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Epidemiology of Sports Injuries

Review

Sodium supplementation during prolonged exercise: effects on plasma sodium and performance

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Abstract

Introduction

The reported incidence of exercise-associated hyponatraemia (plasma sodium concentration <135 mmol/L) in endurance sport has increased notably in recent decades. Sodium supplements have been suggested to attenuate the decline in plasma sodium concentration and hence, prevent the effects of exercise-associated hyponatraemia on performance. This article reviews intervention studies that have assessed the impact of sodium supplements on plasma sodium concentrations and/or performance during endurance exercise.

Conclusion

Despite significant results in some laboratory studies show benefits of sodium supplementation, more recent field studies suggest that sodium supplementation has little impact on plasma sodium concentration during a racing situation and no effect on performance. These discrepancies are likely because of design differences between the studies. However, a well-controlled crossover field trial in a hot environment is still required in order to develop practical recommendations.

Introduction

Exercise-associated hyponatraemia (EAH), is a blood electrolyte disorder induced during the 24 hours, following exercise[1]. It is defined as a plasma-sodium concentration (plasma [Na^+]) less than the normal reference range; for most laboratories it is found to be 135 mmol/L[1].

Mild hyponatraemia (plasma [Na^+] 130 – 135 mmol/L) is often asymptomic[2–5], although mild symptoms can develop[6]. These include bloating, nausea, lethargy, vomiting and headache[1,7–9]. Whilst such symptoms are not life-threatening, they likely have some effect on exercise performance. They are also non-specific, and can often be confused with the symptoms of dehydration. As the athlete's plasma [Na^+] decreases below 130 mmol/L, the symptoms correspondingly increase in severity. This occurs as a result of cerebral oedema, producing symptoms, such as confusion, disorientation, seizures, respiratory distress, coma and even possible death[1,10–13].

The incidence rates of EAH for endurance races has been reported to be up to 30% amongst Ironman triathletes[1,13–17], and in marathon runners up to 13%[3,18–20]. Such endurance events host large participation numbers; the 2010 London marathon, for example, hosted 36,000 runners[21]. With this in consideration, such incidence rates correspond to a large absolute number of EAH sufferers in the sporting population.

Whilst excessive fluid consumption has been a clear causative factor of EAH, there is evidence to suggest that excessive sweat sodium loss may also contribute[7,8]. It has been speculated that sodium supplementation during endurance exercise could attenuate the reduction in plasma [Na^+] in these situations[22], therefore reduce the risk of EAH and potentially improve performance. The research stems from both laboratory and field studies, each with their own advantages and disadvantages. In this review, intervention trials with a high and low sodium trial, which measure plasma sodium during an endurance exercise protocol will be discussed. These studies are summarised in Table 1.

Laboratory studies

One of the first laboratory studies was undertaken in 1991 by Barr, Costill and Fink[23]. Eight participants completed a crossover intervention of three cycling trials, at 55% VO_2max for six hours. Participants consumed fluid to match their sweat rate, with either water and/or a 25 mmol/L (29 mmol/h) NaCl saline solution, or they ingested no fluid during the three trials. Barr et al.[23] observed no significant differences in plasma [Na^+] between the two 'fluid' trials (p = 0.27).

These results were similar to those seen in another crossover study by Sanders and colleagues[24]. Six endurance trained male cyclists participated in three trials, again cycling at 55% $\dot{V}O_2$max, but for a shorter time for 4 hours as compared to Barr et al. This time the participants ingested 4.6 mmol/L (4 mmol/h), 50 mmol/L (48 mmol/h) and 100 mmol/L (96 mmol/h) of a sodium carbohydrate drink (~0.965 L/h).

Similar to the findings from Barr et al.[23], plasma [Na^+] was maintained regardless of whether participants were supplemented with sodium. However, Sanders et al.[24] built on these findings to suggest why plasma [Na^+] was maintained. When salt tablets were consumed, the intracellular fluid (ICF) moved into the extracellular fluid (ECF), expanding

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Table 1 Summary of sodium supplement intervention trials.				
Year, Author, Title	**Aim, Design**	**Methods**	**Results**	**Comments**
1991 Barr, Costill, Fink. Fluid replacement during prolonged exercise: effects of water, saline, or no fluid[9].	Randomised crossover design. Effect of water, saline or no fluid on plasma [Na^+] and performance.	8 participants. 3 × 6 h cycling at 55% $\dot{V}O_2$max. Saline (25 mmol/L Na), water or no fluid to replace sweat losses.	No difference in plasma [Na^+] between water and saline (p = 0.27), but plasma [Na^+] higher in the no-fluid group.	First controlled study to suggest over-hydration increases risk of EAH.
1999 Vrijens, Rehrer. Sodium-free fluid ingestion decreases plasma sodium during exercise in the heat[15].	Randomised crossover design. Replace sweat losses with either a sodium-free beverage or sodium-containing sports drink during exercise in the heat.	10 trained men (approx. 25 years). 2 × 3 h cycling 55% $\dot{V}O_2$max. Temperature 34°C, 65% RH. Sports drink (S) (18 mmol/L Na) or water (C) given equal to sweat rate. Sports drink trial ingested around 61 mmol Na^+.	4 of 10 subjects completed 3 h cycling in both trials. Plasma sodium decrease was greater with C than S (p = 0.02). Time to exhaustion decreased with lower plasma [Na^+] (p = 0.02).	One subject diagnosed with hyponatraemia in the water trial. Consumed 1 L/hr. Higher rate of weight loss than the other participants.
1999 Sanders, Noakes, Dennis. Water and electrolyte shifts with partial-fluid replacement during exercise.	Should athletes who typically replace only. ~50% of fluid loss are required replace their Na^+? Randomised crossover design.	6 cyclists , 3 × 1.5 h rides at 65% VO_2 peak Participants ingested no fluid 4.6 mmol/L (tap water), 100 mmol/L (~80 mmol/h) of NaCl in tablets. Fluid was ingested as a 400 mL bolus at the start of exercise and then 100 mL every 10 minutes until the 80th minute. Temperature 20°C, 55% RH.	Water trial resulted in a lower plasma [Na^+] compared to no fluid and saline trials. Similar plasma [Na^+] was observed between the no fluid and saline trials.	Exercise duration is shorter than events reporting incidence of hyponatraemia.
2001 Sanders, Noakes, Dennis. Sodium replacement and fluid shifts during prolonged exercise in humans[12].	Randomised crossover design. Electrolyte replacement effects on fluid shifts during exercise.	6 cyclists. 3 × 4 h rides 55% $\dot{V}O_2$max. 900 mL/h CHO + Na^+ solution (4.6 mmol/L) (C) or (C) + Na^+ tablets with 400 mg or 800 mg Na^+. (Hence, 4.6, 50, 100 mmol/L for each trial)	Plasma [Na^+] maintained in all interventions (p > 0.05). In high Na^+, ICF is contracted to conserve ECF and expand plasma volume. Reduced renal-free water clearance in high Na^+ to decrease urine production.	Similar to Barr et al. (1991). Less rapid declines in plasma [Na^+] than Vrijens and Rehrer (1999), less extreme climate and less fluid intakes. First study to 'blind' participants using tablets.
2002 Speedy, Thompson, Rodgers, Collins, Sharwood. Oral Salt supplementation during ultradistance exercise[13].	Non-randomised intervention trial. Influence of oral sodium supplementation on body mass, plasma [Na^+] and plasma volume in athletes during the Cape Town Ironman triathlon.	38 athletes (2 female athletes) provided with 700 mg/h salt tablets, all ingested more than 4 g during the race (S). Compared against 133 controls, not given additional sodium (C). Controls matched post-hoc for 1. Body mass change, 2. Pre-race plasma [Na^+] Temperature 22°C, 55% RH	Matched for body mass: S had a significantly greater decrease in haematocrit, and an almost significantly greater increase than C in plasma [Na^+] (p = 0.08). Matched for pre-race plasma [Na^+]: body mass change was significantly less in S, but no difference in change in plasma [Na^+] (p > 0.05).	Not randomised. Did not include athletes who failed to complete the race. Did not control for athletes in control group that could have taken their own salt supplementation. Small changes mirrored those seen in the laboratory with Sanders et al. (2001). Only one gained EAH, could not assess the risk.

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Table 1 Continued

Year, Author, Title	Aim, Design	Methods	Results	Comments
2003 Twerenbold, Knechtle, Kakebeeke et al. Effects of different sodium concentrations in replacement fluids during prolonged exercise in women[14].	Randomised crossover design. Different concentrations of electrolyte replacement in exercise performance and physiology.	13 trained women runners. 3 × 4 h runs on 400 m track. Ingested 1 L/h high Na^+ (25 mmol/L), low Na^+ (15 mmol/L), water. Temperature (range) 5.3–13.9°C	Decrease in plasma [Na^+] smaller in high Na^+ than water ($p < 0.001$). Greater incidence of EAH in water trial. Performance not different.	Similar results to Vrijens and Rehrer (1999). No observed fluid shifts as seen in Sanders et al. (2001).
2006 Hew-Butler, Sharwood, Collins, Seedy, Noakes. Sodium supplementation is not required to maintain serum sodium concentrations during an Ironman triathlon[11].	Randomised trial. Ingestion of additional Na^+ during Cape Town Ironman on plasma [Na^+], risk of EAH, performance.	145 triathletes competing in the Cape Town Ironman. Control group was randomised to 40 placebo tablets of starch (C). Experimental group given 40 identical looking tablets, with 244 mg Na^+ (10.6 mmol) (S). Asked to consume 1–4 (244–1000 mg) tablets every hour. Temperature 17°C, 63% RH	No significant differences ($p > 0.05$) between C and S in finishing time, Na^+ before and after the race, weight before the race, weight change, prevalence of medical care. No salt cravings from either group.	Food and fluid intakes were allowed ad libitum, meaning athletes could eat other salt products; not considered in this analysis. One subject developed hyponatraemia (in the control group), over drank.
2009 Anastasiou, Kavouras, Arnaoutis, Gioxari, Kollia, Botoula, Sidossis. Sodium replacement and plasma sodium drop during exercise in the heat when fluid intake matches fluid loss[8].	Randomised crossover design. Different levels of Na^+ intake on maintaining plasma volume and preventing EAH during prolonged exercise in the heat.	13 untrained men. Cycling/walking for 3 h followed by 5 min rest, calf raisers and steep walking at 5.5 km/h for 45 min. Replace sweat loss during first 3 h, then 150 ml every 15 min during the walking (ACSM guidelines). 4 trials: 1. Low Na CHO drink (20 mmol/L). 2. High Na^+ CHO drink (36 mmol/L). 3. Placebo. 4. Water. Temperature 30°C	HNa and LNa trials resulted in stable plasma volume. Pl and W trials tended to decrease plasma volume over time. Plasma osmolarity levels were higher in HNa than Pl and W after phase 1 and thereafter. LNa after phase 2, 3 ($p < 0.05$). Plasma [Na+] was significantly higher in HNa and LNa compared to Pl and W ($p < 0.05$). Pl and W entered HN states towards the end of the trial.	Moderate levels of sodium attenuate the decline in plasma [Na^+] and preserve plasma volume. Agree with Vrijens and Rehrer (1999). HNa did not show any additional advantage than LNa.
2013 Cosgrove, Black. Sodium supplementation has no effect on endurance performance during a cycling time-trial in cool conditions.	Randomised crossover design. Impact of sodium supplementation on performance and plasma [Na^+] during a 72 km cycling time-trial.	9 well-trained cyclists (5 male cyclists, 4 female cyclists) cycling 72 km hilly road course three times, separated by 7–14 days. First trial is familiarisation, second and third trials are 700 mg/h sodium supplement or identical placebo. Drink ad libitum. Temperature 14°C, 63% RH	No difference in performance ($p = 0.46$) and relative plasma [Na^+] change pre-race to post-race ($p = 0.60$).	Athletes on sodium supplements consumed significantly more fluid ad libitum ($p = 0.02$).

ACSM, American College of Sports Medicine; EAH, exercise-associated hyponatraemia; ECF, extracellular fluid; ICF, intracellular fluid; RH, relative humidity; Temperature, environmental temperature

plasma volume and preventing any large increase in plasma [Na^+] levels. In contrast, when salt tablets were not consumed, the ECF was reduced and the ICF was maintained. Thus, plasma volume decreased and plasma [Na^+] and osmolality were maintained. This phenomenon was further explained by a significant decrease in

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renal-free water clearance during the sodium capsule trials compared to the solution-only trial ($p < 0.05$), highlighted by differences in urine output, and conservation of ECF when sodium was consumed. Interestingly neither Barr et al. nor Sanders et al. reported a significant difference in sweat sodium concentration between their respective high- and low-sodium trials.

The results of these laboratory studies suggested that sodium supplementation may have little effect in preventing EAH[23,24]. However, other studies challenged this view. A crossover intervention trial by Vrijens and Rehrer in 1999[25] demonstrated that sodium could play an important role in the prevention of EAH. Again cycling at 55% VO_2max, but only for three hours, and as with the Barr et al.[23] study, ingesting fluid equal to sweat rates. The participants ingested either water, or Gatorade (18 mmol/L Na^+). In contrast to Barr et al.[23] and Sanders et al.[24], the Gatorade intervention significantly attenuated plasma [Na^+] reduction compared to water (water vs. Gatorade = -2.48 mmol/L/h vs. -0.86 mmol/L/h). Unlike the previous two studies, this study reported that one participant became hyponatraemic (plasma sodium 128 mmol/L) during the water trial, interestingly with a fluid intake rate lower than the mean fluid intake rate for the group. It should be noted, as the intervention in the Vrijins and Rehrer study consisted of consuming Gatorade, differences other than sodium were present between the trials, for example carbohydrate and other electrolytes. They also observed that time to exhaustion decreased with lower plasma sodium concentrations. This suggested a role for sodium supplementation in performance, although more likely to be because of the consumption of carbohydrates.

Despite these apparent limitations in the Vrijens and Rehrer study[23], a later study performed by Anastasiou and colleagues[22] agreed with their results. This trial involved 13 untrained men completing a multi-disciplinary intervention of cycling, walking and calf raises for three hours. Again fluid was ingested equal to sweat loss, and four trials were completed, including high sodium (Gatorade thirst Quencher 36.2 mmol/L), a low sodium (Gatorade Endurance 19.9 mmol/L), water or an artificially flavoured placebo. Both the low and high sodium solutions attenuated the decline in plasma [Na^+] during the exercise intervention, compared to the water and placebo and some of the participants on the water and placebo had plasma sodium concentrations less than 135 mmol/L at the end of the exercise protocol. Again discrepancies in carbohydrate ingestion existed between the two sodium and two non-sodium trials, and as differences were only observed between the sodium and non-sodium trials, and not between the low- and high-sodium trials, it suggested that something other than sodium could be responsible.

These four studies provide conflicting results on the ability of sodium ingestion to influence blood-sodium concentrations, which can be attributed to differences in methodology. Sanders et al.[24] suggested that the steeper declines in plasma [Na^+] observed in the Vrijens and Rehrer study[25] could also be because of differences in urine output, which were considerably lower in the Vrijens and Rehrer study. This is likely due to anti-diuretic hormone secretion in response to exercise stress to conserve plasma volume[26]. When combined with greater sweat sodium losses and large fluid intakes, it is understandable why Vrijens and Rehrer reported a greater decrease in plasma [Na^+].

These studies also highlight some limitations with laboratory-based data collection, particularly in terms of exercise prescription. Exercising at a set VO_2max may stimulate physiological responses to endurance exercise, but it is not applicable to a racing situation, where the degree of exercise intensity is higher and constantly being modified. Twerenbold et al.[27] attempted to address some of the limitations associated with the laboratory-based studies in a 2003 crossover-intervention study, recruiting 13 well-trained, healthy female runners to participate in three four-hour running time trials around a 400 m track. The participants consumed 1 L/h of a high sodium-carbohydrate solution (25 mmol/L), low sodium-carbohydrate solution (15 mmol/L), or water for each of the respective three trials.

The change in plasma [Na^+] from pre-run to post-run was significantly smaller in the high-sodium trial compared to the water trial (sodium vs. water, -2.5mmol/L vs. -6.2mmol/L; $p = 0.001$), which supports the changes observed by Vrijens and Rehrer[25]. This was further reflected in the proportion of participants that developed mild EAH in each trial (46% when consuming high-sodium beverage vs. 92% when consuming water). Despite these large differences in plasma [Na^+], there were no significant effects on performance. This suggested that the body is able to cope with sodium imbalances when exercising in cool conditions.

Although the Twerenbold et al. study[27] used a race-like exercise prescription to assess sodium supplementation, it has been criticised, alongside Vrijens and Rehrer[25] and Anastasiou et al.[22] of over-hydrating their participants and inducing dilutional EAH[28]. The standardised-fluid intakes of 1 L/h[27] were shown to elicit dilutional hyponatraemia (below 135 mmol/L) in 69% of their participants, despite being within the recommended fluid intake guidelines for athletes at that time[29]. Indeed, voluntary fluid consumption tends to be about half an athlete's sweat rate for most sports[30], which highlights

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the importance of investigating the effects of sodium supplementation when athletes consume fluids ad libitum, such as during field studies.

Field studies

The first field study was conducted by Speedy et al.[31], which investigated the effects of sodium supplementation during the 2000 Cape Town Ironman Triathlon. Thirty-eight athletes were recruited at race registration, three days prior to the race. These participants were issued with sufficient salt tablets to provide 700 mg/h of sodium, during the race, which lasted for approximately 12.5 hours. Some of the tablets also contained carbohydrate, but this was not the case for all the salt tablets. The control group (n = 133) was not given any salt tablets, but completed the same pre- and post-race measures as the intervention group. As the participants were not randomised to receive the intervention, the two groups were matched during analysis, according to both body mass change during the race and pre-race plasma sodium. However, despite the matched analysis, this is still a significant limitation for the study, especially as neither group was blinded for the intervention and measures of pre-race body mass and sodium concentration were taken 1–3 days prior to the race and no dietary standardisation or measurements were taken.

There were no significant differences in the change in plasma [Na^+], when matched for pre-race plasma [Na^+], neither was there any difference in performance between the groups. Interestingly, the prevalence of EAH during this race was particularly low; only one athlete developed asymptomatic hyponatraemia out of the entire field. Previous prevalence rates have been much higher; up to 29% of race finishers were reported to have developed EAH by Hiller et al. in the Hawaiian Ironman Triathlon[17], and Speedy et al. reported 18% of race finishers in the New Zealand Ironman Triathlon[15], despite similar temperatures and relative humidity[15]. The low prevalence rates of EAH observed in this Ironman Triathlon could therefore reflect the results gathered in this study; plasma [Na^+] increased and weight decreased in both the control and supplementation groups, which suggested that participants became hypohydrated, and did not over consumed the fluid.

A similar trial was conducted by Hew-Butler et al.[28] a year later during the 2001 Cape Town Ironman, in which 145 triathletes, with a finish time around 12.5 hours were randomised to receive either salt tablets (10.6 mmol Na^+ per tablet, between 10.6–42.4 mmol/h) or a placebo tablet (596 mg starch per tablet). The randomisation and blinding protocol was advantageous compared to the study by Speedy et al.[31], as it reduced selection-bias in analysis, and allowed a much more comparable control group. Despite this, the results of the Hew-Butler et al. study[28] supported the findings of Speedy et al. and showed no significant differences in plasma [Na^+], body mass change, performance or prevalence of medical care between the intervention and placebo groups.

Hew-Butler et al. observed a number of participants developing mild hyponatraemia (post-race [Na^+] 130–135 mmol/L), even though they lost weight during Cape Town race. This should be interpreted with caution as pre-race weight was determined three days prior to the start of the race, so it cannot be confirmed that the loss of body mass occurred during the race and not before the race. This highlights that although over consuming fluid is an important causative factor, other factors can play a role in the development of EAH[32]. There was no difference in post-race plasma [Na^+] between the intervention and control groups in both the field studies[28,31], however this may be reflective of the inherent limitations with these study designs, where dietary intakes before and during the race are not controlled. The Cape Town Ironman provided all athletes with sports drink (Energade, [Na^+] = 18 mmol/L), every 20 km in the cycling leg and every 2.5 km in the running leg. It is therefore, probable that the control group consumed sodium-containing sports foods, with a similar consistency to those in the study by Vrijens and Rehrer[25] and hence, it is difficult to directly compare the results observed in the intervention group to the control group. Further, the fact that both these races were undertaken in South Africa and the low prevalence of hyponatraemia that is reported could indicate that the ad-libitum food and fluid intakes in this country are different to other races, where much higher prevalence of hyponatraemia have been reported.

Cosgrove and Black[33] tried to address some of these control issues by conducting a blinded, randomised-crossover study of sodium supplementation in a 72 km road cycling time-trial. Nine well-trained cyclists (5 male cyclists, 4 female cyclists) consumed either a 30 mmol/h sodium, or a placebo.

In line, with the research at the Cape Town Ironman, Cosgrove and Black[33] found that sodium supplementation had no effect on plasma [Na^+] change (relative change pre-race to post-race, salt = 0.56%, placebo = 0.47%, p = 0.7) during the time-trial, with neither of the participant becoming hyponatraemic on either trial, nor was there any difference in the time taken to complete the time trial between the supplemented and placebo groups. The authors suggested the mildly-cold conditions of the time-trials (14°C) that did not elicit large enough sweat sodium losses to warrant sodium replacement in the time-trials.

Discussion

In this review, the authors have referenced some of their own studies. These referenced studies have been

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conducted in accordance with the Declaration of Helsinki (1964) and the protocols of these studies have been approved by the relevant ethics committees associated to the institution in which they were performed. All human subjects, in these referenced studies, gave informed consent to participate in the studies.

It is interesting that all of the studies, which have shown a beneficial effect have been between the trials containing carbohydrate-electrolyte beverage and water trials. This raises the question as to whether it is the sodium per-se or another substance within the drink, which is eliciting the effect. Indeed, the ingestion of carbohydrate is likely to attenuate the stress of exercise. However, as these carbohydrate electrolyte beverages contain more than just sodium and carbohydrate, any of the drink components could have influenced the results. However, as they were not separately assessed, the exact reasoning for these results cannot be confirmed.

The obvious criticism that the studies reporting beneficial effects are because of the over-consumption of fluids that may well be true, but both Barr et al. and Sanders et al. replaced sweat losses, but yet did not see any differences in plasma sodium between the water and sodium containing trials. Nearly all of the studies have failed to result in high rates of hyponatraemia, even when large amounts of sodium-free fluid have been ingested, this conflicts the observational data from endurance races, where up to 30% of athletes have been reported to have EAH. Future research is required to concentrate on those with a history of hyponatraemia to determine why some athletes are more susceptible to hyponatraemia. This sub-population may benefit from sodium supplements, but before conclusions can be made further investigations are necessary.

Conclusion

Whilst there is some suggestion from laboratory studies that sodium supplementation could reduce EAH incidence, and improve performance, in particular during exercise in the heat, recent field trials have demonstrated that sodium supplementation has no effect on plasma sodium concentrations during a racing situation.

Abbreviations list

EAH, exercise-associated hyponatraemia; ECF, extracellular fluid; ICF, intracellular fluid.

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