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CAUSES OF HYPONATREMIA IN NEW ZEALAND
FEMALE ULTRADISTANCE TRIATHLETES

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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 $< 135\text{mmol/L}$) has been recognised as a serious complication of prolonged exercise, the aetiology 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 ± 1.1 kg ($p = < 0.001$) equated to a percentage dehydration of $2.4 \pm 1.8\%$. One athlete from the study group had asymptomatic hyponatremic (post-race plasma sodium concentration 134 mmol/L). The athlete was the smallest subject in the study (53.4 kg), finished the race 1 kg 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.

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