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

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Abstract

**Purpose:** Exercise in hot environments increases body temperature and thermoregulatory strain. However, little is known regarding the magnitude of effect that ambient temperature (T<sub>a</sub>), relative humidity (RH) and solar radiation (SR) individually have on team sport athletes. We aimed to determine the effect of these individual heat stress variables on team-sport training performance and recovery. **Methods:** Professional Australian Rules Football (ARF) players (n=45) undertook eight-weeks pre-season training producing a total of 579 outdoor field-based observations with T<sub>a</sub>, RH and SR recorded at every training session. External load (distance covered, m.min<sup>-1</sup>; percent high speed running >14.4 km.h<sup>-1</sup>; %HSR) was collected via a global positioning system. Internal load (ratings of perceived exertion (RPE), heart rate (HR)), and recovery (subjective ratings of wellbeing and heart rate variability (rMSSD)) were monitored throughout the training period. Mixed effects linear models analysed relationships between variables using standardised regression coefficients.

**Results:** Increasing SR exposure was associated with reduced distance covered (-19.7 m.min<sup>-1</sup>, β=-0.909, p<0.001), %HSR (-10%, β=-0.953, p<0.001) during training, and rMSSD 48 h post-training (-16.9ms, β=-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%, β=0.260, p<0.001), RPE (1.8AU, β=0.968, p<0.001) and self-reported stress 24 h post-training (-0.11AU, β=-0.24, P=0.002). In contrast, higher Ta was associated with increased distance covered (19.7 m.min<sup>-1</sup>, β=0.911, p<0.001) and %HSR (3.5%, β=0.338, p=0.005). **Conclusions:** We show the importance of considering the individual factors contributing to thermal load in isolation for team sport athletes, and that SR and RH reduce work capacity during team sport training and have potential to slow recovery between sessions.
Introduction

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

The vast majority of previous research on thermoregulation during exercise in hot environments has focused on the endurance athlete, and there is a paucity of available data examining the effect of exercise in the heat on the team-sport athlete \(^6\). Some previous studies have examined physiological changes to heat stress in team sport players, including characterizing responses during match play at various wet bulb globe temperatures \(^7\). Many team-sports undertake physical preparation outdoors during summer months at geographical locations characterised by high \(T_a\), RH and SR \(^6\). Repeatedly training in hot environments may promote long-term adaptations such as improved sweat response and decreased cardiovascular strain with the potential to convey a benefit for team sport performance \(^8\). Conversely, the acute thermoregulatory responses to exercise in hot conditions may attenuate physical work capacity and the quality of training sessions, while frequent heat exposure during high-
intensity intermittent team sport training may lead to overreaching and adverse effects on athlete wellness and performance. Understanding the physiological effects of recurring exposure to heat stress during training or competition is necessary to optimise physical preparation and well-being in team sport athletes. Moreover, there is currently no data on varying environmental conditions that consider the individual effects of T_a, RH and SR on external load, internal load and recovery from intermittent, high intensity team-sport training activities. Thus, the primary aim of this study was to determine the individual effects of T_a, RH and SR on professional team-sport training sessions undertaken in hot conditions. Our secondary aim was to establish the individual effect of these heat variables on recovery during the 48 h period following training bouts. We hypothesized that with increasing exposure to each individual heat stress variable, external work completed during training would decrease concomitant with higher internal loads and impaired recovery.

Materials and Methods

Participants

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

Experimental Protocol

Internal and external training loads were captured throughout an eight-week training period of the pre-season preparation phase spanning January and February of the Australian summer. Upon removal of ‘stationary skill’ 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.

**External training load**

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

**Internal training load**

Ratings of perceived exertion (RPE) were obtained 10-30 min following the completion of each training session using Borg’s CR-10 scale. Heart Rate (HR) data was collected via chest strap HR monitors (T34, Polar Electro, Espoo, Finland). HR data was analysed by quantifying the percent of total duration within specific ‘zones’ (Zone 3 = 65-74%, Zone 4 = 75-84%, Zone 5 = >85% HRmax).

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. Objective measures of heart-rate variability (HRV) were assessed upon waking each morning by R-R series recording via photoplethysmography.
using a valid and reliable, commercially available smartphone application (HRV4Training)\textsuperscript{14}. HRV data was subsequently analysed for the root mean sum of the squared differences (rMSSD) between each successive heartbeat on recovery days +1 (24 h) and +2 (48 h) after training sessions, and compared to rolling baseline data obtained from a minimum of four readings in the prior 7-days\textsuperscript{15}. rMSSD was chosen as the HRV variable of interest due to the relationship with vagal activity\textsuperscript{16} and greater reliability compared to other spectral indices\textsuperscript{17}.

**Environmental Monitoring**

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

**Statistical Analyses**

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

Relationships between internal and external load and the training environment were analysed using mixed effects linear models via the *Lme4* package in *R Studio* statistical computing software (V. 1.1.442). Mixed-effect linear models were applied to training and recovery variables incorporating the individual as a random effect and heat stress variables as fixed effects using the equation:
\[ y_i = \beta_1 x_{1i} + \beta_2 x_{2i} + \beta_3 x_{3i} + b_i + \epsilon_i \]

where \( y_i \) is the value of the outcome variable of interest \( i \), \( \beta_1 \) through \( \beta_3 \) are fixed effect coefficients, \( x_{1i} \) through \( x_{3i} \) are fixed effect variables for observation \( i \), \( b_i \) is the random effects assumed to be multivariate normally distributed, and \( \epsilon_i \) is the error for case \( i \) which is also assumed to be multivariate normally distributed. Normality assumptions were validated using residual and QQ-plots, and the adequacy of the model structures was determined via residual plots and quantified using standard measures of intraclass correlations and coefficients of determination. All variables of interest in the model are reported using standardised regression coefficients (standardised beta (\( \beta \))), allowing assessment of practical significance. Standardised regression coefficients for each variable were multiplied by the standard deviation of the change in dependent variable to obtain the absolute change in the units of measurement. Qualitative descriptors for reporting of standardised beta scores were adopted using a comparable approach to effect size statistics to interpret the magnitude of the association between heat stress, training and recovery variables. We interpreted the standardised beta using threshold values of 0.2 as a small effect, 0.5 as a moderate effect, and 0.8 as a large effect. Multicollinearity between heat stress variables was established through Spearman’s correlation matrix analysis and in the event that any variables had a multicollinearity \( r > 0.8 \) they were excluded from the model.

**Results**

**Environmental Conditions**

Mean environmental conditions during the experimental period were 30.9 ± 2.1 °C Ta (Range: 26.7 to 34.4 °C), 61 ± 6% RH (Range: 52 to 75 %) and 718 ± 224 W/m² SR (Range: 239 to 1001 W/m²). Multicollinearity did not exist between heat stress variables. Wet-bulb globe temperature (WBGT) during the experimental period was 29 ± 2.5 °C (Range: 24.4 to 32.9 °C). There was no significant difference in variables of environmental...
conditions (Table 1) or training load (Table 2) between training sessions (n=20) throughout the eight-week experimental period.

Insert Table 1 Here.

Insert Table 2 Here.

**External Training Load**

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 relationship between changes in RH and m.min\(^{-1}\) at each training session (Figure 1a). In contrast, increasing \(T_a\) exposure was associated with an increase in distance covered (19.7 m.min\(^{-1}\), β= 0.911, \(p < 0.001\)). There were divergent effects on the change in %HSR completed during training sessions by each of the individual heat stress variables (Figure 1b). Specifically, an increase in SR exposure was associated with a large decrease in %HSR completed during training (-10%, β= -0.953, \(p < 0.001\)). 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 \(T_a\) exposure during training was related to a small increase in percent %HSR (3.5%, β= 0.338, \(p=0.005\)) (Figure 1b).

**Internal Training Load**

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

Increasing RH exposure was associated with an increase in RPE (1.8AU, $\beta=0.968$, $p<0.001$). In addition, increased T\textsubscript{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.

**Recovery**

There was no effect of SR exposure on the BM change from pre- to post-session during training, but there was an associated increase in BM loss with increasing RH (-215 g, $\beta=-0.25$, $p<0.001$) and T\textsubscript{a} (-160 g, $\beta=-0.35$, $p=0.026$). No effect of any heat stress variable on self-reported Overall Wellness at 24 h ($\beta=-0.198-0.003$) and 48 h ($\beta=-0.226-0.172$) was evident. However, increasing RH exposure was associated with higher self-reported stress 24 h post-training (-0.11AU, $\beta=-0.24$, $p=0.002$). Increasing RH exposure was not associated with self-reported recovery for the individual variables of Fatigue, Sleep Quality and Mood ($\beta=-0.24-0.26$). Increasing T\textsubscript{a} exposure was associated with a decrease in self-reported Sleep Quality 48 h post-training (-0.57AU, $\beta=-0.58$, $p=0.03$) but no other self-reported recovery variable 48 h post-training was associated with heat stress variables ($\beta=-0.15-0.39$). There was no effect of any heat stress variable on rMSSD 24 h post-training ($\beta=-0.152-0.072$; Figure 3a).

However, increasing SR exposure was associated with reduced rMSSD 48 h post-training (-16.9ms, $\beta=-0.277$, $p=0.019$) but neither RH nor T\textsubscript{a} exposure generated any significant effect on rMSSD 48 h post-training ($\beta=-0.129-0.288$; Figure 3b).

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

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

The associated decrease in high-intensity work performed with increased SR may be related to $T_{sk}$ which is heavily influenced by the external environment\textsuperscript{3}. SR exposure has been shown to increase $T_{sk}$ in a dose-response manner in thermoneutral\textsuperscript{23,24} and hot\textsuperscript{22,25} environments, while having little meaningful effect on core temperature\textsuperscript{5,24}. The athletes in the current study routinely undertook training under SR intensities equivalent to those reported in previous studies showing increased $T_{sk}$. Consequently, we suggest that elevated $T_{sk}$ with high SR exposure in the present study was likely a primary factor attenuating the capacity for prolonged high
intensity, intermittent exercise above that associated with high $T_a$ alone. When $T_{sk}$ increases, the core-to-skin temperature gradient narrows and promotes increases in skin blood flow, a decrease in stroke volume, and compromised cardiac output. During self-paced team-sport training that includes repeated high-speed running in hot conditions, the thermoregulatory response limits exercise intensity to reduce metabolic heat production so that levels of compensable heat strain can be maintained. While we cannot ascribe cause-and-effect from our data, increased $T_{sk}$ and thermal perceptions of hot skin has previously been associated with a decrease in intensity of aerobic exercise performance. Therefore, we propose that the team-sport athletes in the current study downregulated effort and intensity of work in response to higher $T_{sk}$ with increasing SR exposure.

A second major finding of the present study was that RH was also associated with compromised quality of external work and higher internal stress. It is well-established that when RH is low, evaporative cooling is an efficient cooling mechanism and that increasing humidity limits evaporation, as sweat secreted to the skin surface is not easily dissipated to the external environment. Moreover, in hot conditions, the body will also gain heat from the environment through radiation and conduction. In the present study, increasing RH exposure was associated with a small-to-moderate effect on work performed during training but had the largest effect on an individual’s perception of exertion. When considering the typical training environment of the present study was hot and humid, the effect of RH on perceived effort and heart rate may have been expected. However, this effect did not appear to decrease external work output to the same extent as SR, indicating there may be incongruence between external and internal load parameters during preparation for team-sport in hot environments with potential implications for training load monitoring of 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
that the effects of an increasing individual heat stress variable are
determined within a paradigm where the other heat variables in the model
remain constant. Under conditions of moderate-to-high $T_a$ ($\sim$30-35 °C), it
has been purported that the increased muscle temperature with passive
heat exposure may promote improved performance capacity for repeated,
high-intensity efforts similar to those undertaken in team-sport training $^{31}$. This phenomenon may, at least in part, explain the association between
increasing $T_a$ and running performance in the present study. While the
associated increase in heart rate with RH likely reflects the effects of heat
strain, the association between elevated heart rate and increased $T_a$ may be
related to the greater work performed with increasing $T_a$. Moreover, high
levels of aerobic fitness are closely associated with effective heat
mitigation, and together with heat acclimation may represent the primary
strategies for enhancing exercise capacity in hot conditions $^{32}$. Participants
in our study were heat acclimatised professional team-sport athletes with
a high level of aerobic fitness, and a well-developed capability to tolerate
stressful training environments. As such, it seems reasonable to suggest
that increased muscle temperature could be achieved in trained athletes
without or in spite of an excessive rise in core temperature, a response that
may also be a prerequisite for improved performance of repeated high-
intensity efforts in the heat $^{33}$. However, it is possible the magnitude of
heat stress from increasing $T_a$ exposure in the present study was
insufficient to elicit detrimental effects more commonly associated with
heat stress protocols in laboratory settings. Moreover, it is unclear if
similar responses to increasing $T_a$ exposure would be observed in different
athlete cohorts, geographical locations or whilst undertaking different
training protocols.

Intuitively, the physiological strain of undertaking high-intensity team-
sport 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% $VO_2$peak) 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
heat stress variables and most subjective and objective recovery measures after 24 h recovery was unexpected, although there was a modest association between RH and self-reported stress the day after training. However, a recently published study investigating the recovery time-course in Australian Football has shown that the stress response appears to reach a peak after ~40 h recovery from competition. Our data show a small associated decrease in rMSSD 48 h after training with increasing SR exposure, an effect that was not apparent in response to increasing T_a or RH. In addition, T_a was associated with a decrease in self-reported sleep quality 48 h post-training with no other subjective recovery measures associated with heat stress variables. It could be that quantification of acute HRV has limited capacity to detect a meaningful change in the current study and chronic responses analysed over prolonged periods may be more informative. Our varied findings may also simply reflect the complexity of interactions in subjective and objective measures of recovery, and the most representative sample to detect important relationships is also unknown. Further research is required to determine the relationships between team-sport athlete training load, measures of recovery and heat stress variables, and the effect on athlete well-being during preparation for competition.

**Practical Applications**

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

**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 team-
sport 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.

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


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

<table>
<thead>
<tr>
<th>Heat Stress Variable</th>
<th>Weeks 1-8 Average</th>
<th>Weeks 1-8 Median</th>
<th>Weeks 1-8 Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ambient Temperature (°C)</td>
<td>30.9 ± 2.1</td>
<td>31.4</td>
<td>26.7 to 34.4</td>
</tr>
<tr>
<td>Relative Humidity (%)</td>
<td>61.7 ± 6.2</td>
<td>61.2</td>
<td>52 to 75</td>
</tr>
<tr>
<td>Solar Radiation (W/m²)</td>
<td>718 ± 224</td>
<td>789</td>
<td>239 to 1001</td>
</tr>
<tr>
<td>Wind Speed (Kph)</td>
<td>4.0 ± 1.3</td>
<td>4.1</td>
<td>1.9 to 6.5</td>
</tr>
<tr>
<td>WBGT (°C)</td>
<td>29.0 ± 2.5</td>
<td>29.6</td>
<td>24.4 to 32.9</td>
</tr>
</tbody>
</table>

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 ± standard deviation

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

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; Ta, ambient temperature; %HSR, percentage high-speed running. *P < .05. **P < .01. ***P < .001.
Figure 2 — Standardized coefficient relationship between (A) HR_{mean} and heat-stress variables and (B) percentage duration above 85% of maximal heart rate and heat-stress variables during an 8-week experimental period for players preparing for Australian Football competition (N = 45; 20 training sessions). HR_{mean} indicates mean heart rate; SR, solar radiation; RH, relative humidity; T_a, ambient temperature. ***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; Ta, ambient temperature; rMSSD, root mean sum of the squared differences. *P < .05.