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Effect of individual environmental heat stress variables on training and recovery in professional team sport

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1	Running Head: Thermoregulation and team-sport training			
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30 Abstract

31 Purpose: Exercise in hot environments increases body temperature and 32 thermoregulatory strain. However, little is known regarding the magnitude 33 of effect that ambient temperature (T_a), relative humidity (RH) and solar 34 radiation (SR) individually have on team sport athletes. We aimed to 35 determine the effect of these individual heat stress variables on team-sport 36 training performance and recovery. Methods: Professional Australian 37 Rules Football (ARF) players (n=45) undertook eight-weeks pre-season 38 training producing a total of 579 outdoor field-based observations with T_a, 39 RH and SR recorded at every training session. External load (distance 40 covered, m.min⁻¹; percent high speed running >14.4 km.h⁻¹; %HSR) was 41 collected via a global positioning system. Internal load (ratings of 42 perceived exertion (RPE), heart rate (HR)), and recovery (subjective 43 ratings of wellbeing and heart rate variability (rMSSD)) were monitored 44 throughout the training period. Mixed effects linear models analysed 45 relationships between variables using standardised regression coefficients. 46 Results: Increasing SR exposure was associated with reduced distance covered (-19.7 m.min⁻¹, β=-0.909, p<0.001), %HSR (-10%, β=-0.953, 47 48 p<0.001) during training, and rMSSD 48 h post-training (-16.9ms, β =-49 0.277, p=0.019). Greater RH was associated with decreased %HSR (-3.4%, β =-0.319, p=0.010), but increased % duration >85% HRmax (3.9%, 50 51 β =0.260, p<0.001), RPE (1.8AU, β =0.968, p<0.001) and self-reported 52 stress 24 h post-training (-0.11AU, β =-0.24, P=0.002). In contrast, higher 53 Ta was associated with in increased distance covered (19.7 m.min⁻¹, β =0.911, p<0.001) and %HSR (3.5%, β =0.338, p=0.005). Conclusions: 54 55 We show the importance of considering the individual factors contributing 56 to thermal load in isolation for team sport athletes, and that SR and RH 57 reduce work capacity during team sport training and have potential to slow 58 recovery between sessions.

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63 Introduction

64 Training and competition in the heat can provide additional physiological 65 demands with potential to limit the intensity/duration of exercise, and 66 increase the rating of perceived exertion (RPE) compared with exercise in 67 thermoneutral conditions¹. Indeed, there is a large body of evidence 68 examining the effects of hot environments whilst undertaking steady-state 69 aerobic exercise ^{1,2}. For example, Galloway and Maughan ³ previously 70 showed that under laboratory conditions of increasing ambient 71 temperature (T_a) with fixed RH there are significant increases in heart rate 72 (HR), core temperature, skin temperature (T_{sk}), sweat rate, RPE and 73 impaired exercise capacity. Similarly, when increasing RH with fixed hot 74 T_a there is also increased T_{sk}, sweat rate, and RPE, and decreased exercise 75 performance ⁴. Finally, Otani and colleagues ⁵ showed that the effects of 76 increased solar radiation (SR) exposure together with constant T_a and RH 77 elevates Tsk, reduces the core-to-skin temperature gradient, and 78 subsequently leads to decreased endurance capacity. Accordingly, each 79 heat stress variable contributing to total heat strain may have individual 80 effects with the potential to reduce physical performance in hot conditions.

81 The vast majority of previous research on thermoregulation during 82 exercise in hot environments has focused on the endurance athlete, and 83 there is a paucity of available data examining the effect of exercise in the 84 heat on the team-sport athlete ⁶. Some previous studies have examined 85 physiological changes to heat stress in team sport players, including 86 characterizing responses during match play at various wet bulb globe 87 temperatures ⁷. Many team-sports undertake physical preparation outdoors 88 during summer months at geographical locations characterised by high T_a, 89 RH and SR⁶. Repeatedly training in hot environments may promote long-90 term adaptations such as improved sweat response and decreased 91 cardiovascular strain with the potential to convey a benefit for team sport 92 performance⁸. Conversely, the acute thermoregulatory responses to 93 exercise in hot conditions may attenuate physical work capacity and the quality of training sessions, while frequent heat exposure during high-94

62

95 intensity intermittent team sport training may lead to overreaching and
96 adverse effects on athlete wellness and performance ⁹.

97 Understanding the physiological effects of recurring exposure to heat 98 stress during training or competition is necessary to optimise physical 99 preparation and well-being in team sport athletes. Moreover, there is 100 currently no data on varying environmental conditions that consider the 101 individual effects of T_a, RH and SR on external load, internal load and 102 recovery from intermittent, high intensity team-sport training activities. 103 Thus, the primary aim of this study was to determine the individual effects 104 of T_a, RH and SR on professional team-sport training sessions undertaken 105 in hot conditions. Our secondary aim was to establish the individual effect 106 of these heat variables on recovery during the 48 h period following 107 training bouts. We hypothesized that with increasing exposure to each 108 individual heat stress variable, external work completed during training 109 would decrease concomitant with higher internal loads and impaired 110 recovery.

111 Materials and Methods

112 **Participants**

113 A convenience sample of forty-five professional male athletes (mean \pm 114 standard deviation [SD]; age: 22.9 ± 3.8 yrs, height: 188.4 ± 8.3 cm, body 115 mass: 86.9 \pm 9.4 kg, maximal aerobic speed 17.6 \pm 0.8 km/h) from one 116 professional football club competing in the Australian Football League 117 participated in this study. Athletes completed a minimum of five weeks 118 pre-season training in a hot environment before the data collection period 119 and were deemed to be heat acclimated. Ethical approval was granted by 120 Bond University Human Research Ethics Committee (FO00007).

121

122 Experimental Protocol

123 Internal and external training loads were captured throughout an eight-124 week training period of the pre-season preparation phase spanning January 125 and February of the Australian summer. Upon removal of 'stationary skill' 126 sessions that were completed during the experimental period, an average 16 ± 3 sessions were recorded per player (range: 4 to 20), resulting in 579
outdoor field-based training observations in the final analysis. Participants
wore the same clothing (singlet and shorts) during each training session.
Pre- and post- each training session, body mass (BM) was recorded with
participants wearing the same clothing (running shorts) using standardised
weighing scales (Excell Precision, New Taipei, Taiwan) to the nearest 100
g for calculation of body mass changes from pre- to post-session.

134

135 External training load

136 External training loads for each participant were collected and downloaded 137 in accordance with previously described methods ¹⁰. Participants used the 138 same global positioning system (GPS) device (S5, Catapult Sports, 139 Melbourne, Australia) for each session to mitigate inter-unit measurement 140 errors ¹¹. Distance covered per minute (m.min⁻¹) and percent of total 141 distance completed above 14.4 km/h (% high speed running; %HSR) were 142 selected and used in subsequent statistical analyses, in part, to reduce 143 potential issues of multicollinearity. Individual training session data was 144 only included in the analysis if the athlete had completed a minimum of 145 80% of each prescribed training session.

146

147 Internal training load

148Ratings of perceived exertion (RPE) were obtained 10-30 min following149the completion of each training session using Borg's CR-10 scale 12. Heart150Rate (HR) data was collected via chest strap HR monitors (T34, Polar151Electro, Espoo, Finland). HR data was analysed by quantifying the percent152of total duration within specific 'zones' (Zone 3 = 65-74%, Zone 4 = 75-15384%, Zone 5 = >85% HR_{max}).

154

Individual self-reported ratings of wellbeing were assessed via a psychometric questionnaire on a 10-point Likert scale with 1 representing 'the worst I could possibly feel' and 10 representing 'the best I could possibly feel' in accordance with methods described previously ¹³. Objective measures of heart-rate variability (HRV) were assessed upon waking each morning by R-R series recording via photoplethysmography

161 using a valid and reliable, commercially available smartphone application 162 (HRV4Training)¹⁴. HRV data was subsequently analysed for the root 163 mean sum of the squared differences (rMSSD) between each successive 164 heartbeat on recovery days +1 (24 h) and +2 (48 h) after training sessions, 165 and compared to rolling baseline data obtained from a minimum of four readings in the prior 7-days ¹⁵. rMSSD was chosen as the HRV variable 166 167 of interest due to the relationship with vagal activity ¹⁶ and greater reliability compared to other spectral indices ¹⁷. 168

169

170 Environmental Monitoring

171 The training location at which data were collected was a coastal, sub-172 tropical region (28° S, 153° E) in Australia. The T_a (°C) and RH (%) were 173 measured via a portable weather station (Kestrel 5000, Kestrel 174 Instruments, Pennsylvania, USA), while SR (W/m²) was recorded via 175 pyranometer (MP-100, Apogee Instruments, Utah, USA) at 15-minute 176 intervals during each field-based training session. The devices were 177 mounted on a level tripod 1.5 m above ground in the same location 178 adjacent to the training field. After completion of each training session, 179 data was downloaded to a custom Microsoft Excel spreadsheet. To account 180 for the varying duration of training sessions, environmental 'exposure' 181 was quantified by multiplying session duration by the mean of recorded 182 T_a, RH and SR with data expressed as session means.

183

184 Statistical Analyses

185 Training environment data during the experimental period were analysed 186 using one-way analysis of variance (IBM SPSS Statistics, V. 25). Data are 187 presented as mean \pm standard deviation with statistical significance set at 188 p < 0.05.

189 Relationships between internal and external load and the training
190 environment were analysed using mixed effects linear models via the
191 *Lme4* package in *R Studio* statistical computing software (V. 1.1.442).
192 Mixed-effect linear models were applied to training and recovery variables
193 incorporating the individual as a random effect and heat stress variables as
194 fixed effects using the equation:

195	$y_i = \beta_1 x_{1i} + \beta_2 x_{2i} + \beta_3 x_{3i} + b_i + \varepsilon_i$
196	where y_i is the value of the outcome variable of interest <i>i</i> , β_1 through β_3
197	are fixed effect coefficients, x_{1i} through x_{3i} are fixed effect variables for
198	observation i, b_i is the random effects assumed to be multivariate normally
199	distributed, and ε_i is the error for case <i>i</i> which is also assumed to be
200	multivariate normally distributed 18. Normality assumptions were
201	validated using residual and QQ-plots, and the adequacy of the model
202	structures was determined via residual plots and quantified using standard
203	measures of intraclass correlations and coefficients of determination. All
204	variables of interest in the model are reported using standardised
205	regression coefficients (standardised beta (β)), allowing assessment of
206	practical significance. Standardised regression coefficients for each
207	variable were multiplied by the standard deviation of the change in
208	dependent variable to obtain the absolute change in the units of
209	measurement ¹⁹ . Qualitative descriptors for reporting of standardised beta
210	scores were adopted using a comparable approach to effect size statistics
211	to interpret the magnitude of the association between heat stress, training
212	and recovery variables. We interpreted the standardised beta using
213	threshold values of 0.2 as a small effect, 0.5 as a moderate effect, and 0.8 $$
214	as a large effect. Multicollinearity between heat stress variables was
215	established through Spearman's correlation matrix analysis and in the
216	event that any variables had a multicollinearity $r = > 0.8$ they were
217	excluded from the model ^{20,21} .

218

219 Results

220 Environmental Conditions

221 Mean environmental conditions during the experimental period were 30.9 222 ± 2.1 °C T_a (Range: 26.7 to 34.4 °C), $61 \pm 6\%$ RH (Range: 52 to 75 %) and 223 718 ± 224 W/m² SR (Range: 239 to 1001 W/m²). Multicollinearity did not 224 exist between heat stress variables. Wet-bulb globe temperature (WBGT) 225 during the experimental period was 29 ± 2.5 °C (Range: 24.4 to 32.9 °C). 226 There was no significant difference in variables of environmental

Accepted author manuscript version reprinted, by permission, from International Journal of Sports Physiology and Performance, 15(10), 1393-1399, https://doi.org/10.1123/ijspp.2019-0837 © Human Kinetics, Inc. 227 conditions (Table 1) or training load (Table 2) between training sessions

Insert Table 1 Here.

228 (n=20) throughout the eight-week experimental period.

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230

231

- Insert Table 2 Here.
- 232

233 External Training Load

234 Increasing SR exposure was associated with a decrease in distance covered (-19.7 m.min⁻¹, β = -0.909, p <0.001), but there was no significant 235 relationship between changes in RH and m.min⁻¹ at each training session 236 237 (Figure 1a). In contrast, increasing T_a exposure was associated with in an increase in distance covered (19.7 m.min⁻¹, β = 0.911, p <0.001). There 238 239 were divergent effects on the change in %HSR completed during training 240 sessions by each of the individual heat stress variables (Figure 1b). 241 Specifically, an increase in SR exposure was associated with a large 242 decrease in %HSR completed during training (-10%, β = -0.953, *p* < 0.001). 243 There was also a small effect of increasing RH that was associated with lower %HSR (-3.4%, β = -0.319, p =0.010). However, increasing T_a 244 245 exposure during training was related to a small increase in percent %HSR 246 $(3.5\%, \beta = 0.338, P=0.005)$ (Figure 1b).

247 Internal Training Load

248 Increasing T_a exposure during training sessions was associated with a 249 concomitant increase in mean HR (4.8 bpm, β = 0.449, p <0.001). 250 However, there was no relationship between increases in SR or RH 251 exposure and mean HR (Figure 2a). There were no significant 252 relationships between the percent duration of training completed between 253 65-74% HR_{max} and any heat stress variables during the experimental 254 period ($\beta = -0.04 - 0.12$). Percent duration of training completed between 255 75-84% HR_{max} was associated with higher RH exposure (4.6%, β = 0.28, p 256 =0.002), but there was no effect of either SR or T_a exposure on the percent 257 duration of training completed between 75-84% HR_{max}. When RH exposure increased, there was an associated increase in percent duration of training being completed above 85% of HR_{max} (3.9%, β = 0.260, *p* <0.001) with a similar result evident with T_a exposure (4.3%, β = 0.287, *p* =0.001). In contrast, the percent duration of training sessions completed above 85% HR_{max} underwent and associated decrease with increasing SR exposure (-2.9%, β = -0.192, *p* <0.001; Figure 2b).

Increasing RH exposure was associated with an increase in RPE (1.8AU, $\beta = 0.968, p < 0.001$). In addition, increased T_a exposure was also associated with an increase in RPE, but this was a small effect (0.3AU, $\beta = 0.153, p$ =0.019). There were no significant relationships between RPE and the level of SR exposure during training sessions.

269 Recovery

270 There was no effect of SR exposure on the BM change from pre- to post-271 session during training, but there was an associated increase in BM loss 272 with increasing RH (-215 g, β = -0.25, *p* <0.001) and T_a (-160 g, β = -0.35, 273 p = 0.026). No effect of any heat stress variable on self-reported Overall 274 Wellness at 24 h (β =-0.198 – 0.003) and 48 h (β = -0.226 – 0.172) was 275 evident. However, increasing RH exposure was associated with higher 276 self-reported stress 24 h post-training (-0.11AU, β = -0.24, p =0.002). 277 Increasing RH exposure was not associated with self-reported recovery for 278 the individual variables of Fatigue, Sleep Quality and Mood (β =-0.24 – 279 0.26). Increasing T_a exposure was associated with a decrease in self-280 reported Sleep Quality 48 h post-training (-0.57AU, β = -0.58, p =0.03) but 281 no other self-reported recovery variable 48 h post-training was associated 282 with heat stress variables (β =-0.15 – 0.39). There was no effect of any heat 283 stress variable on rMSSD 24 h post-training (β = -0.152-0.072; Figure 3a). 284 However, increasing SR exposure was associated with reduced rMSSD 48 285 h post-training (-16.9ms, β = -0.277, p =0.019) but neither RH nor T_a 286 exposure generated any significant effect on rMSSD 48 h post-training (β = 287 -0.129-0.288; Figure 3b).

288

289 Discussion

290 This study aimed to determine the individual effects of T_a, RH and SR 291 during physical preparation for professional team-sport. Our data show the 292 importance of considering the impact of the individual heat stress variables 293 contributing to thermal load in isolation. Specifically, we show for the first 294 time that SR is associated with profound effects on the quality of training 295 in team-sport athletes related to reduced self-paced high intensity work 296 performed during the preparation phase of a competitive season. Our 297 findings also show RH is associated with reductions in the level of high-298 speed running during team-sport training and exerts the largest effect for 299 increasing players rating of perceived exertion. In contrast, increasing T_a 300 was associated with higher work capacity during pre-season training as 301 evidenced by greater external training loads. Accordingly, the novel data 302 from the present study indicates SR and RH each appear to have negative 303 effects on the team-sport athlete, with associated reductions in intermittent 304 high-intensity running capacity and potential to slow recovery between 305 training sessions.

306 Our data are in agreement with the limited number of previous studies 307 investigating the effects of SR on exercise intensity or duration ^{5,22}. Otani 308 and colleagues have previously shown time-to-exhaustion during 309 prolonged endurance exercise is reduced even at moderate SR intensity $(500 \text{ W/m}^2)^{5}$ and that self-selected exercise intensity decreases when 310 exposed to increasing levels of SR in hot environments ²². We extend on 311 312 these findings to show high SR exposure is closely related to impaired 313 high-intensity work capacity during training for a professional team-sport.

314 The associated decrease in high-intensity work performed with increased 315 SR may be related to T_{sk} which is heavily influenced by the external 316 environment³. SR exposure has been shown to increase T_{sk} in a doseresponse manner in thermoneutral ^{23,24} and hot ^{22,25} environments, while 317 318 having little meaningful effect on core temperature ^{5,24}. The athletes in the 319 current study routinely undertook training under SR intensities equivalent 320 to those reported in previous studies showing increased T_{sk}. Consequently, 321 we suggest that elevated T_{sk} with high SR exposure in the present study 322 was likely a primary factor attenuating the capacity for prolonged high

323 intensity, intermittent exercise above that associated with high T_a alone. 324 When T_{sk} increases, the core-to-skin temperature gradient narrows and 325 promotes increases in skin blood flow, a decrease in stroke volume, and 326 compromised cardiac output ²⁶. During self-paced team-sport training that 327 includes repeated high-speed running in hot conditions, the 328 thermoregulatory response limits exercise intensity to reduce metabolic 329 heat production so that levels of compensable heat strain can be maintained ²⁷. While we cannot ascribe cause-and-effect from our data, 330 increased T_{sk}^{28} and thermal perceptions of hot skin ²⁹ has previously been 331 332 associated with a decrease in intensity of aerobic exercise performance. 333 Therefore, we propose that the team-sport athletes in the current study 334 downregulated effort and intensity of work in response to higher T_{sk} with 335 increasing SR exposure.

336 A second major finding of the present study was that RH was also 337 associated with compromised quality of external work and higher internal 338 stress. It is well-established that when RH is low, evaporative cooling is 339 an efficient cooling mechanism and that increasing humidity limits 340 evaporation, as sweat secreted to the skin surface is not easily dissipated 341 to the external environment ³⁰. Moreover, in hot conditions, the body will 342 also gain heat from the environment through radiation and conduction ¹. 343 In the present study, increasing RH exposure was associated with a small-344 to-moderate effect on work performed during training but had the largest 345 effect on an individual's perception of exertion. When considering the 346 typical training environment of the present study was hot and humid, the 347 effect of RH on perceived effort and heart rate may have been expected. 348 However, this effect did not appear to decrease external work output to the 349 same extent as SR, indicating there may be incongruence between external 350 and internal load parameters during preparation for team-sport in hot 351 environments with potential implications for training load monitoring of 352 athletes in the heat.

In contrast to the effects of SR and RH, higher T_a was associated with
increased work performed and internal load during training sessions.
Importantly, an operational construct of the statistical model employed is

Accepted author manuscript version reprinted, by permission, from International Journal of Sports Physiology and Performance, 15(10), 1393-1399, https://doi.org/10.1123/ijspp.2019-0837 © Human Kinetics, Inc. 356 that the effects of an increasing individual heat stress variable are 357 determined within a paradigm where the other heat variables in the model 358 remain constant. Under conditions of moderate-to-high T_a (~30-35 °C), it 359 has been purported that the increased muscle temperature with passive 360 heat exposure may promote improved performance capacity for repeated, high-intensity efforts similar to those undertaken in team-sport training ³¹. 361 362 This phenomenon may, at least in part, explain the association between 363 increasing T_a and running performance in the present study. While the 364 associated increase in heart rate with RH likely reflects the effects of heat 365 strain, the association between elevated heart rate and increased T_a may be 366 related to the greater work performed with increasing T_a. Moreover, high 367 levels of aerobic fitness are closely associated with effective heat 368 mitigation, and together with heat acclimation may represent the primary 369 strategies for enhancing exercise capacity in hot conditions ³². Participants 370 in our study were heat acclimatised professional team-sport athletes with 371 a high level of aerobic fitness, and a well-developed capability to tolerate 372 stressful training environments. As such, it seems reasonable to suggest 373 that increased muscle temperature could be achieved in trained athletes 374 without or in spite of an excessive rise in core temperature, a response that 375 may also be a prerequisite for improved performance of repeated high-376 intensity efforts in the heat ³³. However, it is possible the magnitude of 377 heat stress from increasing T_a exposure in the present study was 378 insufficient to elicit detrimental effects more commonly associated with 379 heat stress protocols in laboratory settings. Moreover, it is unclear if 380 similar responses to increasing T_a exposure would be observed in different 381 athlete cohorts, geographical locations or whilst undertaking different 382 training protocols.

Intuitively, the physiological strain of undertaking high-intensity teamsport training in hot environments would elicit a significant effect on psychometric wellbeing and parasympathetic activity that is reflected in rMSSD ³⁴. Indeed, high-intensity exercise (>50% VO_{2peak}) has been shown to progressively decrease parasympathetic tone ³⁵ with potential to increase subjective feelings of fatigue and decrease sleep quality ³⁵ during recovery from exercise in hot conditions. The lack of association between 390 heat stress variables and most subjective and objective recovery measures 391 after 24 h recovery was unexpected, although there was a modest 392 association between RH and self-reported stress the day after training. 393 However, a recently published study investigating the recovery time-394 course in Australian Football has shown that the stress response appears to reach a peak after ~40 h recovery from competition ³⁶. Our data show a 395 396 small associated decrease in rMSSD 48 h after training with increasing SR 397 exposure, an effect that was not apparent in response to increasing T_a or 398 RH. In addition, T_a was associated with a decrease in self-reported sleep 399 quality 48 h post-training with no other subjective recovery measures 400 associated with heat stress variables. It could be that quantification of 401 acute HRV has limited capacity to detect a meaningful change in the 402 current study and chronic responses analysed over prolonged periods may 403 be more informative ³⁷. Our varied findings may also simply reflect the 404 complexity of interactions in subjective and objective measures of 405 recovery ³⁸, and the most representative sample to detect important 406 relationships is also unknown. Further research is required to determine 407 the relationships between team-sport athlete training load, measures of 408 recovery and heat stress variables, and the effect on athlete well-being 409 during preparation for competition.

410 Practical Applications

411 Measuring environmental parameters individually may be more sensitive 412 when assessing the magnitude of heat stress during team-sport training and 413 recovery. Moreover, heat management strategies should not be limited to 414 competition but should include the physical preparation period if 415 maximising the quality and quantity of work completed within team-sport 416 training is desired.

417 Conclusion

In conclusion, our data is the first to show the effects of individual heat
stress variables on team-sport training and the associated acute negative
effects on physical performance and recovery parameters with exposure to
increasing SR and RH. Indeed, we have demonstrated these effects in heat-

acclimatised athletes indicating that heat acclimation alone may be unable
to ameliorate reduced acute detrimental responses associated with teamsport training undertaken in hot conditions. Given the reality of a warming
climate and the increased prevalence of environmental extremes in many
geographical locations, mitigating the effects of heat for team-sport
athletes will be increasingly important.

428

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432 Conflict of interest: Fergus O'Connor, Steven Stern, Thomas Doering,
433 Geoffrey Minett, Peter Reaburn, Jonathan Bartlett, and Vernon Coffey
434 declare no direct or indirect conflicts of interest relevant to the content of
435 this study.

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Heat Stress Variable	Weeks 1-8 Average	Weeks 1-8 Median	Weeks 1-8 Range
Ambient Temperature (°C)	30.9 ± 2.1	31.4	26.7 to 34.4
Relative Humidity (%)	61.7 ± 6.2	61.2	52 to 75
Solar Radiation (W/m ²)	718 ± 224	789	239 to 1001
Wind Speed (Kph)	4.0 ± 1.3	4.1	1.9 to 6.5
WBGT (°C)	29.0 ± 2.5	29.6	24.4 to 32.9

Table 1. Environmental conditions during the eight-week data collection period. Data are mean \pm standard deviation (n=20 training sessions).

Table 2. Training session load quantified via GPS, electronic heart monitoring and participant ratings of exertion during the eight-week experimental period for players preparing for Australian Football competition (n=45). Data are mean \pm standard deviation

Training Variable	Weeks 1-8	Median	Range
Session Duration (min)	70.8 ± 24.1	60.4	26.7 to 97.7
Session Distance covered (m)	7323 ± 2853	6417	26.4 to 10197
Average speed (m.min ⁻¹)	102.7 ± 21.1	102.9	65.3 to 210.9
High-Speed Running (%)	25.0 ± 10.6	26.7	7.1 to 39.6
Mean HR (bpm)	155.7 ± 10.7	154.6	140.4 to 163.3
Time >85% HR _{max} (%)	16.9 ± 15.1	15.5	16.4 to 53.6
RPE (AU)	6.4 ± 1.9	6.8	2.7 to 9.0
rMSSD+1 (ms)	140 ± 95	135.7	60.3 to 183.4
rMSSD +2 (ms)	140 ± 95	136.6	60.3 to 183.4

HR, heart rate; RPE, rating of perceived exertion; rMSSD, root mean square of the successive differences.



Figure 1 — Standardized coefficient relationships between (A) distance covered (in m/min) and heat-stress variables and (B) %HSR and heat-stress variables during an 8-week experimental period for players preparing for Australian Football competition (N = 45; 20 training sessions). SR indicates solar radiation; RH, relative humidity; T_a, ambient temperature; %HSR, percentage high-speed running. *P < .05. **P < .01. ***P < .001.







Figure 3 — Standardized coefficient relationship between heart-rate variability (rMSSD) and heat-stress variables following (A) 24- and (B) 48-hour recovery from training sessions during an 8-week experimental period for players preparing for Australian Football competition (N = 45; 20 training sessions). SR indicates solar radiation; RH, relative humidity; T_a , ambient temperature; rMSSD, root mean sum of the squared differences. *P < .05.