

This article was downloaded by:[Universidad De Extremadura]
[Universidad De Extremadura]

On: 7 May 2007

Access Details: [subscription number 776098510]

Publisher: Routledge

Informa Ltd Registered in England and Wales Registered Number: 1072954

Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



Journal of Sports Sciences

Publication details, including instructions for authors and subscription information:

<http://www.informaworld.com/smpp/title-content=t713721847>

Drinking guidelines for exercise: What evidence is there that athletes should drink "as much as tolerable", "to replace the weight lost during exercise" or "ad libitum"?

To cite this Article: , 'Drinking guidelines for exercise: What evidence is there that athletes should drink "as much as tolerable", "to replace the weight lost during exercise" or "ad libitum"?', Journal of Sports Sciences, 25:7, 781 - 796

To link to this article: DOI: 10.1080/02640410600875036

URL: <http://dx.doi.org/10.1080/02640410600875036>

PLEASE SCROLL DOWN FOR ARTICLE

Full terms and conditions of use: <http://www.informaworld.com/terms-and-conditions-of-access.pdf>

This article maybe used for research, teaching and private study purposes. Any substantial or systematic reproduction, re-distribution, re-selling, loan or sub-licensing, systematic supply or distribution in any form to anyone is expressly forbidden.

The publisher does not give any warranty express or implied or make any representation that the contents will be complete or accurate or up to date. The accuracy of any instructions, formulae and drug doses should be independently verified with primary sources. The publisher shall not be liable for any loss, actions, claims, proceedings, demand or costs or damages whatsoever or howsoever caused arising directly or indirectly in connection with or arising out of the use of this material.

© Taylor and Francis 2007

Drinking guidelines for exercise: What evidence is there that athletes should drink “as much as tolerable”, “to replace the weight lost during exercise” or “*ad libitum*”?

T. D. NOAKES

UCT/MRC Research Unit for Exercise Science and Sports Medicine, University of Cape Town, Cape Town, South Africa

(Accepted 16 June 2006)

Abstract

The most recent (1996) drinking guidelines of the American College of Sports Medicine (ACSM) propose that athletes should drink “as much as tolerable” during exercise. Since some individuals can tolerate rates of free water ingestion that exceed their rates of free water loss during exercise, this advice has caused some to overdrink leading to water retention, weight gain and, in a few, death from exercise-associated hyponatraemic encephalopathy. The new drinking guidelines of the International Olympic Committee (IOC), recently re-published in this Journal, continue to argue that athletes must drink enough to replace all their weight lost during exercise and to ingest sodium chloride since sodium is “the electrolyte most critical to performance and health”. In this rebuttal to that Consensus Document, I argue that these new guidelines, like their predecessors, lack an adequate, scientifically proven evidence base. Nor have they been properly evaluated in appropriately controlled, randomized, prospective clinical trials. In particular, these new guidelines provide erroneous recommendations on five topics.

If novel universal guidelines for fluid ingestion during exercise are to be promulgated by important international bodies including the IOC, they should first be properly evaluated in appropriately controlled, randomized, prospective clinical trials conducted under environmental and other conditions that match those found in “out-of-doors” exercise. This, and the potential influence of commercial interests on scientific independence and objectivity, are the two most important lessons to be learned from the premature adoption of those 1996 ACSM drinking guidelines that are not evidence-based. These concerns need to be addressed before the novel IOC guidelines are accepted uncritically. Otherwise the predictable consequences of the premature adoption of the 1996 ACSM guidelines will be repeated.

Keywords: *Fluids, exercise-associated hyponatraemia, dehydration, exercise performance, hyperthermia, heat illness*

Introduction

The International Olympic Committee (IOC) has published a Consensus on Sports Nutrition (Maughan, Burke, & Coyle, 2004), the result of a three-day meeting of 30 scientists in June 2003. Included in that consensus is a chapter entitled “Fluid and fuel during exercise”. That chapter was re-published in this Journal (Coyle, 2004). For several reasons, that Consensus Statement invites comment.

Before the adoption of the IOC Consensus Document, there were two opposing fluid guidelines in use internationally; those of the American College of Sports Medicine (Armstrong *et al.*, 1996; Convertino *et al.*, 1996), which differ only marginally from the new IOC guidelines, and those of the International Marathon Medical Directors Association (IMMDA) (Noakes, 2003b), which have recently been adopted

by United States of America Track and Field (USATF). The latter, which include research findings that are infrequently acknowledged by those who advise the ACSM, the National Athletic Trainers Association (Casa, Armstrong, & Hillman, 2000) or the US Military (Montain, Latzka, & Sawka, 1999) and now apparently the IOC, differ in a number of crucial ways from those presented in the original ACSM guidelines as now slightly modified in the IOC Consensus Document. It is my argument that the IOC Consensus Document advances the original ACSM guidelines hardly at all, since it again fails to include, as is the case with the 1996 ACSM guidelines (Noakes, 2003b, 2003c, 2004b), an adequate evidence base of rigorous scientific findings that support this new consensus. Nor, before their adoption, were they evaluated in properly controlled, randomized, prospective clinical trials.

The draftees of the IOC Consensus Document argue that they wish to be systematic and to provide a “reality check” by reviewing the “vast literature that has grown exponentially in the past two decades” with a “heavy reliance upon controlled laboratory studies as well as careful study of athletes in the field during training and competition”. Yet in my view, only arguments that will continue to support the popular dogma of the ACSM guidelines are considered and that body of evidence (Noakes, 1995, 2000, 2001, 2002, 2003a, 2003b, 2003c, 2004a, 2004b, 2004c; Noakes *et al.*, 1988; Noakes & Glace, 2004) which might support alternate conclusions is ignored. Fortunately, there are scientists other than myself, including some in North America, who are also now beginning to question the validity of those guidelines (Hosey & Glazer, 2004; Hsieh, 2004).

Instead of exhaustively interrogating the appropriateness of the evidence base that the Consensus Document uses to support these new conclusions, since that argument has been advanced elsewhere (Noakes, 2000, 2003b, 2004b), it is perhaps more appropriate again to question what scientific evidence there is to support the guidelines in those four specific areas where the respective guidelines of the ACSM and the IOC, and IMMIDA/USATF, would appear to be most at odds.

Finally, I address the issue of potential conflicts of interest between scientists, scientific bodies, and the sports drink industry in the conduct of the science that purportedly “supports” these guidelines, and in their subsequent promulgation and the fact that the main target for the sale of sports drinks is recreational athletes who usually exercise for too short a duration to require the consumption of a sports drink.

Issue 1

Both the ACSM guidelines and the IOC Consensus Document are based on the belief that only the full replacement of fluid during exercise can optimize performance and minimize health risks. The important questions that need to be addressed by both the research community and those who provide advice on fluid replacement during exercise are as follows:

(i) What evidence is there from *appropriately controlled, prospective clinical trials* that athletes competing in out-of-doors competition benefit by replacing *all* the weight they lose during exercise? Since the ACSM/IOC guidelines are most usually applied to out-of-doors exercise and competition, it is important that this question is addressed. Definitive outcome measures would presumably include proof that this specific drinking regimen improves exercise performance and reduces the incidence of heat stroke – the

only important heat illness (Noakes, 2003a) – more than does any other possible drinking behaviour during exercise. Discussion of the effects of fluid ingestion on “heat illnesses” other than heatstroke is unhelpful since these conditions are usually defined in terms of a variety of symptoms (Armstrong, 2000), none of which can be diagnostic of a specific “heat illness”. Indeed, the diagnosis of “illness” on the basis of non-specific symptoms violates a core teaching in modern medicine, which is that any illness must have one or more distinctive features that separate it from all other diseases. But the “heat illnesses” other than heatstroke do not have defining characteristics that allow each to be identified as a separate medical condition with a unique causation and presentation, definitive diagnostic feature(s), and a specific treatment. Rather, the current definitions of the different heat illnesses include so many overlapping signs and symptoms (Armstrong, 2000) that such distinction is impossible.

Furthermore, comparison of the effects of drinking to replace all the weight lost during exercise to these in which no fluid is ingested during exercise would obviously not be appropriate since no-one claims that not drinking is the most appropriate behaviour for athletes. Rather, the control condition needs to be drinking “*ad libitum*”, as this is the naturally chosen human behaviour.

Experimental variables that would need to be considered include the exercise intensity and duration, and the environmental conditions in which the exercise is undertaken, as well as the nature of the exercise, in particular whether it involved weight-bearing, typically running, or non-weight-bearing exercise, typically cycling (on the flat). This is important since some weight loss during exercise is likely to have an ergogenic effect, particularly during weight-bearing exercise, such as running or cycling uphill, since it reduces the energy cost of movement. The magnitude of this potential effect needs to be determined.

Answers to these questions would need also to include findings from published studies showing that individuals who attempt to replace all the weight they lose during prolonged exercise might develop adverse consequences, such as impaired exercise performance, the development of specific, typically gastrointestinal, symptoms and an increased probability for a reduced serum sodium concentration. Besides the extensive literature on the dangers of gaining weight during exercise (Noakes, 2002, 2003b, 2003c; Noakes *et al.*, 2005), several recent studies (Glace, Murphy, & McHugh, 2002; Robinson *et al.*, 1995; Stuempfle *et al.*, 2002; Stuempfle, Lehmann, Case, Hughes, & Evans, 2003; Twerenbold *et al.*, 2003) invite critical attention since they suggest that those who do not lose

some weight during exercise may also be more likely to develop uncomfortable symptoms.

(ii) If it is crucial to replace all the fluid lost during exercise, why is it that the best athletes in the world seem religiously to avoid this advice? I refer to the apparently low rates of fluid intake by the world's best marathoners and cyclists during exercise of 1–3 h duration and which seem to come nowhere remotely close to the $1200 \text{ ml} \cdot \text{h}^{-1}$ guideline of the ACSM as endorsed by the Gatorade Sports Science Institute (Murray, 1996). It was of interest, for example, to watch the 2004 Athens Women's Olympic Marathon during which both the pre-race favourite, Englishwoman Paula Radcliffe, and the winner, Japanese runner Mizuki Noguchi, both appeared to drink modest amounts. It is difficult to believe that Ms Noguchi, competing in one of the hottest marathons in recent history (start temperature = 35°C), was not significantly "dehydrated" at the end of the race because she had spent a total of only about 30 s (equivalent to 0.3% of her total running time of 8780 s) drinking during the race (M. Boddington *et al.*, unpublished data). Yet she won the race. How is this possible if a failure to drink 1.2 litres per hour, especially during exercise in the heat, is so detrimental for performance and for health, as laid out in the ACSM guidelines? Recent evidence for low rates of fluid intake before, during, and after exercise in elite runners comes from studies of Kenyan athletes training in Eldoret, Kenya (B. Fudge *et al.*, unpublished data).

Many similar historical examples exist (Buskirk & Beetham, 1960; Muir, Percy-Robb, Davidson, Walsh, & Passmore, 1970; Noakes, 1995, 2003a; Pugh, Corbett, & Johnson, 1967; Wyndham & Strydom, 1969), especially in athletes competing before 1969, during the era when athletes were specifically advised not to drink during any form of exercise, regardless of its duration or intensity or the environmental conditions in which the exercise was undertaken (Noakes, 1993, 2003a). No-one has yet explained how, if "dehydration" is so dangerous, these athletes were ever able to survive this practice, let alone produce remarkable athletic performances without any apparent risk to their health in many thousands of competitions before the adoption of the first ACSM guidelines in 1975 (American College of Sports Medicine, 1975).

Indeed, in the paper referenced by the IOC Consensus Document, Chevront, Carter, and Sawka (2003) reported a linear relationship between average running speed and dehydration in 42-km marathon runners so that those athletes who are the most dehydrated ran the fastest. Yet the authors caution that "this should not be wrongly interpreted as support for an ergogenic effect of dehydration" (p. 204). But the authors provide no explanation of

why it is wrong to conclude that dehydration might be ergogenic in weight-bearing activities such as long-distance running. Clearly, their interpretation is based on what is their particular model of reality (Noakes & Speedy, 2006) and the predictions it makes of what should happen. They merely acknowledge that "the question of how competitive runners perform so well when dehydrated still remains unclear". Perhaps it is because humans evolved to run long distances in the heat without drinking and that this ability provided a profound advantage for the subsequent evolution of humans (Heinrich, 2001; Noakes & Speedy, 2006).

(iii) What evidence is there to show that "replacing all the water lost through sweating", the current ACSM and IOC guideline, improves performance more and reduces the risk of heat stroke more effectively than does drinking *ad libitum* – that is, according to the dictates of thirst – the current IMMDA/USATF guideline. I am personally unaware of any such evidence. Rather, separate studies (Chevront & Haymes, 2001; Daries, Noakes, & Dennis, 2000; McConell, Burge, Skinner, & Hargreaves, 1997) suggest that *ad libitum* drinking is at least as effective as is the current ACSM guideline. Drinking *ad libitum* substantially reduces (but does not absolutely negate) the risk that athletes will voluntarily overdrink sufficiently to induce exercise-associated hyponatraemic encephalopathy, thereby risking death, or that they will develop uncomfortable symptoms during exercise.

Indeed, it is most interesting that one of the original industry-sponsored foundation studies (Costill, Kammer, & Fisher, 1970) on which the current ACSM guidelines are based reported that athletes attempting to drink a litre an hour (the current ACSM/IOC guidelines) for as little as 2 h developed disabling gastrointestinal symptoms: "All of the runners experienced extreme sensations of fullness during the final five or six feedings [i.e. after 75 minutes running]. At the end of 100 minutes of running and feeding, it became apparent that further attempts to ingest fluids would have been intolerable". These symptoms were caused by the large residual of unabsorbed fluid present in the stomachs of these athletes at the end of exercise and which would have been even larger when they terminated drinking 20 min earlier. It is surprising that this finding seems to have been "overlooked" in the formulation of the current ACSM guidelines and now in these newly modified guidelines of the IOC.

The IOC Consensus Document correctly places the appropriate emphasis on the thorough recent review of Chevront and colleagues (Chevront *et al.*, 2003) of the important studies that establish the effects of dehydration on exercise performance. In Table I of that review, the authors list 13 studies in

Table I. Effects on exercise performance of drinking fluid ("some", "ad libitum" or "as much as tolerable") versus not drinking during exercise.

Study	Sample size (n)	Exercise	Environment,		Drink conditions	Dehydration (% body mass)	Performance results	My conclusions
			°C (relative humidity, %)	humidity, %)				
Pitts <i>et al.</i> (1944)	6	Walk 3.5 mph, 2.5% grade for 2 h in the laboratory (10 min rest per hour)	35 (83)		NF FF	No data	NF = 10/16 failed to complete 2 h FF = 22/22 completed 2 h	Fluid ingestion increased probability of task completion
Brown (1947)	70 NF 59 FF	2.5–19 mile desert marches	30–45		NF AL	NF = 8% AL = ~4%	NF = 11/70 (16%) failed to complete hike AL = 1/59 (1.7%) failed to complete hike	10-fold greater probability of completing task when fluid was ingested
Ladell (1995)	4	Bench stepping for 160 min	38 (30) 38 (78)		NF FF	No data	NF = 3/12 completed 160 min FF = 38/41 completed 160 min	Fluid ingestion increased probability of task completion
Strydom <i>et al.</i> (1966)	60	29-km (18-mile) route march	21–32		SF AL	SF = 4.8% AL = 2.9%	SF = 20% greater probability of failing to complete the route march	Fluid ingestion increased probability of task completion
Maughan <i>et al.</i> (1989)	6	Cycle ergometer exercise at 70% $\dot{V}O_{2max}$ to exhaustion in the laboratory	Laboratory		NF SF	NF = 1.8% SF = 2.0%	No differences in time to exhaustion	Fluid ingestion did not increase endurance at a fixed work rate
Barr <i>et al.</i> (1991)	8	Cycle ergometer exercise at 55% $\dot{V}O_{2max}$ for 6 h in the laboratory	30 (50)		NF SF	NF = 6.4% SF = 1.2%	NF = 25% decrease in time to exhaustion	Fluid ingestion increased endurance at a fixed work rate
Walsh <i>et al.</i> (1994)	6	Cycle ergometer exercise at 70% $\dot{V}O_{2max}$ followed by performance ride or run at 90% $\dot{V}O_{2max}$	32 (60)		NF FF	NF = 1.8% FF = 0.2%	NF = 31% decrease in time to exhaustion	Fluid ingestion increased endurance at a fixed work rate
Robinson <i>et al.</i> (1995)	8	Cycle ergometer exercise "at maximum power output" for 60 min	20 (60)		NF FF	NF = 2.3% FF = 0.9%	FF = significant 1.7% decrease in performance ride or run	Full fluid replacement impaired exercise performance
Fallowfield <i>et al.</i> (1996)	8	Treadmill run at 70% $\dot{V}O_{2max}$ to exhaustion	20		NF SF	NF = 2.0% SF = 2.7%	NF = 25% decrease in time to exhaustion	Fluid ingestion increased endurance at a fixed work rate
Mudambo <i>et al.</i> (1997)	18 NF 6 SF	Walk/run/obstacle course lasting 3 h out-of-doors	39 (28)		NF SF	NF = 7% SF = 2.8%	NF = 6/18 participants failed to complete exercise bout	Fluid ingestion increased probability of task completion

(continued)

Table I. (Continued)

Study	Sample size (<i>n</i>)	Exercise	Environment, °C (relative humidity, %)	Drink conditions	Dehydration (% body mass)	Performance results	My conclusions
McConell <i>et al.</i> (1997)	7	Cycle ergometer exercise at 69% $\dot{V}O_{2max}$ for 120 min, then 90% $\dot{V}O_{2max}$ to exhaustion in the laboratory	21	NF SF FF	NF = 3.2% SF = 1.8% FF = 0.1%	SF = 6/6 completed exercise bout NF = 48% decrease in performance ride or run vs. FF No difference in performance ride or run between SF and FF	Fluid ingestion improved exercise performance. No additional benefit of drinking more than "some" fluid
McConell <i>et al.</i> (1998)	8	Cycle ergometer exercise at 80% $\dot{V}O_{2max}$ for 45 min, then 15 min performance ride or run in the laboratory	21 (41)	NF SF FF	NF = 1.9% SF = 1.0% FF = 0.0%	No differences in performance ride or run among trials	Fluid ingestion had no measurable effect on exercise performance in exercise of ~60 min
Bachle <i>et al.</i> (2001)	11	Cycle ergometer exercise "at maximum power output" for 60 min	21 (72)	NF FF	NF = 1.0% FF = +0.7%	No difference in performance ride or run between trials	Fluid ingestion did not increase power output during exercise of a fixed duration
Kay and Marino (2003)	7	60 min self-paced cycle ergometer time-trial	19.8 (63%) 33.2 (63%)	NF FF	NF = 1.3–1.6% FF = +0.1–0.1%	No difference in performance ride or run between trials	Fluid ingestion did not increase time-trial performance
J. P. Dugas <i>et al.</i> (unpublished)	6	80-km self-paced cycle ergometer time trial	30 (50)	NF MR AL SF FF	NF = 4.3% MR = 3.9% Less than AL = 2.9% AL = 2.1% SF = 1.9% FF = 0.5%	Less than AL (including MR) = 2% decrease in performance ride or run No difference in performance between AL, SF, and FF	Fluid ingestion equal to <i>ad libitum</i> or greater improved time-trial performance. Full fluid replacement was no more effective than <i>ad libitum</i> drinking. Mouth rinsing was associated with impaired performance. Hence <i>ad libitum</i> fluid ingestion, not the act of drinking, improves exercise performance

Abbreviations: NF = no fluid; MR = mouth rinsing only; AL = *ad libitum* fluid intake; SF = some fluid intake (> NF, < FF); FF = fluid > sweat losses.

which there is a reasonable measure of exercise performance in participants who completed different exercise bouts with varying states of dehydration. Athletes ingested either nothing or varying amounts of fluid during different exercise bouts thereby developing different states of dehydration during exercise. This study design is the only reasonable one from which it is possible to draw conclusions about the effects of different levels of exercise-induced dehydration as opposed to some other form of dehydration on exercise performance. For example, studies in which dehydration is induced before exercise, either by prior exposure to exercise and heat (Armstrong *et al.*, 1997; Sawka, Young, Francesconi, Muza, & Pandolf, 1985) or the use of diuretics (Armstrong, Costill, & Fink, 1985), cannot exclude the possibility that the dehydration-inducing intervention and not the resulting dehydration was the real cause of the impaired exercise performance. Unfortunately, these studies are frequently quoted as primary evidence that “dehydration” impairs exercise performance (Gisolfi, 1996; Sawka & Montain, 2000).

In ten of the studies reviewed by Chevront *et al.* (2003), exercise performance was impaired in those who drank nothing during exercise and was improved by fluid ingestion during exercise; in three, fluid ingestion had no effect. Notably, studies which failed to show a beneficial effect of fluid ingestion were of relatively short duration and were performed in cool conditions. Thus those data clearly support the conclusion that not drinking *at all* during exercise is likely to impair performance especially during more prolonged exercise in the heat.

However, since there was no control for the act of drinking in those studies, the studies (technically) do not disprove the alternative hypothesis that the act of drinking, rather than that of fluid ingestion, improves performance. However, this hypothesis seems improbable since it is clear that fluid ingestion does produce significant physiological effects that could aid performance and which do not likely result purely from the passage of water through the mouth and throat. Rather, these effects probably require that the fluid is ingested *and* assimilated into the body. Furthermore, that alternative hypothesis has recently been disproved (J. P. Dugas *et al.*, unpublished data).

But the question under debate is not whether fluid ingestion usually improves athletic performance; that is quite clear and does not require further argument (Noakes, 2003b). Rather, the relevant question is how much fluid needs to be ingested to enhance performance during exercise. Thus the important question that has not been addressed by either Chevront *et al.* (2003) or the IOC Consensus Statement, is whether there is an additional performance benefit of drinking “as much as tolerable”, or

“to replace sweat losses”, compared with drinking “some” fluid during exercise.

Here I add some additional relevant studies to those already identified by Chevront *et al.* (2003) to address the question: Is there any published evidence to show that an added performance advantage is gained by drinking more than *ad libitum* during exercise?

Table I includes 15 studies in which fluid ingestion was compared with no fluid ingestion during exercise. In nine of those studies, there was a clear benefit of fluid ingestion and in only one was exercise performance impaired by (full) fluid replacement (Robinson *et al.*, 1995) due to the development of gastrointestinal symptoms resulting from excessive fluid consumption, similar to those reported in the classic study by Costill and colleagues (1970). It is also clear that the benefits of fluid ingestion increase with the duration of the exercise and are least apparent in exercise of short duration.

Thus these data clearly support the conclusion that some drinking during exercise improves exercise performance compared with not drinking anything during exercise. The work of Dugas and colleagues (unpublished data) also confirmed that this effect is not due simply to the effects of fluid acting on receptors in the mouth. For the benefit of fluid ingestion to be achieved, the fluid must be swallowed (and presumably absorbed).

Table II compares the six studies in which the effects on exercise performance of “some” fluid replacement are compared with those of full replacement. In none of these studies was full fluid replacement superior to *ad libitum* drinking. The studies of Daries *et al.* (2000) and Dugas *et al.* (unpublished data) are especially relevant. In the study of Daries *et al.* (2000), in comparison to full fluid replacement, *ad libitum* drinking was associated with superior performance during a 30-min running time trial but small numbers mitigated against a significant finding. Dugas and colleagues (unpublished data) compared the effects on performance during an 80-km (non-weight-bearing) cycling time trial in individuals who followed six different fluid replacement regimes on separate occasions: (1) no fluid; (2) mouth washing without fluid ingestion; (3) replacing 33% of fluid losses; (4) *ad libitum* drinking (replacing ~55% of fluid losses); (5) replacing 66% of fluid losses; (6) replacing 100% of fluid losses. Analysis showed that there was no significant advantage of drinking more than *ad libitum*, but that drinking less than *ad libitum* was associated with a 2% impairment of performance compared with drinking *ad libitum* or more.

Thus these studies confirm the conclusion of the IOC Consensus Statement that not drinking at all during exercise is more likely to impair performance

Table II. Effects on exercise performance of drinking some fluid ("some" or "ad libitum") versus full fluid replacement.

Study	Sample size (<i>n</i>)	Exercise	Environment, °C (relative humidity, %)	Drink conditions	Dehydration (% body mass)	Performance results	My conclusions
Below <i>et al.</i> (1995)	8	Cycle ergometer exercise at 50% $\dot{V}O_{2max}$ for 50 min, then performance ride or run	31 (54)	LF FF	LF = 2.0% FF = 0.5%	LF = 7% decrease in performance ride or run	Less than <i>ad libitum</i> drinking impairs exercise performance
McConell <i>et al.</i> (1997)	7	Cycle ergometer exercise at 69% $\dot{V}O_{2max}$ for 120 min, then 90% $\dot{V}O_{2max}$ to exhaustion in the laboratory	21	NF SF FF	NF = 3.2% SF = 1.8% FF = 0.1%	NF = 48% decrease in performance ride or run vs. FF No difference in performance ride or run between SF and FF	No additional benefit of drinking more than "some" fluid
McConell <i>et al.</i> (1998)	8	Cycle ergometer exercise at 80% $\dot{V}O_{2max}$ for 45 min, then 15 min performance ride or run in the laboratory	21 (41)	NF SF FF	NF = 1.9% SF = 1.0% FF = 0.0%	No differences in performance ride or run among trials	No benefit of any volume of fluid ingestion on performance during exercise of ~60 min
Daries <i>et al.</i> (2000)	8	Treadmill run at 65% $\dot{V}O_{2max}$ then "as far as possible" in 30 min	25 (55)	AL SF FF	AL = 1.95% SF = 1.59% FF = 0.75%	AL = 15.8 km · h ⁻¹ for 30 min SF = 15.6 km · h ⁻¹ for 30 min FF = 15.4 km · h ⁻¹ for 30 min	<i>Ad libitum</i> drinking produced insignificantly faster performance in the 30-min performance ride or run than did either "some" or "full fluid" replacement
Backx <i>et al.</i> (2003)	8	Cycle ergometer time-trial	20 (70)	LF MF AL FF NF MR	LF = 1.25% MF = 1.0% AL = 0.7% FF = 0.5% NF = 4.3% MR = 3.9	No differences in performance ride or run among trials	No benefit of drinking more than "little" fluid during exercise
J. P. Dugas <i>et al.</i> (unpublished)	6	80-km self-paced cycle ergometer time-trial	33 (50)	Less than AL AL SF FF	Less than AL = 2.9% AL = 2.1% SF = 1.9% FF = 0.5%	Less than AL (including MR) = 2% decrease in performance ride or run No difference in performance between AL, SF, and FF	No benefit of drinking more than <i>ad libitum</i> during exercise

Abbreviations: NF = no fluid; MR = mouth rinsing only; LF = little fluid (< AL); AL = *ad libitum* fluid intake; SF = some fluid intake (> NF, < FF); FF = fluid > sweat losses.

than ingesting some fluid. But it extends that conclusion by showing that there is no published evidence to suggest that drinking in excess of *ad libitum* provides an additional advantage.

However, this finding that *ad libitum* or “some” fluid ingestion during exercise appears to be at least as effective as “drinking as much as tolerable” has one important intellectual consequence: It suggests that it is not the level of dehydration that determines the extent to which performance is affected by fluid ingestion or its avoidance during exercise. Rather, it may be that performance will be optimized regardless of the amount of dehydration that develops, provided that sufficient fluid is ingested to prevent the development of thirst during exercise. This hypothesis invites scientific scrutiny.

If true, this would explain why it is possible for elite athletes to perform well while drinking sparingly during exercise, for the fastest marathon runners to be among the most dehydrated – as found both in classic (Buskirk & Beetham, 1960; Muir *et al.*, 1970; Pugh *et al.*, 1967; Wyndham & Strydom, 1969) and modern (Chevront *et al.*, 2003) studies – and for athletes who lose in excess of 10% of body mass to be among the top finishers in 226-km Ironman triathlons (Sharwood, Collins, Goedecke, Wilson, & Noakes, 2002, 2004).

Issue 2

Those who argue for the “drink as much as tolerable” or “to replace all the weight lost” guideline usually equate “dehydration” with “hyperthermia”, as if dehydration cannot occur without hyperthermia or vice versa. Here the questions that require attention are as follows:

(i) What evidence is there to show that dehydration is the most important determinant of rectal temperature as well as the risk of collapse, in out-of-doors, self-paced exercise? As we have argued consistently for the past decade (Noakes, 1995), almost all the evidence for this relationship comes from industry-sponsored research conducted indoors in laboratories that were not designed to provide avenues for an adequate convective cooling (Noakes, 2003b) and in which an unvarying exercise intensity is imposed on the athlete. When adequate convective cooling is provided in these experiments, the small and biologically insignificant difference (approximately 0.2°C) in the rectal temperature response produced as a result of drinking “as much as tolerable” (Montain & Coyle, 1992) compared with *ad libitum* drinking disappears (Noakes, 2004b; Saunders, Dugas, Tucker, Lambert, & Noakes, 2005).

(ii) What evidence is there from *appropriately controlled, randomized, prospective clinical trials* to show

that dehydration increases the risk of heat stroke so that the avoidance of “dehydration” protects against the development of this serious condition? To my knowledge, there are no such clinical trials reported in the scientific or medical literature (Noakes, 1995). In the absence of such clinical trials, it is no longer defensible to argue (1) that dehydration causes heat stroke or (2) that drinking more during exercise will prevent heat stroke or indeed those symptom-defined conditions inappropriately termed the “heat illnesses” (Armstrong, 2000).

It is interesting that these ideas are based on a novel paradigm, popularized simultaneously with the growth of the sports drink industry, and which may be termed the Cardiovascular Model of Thermo-regulation (Noakes & Speedy, 2006). This model proposes that dehydration affects thermal regulation during exercise by impairing cardiovascular function, thereby reducing the skin blood flow and hence the ability to lose heat by sweating (Chevront *et al.*, 2003; Stover, Zachwieja, Stofan, Murray, & Horswill, 2006). This model appears to have arisen from early studies which showed that individuals who did not drink during exercise developed elevated heart rates (Brown, 1947; Brown & Towbin, 1947; Ladell, 1955; Pitts, Johnson, & Consolazio, 1944) and low stroke volumes (Nadel, Fortney, & Wenger, 1980). As a result, “Acute dehydration apparently limits man’s ability to work, largely through impaired cardiovascular function” (Buskirk, Iampietro, & Bass, 1958).

This model is now widely accepted. Thus a recent scientific publication from the Gatorade Sports Science Institute (GSSI) is based on the hypothesis that: “[as a consequence of dehydration] Cardiac output is reduced, the increase in skin blood flow is attenuated, and core temperature rises at an accelerated rate with progressive dehydration and continued exercise. Hypohydration . . . also has negative consequences on cardiovascular function and thermoregulation . . . and [can] put the athletes at higher risk for heat exhaustion or heatstroke, the latter being a potentially catastrophic event” (Stover *et al.*, 2006, p. 330).

In addition during this process, a novel medical condition, “dehydration”, entered the popular lexicon. Formerly understood as a physiological state in which the total body water is reduced, the term has since been corrupted to brand a life-threatening disease with a multitude of signs and symptoms that begin to develop the instant some fluid is lost from the body. This model is particularly attractive to the sports drink industry because it provides a direct (causal) link between their product and cardiovascular function, the control of sweating, and the risk of heat illness during exercise. Thus it allows sports drinks to be promoted on medicinal grounds, in

particular because of an (assumed) capacity to prevent heat illness during exercise.

Yet as noted as early as 1955 (Ladell, 1955), the classic early studies (Adolph, 1947a, 1947b; Eichna, Bean, Ashe, & Nelson, 1945) did not show that individuals who did not drink during exercise risked their lives or that they had lower sweat rates than when they ingested fluid at higher rates. Thus Ladell (1955) concluded that: "Abstinence from water had no effect on the sweat rate, until water deficits of more than 2.5 kg had been incurred" (p. 43). This would equate to a body mass loss of 3.6% in a 70-kg male athlete. Indeed, in the first industry-sponsored classic study, Costill and colleagues (1970) reported that even when they did not drink during 2 h of exercise in the heat, "the runners' skin was sufficiently wetted by sweating to permit maximal evaporation". Even the industry-funded classic paper by Montain and Coyle (1992) reported that sweat rates were the same in individuals who drank either nothing or 2.4 litres of fluid during 2 h of exercise in moderately severe heat. The authors concluded that the higher end-exercise rectal temperature in the no-drink condition was due to a reduced skin blood flow. But this explanation does not make physiological sense. For if sweat rates were not *reduced* in those who were not allowed to drink during exercise, they could presumably have been increased to offset any effect of a reduced skin blood flow on heat balance under those specific experimental conditions. That sweat rates were not increased suggests that the increased core temperature in the no-drink condition is a deliberately chosen bodily ploy.

For example, Schmidt-Nielsen (1964), whose lifelong studies of human and animal physiology led him to conclude that "Man is an excellent temperature regulator" (p. 2), has concluded that the leading endurance mammal on the African continent, the African hunting dog,

achieves considerable savings in use of water by permitting its body temperature to increase (during exercise) . . . thus facilitating heat loss by conduction . . . On previous occasions I have often emphasized that an increased body temperature during exercise cannot be considered a measure of a failing temperature regulation. We now see that in running animals as well, an increase in body temperature can readily be interpreted as a means for reducing the loss of water, rather than as an expression of inadequate heat dissipation. (Schmidt-Nielsen, 1972, pp. 63–64)

He continues:

It has been known for many years that the body temperature of man during exercise is increased,

and that the increase is closely related to the intensity of exercise. When steady state has been attained, the rectal temperature is accurately maintained at the higher level, the exact level being determined by the intensity of the exercise. The usual interpretation is that the increased body temperature speeds up all physiological processes and therefore gives an advantage in performance. I should like to propose that, for an organism that has evolved as a hunter pursuing its prey by running it down in open country, the advantages of increased body temperature to the water economy may be a consideration of no less importance. (Schmidt-Nielsen, 1972, pp. 64–65)

Thus a rather more plausible explanation is that the higher rectal temperature during the no-drink condition is the result of a bodily adaptation to optimize heat loss when the body "knows" that no fluid is to be ingested during exercise (J. P. Dugas *et al.*, unpublished data).

But more to the point, the sweating response is regulated by neural mechanisms that are not dependant on the cardiovascular response to exercise (Shibasaki, Kondo, & Crandall, 2003), a point apparently ignored by the modern generation of thermal physiologists. Thus the principal determinant of the sweating response to exercise is the neural and not the cardiovascular reaction.

Issue 3

Proponents of the "drink to replace all the weight lost during exercise" dogma usually argue that dehydration impairs exercise performance as a linear function of the level of dehydration and that this effect is further influenced by the environmental temperature in which the exercise is undertaken (see, for example, Figure 1 in Coyle, 2004). This conclusion is not however supported by the findings reported in Table II, which show that performance is not optimized by preventing "dehydration" but rather by drinking *ad libitum* during exercise, regardless of the level of dehydration that develops. Thus the questions that require answers are as follows:

(i) What evidence is there from *appropriately controlled, randomized, clinical trials* to show that exercise performance (a) deteriorates as a linear function of the level of dehydration and (b) that this relationship becomes steeper as the environmental conditions become increasingly more severe?

Another reason for this question is that Figure 1 in the IOC Consensus Document (Coyle, 2004) lacks any units for the estimated performance decrements with increasing "dehydration". Therefore, the question requiring an answer is: What are the units and

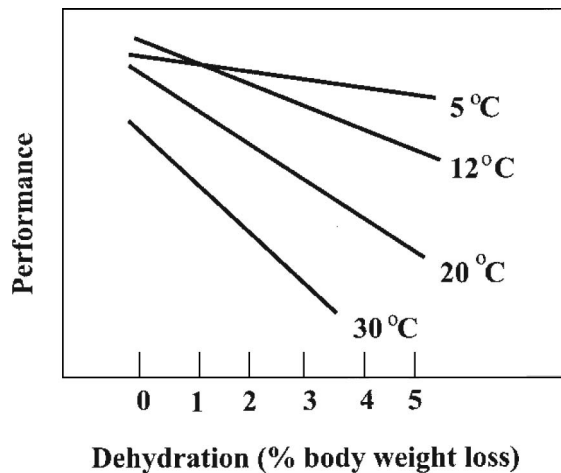


Figure 1. This figure of the theoretical relationship between performance impairment and dehydration (% body weight loss) during exercise at different environmental temperatures previously published in this journal (Coyle, 2004), lacks any units for the performance impairment.

Source: (Coyle E. F. (2004). Fluid and fuel intake during exercise. *Journal of Sports Sciences*, 22, 39–55. Taylor and Francis Ltd., <http://www.tandf.co.uk/journals>, reprinted with permission of the publisher.).

how were they derived? Or is the figure a “teaching concept” for which data are still in the process of being collected? It is important that concept slides should be presented as such and not as definitive findings that carry the weight of scientific proof. Such figures soon become the basis for “foundation myths” and unchallengeable dogmas (Noakes, 2004b).

The second reason for this question is the consistent finding that athletes who win (Buskirk & Beetham, 1960; Muir *et al.*, 1970; Pugh *et al.*, 1967; Wyndham & Strydom, 1969) or finish near the top of competitive endurance events are frequently quite markedly dehydrated, by up to 8–10% in the case of some Ironman triathletes (Sharwood *et al.*, 2004). The finding of Chevront and colleagues (2003) that the fastest marathon runners are also those who lose the most body mass during exercise was discussed earlier. If it is true that dehydration causes a linear reduction in exercise performance, then all these athletes could have finished those races hours faster if they had simply drunk “to prevent any weight loss during exercise”. Yet such performances would be impossible. Thus common sense indicates that the basis for the prediction of the IOC Consensus Document must be erroneous.

The explanation could be that the level of dehydration is not the determinant of the impaired exercise performance. Rather, it is linked in some way to the increased thirst sensations caused by inadequate fluid replacement. Or perhaps to an anticipatory response when the body “knows” that it will not be

receiving adequate fluid replacement during exercise (J. P. Dugas *et al.*, unpublished data).

Issue 4

The ACSM guidelines promote sodium ingestion during exercise despite a large body of contrary evidence (Hew, Sharwood, Speedy, & Noakes, 2006; Noakes, 2004a; Sanders, Noakes, & Dennis, 1999, 2001; Speedy *et al.*, 2002), including the conclusion of the Institute of Medicine for the National Academies (2004) that “the current intake of sodium for most individuals in the United States and Canada greatly exceeds both the Adequate Index (AI) (1.5 g (65 mmol)/day) and the Tolerable Upper Intake Level (UL) (2.3 g (100 mmol)/day)” (p. 6–112) so that interventions to reduce sodium intake are required, particularly to reduce the risk that hypertension will develop.

The Institute of Medicine further states that the adequate index for sodium of $1.5 \text{ g} \cdot \text{day}^{-1}$ “are for individuals who are moderately active in temperate climates”. They do however caution that “high levels of activity or exposure to high temperature or humidity results in increased needs for water to replace sweat losses. In these settings, additional sodium intake above the AI may be required as well, *but experimental data are lacking*, especially at dietary intakes of 1.5 g (65 mmol) per day. Still for most physically active individuals, the AI (1.5 g (65 mmol) per day) should be adequate” (p. 6–42; emphasis added).

Thus the conclusion of the Institute of Medicine is that there is no scientific evidence to show that the adequate index is not suitable also for those who are physically active. But to avoid a dogma that may be incorrect, they add the proviso that “the AI might not apply to highly active individuals” (p. 6–42).

This is compatible with the conclusion of the 2005 International Consensus Conference on Exercise-Associated Hyponatraemia (Hew-Butler *et al.*, 2005), which found that only during very prolonged exercise (such as the 226-km Ironman triathlon) undertaken in more extreme environmental conditions, *might* an acute sodium deficit contribute to exertional hyponatraemia. Thus the consensus opinion is that only under those unique conditions of very, very prolonged exercise in more severe environmental conditions, might an increased sodium intake *during* exercise be beneficial. However, there currently are no studies to support this hypothesis and two that actively contradict it (Hew *et al.*, 2006; Speedy *et al.*, 2002).

The finding that the osmotically inactive but exchangeable sodium stores in the body likely play a major role in the maintenance of serum sodium concentrations during exercise, regardless of the

nature or volume of fluids ingested during exercise (Noakes *et al.*, 2005), suggests that it is not the sodium ingested during exercise that is the key determinant of whether the serum sodium concentration will fall during exercise. Rather, it could be the individual athlete's ability to mobilize osmotically inactive sodium, or alternatively to resist osmotic inactivation of circulating Na^+ , that are the more important determinants (Noakes *et al.*, 2005).

On the other hand, the IOC Consensus Document states that sodium is "the electrolyte most critical to performance and health", so that it is important that "sodium be included in fluids ingested during exercise". Here the questions that invite answers are as follows:

(i) What evidence is there from *appropriately controlled, randomized, prospective clinical trials* to show that "it is especially important for athletes to include sodium in fluid replacement solutions to minimize hyponatraemia"? In fact, all the evidence proves this statement to be wrong.

Since all the currently published evidence shows that exercise-associated hyponatraemia and exercise-associated hyponatraemic encephalopathy are conditions of inadequate free water clearance by the kidneys in the face of high rates of free water ingestion leading to fluid overload (Speedy *et al.*, 2001) and to which any acute sodium deficiency plays only a minor role, if any (Hew *et al.*, 2006; Hew-Butler *et al.*, 2005; Noakes, 2002; Noakes *et al.*, 2005; Weschler, 2005), it is not immediately clear how the ingestion of sports drinks with low sodium content can "minimize hyponatraemia". Rather, since the condition is essentially one of inappropriate ADH secretion (SIADH), in which renal excretion of sodium is unimpaired whereas that of water is inhibited (Schwartz, Bennett, Curelop, & Bartter, 1957; Zerbe, Stropes, & Robertson, 1980), either the ingestion or intravenous infusion of excessive volumes of any saline solutions, other than those that are markedly hypertonic, will compound the hyponatraemia (Noakes, 2004a, 2004c; Noakes & Glace, 2004; Weschler, 2005). In fact, it is absolutely contraindicated for persons suffering from exercise-associated hyponatraemia and exercise-associated hyponatraemic encephalopathy to either ingest or be infused with hypotonic or isotonic saline solutions (Ayus, Arieff, & Moritz, 2005).

In this context, the specific choice of language by the IOC Consensus Committee is revealing since it acknowledges that sodium ingestion during exercise cannot prevent exercise-associated hyponatraemia, only "minimize" it. But the avoidance of overdrinking can absolutely prevent the development of exercise-associated hyponatraemic encephalopathy (Almond *et al.*, 2005; Noakes, Sharwood, Collins, & Perkins, 2004; Noakes *et al.*, 2005; Speedy *et al.*,

2000). That sodium plays an integral part in the development of exercise-associated hyponatraemia and exercise-associated hyponatraemic encephalopathy is accepted (Noakes *et al.*, 2005). However, the real determinant of the serum sodium concentration during exercise is unlikely to be the amount lost in sweat and urine or the amount ingested in hypotonic, electrolyte-poor sports drinks. Rather, it is more likely to be a result of (1) the extent to which the osmotically inactive, exchangeable sodium stores are mobilized in response to excessive fluid consumption, or (2) the magnitude of the reverse reaction – the conversion of osmotically active circulating Na^+ into osmotically inactive, intracellular sodium.

Indeed, it has been known since 1994 that the ingestion of an electrolyte-containing sports drink instead of water does not prevent the development of hyponatraemia in psychiatric patients with polydipsia (Goldman, Nash, Blake, & Petkovic, 1994; Reeves, 2004). The clear explanation was that the psychiatric patients preferred the taste of the sports drink to water; thus they were more likely to ingest greater volumes of the sports drinks than of water. As a result, the ingestion of an hypotonic sports drink increased their free water load, thereby compounding the hyponatraemia in the presence of the water-retaining effects of SIADH and the sodium-losing effects of the resulting increased interstitial pressure in the kidneys (Bartter & Schwartz, 1967; Schwartz *et al.*, 1957; Verbalis, 2003; Zerbe *et al.*, 1980).

Similarly, the pioneering study by Dr Cade, the inventor of Gatorade, and his colleagues (1992) showed that serum sodium concentrations were maintained at or above resting values in 42-km marathon runners whether they drank water or half- or full-strength Gatorade. And this was despite acute sodium losses calculated at 400 mmol. This acute sodium loss exceeds that measured in most individuals with exercise-associated hyponatraemic encephalopathy (Dugas & Noakes, 2005; Irving *et al.*, 1991; Noakes, 2002, 2004b; Noakes *et al.*, 2004, 2005). Others from Dr Cade's university have also failed to show that sodium ingestion is necessary to maintain the serum sodium concentrations during prolonged exercise (Powers *et al.*, 1990). Indeed, work from Dr Costill's laboratory at the same time (Barr, Costill, & Fink, 1991) showed that unreplaced sodium losses of 200–260 mmol "were not large enough to present a risk of hyponatremia during 6 h of exercise" (p. 816) so that "sodium replacement does not appear to be necessary during events of moderate intensity and less than 6 h duration; nevertheless, sodium losses were substantial" (p. 817). Costill (1986) later wrote that: "Electrolytes . . . have long been touted as important ingredients in sports drinks. But research shows that such claims are unfounded" (p. 76).

The continuing inference of the Gatorade Sports Science Institute including its director (Murray, Stofan, & Eichner, 2003; Murray & Eichner, 2004), the ACSM (Armstrong *et al.*, 1996; Convertino *et al.*, 1996), the US Army Research Institute of Environmental Medicine (Montain, Sawka, & Wenger, 2001), and now the IOC Consensus Document that the ingestion of sports drinks can prevent or “minimize” hyponatraemia, even though:

- (a) studies refuting this conclusion are in the public domain, including the initial study published in 1991 showing that ultramarathon runners with exercise-associated hyponatraemic encephalopathy are no more sodium deficient than are those finishing ultramarathon races with normal serum sodium concentrations (Irving *et al.*, 1991);
- (b) studies conducted by the inventor of Gatorade himself (Cade *et al.*, 1992) showed that marathon runners who ingested only water maintained their serum sodium concentrations, even when they developed acute sodium deficits of 400 mmol, among the highest yet recorded in endurance athletes (Noakes, 2002);
- (c) the inquest into the cause of the death from exercise-associated hyponatraemic encephalopathy of Dr Cynthia Lucero in the 2002 Boston Marathon concluded that she died because she had ingested large volumes of the sports drink, Gatorade (Smith, 2002); and
- (d) the recent finding, published in the *New England Journal of Medicine* (Almond *et al.*, 2005), that “the composition of the fluids consumed (plain water rather than sports drinks that contain electrolytes” was not a factor explaining why exercise-associated hyponatraemia developed in 62 (13%) of 488 runners studied in the same Boston Marathon (at which Gatorade was also the official sports drink).

All raise sober questions about the ability of the members of the IOC Consensus Document to draw defensible conclusions from the published data.

(ii) What evidence is there from appropriately controlled clinical trials to show that the “excessive loss of sodium during exercise . . . might cause them to fatigue due to development of muscle weakness or cramps”? My understanding is that sodium deficiency has never been linked to the development of muscle cramps or muscle “weakness” (Maughan, 1986; Schweltnus, Nicol, Laubscher, & Noakes, 2004; Sulzer, Schweltnus, & Noakes, 2005).

(iii) Indeed the question may be asked: What is the evidence that muscle cramping is a disease of sodium

deficiency? Are there any other credible explanations for the aetiology of this condition (Schweltnus, Derman, & Noakes, 1997) that should also be included in a review article that claims to be exhaustive and definitive?

Issue 5

The final issue that requires attention relates to the potential influence of possible conflicts of interest when scientists who receive funding from a particular industry, then write position statements or guidelines that might favour the sale of the products of that industry (*Nature*, 2005; Taylor & Giles, 2005). This is compounded when the organizations on whose behalf those guidelines are promulgated are themselves reliant on funding from that industry for their sustainability or growth, or both.

These concerns are clearly applicable to the drinking guidelines drawn up by the ACSM as advocated by the Gatorade Sports Science Institute (GSSI). Gatorade and the GSSI are the only two “platinum” sponsors of the ACSM. The ACSM drinking guidelines promote the twin concepts that (1) sports drinks containing salt (and glucose) are more beneficial during exercise than water and (2) that drinking “as much as tolerable” is the preferred option during exercise. Both these conclusions favour the promotion of sports drinks, including Gatorade, over water for all who participate in physical activity. Indeed, research sponsored by Gatorade and the GSSI consistently attempts to “prove” that electrolyte-containing sports drinks are superior to water (Baker, Munce, & Kenney, 2005; Gatorade Sports Science Institute, 2000) despite findings that might sometimes be interpreted to support the opposite conclusion (Dugas, 2006; Noakes, 2006).

The more outspoken opinion of Goldman (2001, p. 22) is that:

“Unfortunately, the late 20th century marketing success of “sports drinks” which contain large [*sic*] amounts of electrolytes and glucose, seem certain to continue to provide funds to (i) support research proving the benefits of such drinks and (ii) attempt to convince the military that purchase of such drinks would be beneficial to troops. These commercial marketing attempts may well prevail despite ample evidence that such drinks represent unnecessary – and possibly detrimental – supplements to a normal military diet, which typically contains large amounts of salt”.

The point is that the industry-favourable guidelines of the ACSM and the GSSI fail to warn (a) that they have been developed by organizations that

receive funding from the sports drink industry, and (b) that there is no scientific evidence that recreational athletes who exercise for less than 60 min per session, and who constitute the main target market for sports drinks, derive any unique physiological benefit by ingesting any fluids – water or sports drink – during exercise of such short duration (Table I), although they will likely feel less thirst if they do.

Thus, the ACSM drinking guidelines contain no warnings of potential conflicts of interest if either the ACSM or any of the authors of the guidelines receive financial or other “rewards” from the sports drinks industry. Similarly, the IOC document does not include any statement regarding conflicts of interest nor does it list which draftees, if any, of the IOC Consensus Statement receive funding or other largesse from the sports drink industry either in North America or Europe. Given the recent concerns about the effect of such funding on the scientific objectivity of those who benefit (*Nature*, 2005; Taylor & Giles, 2005), it is perhaps now opportune that such a declaration should, in future, always accompany any such consensus guidelines.

One might perhaps argue that scientists, the ACSM, and the IOC have every right to seek sponsorship from whomever they choose. However, by accepting the manufacturer of a sports drink as its sole “platinum sponsor”, the ACSM, in my view, foregoes its intellectual independence in this matter and thus the privilege of producing drinking guidelines for the global community. Since the research that we undertake on fluids and exercise is also funded by the sports drink industry (see voluntary disclosure below), the same applies to the guidelines I have developed (Noakes, 2003b). However, it is more difficult to understand how those guidelines, which promote *ad libitum* drinking, could be construed as unfairly advantageous to the sale of sports drinks.

I would like to conclude as follows:

- (1) It is essential that future guidelines for drinking during exercise should be based on the findings from randomized, appropriately controlled prospective clinical trials conducted under environmental and other conditions that match those found in “out-of-doors” exercise; and
- (2) that such guidelines must be developed by organizations and individuals who have no obligations to, and hence who are independent of, the sports drinks industry; or
- (3) who are prepared fully to declare any possible conflicts of interest that could be a result of the funding or any other largesse they receive from the sports drink industry.

Finally, to ensure that there is no publication bias in this field of research, in future journal editors should mandate (1) that all their chosen reviewers with contacts to the sports drink industry should declare those potential conflicts of interests, and (2) that the review of journal submissions that may appear to conflict with the currently accepted, industry-favourable dogma that promotes drinking either “as much as tolerable” or “to replace sweat losses” during exercise, should be entrusted to reviewers, most of whom have no links to the sports drink industry.

Journal editors who oversee the disposal of such articles should themselves also be required to make the public declaration of the extent to which they enjoy such links.

Voluntary declaration of potential conflict of interest

The author’s research group receives an annual financial research grant from Bromor (Pty) Ltd, manufacturers of the sports drink, Energade, available mainly in Southern Africa. The Sports Science Institute of South Africa of which the author is a Board member and Executive Co-Director, also receives financial support from Bromor (Pty) Ltd for its endorsement of the product, Energade. The author receives no personal financial benefit, either at present or promised in the future, as a result of this relationship.

Acknowledgements

The author’s research on which this review is based is funded by the Medical Research Council of South Africa, the Harry Crossley and Nellie Atkinson Staff Research Funds of the University of Cape Town, Discovery Health, Bromor Foods, and the National Research Foundation of South Africa through its THRIP initiative.

References

- Adolph, E. F. (1947a). *Physiology of man in the desert*. New York: Interscience Publishers, Inc.
- Adolph, E. F. (1947b). Water metabolism. *Annual Review of Physiology*, 9, 381–408.
- Almond, C. S., Shin, A. Y., Fortescue, E. B., Mannix, R. C., Wypij, D., Binstadt, B. A. *et al.* (2005). Hyponatremia among runners in the Boston Marathon. *New England Journal of Medicine*, 352, 1550–1556.
- American College of Sports Medicine (1975). The American College of Sports Medicine position statement on prevention of heat injuries during distance running. *Medicine and Science in Sports*, 7, VII–VIX.
- Armstrong, L. E. (2000). *Performing in extreme environments*. Champaign, IL: Human Kinetics.
- Armstrong, L. E., Costill, D. L., & Fink, W. J. (1985). Influence of diuretic-induced dehydration on competitive running performance. *Medicine and Science in Sports and Exercise*, 17, 456–461.

- Armstrong, L. E., Epstein, Y., Greenleaf, J. E., Haymes, E. M., Hubbard, R. W., Roberts, W. O. *et al.* (1996). American College of Sports Medicine position stand: Heat and cold illnesses during distance running. *Medicine and Science in Sports and Exercise*, 28, i–x.
- Armstrong, L. E., Maresh, C. M., Gabaree, C. V., Hoffman, J. R., Kavouras, S. A., Kenefick, R. W. *et al.* (1997). Thermal and circulatory responses during exercise: Effects of hypohydration, dehydration, and water intake. *Journal of Applied Physiology*, 82, 2028–2035.
- Ayus, J. C., Arieff, A., & Moritz, M. L. (2005). Hyponatremia in marathon runners. *New England Journal of Medicine*, 353, 427–428.
- Bachle, L., Eckerson, J., Albertson, L., Ebersole, K., Goodwin, J., & Petzel, D. (2001). The effect of fluid replacement on endurance performance. *Journal of Strength and Conditioning Research*, 15, 217–224.
- Backx, K., van Someren, K. A., & Palmer, G. S. (2003). 1 h cycling performance is not affected by ingested fluid volume. *Journal of Sports Sciences*, 13, 333–342.
- Baker, L. B., Munce, T. A., & Kenney, W. L. (2005). Sex differences in voluntary fluid intake by older adults during exercise. *Medicine and Science in Sports and Exercise*, 37, 789–796.
- Barr, S. L., Costill, D. L., & Fink, W. J. (1991). Fluid replacement during prolonged exercise: Effects of water, saline, or no fluid. *Medicine and Science in Sports and Exercise*, 23, 811–817.
- Barter, F. C., & Schwartz, W. B. (1967). The syndrome of inappropriate secretion of antidiuretic hormone. *American Journal of Medicine*, 42, 790–806.
- Below, P. R., Mora-Rodriguez, R., Gonzalez-Alonso, J., & Coyle, E. F. (1995). Fluid and carbohydrate ingestion independently improve performance during 1 h of intense exercise. *Medicine and Science in Sports and Exercise*, 27, 200–210.
- Brown, A. H. (1947). Dehydration exhaustion. In E. F. Adolph & Associates (Eds.), *Physiology of man in the desert* (pp. 208–225). New York: Interscience Publishers, Inc.
- Brown, A. H., & Towbin, E. J. (1947). Relative influences of heat, work, and dehydration on blood circulation. In E. F. Adolph & Associates (Eds.), *Physiology of man in the desert* (pp. 197–207). New York: Interscience Publishers, Inc.
- Buskirk, E. R., & Beetham, W. P. J. (1960). Dehydration and body temperature as a result of marathon running. *Medicina Sportiva*, XIV, 493–506.
- Buskirk, E. R., Iampietro, P. F., & Bass, D. E. (1958). Work performance after dehydration: Effects of physical conditioning and heat acclimatization. *Journal of Applied Physiology*, 12, 189–194.
- Cade, R., Packer, D., Zauner, C., Kaufmann, D., Peterson, J., Mars, D. *et al.* (1992). Marathon running: Physiological and chemical changes accompanying late-race functional deterioration. *European Journal of Applied Physiology and Occupational Physiology*, 65, 485–491.
- Casa, D. J., Armstrong, L. E., & Hillman, S. K. (2000). National Athletic Trainers Association Position Statement: Fluid replacement for athletes. *Journal of Athletic Training*, 35, 212–224.
- Cheuvront, S. N., Carter, R., III, & Sawka, M. N. (2003). Fluid balance and endurance exercise performance. *Current Sports Medicine Reports*, 2, 202–208.
- Cheuvront, S. N., & Haymes, E. M. (2001). *Ad libitum* fluid intakes and thermoregulatory responses of female distance runners in three environments. *Journal of Sports Sciences*, 19, 845–854.
- Convertino, V. A., Armstrong, L. E., Coyle, E. F., Mack, G. W., Sawka, M. N., Senay, L. C., Jr. *et al.* (1996). American College of Sports Medicine Position Stand: Exercise and fluid replacement. *Medicine and Science in Sports and Exercise*, 28, i–vii.
- Costill, D. L. (1986). *Inside running: Basics of sports physiology*. Indianapolis, IN: Benchmark Press.
- Costill, D. L., Kammer, W. F., & Fisher, A. (1970). Fluid ingestion during distance running. *Archives of Environmental Health*, 21, 520–525.
- Coyle, E. F. (2004). Fluid and fuel intake during exercise. *Journal of Sports Sciences*, 22, 39–55.
- Daries, H. N., Noakes, T. D., & Dennis, S. C. (2000). Effect of fluid intake volume on 2-h running performances in a 25 degrees C environment. *Medicine and Science in Sports and Exercise*, 32, 1783–1789.
- Dugas, J. P. (2006). Sodium ingestion and hyponatraemia: Sports drinks do not prevent a fall in serum sodium concentration during exercise. *British Journal of Sports Medicine*, 40, 372–374.
- Dugas, J. P., & Noakes, T. D. (2005). Hyponatraemic encephalopathy despite a modest rate of fluid intake during a 109 km cycle race. *British Journal of Sports Medicine*, 39, e38.
- Eichna, L. W., Bean, W. B., Ashe, W. F., & Nelson, N. (1945). Performance in relation to environmental temperature. *Bulletin of Johns Hopkins Hospital*, 76, 25–58.
- Fallowfield, J. L., Williams, C., Booth, J., Choo, B. H., & Grouns, S. (1996). Effect of water ingestion on endurance capacity during prolonged running. *Journal of Sports Sciences*, 14, 497–502.
- Gatorade Sports Science Institute (2000). *Fluids 2000. Sports drinks: How they work. Why water leaves you empty*. Sports Science Centre. Sports Science Topics (available from: <http://www.gssiweb.com>).
- Gisolfi, C. V. (1996). Fluid balance for optimal performance. *Nutrition Reviews*, 54, S159–S168.
- Glance, B., Murphy, C., & McHugh, M. (2002). Food and fluid intake and disturbances in gastrointestinal and mental function during an ultramarathon. *International Journal of Sport Nutrition, and Exercise Metabolism*, 12, 414–427.
- Goldman, M. B., Nash, M., Blake, L., & Petkovic, M. S. (1994). Do electrolyte-containing beverages improve water imbalance in hyponatremic schizophrenics? *Journal of Clinical Psychiatry*, 55, 151–153.
- Goldman, R. F. (2001). Introduction to heat-related problems in military operations. In K. B. Pandolf & R. E. Burr (Eds.), *Medical aspects of harsh environments* (pp. 3–49). Washington, DC: Office of the Surgeon General Department of the Army, USA.
- Heinrich, B. (2001). *Racing the antelope*. New York: Harper Collins.
- Hew, T. D., Sharwood, K. A., Speedy, D. B., & Noakes, T. D. (2006). *Ad libitum* sodium ingestion does not influence serum sodium concentrations during an Ironman triathlon. *British Journal of Sports Medicine*, 40, 255–259.
- Hew-Butler, T. D., Almond, C. S., Ayus, J. C., Dugas, J. P., Meeuwisse, W. H., Noakes, T. D. *et al.* (2005). Consensus Document of the 1st International Exercise-Associated Hyponatremia (EAH) Consensus Symposium, Cape Town, South Africa 2005. *Clinical Journal of Sports Medicine*, 15, 207–213.
- Hosey, R. G., & Glazer, J. L. (2004). The ergogenics of fluid and electrolyte balance. *Current Sports Medicine Reports*, 3, 219–223.
- Hsieh, M. (2004). Recommendations for treatment of hyponatraemia at endurance events. *Sports Medicine*, 34, 231–238.
- Institute of Medicine for the National Academies (2004). *Dietary reference intakes for water, potassium, sodium, chloride, and sulphate*. Washington, DC: The National Academies Press.
- Irving, R. A., Noakes, T. D., Buck, R., van Zyl, S. R., Raine, E., Godlonton, J. *et al.* (1991). Evaluation of renal function and fluid homeostasis during recovery from exercise-induced hyponatremia. *Journal of Applied Physiology*, 70, 342–348.

- Kay, D., & Marino, F. E. (2003). Failure of fluid ingestion to improve self-paced exercise performance in moderate-to-warm humid environments. *Journal of Thermal Biology*, 28, 29–34.
- Ladell, W. S. (1955). The effects of water and salt intake upon the performance of men working in hot and humid environments. *Journal of Physiology*, 127, 11–46.
- Maughan, R. J. (1986). Exercise-induced muscle cramp: A prospective biochemical study in marathon runners. *Journal of Sports Sciences*, 4, 31–34.
- Maughan, R. J., Burke, L. M., & Coyle, E. F. (Eds.) (2004). *Food, nutrition and sports performance II. The International Olympic Committee Consensus on Sports Nutrition*. London: Routledge.
- Maughan, R. J., Fenn, C. E., & Leiper, J. B. (1989). Effects of fluid, electrolyte and substrate ingestion on endurance capacity. *European Journal of Applied Physiology and Occupational Physiology*, 58, 481–486.
- McConnell, G. K., Burge, C. M., Skinner, S. L., & Hargreaves, M. (1997). Influence of ingested fluid volume on physiological responses during prolonged exercise. *Acta Physiologica Scandinavica*, 160, 149–156.
- McConnell, G. K., Stephens, T. J., & Canny, B. J. (1998). Fluid ingestion does not influence intense 1-h exercise performance in a mild environment. *Medicine and Science in Sports and Exercise*, 386–392.
- Montain, S. J., & Coyle, E. F. (1992). Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *Journal of Applied Physiology*, 73, 1340–1350.
- Montain, S. J., Latzka, W. A., & Sawka, M. N. (1999). Fluid replacement recommendations for training in hot weather. *Military Medicine*, 164, 502–508.
- Montain, S. J., Sawka, M. N., & Wenger, C. B. (2001). Hyponatremia associated with exercise: Risk factors and pathogenesis. *Exercise and Sport Sciences Reviews*, 29, 113–117.
- Mudambo, K. S., Leese, G. P., & Rennie, M. J. (1997). Dehydration in soldiers during walking/running exercise in the heat and the effects of fluid ingestion during and after exercise. *European Journal of Applied Physiology and Occupational Physiology*, 76, 517–524.
- Muir, A. L., Percy-Robb, I. W., Davidson, I. A., Walsh, E. G., & Passmore, R. (1970). Physiological aspects of the Edinburgh Commonwealth Games. *Lancet*, 2, 1125–1128.
- Murray, B. (1996). Fluid replacement: The American College of Sports Medicine position stand. *Sports Science Exchange*, 9, 1–5 (online journal).
- Murray, B., & Eichner, E. R. (2004). Hyponatremia of exercise. *Current Sports Medicine Reports*, 3, 117–118.
- Murray, B., Stofan, J., & Eichner, E. R. (2003). Hyponatremia in athletes. *Sports Science Exchange*, 16, 1–5 (online journal).
- Nadel, E. R., Fortney, S. M., & Wenger, C. B. (1980). Effect of hydration state on circulatory and thermal regulations. *Journal of Applied Physiology*, 49, 715–721.
- Nature (2005). Editorial: An unhealthy practice. *Nature*, 437, 1065–1066.
- Noakes, T. D. (1993). Fluid replacement during exercise. *Exercise and Sport Science Reviews*, 21, 297–330.
- Noakes, T. D. (1995). Dehydration during exercise: What are the real dangers? *Clinical Journal of Sports Medicine*, 5, 123–128.
- Noakes, T. D. (2000). Hyponatremia in distance athletes. Pulling the IV on the dehydration myth. *Physician and Sports Medicine*, 28, 71–76.
- Noakes, T. D. (2001). Hyponatremia or hype? *Physician and Sports Medicine*, 29, 22–40.
- Noakes, T. (2002). Hyponatremia in distance runners: Fluid and sodium balance during exercise. *Current Sports Medicine Reports*, 1, 197–207.
- Noakes, T. D. (2003a). *Lore of running*. Champaign, IL: Human Kinetics.
- Noakes, T. D. (2003b). Fluid replacement during marathon running. *Clinical Journal of Sports Medicine*, 13, 309–318.
- Noakes, T. D. (2003c). Overconsumption of fluids by athletes. *British Medical Journal*, 327, 113–114.
- Noakes, T. (2004a). Sodium ingestion and the prevention of hyponatremia during exercise. *British Journal of Sports Medicine*, 38, 790–792.
- Noakes, T. D. (2004b). Can we trust rehydration research? In M. McNamee (Ed.), *Philosophy and sciences of exercise, sports and health* (pp. 144–168). Abingdon, UK: Taylor & Francis.
- Noakes, T. D. (2004c). Response to the Letter to the Editor by Dr D.W. Stoddard. *Clinical Journal of Sports Medicine*, 14, 248–250.
- Noakes, T. D. (2006). Sports drinks: Prevention of “voluntary dehydration” and development of exercise-associated hyponatremia. *Medicine and Science in Sports and Exercise*, 38, 193.
- Noakes, T. D., Adams, B. A., Myburgh, K. H., Greeff, C., Lotz, T., & Nathan, M. (1988). The danger of an inadequate water intake during prolonged exercise: A novel concept revisited. *European Journal of Applied Physiology and Occupational Physiology*, 57, 210–219.
- Noakes, T. D., & Glace, B. (2004). Letters to the Editors. *International Journal of Sport Nutrition, and Exercise Metabolism*, 14, 249–254.
- Noakes, T. D., Sharwood, K., Collins, M., & Perkins, D. R. (2004). The dipsomania of great distance: Water intoxication in an Ironman triathlete. *British Journal of Sports Medicine*, 38, E16.
- Noakes, T. D., Sharwood, K., Speedy, D., Hew, T., Reid, S., Dugas, J. et al. (2005). Three independent biological mechanisms cause exercise-associated hyponatremia: Evidence from 2,135 weighed competitive athletic performances. *Proceedings of the National Academy of Sciences USA*, 102, 18550–18555.
- Noakes, T. D., & Speedy, D. B. (2006). Case proven: Exercise-associated hyponatremia is due to overdrinking. So why did it take 20 years before the original evidence was accepted? *British Journal of Sports Medicine*, 40, 567–572.
- Pitts, G. C., Johnson, R. E., & Consolazio, F. C. (1944). Work in the heat as affected by intake of water, salt and glucose. *American Journal of Physiology*, 142, 253–259.
- Powers, S. K., Lawler, J., Dodd, S., Tulley, R., Landry, G., & Wheeler, K. (1990). Fluid replacement drinks during high intensity exercise: Effects on minimizing exercise-induced disturbances in homeostasis. *European Journal of Applied Physiology and Occupational Physiology*, 60, 54–60.
- Pugh, L. G., Corbett, J. L., & Johnson, R. H. (1967). Rectal temperatures, weight losses, and sweat rates in marathon running. *Journal of Applied Physiology*, 23, 347–352.
- Reeves, R. R. (2004). Worsening of hyponatremia with electrolyte-containing beverage. *American Journal of Psychiatry*, 161, 374–375.
- Robinson, T. A., Hawley, J. A., Palmer, G. S., Wilson, G. R., Gray, D. A., Noakes, T. D. et al. (1995). Water ingestion does not improve 1-h cycling performance in moderate ambient temperatures. *European Journal of Applied Physiology and Occupational Physiology*, 71, 153–160.
- Sanders, B., Noakes, T. D., & Dennis, S. C. (1999). Water and electrolyte shifts with partial fluid replacement during exercise. *European Journal of Applied Physiology and Occupational Physiology*, 80, 318–323.
- Sanders, B., Noakes, T. D., & Dennis, S. C. (2001). Sodium replacement and fluid shifts during prolonged exercise in humans. *European Journal of Applied Physiology*, 84, 419–425.
- Saunders, A. G., Dugas, J. P., Tucker, R., Lambert, M. I., & Noakes, T. D. (2005). The effects of different air velocities on heat storage and body temperature in humans cycling in a hot, humid environment. *Acta Physiologica Scandinavica*, 183, 241–255.

- Sawka, M. N., & Montain, S. J. (2000). Fluid and electrolyte supplementation for exercise heat stress. *American Journal of Clinical Nutrition*, 72, 564S–572S.
- Sawka, M. N., Young, A. J., Francesconi, R. P., Muza, S. R., & Pandolf, K. B. (1985). Thermoregulatory and blood responses during exercise at graded hypohydration levels. *Journal of Applied Physiology*, 59, 1394–1401.
- Schmidt-Nielsen, K. (1964). *Desert animals*. Oxford: Oxford University Press.
- Schmidt-Nielsen, K. (1972). *How animals work*. Cambridge: Cambridge University Press.
- Schwartz, W. B., Bennett, W., Curelop, S., & Bartter, F. C. (1957). A syndrome of renal sodium loss and hyponatremia probably resulting from inappropriate secretion of antidiuretic hormone. *American Journal of Medicine*, 23, 529–542.
- Schwellnus, M. P., Derman, E. W., & Noakes, T. D. (1997). Aetiology of skeletal muscle “cramps” during exercise: A novel hypothesis. *Journal of Sports Sciences*, 15, 277–285.
- Schwellnus, M. P., Nicol, J., Laubscher, R., & Noakes, T. D. (2004). Serum electrolyte concentrations and hydration status are not associated with exercise associated muscle cramping (EAMC) in distance runners. *British Journal of Sports Medicine*, 38, 488–492.
- Sharwood, K., Collins, M., Goedecke, J., Wilson, G., & Noakes, T. (2002). Weight changes, sodium levels, and performance in the South African Ironman Triathlon. *Clinical Journal of Sports Medicine*, 12, 391–399.
- Sharwood, K. A., Collins, M., Goedecke, J. H., Wilson, G., & Noakes, T. D. (2004). Weight changes, medical complications, and performance during an Ironman triathlon. *British Journal of Sports Medicine*, 38, 718–724.
- Shibasaki, M., Kondo, N., & Crandall, C. G. (2003). Non-thermoregulatory modulation of sweating in humans. *Exercise and Sport Sciences Reviews*, 31, 34–39.
- Smith, S. (2002). Marathon runner's death linked to excessive fluid intake. *Boston Globe*, 13 August, p. A1.
- Speedy, D. B., Noakes, T. D., Boswell, T., Thompson, J. M., Rehrer, N., & Boswell, D. R. (2001). Response to a fluid load in athletes with a history of exercise induced hyponatremia. *Medicine and Science in Sports and Exercise*, 33, 1434–1442.
- Speedy, D. B., Rogers, I. R., Noakes, T. D., Thompson, J. M., Guirey, J., Safih, S. *et al.* (2000). Diagnosis and prevention of hyponatremia at an ultradistance triathlon. *Clinical Journal of Sports Medicine*, 10, 52–58.
- Speedy, D. B., Thompson, J. M., Rodgers, I., Collins, M., Sharwood, K., & Noakes, T. D. (2002). Oral salt supplementation during ultradistance exercise. *Clinical Journal of Sports Medicine*, 12, 279–284.
- Stover, E. A., Zachwieja, J., Stofan, J., Murray, R., & Horswill, C. A. (2006). Consistently high urine specific gravity in adolescent american football players and the impact of an acute drinking strategy. *International Journal of Sports Medicine*, 27, 330–335.
- Strydom, N. B., Wyndham, C. H., Van Graan, C. H., Holdsworth, L. D., & Morrison, J. F. (1966). The influence of water restriction on the performance of men during a prolonged march. *South African Medical Journal*, 40, 539–544.
- Stuempfle, K. J., Lehmann, D. R., Case, H. S., Bailey, S., Hughes, S. L., McKenzie, J. *et al.* (2002). Hyponatremia in a cold weather ultraendurance race. *Alaska Medicine*, 44, 51–55.
- Stuempfle, K. J., Lehmann, D. R., Case, H. S., Hughes, S. L., & Evans, D. (2003). Change in serum sodium concentration during a cold weather ultradistance race. *Clinical Journal of Sports Medicine*, 13, 171–175.
- Sulzer, N. U., Schwellnus, M. P., & Noakes, T. D. (2005). Serum electrolytes in Ironman triathletes with exercise-associated muscle cramping. *Medicine and Science in Sports and Exercise*, 37, 1081–1085.
- Taylor, R., & Giles, J. (2005). Cash interests taint drug advice. *Nature*, 437, 1070–1071.
- Twerenbold, R., Knechtle, B., Kakebeeke, T. H., Eser, P., Muller, G., von Arx, P. *et al.* (2003). Effects of different sodium concentrations in replacement fluids during prolonged exercise in women. *British Journal of Sports Medicine*, 37, 300–303.
- Verbalis, J. G. (2003). Disorders of body water homeostasis. *Best Practice and Research in Clinical Endocrinology and Metabolism*, 17, 471–503.
- Walsh, R. M., Noakes, T. D., Hawley, J. A., & Dennis, S. C. (1994). Impaired high-intensity cycling performance time at low levels of dehydration. *International Journal of Sports Medicine*, 15, 392–398.
- Weschler, L. B. (2005). Exercise-associated hyponatremia: A mathematical review. *Sports Medicine*, 35, 899–922.
- Wyndham, C. H., & Strydom, N. B. (1969). The danger of an inadequate water intake during marathon running. *South African Medical Journal*, 43, 893–896.
- Zerbe, R., Stropes, L., & Robertson, G. (1980). Vasopressin function in the syndrome of inappropriate antidiuresis. *Annual Review of Medicine*, 31, 315–327.