT IS INVOLVED?

The study will collect nutritional, biochemical, hormonal, and physical information from New Zealand female ultradistance triathletes at the New Zealand Ironman event 3 March 2001. The information gathered will be used to protect the health and may improve the performance of the female triathlete while she competes in ultradistance events.
Participants will be asked to participate in the following procedures:

- Record their food and fluid intake for a period of fourteen (14) days. The first 7-day diary is to be recorded eight (8) weeks from Ironman (approximately 1 January 2001).
- The second 7-day diary is to be recorded while tapering, a week before the event (24 February 2001 – 2 March 2001).
- Have their body composition assessed the day before the event and when they finish the event (weight, bio-impedance and blood pressure).
- Complete a menstrual status questionnaire.
- Have a blood and urine sample taken the day before the event and when they finish the event.

DO I HAVE TO TAKE PART IN THIS STUDY?

Your participation is **entirely voluntary** (your choice). If you agree to take part in the study, you are free to withdraw at any time and have the right to refuse to answer any questions at any time.
In the unlikely event of a physical injury as a result of your participation in this study, you will be covered by the accident compensation legislation with its limitations. If you have any questions about ACC please feel free to ask the researcher for more information before you agree to take part in this trial.
It will not cost you anything to take part in this study.

WHAT WILL HAPPEN TO THE RESULTS?

Data collected from participants is completely confidential. No material, which could personally identify you, will be used in any reports on this study. The results will be stored by code number in a computer in a locked room throughout the study. Information will be stored for 10 years and then destroyed.
You will receive a copy of the analysis of your biochemical, hormonal, nutritional and body composition data.

Appendix 2

Consent Form

Massey University
COLLEGE OF SCIENCES

Institute of Food, Nutrition
and Human Health
Private Bag 102 904,
North Shore Mail Centre,
Auckland,
New Zealand
Telephone: 64 9 443 9700
Facsimile: 64 9 443 9640

Causes of Hyponatremia (low blood salt) in New Zealand female ultradistance triathletes competing in New Zealand Ironman, 3 March 2001

CONSENT FORM

English	I wish to have an interpreter.	Yes	No
Maori	E hiahia ana ahau ki tetahi kaiwhakamaori/kaiwhaka pakeha korero.	Ae	Kao
Samoan	Oute manaó ia iai se faámatala upu.	Ioe	Leai
Tongan	Oku ou fiemaú ha fakatonulea.	Io	Ikai
Cook Island	Ka inangaro au I tetai tangata uri reo.	Ae	Kare
Niuean	Fia manako au ke fakaaoga e taha tagata fakahokohoko kupu.	E	Nakai
	Other languages to be added following consultation with relevant communities.		

I have read and understand the information sheet dated _____ for volunteers taking part in the study designed to find out the causes of Hyponatremia (low blood salt) in New Zealand female ultradistance triathletes competing in the New Zealand Ironman, 3 March 2001. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.

I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future health care.

I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study. I understand that the project will be submitted as part fulfilment of the completion of a Master of Science degree. I understand the compensation provisions for this study.

I have had time to consider whether to take part. I know who to contact if I have any side effects to the study. I know who to contact if I have any questions about the study.

I consent to blood and urine samples being destroyed at the end of the study.

YES / NO

I would like the researcher to discuss the outcomes of the study with me.

YES / NO

I _____(full name), hereby consent to take part in this study.

Date:

Signature:

Full name of researcher:	Nichola (Nikki) Tui Hart
Contact phone number for researcher:	(09) 366 0336
Project explained by:	Nichola (Nikki) Tui Hart

Signature:

Name of witness:

Signature of witness:

(Note: A copy of the consent form to be retained by participant).

Appendix 3

Food Diary

7-Day FOOD DIARY 1

Causes of hyponatremia (low blood salt) in New Zealand female ultradistance triathletes

Participant's Name:_____

Date Began:_____

Date Ended:_____

Causes of hyponatremia (low blood salt) in New Zealand female ultradistance triathletes

This diary is designed to detail your food intake over the period of allocated days. Five out of the seven days are to be weekdays, and the other two weekend days.

INSTRUCTIONS

- Please record all food and drink as shown on the tables in the following pages **just before you eat or drink** NOT from memory at the end of the day.
- Use a new line for each food and drink. You can use more than one line for a food and drink.
- Remember to include all snacks and drinks, even tap water. Include all supplements and medicines.
- Use as many pages of the booklet as you need.
- Check the sample food record to see how it is done.

All information provided in this diary will be treated with the strictest confidence. No one outside the study will have access to it.

Thank-you for participating in this study. I really appreciate the time you are giving!

Nikki Hart (Sports Dietitian)
Ph (09) 366 0336 work
Ph (09) 302 3121 home
Fax (09) 302 3185

Eating Time	Food or Drink (Name)

How much did you eat or drink?	Brand name of food or drink (if any)	Preparation of food e.g boiling, frying, microwaving.

Appendix 4

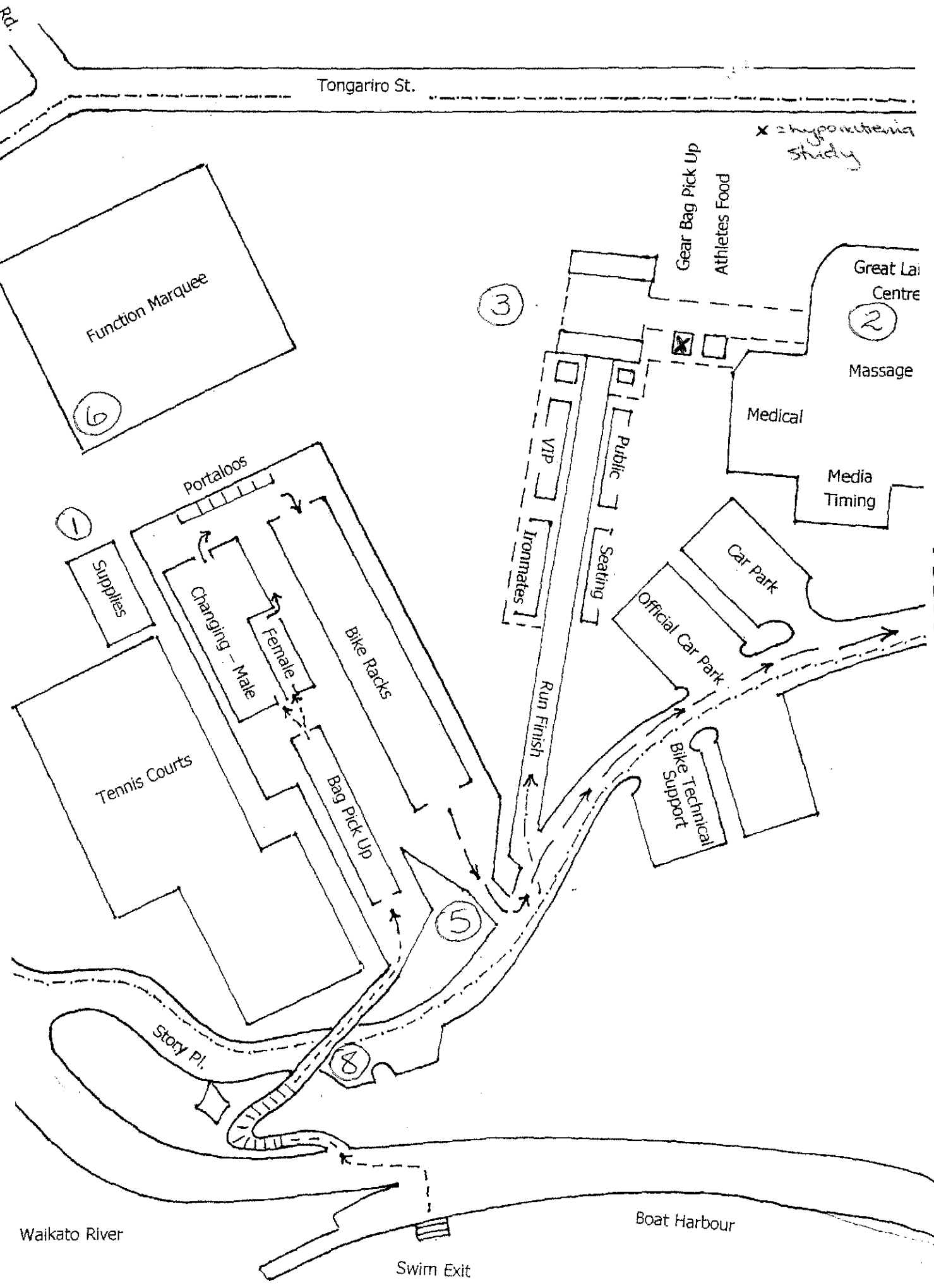
Supplement/Medication List

Athlete	Supplement/Medication
1	Multivitamin, Bee pollen, Vitamin C with Echniacea, Spirulina
2	-
3	Swedish bitters, Orthoplex Q10, Magnesium, Spirulina Brufren, Asprin
4	-
5	Iron & Zinc, L-Glutamine, Viamin C
6	-
7	Iron, Vitamin C, Multivitamin, Magnesium & Potassium
8	-
9	-
10	-
11	Vitamin C, Formula IV, VitaminA, Vitamin B, Multi-mineral, Calcium& Magnesium, Garlic, Zinc & Echinacea
12	Berroca
13	Glucosamine Sulphate (joints), CalciumSandoz, Multivitamin, Echinacea & Vitamin C, Berroca
14	Multivitamin, Magnesium, Horseradish, Garlic, Vitamin C, Opti-zinc Minotab (Antibiotics for skin)
15	Multivitamin Somac (for hernia)
16	Spirulina, Multivitamin, Herbal liver cleanse
17	-
18	Vitamin C Cataflam, Voltaren (2 x 75mg), Panadol x 4 (period arrived race day)
19	Berocca

Appendix 5

Site Map Displaying Location of Testing Facility at Ironman Race

IRONMAN NEW ZEALAND TRIATHLON 2001
Northern Domain Transition / Finish



Appendix 6

Technical Description of the Seac - Bioelectrical Impedance Assessment Technique

Information required:

1. **Gender**
2. **Age (years)**
3. **Height (cm)**
4. **Weight (kg)**

Obesity is a condition in which there is excess body fat. It is usually defined by body mass index (BMI), calculated as follows:

$$BMI = \frac{WEIGHT\ (kg)}{HEIGHT \times HEIGHT\ (m)}$$

A general definition of obesity is where BMI is greater than 30 kg/m^2[4].
Central fat distribution carries significantly greater health risks than peripheral distribution. However there is some evidence that abdominal fat may be easier to lose than sub-cutaneous fat.[5]

Changes in % body fat as a result of dietary, pharmaceutical or exercise intervention can be monitored by the BIM4.

Patient Preparation

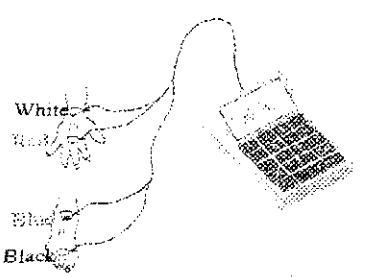
1. The patient should not have eaten for 4-5 hours prior to testing.
2. Alcohol should not be consumed for 24 hours prior to testing.
3. The patient should not have exercised for 12 hours prior to testing.
4. The patient should empty their bladder immediately before testing.

Patient Test

1. The patient should be lying face-up with legs slightly apart and hands resting next to the body palms down. Hands should not be touching any part of the body. The patient's inner thighs should not be in skin to skin contact.

2. Take the patient's right sock and shoe off and place electrodes on to the right foot and the right hand as follows:

   1. White - midpoint between Distal ulna/radius Dorsal (right) wrist
   2. Red - 3rd metacarpal head Dorsum (right) hand
   3. Blue - midpoint between Distal tibia/fibula Dorsal (right) ankle
   4. Black - 2nd metatarsal head Dorsum (right) foot



3. Attach leads to the electrodes.

[4] Seidell, J.C. International Journal of Obesity & Relative Metabolic Disorders. 1995, Mar 19 (3:202-205)
[5] Obesity Resource Information Centre, 40-42 Osnaburgh Street, London NW1 3ND

Appendix 7

Pre-Event/Post-Event Participant Identification Forms

Causes of hyponatremia in NZ female ultradistance triathletes
NZ Ironman 3 March 2001

Participant name:_____

Race Number:_____

PRE-EVENT (Friday 2 March 2001 afternoon)

Physical

Weight:_____ BIA: See Below

Blood Pressure:_____ HEIGHT _____

Blood Test

Plasma sodium
Haemoglobin
Haematocrit
Serum Creatinine
Serum Urea
Total Protein
Albumin
Serum Osmolality

FAT MASS _____
% FAT _____
FAT FREE MASS _____
% FFM _____
TOTAL BODY WATER _____
% TBW _____

Lutenizing Hormone
Progesterone

Urine Test

Osmolality
Sodium
Creatinine

POST-EVENT (Saturday 3 March)

Participant name:_____

Race Number:_____ **Time finished:**_____

Physical

Weight:_____ **BIA:** See Below

Blood Pressure:_____ HEIGHT _____

Blood Test

Plasma sodium
Haemoglobin
Haematocrit
Serum Creatinine
Serum Urea
Total Protein
Albumin
Serum Osmolality

FAT MASS_____
% FAT _____
FAT FREE MASS_____
% FFM _____
TOTAL BODY WATER_____
% TBW _____

Urine Test

Osmolality
Sodium
Creatinine

TEMPERATURE (°C)_____

Nikki Hart NZRD
Ph (09) 366 0336
Ph (09) 302 3121
Ph (021) 139 4918

Appendix 8

Menstrual Status Questionnaire Form

MENSTRUAL STATUS QUESTIONNAIRE

The information collected on this form is for the purpose of providing background and menstrual status information about the participants in the study "Causes of hyponatremia in New Zealand female ultradistance triathletes".

1. Name:

2. Address:

3. Contact phone number:

4. Age:

5. Do you menstruate (have a regular period)?

YES

NO please comment_____

_____

6. What is the average length of your menstrual cycle in days? (Count from the first day of your period to the start of your next period)

7. Has the length of your menstrual cycle changed since training?

YES

Please comment_____

_____

NO

8. While training for Ironman have you experienced a loss of menstruation for longer than 3 months (amenorrhea)?

YES (go to question 9)

NO (go to question 10)

9. How many months has it been since you had a regular menstrual cycle?

More than 3 months

4-5 months

6 months

More than 6 months (please comment)_____

_____

10. Are you taking an oral contraceptive pill?

YES (go to question 11)

NO (go to question 13)

11. What is the name of the oral contraceptive pill you are taking?

12. Do you ever combine 2 months of your oral contraceptive to avoid menstruation on race day?

YES

NO

13. Number of years competing in Ironman?

Thank-you for taking the time to complete this questionnaire!
*Any other comments*_____

Appendix 9

Participant Result Form (Correlated and Tabled Medical, Anthropometrical and Nutritional Data)

Results

Name:
Race Number:
Number of participants in the study: 19
Age: **Mean age:** 37.5 years
Finishing time:
Mean finishing time: 755 minutes (12 hours 35 minutes)

Weight Changes

	Pre-race	Post-race	Difference	% dehydration
Your Result				
Mean	62.88	61.66	-1.56	2.42

Pre-race Bio-electrical Impedance Assessment

	Fat Mass	% Fat	Fat Free Mass (FFM)	% FFM	Total Body Water (TBW)	% TBW
Your Result						
Mean	14.79	23.34	47.99	76.56	34.69	55.34

Urine Test Results

	Pre-race	Post-race	Difference
U. Sodium			
Mean	65.72	37.47	-25.33
U. Creatinine			
Mean	4.78	12.77	8.43
U. Osmolality			
Mean	379.39	568.80	255.93

Blood Test Results

	Pre-race	Post-race	Difference	Normal range
Haemoglobin (Hb)				
Mean	132.89	137.72	4.67	115-155
Haematocrit (Hct)				
Mean	0.40	0.40	0.01	0.33-0.48
Mean Cell Volume (MCV)				
Mean	94.09	93.47	-0.65	80.0-96.0
Mean Cell Hb (MCH)				
Mean	31.56	31.86	0.29	25-33
Total White Blood Cell (TWBC)				
Mean	7.29	16.52	9.18	4-11
Neutrophils				
Mean	4.54	13.87	9.26	2-7
Monocytes				
Mean	0.49	1.03	0.52	0.1-1.2
S. Albumin				
Mean	42.94	45.94	3	40-50
S. Urea				
Mean	5.35	8.74	3.52	3.1-7.5
S. Creatinine				
Mean	0.09	0.11	0.03	0.08-0.13
S. Sodium				
Mean	141.37	140.78	-0.83	138-145
S. Protein				
Mean	70.50	75.67	5.06	65-85
S. Globulin				
Mean	27.56	29.72	2.06	25-35
S. Osmolality				
Mean	292.58	299.06	6.44	280-300

Hormone Test Results

S. Lutenising Hormone	Progesterone	Comment

Food Diary 1

	Energy (kJ)	Energy (kcal)	CHO	g CHO/kg	Protein	g Prot/kg	Fat	Fibre	Na+
Your Result									
Mean									

Food Diary 2

	Energy (kJ)	Energy (kcal)	CHO	g CHO/kg	Protein	g Prot/kg	Fat	Fibre	Na+
Your Result									
Mean									

Appendix 10A

Individual Subjects' Calculated Resting Metabolic Rate (RMR) and Expected Daily Energy Expenditure (EDEE) Table

Athlete	FFM (kg)	Weight (kg)	Height (cm)	Age (years)	RMR (C)	RMR (H-B)	EDEE (C)	EDEE (H-B)
1	55.1	68.6	166	28	7156 kJ (1712 kcal)	6216 kJ (1487 kcal)	17176 kJ (4109 kcal)	14918 kJ (3569 kcal)
2	46.5	59.0	160	43	6366 kJ (1523 kcal)	5493 kJ (1314 kcal)	15278 kJ (3655 kcal)	13184 kJ (3154 kcal)
3	45.2	58.6	163	39	6245 kJ (1494 kcal)	5576 kJ (1334 kcal)	14989 kJ (3586 kcal)	13384 kJ (3202 kcal)
4	53.0	68.2	165	29	6964 kJ (1666 kcal)	6174 kJ (1477 kcal)	16712 kJ (3998 kcal)	14818 kJ (3545 kcal)
5	48.7	67.2	168	27	6567 kJ (1571 kcal)	6195 kJ (1482 kcal)	15759 kJ (3770 kcal)	14868 kJ (3557 kcal)
6	46.2	59.6	176	43	6337 kJ (1516 kcal)	5639 kJ (1349 kcal)	15207 kJ (3638 kcal)	13535 kJ (3238 kcal)
7	45.5	56.8	160	40	6274 kJ (1501 kcal)	5463 kJ (1307 kcal)	15056 kJ (3602 kcal)	13113 kJ (3137 kcal)
8	40.4	54.2	155	32	5806 kJ (1389 kcal)	5476 kJ (1310 kcal)	13936 kJ (3334 kcal)	13142 kJ (3144 kcal)
9	46.9	64.8	168	36	6404 kJ (1532 kcal)	5923 kJ (1417 kcal)	15370 kJ (3677 kcal)	14216 kJ (3401 kcal)
10	63.3	76.2	184	37	7913 kJ (1893 kcal)	6483 kJ (1551 kcal)	18990 kJ (4543 kcal)	15558 kJ (3722 kcal)
11	44.2	53.4	162	26	6153 kJ (1472 kcal)	5618 kJ (1344 kcal)	14768 kJ (3533 kcal)	13485 kJ (3226 kcal)
12	46.1	58.8	160	55	6329 kJ (1514 kcal)	5250 kJ (1256 kcal)	15190 kJ (3634 kcal)	12599 kJ (3014 kcal)
13	52.5	64.8	166	52	6918 kJ (1655 kcal)	5593 kJ (1338 kcal)	16603 kJ (3972 kcal)	13422 kJ (3211 kcal)
14	42.9	58.6	161	29	6036 kJ (1444 kcal)	5760 kJ (1378 kcal)	14488 kJ (3466 kcal)	13823 kJ (3307 kcal)
15	42.8	54.6	163	42	6028 kJ (1442 kcal)	5359 kJ (1282 kcal)	14467 kJ (3461 kcal)	12862 kJ (3077 kcal)
16	51.6	70.6	165	29	6834 kJ (1635 kcal)	6270 kJ (1500 kcal)	16402 kJ (3924 kcal)	15048 kJ (3600 kcal)
17	44.9	58.0	162	53	6220 kJ (1488 kcal)	5271 kJ (1261 kcal)	14927 kJ (3571 kcal)	12649 kJ (3026 kcal)
18	49.7	66.0	168	40	6659 kJ (1593 kcal)	5894 kJ (1410 kcal)	15980 kJ (3823 kcal)	14145 kJ (3384 kcal)
19	46.4	76.8	153	38	6358 kJ (1521 kcal)	6249 kJ (1495 kcal)	15257 kJ (3650 kcal)	14998 kJ (3588 kcal)

(C) = Cunningham Predictive Equation

(H-B) = Harris-Benedict Predictive Equation

Appendix 10B

Individual Subjects' EDEE Values Compared with Mean Daily Energy Intake (MDEI) Results for the First, and Second 7-day Food Diaries

Athlete	EDEE (C)	EDEE (H-B)	1st Food Diary (MDEI)	Diff (C)	Diff (H-B)	2nd Food Diary (MDEI)	Diff (C)	Diff (H-B)
1	**17176 kJ (4109 kcal)**	**14918 kJ (3569 kcal)**	**12847 kJ** (3172 kcal)	**4329 kJ** 25%	**2071 kJ** 14%	**13829 kJ** (3353 kcal)	**3347 kJ** 19%	**1089 kJ** 7%
2	**15278 kJ** (3655 kcal)	**13184 kJ** (3154 kcal)	**6203 kJ** (1795 kcal)	**9075 kJ** 59%	**6981 kJ** 53%	**8011 kJ** (2119 kcal)	**7267 kJ** 48%	**5173 kJ** 39%
3	**14989 kJ** (3586 kcal)	**13384 kJ** (3202 kcal)	**11530 kJ** (2760 kcal)	**3459 kJ** 23%	**1854 kJ** 14%	**9713 kJ** (2372 kcal)	**5276 kJ** 35%	**3671 kJ** 27%
4	**16712 kJ** (3998 kcal)	**14818 kJ** (3545 kcal)	**12435 kJ** (2980 kcal)	**4277 kJ** 26%	**2383 kJ** 16%			
5	**15759 kJ** (3770 kcal)	**14868 kJ** (3557 kcal)	**10940 kJ** (2957 kcal)	**4819 kJ** 31%	**3928 kJ** 26%	**8108 kJ** (2283 kcal)	**7651 kJ** 49%	**6760 kJ** 45%
6	**15207 kJ** (3638 kcal)	**13535 kJ** (3238 kcal)	**10838 kJ** (2781 kcal)	**4369 kJ** 29%	**2697 kJ** 20%	**9835 kJ** (2576 kcal)	**5372 kJ** 35%	**3700 kJ** 27%
7	**15056 kJ** (3602 kcal)	**13113 kJ** (3137 kcal)	**8921 kJ** (2208 kcal)	**6135 kJ** 41%	**4192 kJ** 32%	**10901 kJ** (2614 kcal)	**4155 kJ** 28%	**2212 kJ** 17%
8	**13936 kJ** (3334 kcal)	**13142 kJ** (3144 kcal)	**8260 kJ** (2023 kcal)	**5676 kJ** 41%	**4882 kJ** 37%	**8669 kJ** (2074 kcal)	**5267 kJ** 38%	**4473 kJ** 34%
9	**15370 kJ** (3677 kcal)	**14216 kJ** (3401 kcal)	**9556 kJ** (2289 kcal)	**5814 kJ** 38%	**4660 kJ** 33%	**10087 kJ** (2414 kcal)	**5283 kJ** 34%	**4129 kJ** 29%
10	**18990 kJ** (4543 kcal)	**15558 kJ** (3722 kcal)	**10315 kJ** (2739 kcal)	**8675 kJ** 46%	**5243 kJ** 34%			
11	**14768 kJ** (3533 kcal)	**13485 kJ** (3226 kcal)	**12530 kJ** (3013 kcal)	**2238 kJ** 15%	**955 kJ** 7%	**11975 kJ** (2863 kcal)	**2793 kJ** 19%	**1510 kJ** 11%
12	**15190 kJ** (3634 kcal)	**12599 kJ** (3014 kcal)	**11228 kJ** (2716 kcal)	**3962 kJ** 26%	**1371 kJ** 11%	**10164 kJ** (2485 kcal)	**5026 kJ** 33%	**2435 kJ** 19%
13	**16603 kJ** (3972 kcal)	**13422 kJ** (3211 kcal)	**12246 kJ** (2931 kcal)	**4357 kJ** 26%	**1176 kJ** 9%	**11153 kJ** (2668 kcal)	**5450 kJ** 33%	**2269 kJ** 17%
14	**14488 kJ** (3466 kcal)	**13823 kJ** (3307 kcal)	**8060 kJ** (1929 kcal)	**6428 kJ** 44%	**5763 kJ** 42%	**10376 kJ** (2484 kcal)	**4112 kJ** 28%	**3447 kJ** 25%
15	**14467 kJ** (3461 kcal)	**12862 kJ** (3077 kcal)	**12401 kJ** (3035 kcal)	**2066 kJ** 14%	**461 kJ** 4%	**11512 kJ** (2789 kcal)	**2955 kJ** 20%	**1350 kJ** 11%
16	**16402 kJ** (3924 kcal)	**15048 kJ** (3600 kcal)	**11592 kJ** (2791 kcal)	**4810 kJ** 29%	**3456 kJ** 23%	**11375 kJ** (2721 kcal)	**5027 kJ** 31%	**3673 kJ** 24%
17	**14927 kJ** (3571 kcal)	**12649 kJ** (3026 kcal)	**15945 kJ** (3852 kcal)	**+1018 kJ** -7%	**+3296 kJ** -26%	**11913 kJ** (2848 kcal)	**3014 kJ** 20%	**736 kJ** 6%
18	**15980 kJ** (3823 kcal)	**14145 kJ** (3384 kcal)	**11048 kJ** (2760 kcal)	**4932 kJ** 31%	**3097 kJ** 22%	**8687 kJ** (2114 kcal)	**7293 kJ** 46%	**5458 kJ** 39%
19	**15257 kJ** (3650 kcal)	**14998 kJ** (3588 kcal)	**8520 kJ** (2035 kcal)	**6737 kJ** 44%	**6478 kJ** 43%	**6321 kJ** (1508 kcal)	**8936 kJ** 59%	**8677 kJ** 58%