TITLE:

Why do team sports athletes drink fluid in excess when exercising in cool conditions?

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ABSTRACT

This study assessed the potential physiological and perceptual drivers of fluid intake (FI) and thirst sensation (TS) during intermittent exercise. 10 male rugby players (17 ± 1 years, stature: 179.1 ± 4.2 cm, body mass (BM): 81.9 ± 8.1 kg) participated in 6x6 min small-sided games, interspersed with 2 min rest, where FI was *ad libitum* during rest periods. Pre and post measurements of BM, subjective ratings (thirst, thermal comfort, thermal sensation, mouth dryness), plasma osmolality (POsm), serum sodium concentration ($S[Na^+]$), haematocrit and haemoglobin (to calculate plasma volume change; PV) were taken. FI was measured during rest periods. BM change was -0.17 ± 0.59% and FI was 0.88 ± 0.38L. Pre to post POsm decreased (-3.1 ± 2.3 $mOsm \cdot kg^{-1}$; $p = 0.002$) and $S[Na^+]$ remained similar (-0.3 ± 0.7$mmol \cdot L^{-1}$, $p = 0.193$). ΔPV was 5.84 ± 3.65%. FI displayed a relationship with pre POsm ($r = -0.640$, $p = 0.046$), pre thermal comfort ($r = 0.651$; $p = -0.041$), $\Delta S[Na^+]$ ($r = 0.816$, $p = 0.004$), and ΔPV ($r = 0.740$; $p = 0.014$). ΔTS displayed a relationship with pre mouth dryness ($r = 0.861$, $p = 0.006$) and Δmouth dryness ($r = 0.878$, $p = 0.004$). Yet a weak positive relationship between ΔTS and FI was observed ($r = 0.085$, $p = 0.841$). These data observed in an ambient temperature of 13.6 ± 0.9℃, suggest team sport athletes drink in excess of fluid homeostasis requirements and TS in cool conditions, however this was not influence by thermal discomfort.

KEYWORDS: HYDRATION; HYPONATREMIA; TEAM SPORT; THERMAL DISCOMFORT; THIRST.

INTRODUCTION.

Fluid balance is important for optimal physiological functioning, health and exercise performance (Baker and Jeukendrup 2014). Comprehension of the mechanisms that regulate fluid balance in varying sporting and environmental situations is vital to ensure the implementation of appropriate hydration strategies. Typically during exercise in temperatures less than 25˚C, team sport athletes appear to lose between ~1% and 1.5% of pre exercise body mass (BM) (Garth and Burke 2013). This magnitude of BM loss is well within the 2% threshold often proposed to impair performance in high-intensity, intermittent sport (Judelson et al. 2007), however inconsistencies surrounding this threshold are evident within the literature (Judelson et al. 2007; Sawka et al. 2007; Kraft et al. 2012). Nevertheless, this highlights that team sport athletes in such environmental conditions either do not elicit high fluid losses and/or are capable of offsetting their fluid losses with suitable fluid intake.

In contrast to the plethora of studies reporting fluid losses sufficient to cause a decrease in BM (reported as 'dehydration') (See review: Garth and Burke (2013)) other studies have reported potential cases of over-drinking in team sports (Horswill et al. 2009; MacLeod and Sunderland 2009; Black et al. 2013; Cosgrove et al. 2014; Jones et al. 2015). Over-drinking appears to be reported when athletes consume fluid in the absence of body fluid deficits to magnitudes which would stimulate the physiologically driven responses for compensatory water acquisition (≥2% change in plasma osmolality (POsm) and / or ≥10% change in blood volume) (Cheuvront et al. 2013; Cheuvront and Kenefick 2014) and thus in excess of homeostatic needs.

Over-drinking can lead to exercise induced hyponatremia (EAH), a condition characterised by its low (<135mmol·l^{-1}) sodium concentration ([Na^+]) in the blood. EAH can cause adverse increases in cellular volume known to cause health implications and decreases

in muscle performance (Speedy et al. 2001; Schucany 2007), although limited studies exist to confirm this. Recently, a study by Jones and colleagues (2015) reported observations of EAH on 33, 19 and 16% of observations during match-play, field and gym training in professional rugby players, respectively. While performance was not quantified, this appears to be a suboptimal hydration strategy. Furthermore, observational studies of this nature (O'Hara et al. 2010; Jones et al. 2015; Jones et al. 2016a; Jones et al. 2016b) fail to accurately determine body fluid balance due to the absence of measurement of appropriate physiologically controlled variables (*viz*. POsm) (Tam and Noakes 2013), thus the cause of potential over-drinking is not known.

Thirst is an innate regulatory mechanism that drives fluid intake and maintains body fluid homeostasis (McKinley and Johnson 2004). There is however a complex interplay of homeostatic and non-homeostatic factors that are known to mediate thirst sensation (Ramsay and Booth 1991; McKinley and Johnson 2004), which are yet to be fully investigated within high-intensity, intermittent team sport activities. Recently, the promotion of consuming fluid to the dictation of thirst has been recommended as the safest fluid intake strategy to ensure sufficient fluid balance in endurance activity (Hew-Butler et al. 2015; Hoffman et al. 2016b). However a major concern is the potential for non-homeostatic factors to stimulate inappropriate thirst sensation and consequently fluid intake above homeostatic needs (Hew-Butler et al. 2015), although to date this has received little investigation, particularly in the context of team sports.

Over-drinking in high-intensity intermittent sporting activities may be caused by the drive to consume fluid to attenuate thermal discomfort (Price and Campbell 2003; Black et al. 2013; Jones et al. 2015) similar to observations in some wheelchair athletes who are unable to produce sweat and thus less able to dissipate heat (Goosey-Tolfrey et al. 2008; Black et al. 2013). Although the mechanisms behind this were previously not suggested, this could be

plausible due to the associations between increases in body temperature and water retention mechanisms (Takamata et al. 1995). Increases in body temperature has also been associated with perceptual triggers that may cause behavioural changes such as timing and extent of fluid intake to alleviate the anticipated thermoregulatory onslaught (Phillips et al. 1984; Johnson et al. 2010; Periard et al. 2014). This is similar to that which has been seen between thermal discomfort and pacing strategies in the heat (Cheung 2010; Schlader et al. 2011). However, to date this relationship has not been explored within the context of team sports, with the measurement of key physiological (i.e. POsm) and perceptual (i.e. thermal comfort and thirst sensation) variables to confirm its origin.

To this end, to obtain a more comprehensive understanding of fluid dynamics and investigate the potential drivers of over-drinking in high-intensity intermittent team sport, the aim of this study was to assess the fluid intake, BM change, POsm change and physiological and perceptual variables during a rugby league specific training session in cool environmental conditions. It is hypothesised that (1) a prevalence of over-drinking will occur (2) fluid intake will be driven by thermal discomfort in the absence of dehydration mediated thirst thresholds.

MATERIALS AND METHODS

Study Design

Participants undertook a rugby league specific training session outdoors on a 3G pitch. During training, participants undertook intermittent small-sided games (SSG), which consisted of 6 x 6 minutes bouts of non-contact match play (Mean Total Distance: 4208.7m; Mean relative distance: 116.9m·s^{-1}; Mean High Speed Running (HSR): 37.5m·s^{-1}; Mean heart rate (HR): 164.3 beats.min^{-1}), interspersed with 2 minutes passive rest. The SSG was planned, delivered and refereed by a professional rugby league coach. Prior to and following the SSG, participants had BM measured and blood samples drawn then completed perception scales. During the SSG, fluid intake, specific physiological variables and external load measures were recorded. Players were split by the coach into two equal teams of ten to ensure a comparable number of participants and positional matches. Five players from each team participated in the study.

Participants

Ten male young professional rugby league players volunteered for this study (age: 17 ± 1 years, stature: 179.1 ± 4.2 cm, BM: 81.9 ± 8.1 kg and body surface area (BSA) = 2.01 ± 0.12m^2). To reduce performer bias, the specific purpose of the study was partially disclosed to participants, as the aim to investigate thermoregulatory responses during SSG (Cosgrove et al. 2014). All participants were fully informed of the true purposes after completion of the study. Participants were informed of the experimental procedures and risks and gave written informed consent. Ethics approval was granted by the Leeds Beckett University's Local Research Ethics Committee prior to testing.

Pre-testing requirements

Participants were asked to undergo their usual pre-training habitual routines in regards to fluid intake. In addition, all participants were asked to refrain from consuming alcohol, caffeine-containing products and strenuous exercise in the 24 hours prior to testing. Participants were asked to arrive in an over-night fasted state having been provided with a pre-set evening meal plan that contained foods low in sodium and carbohydrate to ensure limited fluid disturbances and a more balanced metabolic state upon arrival (Colombani et al. 2013). This was discussed with the club's nutritionist as to ensure similarity to their habitual diet and thus would cause no implications to shifts in metabolism.

Procedures

On arrival participants adopted a semi-recumbent position for 20 minutes, prior to two 4mL venous blood samples being drawn from the antecubital fossa vein. Samples were subsequently analysed for POsm and serum [Na^+] (s[Na^+]) to ascertain pre-exercise hydration status, and haemoglobin (Hb), and haematocrit (Hct). A POsm>295 $mOsm \cdot kg^{-1}$ was deemed hypohydrated (Kratz et al. 2004).

Following a urine void, BM was measured to the nearest 0.1 g using calibrated digital scales (Seca, Alpa 770, Hamburg, Germany) in underwear. All participants were then fitted with a skin thermistor (VitalSense® Dermal Patch, EQ-ACC-048, Hidalgo, Cambridge, UK) on their upper left thigh to monitor local skin temperature (T_{sk}) as previously done by Periard et al. (2014). Thigh T_{sk} as well as heart rate (HR) were then transmitted and continuously recorded on a monitoring device (SEMEquivital EQ02 lifemonitor, EQ-02-SEM-007, Hidalgo, Cambridge, UK) positioned on the left of the participants torso via a sensor belt (EQ-02-B1-1-

S Hidalgo, Cambridge, UK). Participants were also fitted with a portable 10-Hz global positioning system (GPS) unit (OptimEye S5, Catapult Sports, Melbourne, Australia) for recording of time motion analysis (absolute distance (m), relative distance ($m \cdot s^{-1}$) and HSR distance ($>5.5 m \cdot s^{-1}$) during the SSG. This was positioned by a vest between the scapulae at the base of the cervical spine (Emmonds et al. 2015). Participants were then asked to record resting subjective scale measures for thermal comfort, thermal sensation, thirst sensation and mouth dryness, which they had previously been familiarised with.

Participants then performed a 5 minute standardised warm-up (low intensity running and dynamic stretches) followed immediately by the SSG. Thigh T_{sk}, HR and time motion analysis data were continuously recorded throughout the SSG. Within each 2-minute rest period, upon cessation of each SSG, fluid (water) was freely available from individually marked bottles (500 ml) for the participants to ingest *ad libitum*. The bottles were weighed to the nearest 1g before and after each rest bout on calibrated portable scales (CS-2000, Ohaus, N.J, USA) to determine fluid consumption. Participants were instructed to not spit out or use their fluid for anything other than fluid intake. Researchers observed compliancy at all times. No participants passed urine or consumed any food during the observational period.

Ambient temperature and relative humidity were measured using a digital weather station (Oregon Scientific, Maidenhead, UK) and wind velocity was measured using a handheld Anemometer (RS, 180-7111, Northamptonshire, UK). These were all recorded at the start and end of the SSG (ambient temperature; $13.55 \pm 0.92^{\circ}C$; relative humidity: $71.45 \pm 1.34\%$; wind velocity: $1.15 \pm 0.35 m \cdot s^{-1}$).

Upon cessation of the SSG, participants immediately recorded all subjective measures and following towel drying were weighed to determine BM change using the equation previous described in O'Hara et al., (2010) and net fluid loss, was calculated as described by King et al.

(2008). Net fluid loss is defined as the balance between all sources of fluid gain and fluid loss, determined by the exact measurement or specific estimation between the initial and final body mass measurements. A final venous blood sample was subsequently drawn following the same procedures previously described for analysis of POsm, S[Na^+], Hct and Hb. Plasma volume was then calculated using the equation of Dill and Costill (1974). Finally, qualitative information was collected surrounding participants' prior hydration and nutritional knowledge through participation within education (further education qualifications e.g. A levels) or relevant workshops.

Statistical Analyses

All statistical analyses were performed using SPSS Statistics for Windows, version 22 (IBM, Armonk, NY, USA), with statistical significance set at $p<0.05$. Prior to analysis, normality and equality of variance was assessed using Kolmogorov-Smirnov test. Pre-post differences were assessed using paired t-tests unless data were non-parametric (thermal sensation) where Wilcoxon signed-rank was used. To identify relationships between key variables deemed closely related to water balance (Mears and Shirreffs 2013), Pearson's product moment correlation analysis was utilised unless data were non-parametric (thermal sensation), when Spearmans rank correlation was performed. Correlation coefficient effect sizes were interpreted as: 0.10 *small*, 0.30 *medium*, 0.50 *large* effect (Cohen 1992). For effect size between pre-post variables, Cohen's *d* was calculated with 95% confidence intervals (CI). This was interpreted as: 0–0.19 *trivial* effect, 0.2–0.59 *small*, 0.6–1.19 *moderate*, 1.2–2.0 *large*, and >2.0 *very large* effect (Hopkins 2000; Batterham and Hopkins 2006). Data are reported as means ± standard deviation (SD).

RESULTS

The physiological and perceptual measures for participants on arrival (Pre), and following the SSG (Post) are shown in Table 1. Total fluid intake was 0.88 ± 0.38 kg with a range of 0.41 – 1.38 kg, whilst net fluid loss was 1.02 ± 0.31 kg with a range of 0.49 - 1.51 kg. Participants had a pre-training POsm of 294.6 ± 4.0 mOsm·kg^{-1}, which decreased from pre to post (p = 0.002), and PV also increased (5.84 ± 3.65%). There was no difference between pre to post BM (p = 0.382), S[Na^+] (p = 0.193; Figure 1), thirst sensation (p = 0.093) or mouth dryness (p = 0.061). Thermal comfort decreased (p = 0.006) pre to post from *slightly comfortably* (+1.00 ± 1.22) to *neutral - slightly uncomfortable* (-0.75 ± +1.62). Whilst thermal sensation increased (p = 0.004) pre to post from *neutral – slightly cool* (-0.30 ± +0.82) to *warm - hot* (+2.50 ± +0.71).

*****INSERT TABLE I NEAR HERE*****

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Relationships

Relationships between physiological and perceptual variables related to water balance (Table 2) revealed that there was a *large* negative correlation between fluid intake and pre POsm (r = - 0.640, p = 0.046). Fluid intake had a *large* positive correlation with PV change (r = 0.740, p = 0.014) BM change (r = 0.767, p = 0.010), S[Na^+] change (r = 0.816, p = 0.004) and pre thermal comfort (r = 0.651, p = 0.041). There was no relationship between fluid intake and any other perceptual variables (thirst sensation, thermal sensation and mouth dryness;

Table 2) or post RPE (r = -0.115, p = 0.751). In addition, there was no relationship between fluid intake and total HSR, total distance, mean relative distance, mean HR and mean Tsk (r range = -0.349 – -0.405, p = 0.246 – 0.777).

There was a *large* positive correlation between change in thirst and pre mouth dryness (r = .861, p = .006) and change in mouth dryness (r = .871, p = .004), although no relationship with other perceptual variables appeared. In addition there was no differences between change in thirst and post RPE, total HSR, total distance, mean relative distance, mean HR and mean Tsk (r range = -0.588 – 0.641, p range = 0.086 – 0.920).

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DISCUSSION

The findings of this study show that during SSG in cool environmental conditions, male rugby league players consumed an average of 0.88 ± 0.38 kg of fluid with a large range of 0.41 – 1.38 kg. On average, players experienced a decrease in POsm, an increase in PV and a weak correlation between fluid intake and thirst was observed. This suggests that team sport athletes consume fluid in excess of homeostatic requirements and thirst sensation. However, fluid intake did not appear to be modulated by a rise in thermal discomfort as previously proposed (Price and Campbell 2003; Black et al. 2013; Jones et al. 2015), nor any other related physiological or perceptual variables and thus our hypothesis is rejected. An unexpected finding was the observation of *large* negative relationships between fluid intake and pre POsm and pre thermal comfort, highlighting that those that arrived 'more hydrated' and more thermally comfortable subsequently ingested more fluid.

This study demonstrated that participants experienced a mean change in BM of -0.17 ± 0.59%, further supporting that of others, in that team sport athletes exercising (e.g. training or match play) within cool environmental conditions (~12˚C) are effective at maintaining hydration to <2% BM loss (Garth and Burke 2013). However, it is apparent within these data that change in BM did not reflect the changes in POsm due to the observation of some BM loss arising without the occurrence of an increase in POsm (Table 1). This observation is likely due to the acknowledged dissociation between BM loss and total body water due to alternative sources for body water gain and BM loss (Maughan et al. 2007; King et al. 2008; Nolte et al. 2011; Tam and Noakes 2013). Consequently, this finding reflects that some degree of BM loss may be essential to maintain POsm (Nolte et al. 2011), given that other (e.g. substrate oxidation, respiratory water loss) mass losses occur during exercise (King et al. 2008). Thus, studies that utilised BM loss as an indicator of hydration status should be interpreted with caution.

Without the observation of POsm, it may be perceived that participants in this study consumed fluid (0.41 – 1.38 kg), sufficient to match or offset net fluid loss (0.49 -1.51 kg), whereas in actuality, participants within this cohort appear to consume fluid in excess of homeostatic requirements, given the observations of a decrease in POsm, increase in PV (Table 1) and weak correlations between POsm change and fluid intake (Table 2). To our knowledge, this is the first study to implement POsm measures as a key determinant of hydration status in high-intensity intermittent team sport activities and thus provides a greater insight into fluid balance than previous studies of this nature (O'Hara et al. 2010; Cosgrove et al. 2014; Jones et al. 2015; Jones et al. 2016a; Jones et al. 2016b). The interpretation of fluid balance through POsm can be confounded by biological variations, such as total body water content, type of exercise and environmental conditions (Cheuvront et al. 2012). For example, during high intensity exercise, regardless of fluid intake, PV decreases (causing POsm to increase) due to hydrostatic pressures exerted on the vascular system (Kargotich et al. 1998). Despite the limited availability of a criterion measure for determining specific fluid shifts, POsm is still deemed to have a higher diagnostic accuracy for assessing intracellular dehydration compared to other assessment methods (e.g. urine osmolality) (Cheuvront and Kenefick 2014).

One explanation for over-drinking may be attributed to the arrival hydration status of certain participants. Given POsm is typically omitted from studies exploring hydration status during team sports (O'Hara et al. 2010; Cosgrove et al. 2014; Jones et al. 2015; Jones et al. 2016a; Jones et al. 2016b), it is unclear to date the *true* arrival hydration status of such individuals. The current study shows 50% of the participants arrived in a hypohydrated state, as determined by POsm values >295mOsmol·kg^{-1}, thus exceeding the reported osmotic threshold for thirst stimulation (290 mOsmol·kg^{-1}) (Phillips et al. 1985). It must be noted that there is, however, substantial inter-individual variation (Robertson 1984) and threshold

inconsistencies within the literature, with ranges of 281 (Thompson et al. 1986) to 299 mOsmol·kg^{-1} (Baylis and Robertson 1980) reported.

If *ad libitum* (e.g. non-thirst related) fluid intake or thirst was driven by osmotic changes, then it would be expected that participants with higher pre POsm would drink more during the training session, as a result of these pre-exercise fluid deficits (Maughan et al. 2005; Silva et al. 2010). However, this does not seem to be the case within the current study, due to no positive correlation being indicated between pre-exercise POsm and fluid intake. Surprisingly, a *large* negative correlation between pre POsm and fluid intake was observed (Table 2), demonstrating that those who arrived 'more hydrated' subsequently drank more. This may be due to an increased awareness and knowledge of the importance of hydration and (perceived) concerns about dehydration that may make certain individuals more prone to over-drinking (Frizzell et al. 1986; Beltrami et al. 2008; Hoffman et al. 2016a).

To partially address this concern, the present study obtained information surrounding participants' prior hydration and nutritional knowledge from participation within education or workshops. Out of the ten participants, only three declared any prior hydration/nutritional knowledge and these did not correspond to those that arrived with low POsm values (pre POsm range: 292 – 297 mOsmol.kg^{-1}) or consumed excessive fluid (fluid intake > fluid loss). Consequently, it does not appear that knowledge of hydration was associated with individuals whom over-drank. However, as to whether individuals within this cohort were habitually 'high drinkers' (Johnson et al. 2016) and/or possess concerns about dehydration independent of educational scientific knowledge, cannot be determined by this study design and warrants further investigation. Nonetheless, these findings may suggest that excessive fluid consumption independent of osmotic driven thresholds may be due to fluids contribution to differing stimuli.

Average post-exercise POsm was within the euhydrated threshold (291.50 ± 4.14 mOsmol.kg^{-1}) (Kratz et al. 2004) despite fluid intake being beyond that required for

homeostatic needs. It is unclear if this would have been the case if exercise was prolonged, given excessive fluid intake has been shown to cause a decrease in POsm and S[Na^+], potentially causing EAH (Hew-Butler et al. 2015). Within the present study, mean post S[Na^+] values were 138.6 ± 1.8 mmol.l^{-1}, indicating that on average, players maintained S[Na^+] to within homeostatic regulatory set points (135 to 145 mmol.l^{-1}) (Kratz and Lewandrowski 1998). Whilst this is therefore not of concern, some participants S[Na^+] decreased, and one participant had a post S[Na^+] of 135 mmol.l^{-1}. This represents borderline asymptomatic EAH (Hew-Butler et al. 2015), likely occurring due to greater fluid consumption compared to fluid loss (1.09 vs. 0.49 kg) and pre SSG borderline hypoosmotic state of 287 mOsmol.kg^{-1}. In addition, a *large* negative relationship existed between pre POsm and S[Na^+] change alongside a *large* positive correlation between fluid intake and S[Na^+] change (Table 2). Consequently, it appears that those who arrived more hydrated and subsequently drank more are at a greater risk of diluting their S[Na^+].

Observations demonstrating participants who arrived more hydrated, drank more, and were at a greater risk of diluting their S[Na^+] is consistent with the findings by Jones et al (2015) and Cosgrove et al (2014), who observed rugby union players during training and match play. In the population observed by Jones et al (2015), 94% of players arrived euhydrated and on 36% of the time, players experienced EAH. In contrast, for the population observed in Cosgrove et al (2014), 89% of players arrived hypohydrated, and there were no observations of EAH. Interestingly, within the current study, those that commenced exercise in a hypohydrated state were more capable of regulating S[Na^+] as no changes from pre to post were observed within these individuals. These observations were again observed when fluid intake exceeded fluid loss.

Although a decrease in POsm occurred, a *large* (ES = 0.96) increase in thirst sensation was reported from pre-post. A decrease in POsm would theoretically suppress thirst and AVP

release (Kumar and Berl 1998). Consequently, thirst appears to be driven by non-osmotic factors causing inappropriate sensations of thirst and/or individuals are wrongly interpreting this thirst sensation (Maughan and Shirreffs 2010). The development of thirst and fluid ingestion before the presence of body fluid deficits has previously been observed in early laboratory based research by Phillips et al. (1984). They concluded that thirst developed in response to subtle oropharyngeal cues and thus provide evidence for anticipatory thirst.

In our study, the increase in thirst was associated with mouth dryness (both pre mouth dryness and mouth dryness change; Table 2). This is not a new notion, as mouth dryness is well reported to be a confounding oropharyngeal variable contributing to the sensation of thirst (Phillips et al. 1985; Brunstrom 2002). However, despite this *large* increase in thirst, no association existed between this and fluid intake. This further elucidates the clear discrepancies between drinking *ad libitum* (as instructed within this study) and drinking to thirst, which are often used interchangeably within the literature (Armstrong et al. 2014). Consequently the behavioural act of fluid consumption appears to be in excess of both osmotic and non-osmotic driven thirst sensations.

Drinking above the dictation of thirst has been reported to be the underlying pathophysiological mechanisms of numerous cases of symptomatic and in some cases fatal EAH (Hew-Butler et al. 2015). Reported cases of symptomatic EAH have involved a variety of temperate to hot environmental conditions (15.2 – 41˚C) and differing activities (e.g. open water swimming (Rogers et al. 2015), Ironman (Hew-Butler et al. 2007) and American football (Dimeff 2006)), whereby EAH has often arisen due to the adoption of wrongful advice to drink ahead of thirst or drink as much as can be tolerated (Hew-Butler et al. 2015). The lack of synergy between thirst and fluid intake in the current study elucidates that other factors were likely involved in influencing fluid consumption beyond changes in POsm and thirst sensation. It is suggested that extrinsic behaviour in relation to fluid availability may override intrinsic

responses in the short term and thus drinking strategies to potentially discourage fluid intake may be beneficial to ensure optimal fluid balance.

There was a weak relationship between fluid intake and thermal comfort change (r = 0.012, p = 0.974) during the SSG. Consequently, these data do not support the proposed theory that fluid intake occurs to attenuate rises in thermal discomfort/strain (Price and Campbell 2003; Black et al. 2013; Jones et al. 2015). The lack of correlation between thermal comfort and thirst (Table 2) also suggests that no physiological mechanism existed in stimulating water retention/acquisition mechanisms (i.e. non-osmotic AVP secretion; Takamata et al., 1995), however, without a direct measurement of AVP, this is merely speculation. This lack of association may be due to the cool conditions of the current study which may not elicit high thermal strains to levels required to stimulate behavioural thermoregulatory responses such as fluid consumption, although this was proposed by Jones et al., (2015) who observed athletes in approximately 10^{o}C. As skin temperature largely determines thermal comfort (Frank et al. 1999), higher ambient temperatures, may play a more pivotal role (rather than metabolic heat accumulation) and augment the requirement of fluid intake to attenuate thermal strain. Consequently, the environmental conditions within the current study may sit below this 'threshold' as fluids may not provide the required thermal comfort stimuli within cold conditions as they would do in hot conditions (Maughan et al. 2005).

In contrast to expectation, despite the lack of association between thermal comfort change and fluid intake, pre thermal comfort showed an association with fluid intake in a positive direction (r = 0.651, p = 0.041). The mechanisms behind this relationship are unknown, however it is assumed that this link may be a result of the corresponding POsm level. Those that had lower POsm upon arrival reported higher thermal comfort levels (r = -0.772, p = 0.009), and as previously mentioned those with lower pre POsm subsequently drank more. Consequently, it is speculated that this would not be a causal factor but one that is integrated

within the POsm-fluid intake relationship. Beyond this, no other factors related to fluid intake (e.g. mouth dryness) or thermal comfort (skin temperature, RPE, thermal sensation) were directly associated with cumulative fluid intake.

It is proposed that the format of the training session (6 x 6 minute SSG, interspersed with 2 minute passive rest in which fluid was available *ad libitum*) may have played a role in the excessive fluid consumption. This could be due to the frequency and length of rest periods and the proximity and volume of fluid availability, in that despite participants not requiring fluid consumptions due to any homeostatic (both thermoregulatory and fluid balance) needs, fluid was then able to be consumed in excess. Thus, high-intensity intermittent team sports that provide fewer opportunities to rehydrate (i.e. soccer) than rugby (union and league) may reduce the prevalence of over-drinking. However, full quantification of drinking behaviours and opportunities have so far not been investigated in such context. Nonetheless it would appear that the availability of fluid during these planned and unplanned break periods may need to be reviewed in both training and match play environments if to ensure individuals better match their homeostatic needs. The practical applications of this study, to be used by practitioners working with similar cohorts are that structured drink breaks during training need to align with the number of breaks during a match and/or consider how much fluid athletes may need, taking into consideration the environmental temperature, training status of athletes, intensity of the session, and their respective arrival hydration status.

The key limitation of this study is the sample size used. This was due to the recruitment of elite rugby league players, thus access to this population was limited. Furthermore, the findings of this study align with the context of the design and method, and should not be extrapolated beyond. For example, to standardise the arrival status of athletes, participants arrived fasted. While this may not be representative of an athlete's habitual arrival status given the large intra- and inter-player variability for habitual arrival hydration status (O'Hara et al.

2010), which can be influenced by food (Sawka et al. 2007), standardisation of the arrival condition of players was considered important in this study. Future research should investigate drinking behaviour in various fed and hydration status' on arrival, as this may influence drinking behaviours. Finally, training sessions that differ from this study by being longer in duration, undertaken in warmer environmental conditions, or when players have differing starting hydration status (for example, if this was the second session of the day, or not an early morning session) may observe different findings, which all warrant further investigation.

In conclusion, based on findings from this study, *ad libitum* fluid intake appears to be in excess of both osmotic (POsm and S[Na^+]) and non-osmotic (thermal comfort) factors. In addition, factors such as thermal comfort do not appear to be either a non-osmotic physiological stimulus or anticipatory behavioural stimulus for thirst sensation and fluid consumption as previously hypothesised. However, individuals appeared to drink above the dictation of thirst sensation. It is proposed that this may be a result of the proximity and frequency of drinks breaks provided. In addition, this raises the question as to whether drinking to the dictate of our innate thirst mechanisms may better guide fluid consumption in high-intensity team sport activities as seen in prolonged endurance exercise (Hew-Butler et al. 2015; Hoffman et al. 2016b), limiting over-hydration and associated dilution of S[Na^+]. However, further exploration is warranted to investigate the mechanisms that dictate fluid balance beyond homeostatic control and tackle drinking behaviours and opportunities to allow for a better synchrony of homeostatic needs, while quantifying the influence on exercise performance.

CONFLICT OF INTEREST

The authors report no conflicts of interest associated with this manuscript.

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TABLES

Table I: Mean ± SD physiological and perceptual variables for participant's pre and post SSG

	Pre	Post	*p* value	Cohens *d*	± 95% CI
BM (kg)	81.94 ± 8.14	81.80 ± 8.16	0.382	0.02	0.88
Net fluid loss (kg)	1.02 ± 0.31		-	-	
Sweat Rate (L/hr)	1.15 ± 0.35				
Fluid Intake (kg)	0.88 ± 0.38		-	-	
Fluid Intake (L/hr)	1.00 ± 0.43				
POsm($mOsm \cdot kg^{-1}$)	294.6 ± 3.98	291.50 ± 4.14	0.002*	0.76	0.91
S[Na+] ($mmol \cdot L^{-1}$)	138.9 ± 1.37	138.6 ± 1.78	0.193	0.19	0.88
PV Δ (%)	5.84 ± 3.65		-	-	
Thirst	4.75±2.12	6.20±1.69	0.093	-0.94	0.81
Thermal Comfort	+1.00 ± 1.22	-0.75 ± +1.62	0.006*	1.22	0.95
Thermal Sensation	- 0.30±+0.82	+2.50±+0.71	0.004*	-3.65	1.43
Mouth Dryness	4.47 ± 2.09	7.23 ± 2.34	0.061	-1.24	0.96

±95% CI: add and subtract this number to the mean effect to obtain the 95% confidence limits for the true difference

Table II: Correlation Coefficients between independent variables and fluid intake and thirst sensation.

Dependent Variable	Independent Variable	*r*	*p*	*r*	*p*
		Pre		*Change*	
Fluid intake	$S[Na^+]$	-0.340	.336	0.816	0.004*
	BM	-	-	0.767	0.010*
	POsm	-0.640	0.046*	-0.075	0.836
	ΔPV	-	-	0.740	0.014*
	Thirst	-0.091	0.830	0.085	0.841
	Thermal Comfort	0.651	0.041*	0.012	0.974
	Thermal Sensation	0.021	0.955	-0.040	0.912
	Mouth Dryness	0.030	0.925	0.246	0.494
Thirst Δ	$S[Na^+]$	0.226	0.591	0.019	0.965
	BM	-	-	0.258	0.539
	POsm	0.244	0.561	-0.322	0.436
	PV	-	-	0.583	0.129
	Thermal Comfort	0.207	0.624	0.302	0.467
	Thermal Sensation	0.415	0.306	0.014	0.974
	Mouth Dryness	0.861	0.006*	0.878	0.004*

*Correlation is significant.

FIGURE CAPTIONS

Figure 1: Individual S[Na+] collected pre and post SSG. Dotted lines represent the reference range for S[Na^{+}] (135 – 145 mmol.l^{-1}) (Kratz and Lewandrowski 1998)