Br J Sports Med 2004;38:790–793

PostScript

LETTER

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Sodium ingestion and the prevention of hyponatraemia during exercise

The study of Twerenbold *et al*[1] is important for a number of reasons, not all of which may have been emphasised sufficiently by the authors.

Firstly, it confirms that a rate of fluid intake of 1000 ml/h is too high for a group of female runners running at ~ 10 km/h and who would therefore complete a 42 km marathon in about 4.25 hours. As the athletes drank 4 litres and gained 2 kg during the trial, their average rate of weight loss (as opposed to sweat rate) was about 500 ml/h. As not all of the weight lost during exercise is sweat and as much as 1–3 kg of this weight loss may result from fuel and water losses that do not contribute to dehydration,[2 3] the absolute maximum rate at which these athletes should have ingested fluid during exercise was probably even less than 500 ml/h. This is substantially less than the drinking guidelines of the American College of Sports Medicine[4 5] and the Gatorade Sports Science Institute,[6] which have promoted rates of fluid ingestion of up to 1200–1800 ml/h. As there is no evidence that gaining weight during exercise improves performance[7 8] whereas there is good evidence that athletes who either lose no weight or who gain weight during exercise are increasingly likely to (*a*) have an impaired performance,[7] (*b*) develop troubling gastrointestinal symptoms,[7–10] or (*c*) finish the race with serum sodium concentrations below about 128 mmol/l causing hyponatraemic encephalopathy,[11–13] it is not immediately clear why the authors chose such high rates of fluid intake in these athletes. Except, perhaps, if they wished to ''prove'' the value of sodium ingestion during exercise. I note, for example, that the study was funded by a commercial company that, I am informed, markets a sports drink containing sodium chloride.

For it seems highly probable that if athletes overdrink so that they retain fluid and gain weight, then the extent to which their serum sodium concentration falls will be influenced, albeit to a quite limited extent, by the sodium content of the ingested fluids. This indeed was shown by the results of this study. But whether that finding has relevance to the sodium requirements of athletes who are specifically advised not to overdrink during exercise to ensure that they do not develop hyponatraemic encephalopathy[13] is an entirely different question, which cannot be answered with the study design chosen by these authors.

For example, the presence of a control group who drank according to the dictates of thirst (''ad libitum'') and not according to the guidelines of influential sports medical[4 5] and commercial[6] organisations, so that they may be less prone to overdrink and so to gain weight during exercise, would have established that athletes who lose more than 1–3 kg during exercise do not develop symptomatic hyponatraemic encephalopathy[11–16] even though they are both dehydrated and sodium deficient. Rather, they are more likely to finish such races with raised serum sodium concentrations.[12–16]

I would rather argue that a fundamental feature of all prospective trials that aim to evaluate a novel intervention such as the role of sodium ingestion in the prevention of hyponatraemia during exercise should be to compare the new intervention with the currently established ''best practice''.

As the currently established best practice is not to ingest fluid at such high rates that weight is gained during exercise, because this practice can produce a fatal outcome,[17] so this study design should, in retrospect, not have been sanctioned. Rather, the control group in the study should have ingested fluid according to guidelines based on the strongest body of current information. It is, for obvious reasons, my biased opinion that the guidelines that come closest to a defendable evidence base are those that have been recently accepted by the United States Track and Field and the International Marathon Medical Directors Association.[16]

Fortunately the data of Twerenbold *et al*[1] do allow some calculations to estimate the likely value of the extra sodium that was ingested by two of their groups. Thus, the athletes in their study had a mean weight of 58 kg. According to the formula of Montain *et al*,[18] their predicted extracellular fluid (ECF) volume would be about 14.5 litres (25% of body weight). As the starting serum (and ECF) sodium concentration ($[Na^+]$) in the three groups of runners was ~137 mmol/l (table 3 of their article[1]), the average total ECF Na^+ content of the three experimental groups was 1989–1993 mmol at the start of the race. As weights increased by 1.8–2.1 kg in the three groups during exercise (table 3 of their article[1]), the increases in ECF volume would have been 450–525 ml in the respective groups, assuming that the ECF increased in proportion to the increase in total body water (TBW). Multiplying this new ECF volume by the serum $[Na^+]$ after the race gives the new total ECF Na^+ content after the race. As shown in table 1, the total ECF Na^+ content increased by 34 mmol in the group that ingested the high salt drink (H) during the race, but fell by 23 mmol in the group drinking water (W). As all groups ran for about four hours, according to these calculations and based on these assumptions, the hourly rates of Na^+ loss would have varied from 6 to 21 mmol/h, giving a sweat $[Na^+]$ of 12–42 mmol/l in the W and H groups respectively (as their total sweat losses were ~2 litres in each group).

The clear paradox identified by the calculations in table 1 is that (*a*) the total Na^+ loss apparently increases with increased Na^+ intake and (*b*) the estimated Na^+ loss in the group who ingested only water during the race (W) is less than one third of that in the group who ingested the most Na^+ (H) during the race.

As these calculations are based on two real measurements (body weight changes and changes in plasma $[Na^+]$), this apparently ludicrous conclusion can only be explained if (*a*) Na^+ ingestion during exercise increases whole body Na^+ losses in sweat and urine or (*b*) the estimated ECF volume in the W group after exercise is less than the value calculated. That is, specifically in the W group, the ECF volume contracted despite an increase in TBW of 1.9 litres. Indeed, this response is to be expected. There is consistent evidence that the response of the ECF and the intracellular fluid (ICF) volumes to fluid ingestion during prolonged exercise are influenced by the Na^+ content of the ingested fluid[19–21] so that the plasma volume is likely to fall the most if no fluid is ingested,[19] to fall less if either water[19] or a dilute Na^+ drink is ingested,[19–21] or to expand if a concentrated (50–100 mmol/l) Na^+ drink is ingested at the same rate that body weight is lost during exercise.[20] In the latter case, any reduction in the TBW appears to come from a reduction in the ICF.[20]

For example, if each group did indeed lose 84 mmol Na^+ as did group H (table 1), a value that seems eminently reasonable as it equates to a quite reasonable sweat $[Na^+]$ of ~40 mmol/l,[22] then the true ECF volume in the W group after the race would have been 14.5 litres—that is, it is unchanged from the starting value. This value (expressed in litres) is calculated as: (pre-race ECF Na^+ content − 84) in mmol divided by post-race serum $[Na^+]$ in mmol/l.

Indeed, if subjects in the W group did lose 84 mmol Na^+ during the race but also had a post-race ECF volume expanded to 14.95 litres, then their post-race $[Na^+]$ would have been even lower (128 mmol/l) than that actually measured after the race (132 mmol/l; table 1). It is probable that, at that low serum $[Na^+]$, they would have exhibited the early symptoms of hyponatraemic encephalopathy.[11] That they did not have such low serum $[Na^+]$ indicates the importance of small changes in ECF volume (in this case only 450 ml or ~3% of the total ECF volume) in determining the extent to which the serum $[Na^+]$ changes during prolonged exercise in which subjects both sweat and ingest fluid to excess.[11]

Unfortunately, the vital importance of these small changes in ECF volume in determining whether hyponatraemic encephalopathy will develop in those who overdrink during exercise[11] continues fastidiously to be ignored by those[1 4–6 18 23–25] who argue incorrectly that it is the Na^+ deficit that determines the extent to which the serum $[Na^+]$ falls in those who develop hyponatraemia during exercise. This calculation elegantly shows why small changes in ECF volume determine whether or not hyponatraemic

Table 1 Sodium balance calculations for three groups of runners running at ~10 km/h for four hours while ingesting solutions with different [Na^+]

	Pre-race [Na^+] (mmol/l) (A)	Pre-race ECF volume (litres) (B)	Pre-race ECF Na^+ content (mmol) (A×B)	Post-race [Na^+] (mmol/l) (C)	Post-race ECF volume (litres) (D)	Post-race ECF Na^+ content (mmol) (C×D)	Post-race Na^+ balance (mmol) (E) (C×D)−(A×B)	Amount of Na^+ ingested (mmol) (F)	Apparent amount (mmol)/rate of Na^+ loss during exercise (mmol/h) ((F−E)/4)
H	137.3	14.50*	1991	134.8	15.02	2025	+34	118†	84/21
L	137.2	14.50	1989	132.8	14.95	1985	−4	71	76/19
W	137.5	14.50	1993	131.8	14.95	1970	−23	0	24/6

H, High sodium intake; L, low sodium intake; W, water during exercise.
*Based on 25% of mean body weight of 57.7 kg for the total group of runners. Weights for different groups were not reported.
†From table 2 of Twerenbold *et al*[1]: to convert mg sodium (table 2) into mmol sodium, divide by the molecular weight of sodium (22.99).

encephalopathy will develop in those who overdrink, regardless of whether or not they also incur a Na^+ deficit either during exercise[3 11–13 15 26 27] or at rest.[28 29] A recent paper[30] confirms these predictions by showing that mathematical modelling supports the argument that changes in TBW exert a much greater effect on serum [Na^+] than does whole body Na^+ content in those who overdrink and hence gain weight during exercise.

Perhaps the point of these calculations is to show that it is not possible to calculate the state of Na^+ balance in athletes during exercise and so to determine whether or not athletes have developed a Na^+ "deficit",[1 6 23–25] simply by measuring serum [Na^+]. This is because the ECF volume will not be the same before, during, and after exercise and will change depending on the nature of the fluid ingested and the extent of any fluid deficit or excess that develops during exercise.[19–21]

But more importantly, these calculations clearly show why the regulation of the TBW and the ECF volume will have a much greater influence on serum [Na^+] than will either the expected Na^+ losses in sweat or the amount of Na^+ ingested from sodium-containing sports drinks.[11 30]

For example a 1 litre (7%) reduction in the ECF volume would "release" 140 mmol Na^+ into the contracted ECF volume. This means that it is possible to lose 140 mmol Na^+ in sweat and urine without any change in serum [Na^+] provided that the ECF volume were to contract by only 7%. If sweat [Na^+] is about 40 mmol/l, as appears to have been the case in this study of Twerenbold *et al*[1] (table 1), then this 140 mmol is the equivalent of the Na^+ content of about 3.5 litres of sweat.

As athletes in this study sweated at a maximum rate of only 500 ml/h when running at 10 km/h, this means that simply by reducing their ECF volume by 1 litre, those athletes could have maintained their pre-race serum [Na^+] while running for seven hours and drinking just sufficient water to allow for a 1 litre reduction in ECF volume and without requiring any Na^+ replacement whatsoever. This simple calculation explains why those endurances athletes who, before about 1969, were advised either not to drink at all, or only sparingly during exercise,[31] always finished their races with raised serum [Na^+][32–39] despite having incurred what might have been quite sizeable Na^+ deficits.

In contrast, athletes in this study who believed the incorrect advice that ingesting Na^+ and fluid at high rates is essential to maintain a normal serum [Na^+] during exercise,[4–6 18 23–25] so they overdrank sufficiently to increase their ECF volume by 1 litre, would need to ingest and retain at least an additional 140 mmol Na^+ in addition to the ~80 mmol lost in sweat (table 1). This is equivalent to the Na^+ content of 12.4 litres of the low and 7.5 litres of the high sodium drinks respectively in this trial. To maintain fluid balance in this four hour trial when drinking at those high rates and sweating at about 500 ml/h, they would then need to urinate at rates of 1375–2600 ml/h. Both of these rates exceed the maximum at which human kidneys are able to produce urine at rest,[28 29] let alone during and after prolonged exercise.[11 13] Drinking at such rates would therefore only lead to progressive fluid accumulation and ultimately death from hyponatraemic encephalopathy.[17]

In summary, these calculations explain (*a*) why contraction of the ECF in athletes who lose body weight during exercise will maintain the serum [Na^+] even in the face of quite large and unreplaced Na^+ loss in sweat, and (*b*) why the ingestion of sodium-containing sports drinks in the vain hope of matching the rates of Na^+ loss in sweat can only lead to fluid retention and progressive hyponatraemia, as elegantly shown by this study.[1] Indeed if this inappropriate behaviour is approached with sufficient vigour, ultimately the result will be death from hyponatraemic encephalopathy,[17] which, as these calculations and this study again show, cannot occur without the presence of distinct fluid overload.[11]

Finally, it is important to note that, even though Na^+ ingestion marginally increased serum [Na^+] in the group that ingested the most concentrated Na^+ drink, this practice was without benefit as running performances were unaltered by Na^+ ingestion, and the incidence of symptoms was no different between the groups as no athletes reportedly developed symptoms. However, the symptoms of mild hyponatraemic encephalopathy are mild and may not have been sought with sufficient diligence. For example, all subjects, myself included, in our study in which mild hyponatraemia was induced by fluid overload at rest,[28] developed quite disabling symptoms at serum [Na^+] of ~136 mmol/l or lower. Indeed it would have been most interesting to determine whether the presence of subtle mental symptoms was different in the three groups in this study, as all had similar degrees of fluid overload despite different serum [Na^+]. If the symptoms in this condition are due purely to fluid overload, then the incidence of symptoms should have been the same in all groups despite different serum [Na^+]. Alternatively, if the symptoms are related to the degree of hyponatraemia, then they should have been most obvious in the W group, who finished with the lowest post-race serum [Na^+]. My bias would be to expect that the extent of any symptoms are more likely related to the degree of fluid overload, and hence the increase in the ICF, than to the level to which the serum [Na^+] has been reduced.

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Conflict of interest: none declared

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BOOK REVIEWS

Tennis

Edited by P A F H Renstrom. London: Blackwell Publishing, 2002, £29.95, pp 318, softcover. ISBN 0632050349

It is widely recognised that each sport has its own unique demands and injuries. Therefore the IOC, ITF, ATP, WTA, and Society for Tennis Medicine and Science should be congratulated on producing, in this publication, a comprehensive overview of tennis sports medicine. Together they have assembled an impressive array of experts in this field to write succinct and relevant chapters.

Every aspect of tennis is covered to cater for a broad range of readers, including players themselves. Some areas are covered in a high level of technical detail to please the biomechanists, in particular. However, some of the sports medicine is basic in concept and lacking significant evidence based validity.

Nevertheless, I would highly recommend this book to any health professional who treats a large number of tennis players. Most chapters provide a clever link between common sports medicine problems and their occurrence in tennis, including conditions that are unique to this sport. At times, some authors are somewhat optimistic with their view of recovery time from surgery—for example, three weeks for arthroscopic debridement of the infrapatellar fat pad.

Overall it is well presented with relevant and useful photographs and diagrams to aid the reader, and each chapter gives a list of further recommended reading. Unfortunately the book does not provide an answer to where 14 million tennis balls go, imported each year into Australia, as discussed by the editor recently!

Rating

• Presentation	16/20
• Comprehensiveness	15/20
• Readability	15/20
• Relevance	16/20
• Evidence basis	13/20
• Total	75/100

T Wood

Dying to win

Edited by B Houlihan. Council of Europe Publishing, 2002, £17.95, softcover, pp 247. ISBN 9287146853

Dying to win gives an eye opening account of the extent to which drugs play a major role in sport. Doping is not new and has been used in sport since ancient Olympic times; it is just that drug use in modern times is at such a level of sophistication, it is now an industry in its own right. The book describes the privileged position sport holds in society, having appeal for both the participant and the spectator. This has led to the massive media interest, commercialism, professionalism, and governmental regulation and manipulation. Economic pressure in the industrialised world and governmental propaganda in the former East Germany, and more recently China, paved the way for the increasing pharmaceutical intervention in sport. With the fall of the GDR, the world saw for the first time what it had long suspected, the extent of systematic doping on a State run basis, and the most interesting fact is that the East Germans kept excellent records! Further, the book takes a look at the next big issue surrounding drugs in sport—genetic engineering.

Dying to win does not just describe the evolution of doping. It explains the complex relation between anti-doping policy, implementation of those policies, and the role of governments, the IOC, and international and national sporting organisations. With the ever increasing involvement of the legal profession, a vicious circle occurs: it becomes too costly for sporting organisations to fight court battles, with their reliance on Government funding depending on results and punishments set in accordance with what will stand up in courts. This all leads to the relative inertia of the governing bodies to be pro-active in the anti-drugs campaign. The inception of the World Anti-Doping Agency (WADA) after the 1998 Tour de France drugs fiasco provided a way forward to standardise and implement anti-doping policy across the world by an independent body.

Problems and solutions to anti-doping policy are addressed. The major problem is inadequate definition of doping—to quote Arthur Gold ''The definition lies not in words but in integrity of character.''. It is interesting to note that those behind the athlete, namely coach, administrators, medical profession, and scientists, all seem to lose perspective along with their ethics and ''integrity of character'' when the race for ''gold'' is on. *Dying to win* suggests that these people should be held just as accountable as the athletes themselves. Another unfortunate aspect of anti-doping policy is the difficulty in detecting some abused drugs and the fact that these strategies often lag way behind the ability of the pharmaceutical industry to develop new drugs, often for genuine medical reasons but with the unfortunate ability to enhance performance. Education is proposed as a key aspect to anti-doping policy, and parallels with its success in the use of recreational drugs are made. Governments also play a role in limiting supply, decreasing demand for drugs, and the implementation of independent bodies to carry out drug testing. The success of anti-doping policy is also hard to measure. Fewer positive tests may simply reflect a move to less detectable methods rather than a decrease in use, and success may be better measured in terms of fewer world records.

Dying to win gives an accurate account of the problem of doping in sport and the difficulties and complexities in finding solutions to the problems. It makes interesting and provocative reading for all those involved in sport, from the athlete and coach to the sport administrator, the medical profession, and governments.

Rating

- Presentation 17/20
- Comprehensiveness 19/20
- Readability 15/20
- Relevance 18/20
- Evidence basis 19/20
- Total 88/100

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CALENDAR OF EVENTS

UK Radiological Congress 2005 (UKRC 2005)

6−8 June 2005, Manchester, UK

The UK Radiological Congress (UKRC) meeting will encompass the medical, scientific, educational, and management issues that are of interest and relevance to all those involved in the diverse fields of radiological sciences and oncology.

The UKRC provides a forum in which to bring together clinicians, scientists, radiographers, technicians, and other professionals to present and discuss the latest developments and challenges in diagnostic imaging, radiotherapy, and allied radiological sciences.

Key subjects to be covered include: diagnostic radiology; ultrasound; nuclear medicine; interventional radiology; veterinary radiology; emerging technologies; image analysis; computer applications; PACS; radiobiology; radiological physics; management & audit; computed tomography; magnetic resonance; equipment development.

Expected attendance (conference and exhibition): 4000

Further details: UKRC 2005 Organisers, PO Box 2895, London W1A 5RS, UK; Website: www.ukrc.org.uk; Fax: +44 (0)20 7307 1414; Conference tel: +44 (0)20 7307 1410, Email: conference@ukrc.org.uk; Exhibition tel: +44 (0)20 7307 1420, Email: exhibition@ukrc.org.uk

1st World Congress on Sports Injury Prevention

23−25 June 2005, Oslo, Norway

This congress will provide the world's leading sports medicine experts with an opportunity to present their work to an international audience made up of physicians, therapists, scientists, and coaches. The congress will present scientific information on sports injury epidemiology, risk factors, injury mechanisms and injury prevention methods with a multidisciplinary perspective. Panel discussions will conclude symposia in key areas providing recommendations to address the prevention issue in relation to particular injuries and sports.

Further details: Oslo Sports Trauma Research Centre and Department of Sports Medicine, University of Sport and Physical Education, Sognsveien 220, 0806 Oslo, Norway. Email: 2005congress@nih.no; website: www.ostrc.no

Osteosynthese International 2005

15−17 September 2005, Curiohaus, Hamburg

Congress-Chairman: Johannes M. Rueger, M.D., Professor and Chair

Topics:

- Innovations in intramedullary osteosynthesis
- New frontiers in osteoporosis and fracture treatment
- Current trauma research
- Special topic: Recent development in pelvic and acetabular fractures

Abstract submission deadline: 31 March 2005

Further details: INTERCONGRESS GmbH, Martin Berndt, Düsseldorfer Str. 101, 40545 Düsseldorf-Germany. Tel: +49 211 585897-80; fax: +49 211 585897-99; email: martin.berndt@intercongress.de; website: www.osteoint2005.de

4th European Sports Medicine Congress

13−15 October 2005, Lemesos, Cyprus

Further details: Email: pyrgos.com@cytanet.com.cy

BASEM Conference 2005

10−12 November 2005, Edinburgh, Scotland

Further details: Email: basemoffice@compuserve.com

BASEM Conference 2006

5−7 October 2006, Oxford, UK

Further details: Email: basemoffice@compuserve.com

CORRECTIONS

doi: 10.1136/bjsm.2004.003921corr1

Cicharro J L, Hoyos J, Gómez-Gallego F, *et al*. Mutations in the hereditary haemochromatosis gene HFE in professional endurance athletes (*Br J Sports Med* 2004;**38**:418–21). The affiliation of P Celaya was published incorrectly. The correct affiliation is Professional Cycling Team US Postal-Berry Floor. We apologise for this error.

doi: 10.1136/bjsm.2004.000044corr1

Dadebo B, White J, George K P. A survey of flexibility training protocols and hamstring strains in professional football clubs in England (*Br J Sports Med* 2004;**38**:388–94). The multiple regression equation within the Abstract section of this paper was published incorrectly. The correct equation is:

$HSR = 37.79-(0.33SHT+10.05SSP+2.24STE) \pm 2.34$.

We apologise for this error.

doi: 10.1136/bjsm.2004.010876corr1

Sran M M. To treat or not to treat: new evidence for the effectiveness of manual therapy (*Br J Sports Med* 2004;**38**:521–5).

The volume number for reference 23 (Sran *et al*) was incorrectly published as 24; the correct volume number is 29.

In Table 2 the results for Giles and Muller should read: Greater short term benefit for back pain with manipulation, but not for neck pain. Acupuncture more effective for neck pain.

In the section "Definitions and search strategy" the first line of paragraph 2 should read: I searched Medline, Cinahl, and Embase databases for randomised clinical trials comparing manual therapy, including spinal joint mobilisation (with or without manipulation) or manipulation only with other conservative treatments for back or neck pain.

We apologise for these errors.