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PRESS RELEASE

Changes in body mass alone explain almost all of the variance in the serum sodium concentrations during prolonged exercise. Has commercial influence impeded scientific endeavour?

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SUMMARY

In 1991, we provided definitive evidence that exercise-associated hyponatraemia (EAH) is caused by abnormal fluid retention in those who overdrink during prolonged exercise, but this finding was ignored. Instead, in 1996, influential guidelines of the American College of Sports Medicine (ACSM) promoted the concept that athletes should drink 'as much as tolerable' during exercise. What followed was an epidemic of cases of EAH and its associated encephalopathy (EAHE). A recent study funded by the sports drink industry confirms our 1991 finding by showing that 95% of the variance in the serum sodium concentration during exercise can be explained by changes in body mass alone. The possibility is that commercial influence delayed the acceptance of our findings for two decades.

INTRODUCTION

Describing the first reported cases of EAH and EAHE in 1985, we postulated that: 'The aetiology of this condition appears to be voluntary hyperhydration with hypotonic solutions combined with moderate sweat sodium chloride losses…advice (on fluid replacement) should be tempered with the proviso that the intake of hypotonic fluids in excess of that required to balance sweat and urine losses may be hazardous in some individuals.'[1]

A study of fluid and sodium balance in eight ultramarathon runners with EAHE published in 1991[2] showed that normalisation of their serum sodium concentrations ([Na^+]) occurred only after all had excreted a fluid excess ranging from 2 to 6 l. None had developed a larger sodium deficit than did a control group of runners without EAH or EAHE. We concluded that: 'This study conclusively resolves this issue (of what causes EAHE). It shows that each of eight subjects who collapsed with the hyponatremia of exercise (mean plasma sodium concentration 122.4±2.2 mM) were fluid overloaded by an amount ranging from 1.22 to 5.92 liters.' As a result, 'the hyponatremia of exercise results from fluid retention in subjects who ingest abnormally large fluid volumes during prolonged exercise.' The article might as well have been written in Latin, for all the impact it has had.

For example, the 2007 ACSM position stand on exercise and fluid replacement[3] includes a section on EAH. There is no mention of our 1991 paper, and the sole reference to our original[1] report, now recognised as seminal,[4] is gratuitous, for the six authors believe that the first description of EAH is in an obscure paper published in the *South African Medical Journal* in 1971.[5] Yet nowhere in that article does the word hyponatraemia appear. Nor did the authors ever claim they had discovered a novel condition. Instead they reported simply fluid intakes and changes in body weight and serum [Na^+] in a group of South African runners who finished the 90 km Comrades Marathon footrace in good health and without symptoms. It is not clear how a novel disease can be identified in a group who are without that disease. Instead it appears that the Na^+ measurements were unreliable as impossibly low serum [Na^+] was reported even before the race in athletes who were without symptoms.[6]

Thus, the troubling question recurs: how is it possible that our definitive conclusions established more than two decades ago continue to be ignored by those who draw up influential drinking guidelines for exercisers?[7]

It is instructive to review the industrial connections of those who wrote the 2007 ACSM Position Stand.[3] Of the six authors, four – Drs. Maughan, Burke, Eichner and Stachenfeld – have direct and longstanding involvement with Gatorade and the Gatorade Sports Science Institute (GSSI), but only three (Drs Maughan, Eichner and Stachenfeld) deemed it necessary to disclose in the Position Stand the existence of that relationship. The two remaining authors – Drs. Sawka and Montain – are employed by the United States Army Research Institute for Environmental Medicine (USARIEM). As described elsewhere,[8] it was a publication by USARIEM scientists in 1984[9] that first advocated very high (1.8 l/h) rates of fluid ingestion, first in the US military[10] and later, following the publication of the 1987[11] and 1996[12 13] ACSM Position Stands, a somewhat lesser rate (1.2 l/h) for the exercising public. While the 1987 ACSM Position Stand[11] is anonymous, all four senior authors of the 1996[12 13] and 2007 ACSM position stands[3 14] were employed either by the US military or by USARIEM, or had trained there. It is perhaps surprising that given the large number of experts available to it, the ACSM should consistently choose the authors of its influential Position Stands from such a narrow selection of group thinkers.

It may be relevant that Gatorade produces special prepacked 'care' packs for US military 'troops in battle fields or boot camps requiring on-the-go hydration to replace nutrients and carbohydrates loss [sic] during exercise and battle.'[15] Indeed:

'Record heat and sales to the military overseas led to a shortage of Gatorade in 2005.'[16] Since the US military provides a captive market for Gatorade products, it would only be sensible for that company to develop a special relationship with relevant groups in the US military as it has with the ACSM, discussed subsequently.

According to this logic, one possible reason why our findings have been ignored could be that they are inconvenient. Our first paper was published 2 years before Quaker Oats purchased the Gatorade brand from Stokely Van-Camp and founded the GSSI. One goal of the GSSI may have been the promotion on a global scale[17–20] of the novel concept that 'dehydration,' and not overhydration, is a potentially fatal complication of exercise.[21]

Our second paper[2] was also published 1 year before the ACSM accepted its first 'sponsorship' from Gatorade in the form of a $250 000 'gift.'[22] Quaker Oats announced that the ACSM is 'the premier organization in sports medicine and exercise science.'[22] For some years, Gatorade and the GSSI were the sole 'platinum' sponsors of the ACSM.

Perhaps emboldened by this support and the 1984 USARIEM study[9] and the 1987 ACSM Position Stand,[11] both of which promoted very much higher rates of fluid intake during exercise than ever before,[23] the next (1996) ACSM Position Stand advanced what I have called the 'zero % dehydration rule.' In future, all athletes would be encouraged to drink 'as much as tolerable' during exercise. The commercial value of this 'zero % dehydration rule' is obvious, for it requires that all must begin to drink as much as they can, the instant they start any form of exercise. What ensued[24] after the 'zero % dehydration rule' took hold was an epidemic of cases of EAH and EAHE.

Fortunately, normal impartial science will deliver the truth. By designing an experiment[25] to distinguish between two opposing hypotheses of what causes EAH, research funded by PepsiCo, the current owners of the Gatorade brand, has confirmed the truth we discovered in 1991. The research design was commercially unwise, since findings that supported our original conclusions would disprove the intellectual underpinning of some significant part of that industry's wealth.

By inducing different degrees of body mass change and net electrolyte loss during prolonged exercise, these authors[25] determined the relative contributions of these two variables to changes in [Na^+] during exercise. Here I reproduce the relevant findings in two figures. The data for serum [Na^+] were calculated from the reported pre-exercise value of 142.4 mmol/l and the change in serum [Na^+] read off figure 2 in their article. The data for net electrolyte change (sodium (Na^+) and potassium (K^+)) are from their table 2.

Figure 1 shows a significant ($p=0.04$; $r^2=0.37$) linear relationship with a negative slope between the postexercise serum [Na^+] and the net (Na^+ and K^+) electrolyte loss so that the lowest postexercise serum [Na^+] occurred in the groups with the smallest net electrolyte losses during exercise. Thus, increasing levels of electrolyte loss were associated with increased, not decreased, serum [Na^+]. There was a small but statistically and biologically insignificant effect (arrows) of sodium ingestion on the serum [Na^+] in the two groups who overdrank so that they either did not lose any weight or gained weight during exercise. This effect was of no biological consequence, since ingesting a solution with a [Na^+] of 30 mmol/l (1.66 times greater than the concentration of Gatorade) reduced the fall in serum [Na^+] caused by overdrinking by between 0.5 and 2 mmol/l. But the avoidance of overdrinking caused the serum [Na^+] to increase by 2–5 mmol/l (figure 1).

Figure 2 shows a significant linear relationship ($p=0.0001$; $r^2=0.95$) with a negative slope between the change in body mass during exercise and the postexercise serum [Na^+]. Thus, 95% of the variance in the serum [Na^+] in these studies is explained solely by the variance in body mass.

Perhaps not unsurprisingly, the authors, employed by the industry, failed to understand their findings, for they concluded that: 'Na^+ ingestion becomes even more critical as the duration of exercise increases' (p. 97).

Instead, these data confirm our original findings, confirmed repeatedly[26–31] but ignored by the 2007 ACSM Position Stand: specifically that the very low postexercise serum [Na^+] in athletes with EAHE is due to body weight gain as a result of fluid retention (figure 2) to which any associated electrolyte, specifically sodium, deficit makes no significant contribution (figure 1). Thus, to prevent EAH, athletes must not gain weight during exercise;[31] ingesting more sodium 'as the duration of

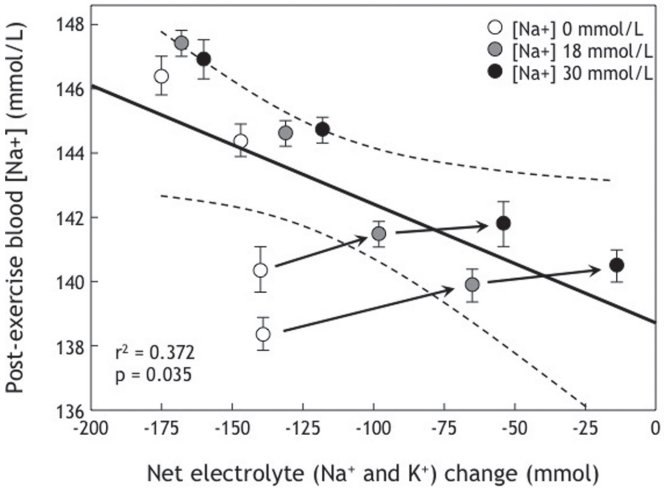

Figure 1 Postexercise serum [Na^+] plotted against net electrolyte (Na^+ and K^+) change in 12 different experimental combinations of weight loss and sodium ingestion during exercise. Arrows indicate the effects of the ingestion of drinks with progressively higher [Na^+] on reducing the extent of the fall of serum [Na^+] in those who overdrink during exercise.

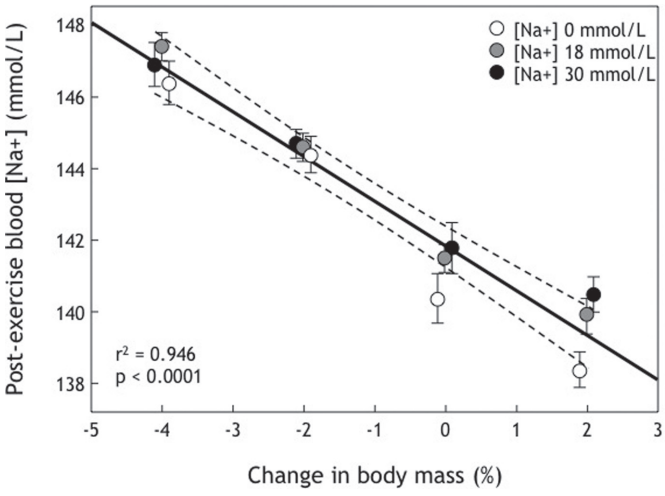

Figure 2 Postexercise serum [Na^+] plotted against change in body mass (%) in 12 different experimental combinations of weight loss and sodium ingestion during exercise.

exercise increases' will have no biologically significant effect as these new data prove convincingly.

Indeed, the small but insignificant effect of sodium ingestion on the serum [Na^+] in those who gained weight during exercise (arrows in figure 1) is probably not the result of a reduced electrolyte deficit, since the lowest serum [Na^+] occurred in those with the smallest sodium deficits. Rather, sodium ingestion must act in some other way, perhaps by influencing sodium release from the postulated internal exchangeable sodium stores.[31]

There are a number of lessons from this sorry saga.[32] First two studies[25 29] funded by a company which has consistently promoted the erroneous concept that sodium ingestion during exercise can prevent EAHE[7] now prove the opposite, specifically that the crucial determinant of the serum [Na^+] during exercise is the extent of the body weight change and not the magnitude of any associated sodium deficit. The industry's erroneous claim was the central theme of a recent symposium also funded by industry, the proceedings[33] of which were provided for free to all members of the ACSM. In the interests of truth, this erroneous information must now be corrected by the relevant bodies.

The results of these studies mean that never again can the claims be made that EAH and EAHE are (1) due to a sodium deficit that can be prevented (2) by the ingestion of a sodium-containing sports drink during exercise. Instead it behoves the industry to warn that the overconsumption of sports drinks, like the overconsumption of water, can cause EAH and EAHE.

Finally, this material is published in the *British Journal of Sports Medicine*, and not in the *Journal of Applied Physiology*, for the reason that a 500-word letter outlining the findings shown in figures 1, 2 was rejected by the latter publication on the grounds that three reviewers had found that figure 1 'misinterpreted' the data of Baker *et al*.[25] A response from Dr Baker articulated the same claim. Neither the author nor the reviewers contested the accuracy of my reproduction of those data. Furthermore, figure 2, the heart of the argument, was not mentioned by either reviewers or author. The American Physiological Society, the publishers of the *Journal of Applied Physiology*, also receives some token financial support from Gatorade through the Gatorade Predoctoral and Beginning Investigator Awards in the Exercise and Environmental Physiology Section.

This experience raises yet again concerns about the independence of the peer review of submissions in sports medicine and the exercise sciences that may have commercial consequences.[34]

Since science does not benefit from the suppression of carefully argued and scientifically proven opinions, one must again ask the question: Then who does?

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Notes