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Physiological strain under different wet bulb temperatures during daylong humid heat exposure in young men

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ABSTRACT

The increasingly severe humid heatwaves worldwide and associated heat stress are posing a growing threat to human health and survival, especially for vulnerable populations who cannot access air conditioning and may experience prolonged heat exposure. This study aimed to evaluate the physiological responses, thermal perceptions and blood inflammatory cell counts during daylong hot-humid exposure. Twelve male participants underwent 8 h exposure under three conditions: thermoneutral environment (26 °C, 50 % RH) as the Control condition, as well as low-level (33.5 °C, 52 % RH) and high-level (36.5 °C, 57 % RH) humid heat environments with wet bulb temperatures of 25.5 °C and 29.0 °C, respectively. During the trials, participants performed sedentary office work. Body temperatures, heart rate, blood pressure, body fluid status, blood cell counts and thermal perceptions were measured. The results indicated that 8 h of high-level exposure elicited significantly increased core temperature (37.7 ± 0.3 °C) and heart rate (102 ± 16 bpm), with decreased mean arterial pressure (67 ± 5 mmHg), noticeable body weight loss (−1.65 % ± 0.67 %) as well as excessively elevated counts of leukocytes and neutrophils. These findings suggested that subjects experienced cardiovascular stress, dehydration and inflammatory responses after the exposure. By contrast, 8 h of low-level exposure caused increases in core temperature (37.3 ± 0.1 °C) and mean skin temperature (35.4 ± 0.3 °C), with mild body weight loss (−1.08 % ± 0.60 %). This study is expected to provide empirical evidence for public health practice and heat protection advice, helping to reduce the potential health risks of hot weather.

1. Introduction

Global ever-rising temperature has inevitably caused a rapid increase in the scale, intensity, frequency and duration of extreme heat events, posing a significant threat to human health [1,2]. According to the World Meteorological Organization (WMO) [3], the global average near-surface temperature from January to September in 2024 was 1.54 °C (± 0.13 °C) higher than the pre-industrial baseline. It is predicted that by the end of the century, the percentage of land areas and human population exposed to deadly humid heatwaves are expected to be 76-times higher than that under a 1.5 °C warming level [4,5]. Furthermore, heatwave events and rapid urbanization have intensified the urban heat island effect, thereby increasing the frequency of hot weather in densely urbanized areas and exacerbating the adverse impacts of heat on human health compared to rural areas [6,7]. Air conditioning is the most widely adopted strategy worldwide to cope with hot weather. However, many of the most vulnerable populations, who face the greatest health risks from heatwaves, cannot afford air conditioning. In addition, unplanned power outages or power restrictions become inevitable due to the sharp rise in energy consumption during heatwaves [8,9], thereby leading to surges in heat-related mortality.

In hot-humid environments, the combination of increased heat production and impaired evaporative cooling significantly increases the risk of heat stroke compared to dry heat environments [10]. The accumulated heat load initially raises core body temperature, which then activates increased cardiac output, vasodilate skin blood vessels, and stimulate sweat gland secretion [11,12]. Meanwhile, if not replenished by adequate fluid intake during exposure, sweat-related fluid losses will lead to dehydration, thereby reducing effective blood volume and cardiac output [12]. Moreover, the decrease in total blood volume

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combined with increase in skin blood flow further lowers central venous pressure and splanchnic blood flow. If the normal thermoregulatory mechanism is overwhelmed, the core temperature will rise to an uncompensatable level, accompanied by subsequent circulatory failure, endotoxin leakage, and systemic inflammatory response [11–13]. Changes in infection or inflammatory processes associated with prolonged heat exposure are typically characterized by elevated concentrations of immune cells (e.g., leukocytes) [13,14] or systemic inflammatory markers (e.g., interleukins) [13–17], which are also critical indicators of heat stroke progression.

Related studies on dry heat exposure indicated that older adults experienced greater increases in heat storage and core temperature than younger adults, with a significant rise in interleukin-6 (IL-6) observed only in older adults, suggesting that older adults may be more susceptible to heat-induced inflammation [15–17]. However, the environmental conditions used in these studies are hot dry environments (40 °C, 9 % RH) with extremely low humidity, which are not applicable to humid heat environments. The prolonged exposure to humid heat environments still warrants further investigation.

Wet-bulb temperature (T_w) is a thermal stress index based on the combination of temperature and humidity, widely used to assess the health effects of humid heat [4,18]. Sherwood and Huber [19] first proposed a T_w of 35 °C as the theoretical survivability limit for humans over six-hour exposure, without considering the physiological principles of thermoregulation, particularly the differences in the limitations of evaporative heat dissipation in extremely humid versus dry environments. Recent studies [20–22] have shown that the T_w associated with uncompensable heat stress is far below this threshold and the livability environmental limit dose not correspond to a fixed T_w under different environmental conditions and activity levels. Studies from the PSU HEAT Project [20,21] used progressive heat stress methods to examine the critical environmental limits during minimal physical activity, above which human thermoregulatory capacity was exceeded and core temperature progressively increased. They revealed that critical T_w varied from 26–31 °C, with higher limits associated with warm-humid environments and lower values noted in hot-dry climates. Likewise, physiological model research from Xu et al. [22] indicated that for any given air temperature, the critical T_w limit was dependent on the humidity, highlighting the importance of considering both ambient temperature and humidity. Therefore, the combination of ambient temperature and humidity is suggested to be more accurate for assessing heat stress and livability levels. Noteworthy, previous studies mainly aimed to determine T_w thresholds based on progressive heat stress tests lasting 1 to 2 hours or physiological model prediction approaches. Few studies have explored the long-term effects of humid heat exposure on human health under real-world meteorological conditions.

The adaptability threshold of T_w sets the upper limit for humid heat environments related to human health. However, there is no internationally unified standard for defining the lower limit of hot humid weather. In this context, the Chinese Meteorological Industry Standard (QX/T 598–2021) [23] defines 'sauna weather' as conditions characterized by high temperatures, high humidity, and low wind speeds, creating a sauna-like environment. Sauna weather is defined as conditions of daily maximum temperature ≥ 33.5 °C, daily mean humidity ≥ 60 % RH and maximum wind force ≤ grade 3. Compared to T_w upper limit, sauna weather conditions define the lower limit of hot humid environments. The effects of prolonged exposure to these moderately hot-humid environments on human health remain largely unexplored, highlighting the need for further research.

As discussed above, the present study aimed to reveal the effects of 8-hour exposure to extreme and moderate humid heat environments on thermoregulatory responses and subjective perceptions in young healthy adults. Representative conditions of high-level (T_w=29.0 °C with 36.5 °C and 57 % RH) and low-level (T_w = 25.5 °C with 33.5 °C and 52 % RH) environments were accordingly selected for heat exposure simulation, based on the meteorological data of Shanghai, China, in summer 2024. It is important to note that these conditions reflected daily averages rather than daily maximums. Moreover, the thermoneutral environment (26 °C and 50 % RH) was set as the Control condition. We hypothesized that after 8 h of high-level humid heat exposure, there would be a significantly elevated core temperature and noticeable hypotension, along with increases in blood inflammatory cell counts. However, we also hypothesized that 8 h of low-level humid heat exposure would elicit an increase in core temperature, but this increase might not be sufficient to induce cardiovascular strain and inflammatory response.

2. Methods

2.1. Participants

An a priori power analysis was performed using G*Power Version 3.1.9.6 (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany), based on conventional values for α (0.05) and β (0.80), and an effect size of 0.8 reported by Mündel et al. for IL-6 as a primary outcome in heat stress versus temperate environments [24]. The point biserial correlation model, which is appropriate for estimating the effect of a continuous predictor (T_{core}) on a categorical outcome (condition) in a repeated-measures design, was used for power calculation, indicating a minimum of six participants would be required to detect a significant effect. However, to enhance statistical robustness and account for inter-individual variability, a larger sample size was included. The power analysis was conducted for the within-subjects main effect of condition in repeated-measures ANOVA, ensuring sufficient power to detect differences across the three conditions (thermoneutral, low-level humid heat condition, and high-level humid heat condition). Additionally, post hoc pairwise comparisons were planned to examine specific differences between conditions. Twelve non-acclimatized healthy male adults participated in this study (mean±SD: age 20.6 ± 1.4 years, height 176.7 ± 6.2 cm, body mass 61.6 ± 5.6 kg, body surface area 1.76 ± 0.09 m^2, body mass index 19.75 ± 1.74 kg/m^2). There was no history of smoking, chronic diseases (hypertension, heart disease, diabetes) or heat-related illnesses among any of the participants. Before the trials, all participants received a detailed explanation of the procedures and signed an informed consent form. This study was approved by a local human ethics committee (XUST-IRB224002) and was conducted in accordance with the Declaration of Helsinki. The recruitment was open to both males and females. However, only two female participants initially volunteered,but both withdrew after the trial began. Further efforts were made to recruit additional female participants, but no more females volunteered.

2.2. Hot-humid environmental conditions

According to the China Weather Network, the mean surface temperature in China during the summer of 2024 reached a record high for the same period, with Shanghai ranking at the top for sauna weather days among cities during the summer of 2024 [23,25]. Therefore, Shanghai was selected as the target city for analyzing humid heatwave conditions. Given that in the absence of solar radiation, indoor T_w is comparable to that outdoors during extreme heat events [26,27], station-level surface temperature (T_a) and relative humidity (RH) were used for selecting environmental conditions. Our analysis focused on the months from June to September, because this period corresponds to the heatwave season in Shanghai and other southern cities of China. Daily T_a and RH for Shanghai during this period were obtained from the timeanddate website [28]. By calculating and analyzing daily maximum T_w (Table A1 in Appendix A) [18], it was found that there were three consecutive days (July 20th to July 22nd) where the daily maximum T_w exceeded 29.0 °C, with July 21st peaking at a daily maximum T_w of 29.8 °C, indicating the occurrence of a humid heatwave [4,29]. The meteorological condition of July 21st, 2024 represented the most extreme

hot-humid environments in the past 30 years (Fig. A1 in Appendix A) [28,30]. In addition, this condition is similar to the critical T_w limits for light physical activity proposed by Vecellio et al. [20], and comparable to those observed in several heat events worldwide, such as the 1995 Chicago heatwave (T_w = 28.8 °C) [31], the 2010 Russian heatwave (T_w=28 °C) [32], as well as the 2024 Saudi Arabia heatwave (T_w = 29.0 °C) [33]. Further analysis of T_w diurnal variations (Table A1 and Fig. A2) indicated that T_w is generally higher between 10 a.m. to 6 p.m. Therefore, to replicate the duration of peak periods of T_w, the average T_a and RH (36.5 °C, 57 % RH) between 10 a.m. and 6 p.m. on July 21st, corresponding to a mean T_w of 29.0 °C, was selected as the high-level humid heat condition in this study.

A total of 59 days from June to September 2024 were found to conform to the definition of sauna weather [23]. By comparing the mean T_a, RH and T_w between 10 a.m. to 6 p.m. for these days (Table A2 in Appendix A), condition of 33.5 °C and 52 % RH with a minimum T_w of 25.5 °C corresponding to the weather in Shanghai on September 3rd, was selected as the low-level hot humid environment. For comparison, the thermoneutral environment (26 °C and 50 % RH) was selected as the Control (CON) condition. The environmental conditions for the 8 h exposure are listed in Table 1. It is important to note that the selected values of 29.0 °C and 25.5 °C represent the average T_w between 10 a.m. and 6 p.m. on specific days, rather than the daily maximum T_w.

All participants underwent three 8 h heat exposure trials under different environmental conditions. All trials were conducted in a counterbalanced, randomized order at the same time each day (between 8 a.m. and 5 p.m.). Successive trials were separated by at least four days to prevent heat acclimation and ensure adequate recovery.

2.3. Experimental procedures

All trials were conducted in autumn (from late September to late October) when the ambient temperature was around 20–25 °C with 30 % RH. These conditions were significantly milder compared to those examined in this study. Participants were instructed to abstain from any form of strenuous exercise, taking medicines, intake of tea, coffee or alcohol for 24 h before each trial. They were also asked to consume at least 500 mL of water the night before and on the morning of each test to ensure proper hydration, verified by measuring urine specific gravity (USG) upon arrival at the laboratory. Throughout all trials, participants wore typical summer clothing (short-sleeved shirt, shorts and sneakers).

Fig. 1 illustrates an overview of the detailed procedure and measurements of the daylong heatwave simulation. Upon arrival at the laboratory, a small urine sample was collected and measured to ensure that the USG was less than 1.020 [34]. If this threshold was exceeded, participants were instructed to consume 500 mL of water and the USG was retested after 30 min. Then they were instructed to remain sedentary for 20 min in the preparation room to stabilize their heart rates and body temperatures. After that, a fingertip blood sample was collected, and nude body weight (BW) was measured. Then they self-inserted a rectal thermistor (10 cm past the anal sphincter) for continuous core temperature (T_{core}) monitoring and were outfitted with iButton temperature sensors and a heart rate belt to record skin temperature (T_{sk}) and heart rate (HR). Following that, participants entered the climate chamber for the 8 h heat exposure.

During the trials, participants were restricted to performing normal office work while seated. To prevent excessive dehydration and electrolyte imbalance due to substantial sweating during sustained exposure [35], they were provided with 2.5 mL/kg body mass of an electrolyte beverage every hour (containing 2.5 g of carbohydrates, 20 mg of sodium and 0.4 mg of zinc per 100 mL). The electrolyte beverage was maintained at 20 °C in an insulated thermos, similar to the temperature of tap water [36]. In addition, they were allowed to wipe the sweat with a towel as needed. Approximately 3 h after the heat exposure began, they consumed a hamburger (491 kcal). At the end of the exposure, participants provided another urine sample, had their nude body weight measured again, and underwent another fingertip blood sample collection. Rectal and skin temperatures as well as heart rate were continuously measured, with subjective perception responses recorded every 30 min and blood pressure recorded every 60 min.

Table 1
Daylong environmental conditions.

No.	Ambient temperature (°C)	Relative humidity (%)	Air velocity (m/s)	Wet bulb temperature (°C)
1	36.5	57 %	≤ 0.15	29.0
2	33.5	52 %	≤ 0.15	25.5
3	26.0	50 %	≤ 0.15	18.8

2.4. Measurements and calculations

2.4.1. Body temperatures

Core temperature (T_{core}) was measured continuously using a rectal thermistor (91,428–05, DigiSense, USA) with a sampling interval of 1 min and an accuracy of ± 0.1 °C. Local skin temperatures were recorded by iButton data-loggers (DS1922L, Maxim Integrated, San Jose, CA) placed on four sites (chest, forearm, thigh, and calf), with a sampling interval of 30 s and an accuracy of ± 0.1 °C. The mean skin temperature (T_{sk}) was calculated based on the following equation [37]:

$$T_{sk} = 0.3 \times (T_{chest} + T_{forearm}) + 0.2 \times (T_{thigh} + T_{calf}) \quad (1)$$

2.4.2. Cardiovascular function

Heart rate (HR) was continuously monitored every minute using a heart rate belt (Polar H10, Polar Electro Oy, Kempele, Finland). The brachial artery blood pressure was measured manually using a blood pressure monitor (U703, OMRON, Dalian, China), in duplicate every hour by the same experienced operator. Mean arterial pressure (MAP) was calculated by the systolic blood pressure (SBP) and diastolic blood pressure (DBP), which is described as

$$MAP = (SBP + DBP \times 2)/3 \quad (2)$$

2.4.3. Blood measures

Capillary blood samples were collected before and after the exposure for blood cell counts using an automatic haematology analyser (DF50CRP, Dymind Inc., Shenzhen, China). Values for leukocytes and neutrophils were analyzed. All markers were corrected for plasma volume change from baseline to the end of exposure, which was estimated from haemoglobin and haematocrit [38].

2.4.4. Body fluid status

Nude body weight (BW) was measured using a high-precision electronic weighing scale (CB 2.2–100, PESA., Switzerland, accuracy: ± 2 g) before and following each exposure simulation. The weights of consumed liquid and food during the exposure were measured using an electronic balance (ME104, Mettler Toledo, USA, accuracy: ± 0.1 mg). The amount of urine was calculated through weight changes before and after urination. Average sweating rate was indicated as the average hourly change in body weight corrected for the fluid and food consumption as well as the urination. USG was measured using a digital optical refractometer (HT312ATC, Zhongyuan, Dezhou, China) before and after the exposure.

2.4.5. Perceptions

Participants' perceptual responses were collected using an online questionnaire every 30 min throughout the exposure, including thermal comfort, thermal sensation, skin wetness sensation and thirst sensation. The voting scales for thermal perceptions are summarized in Fig. 2 [39–42].

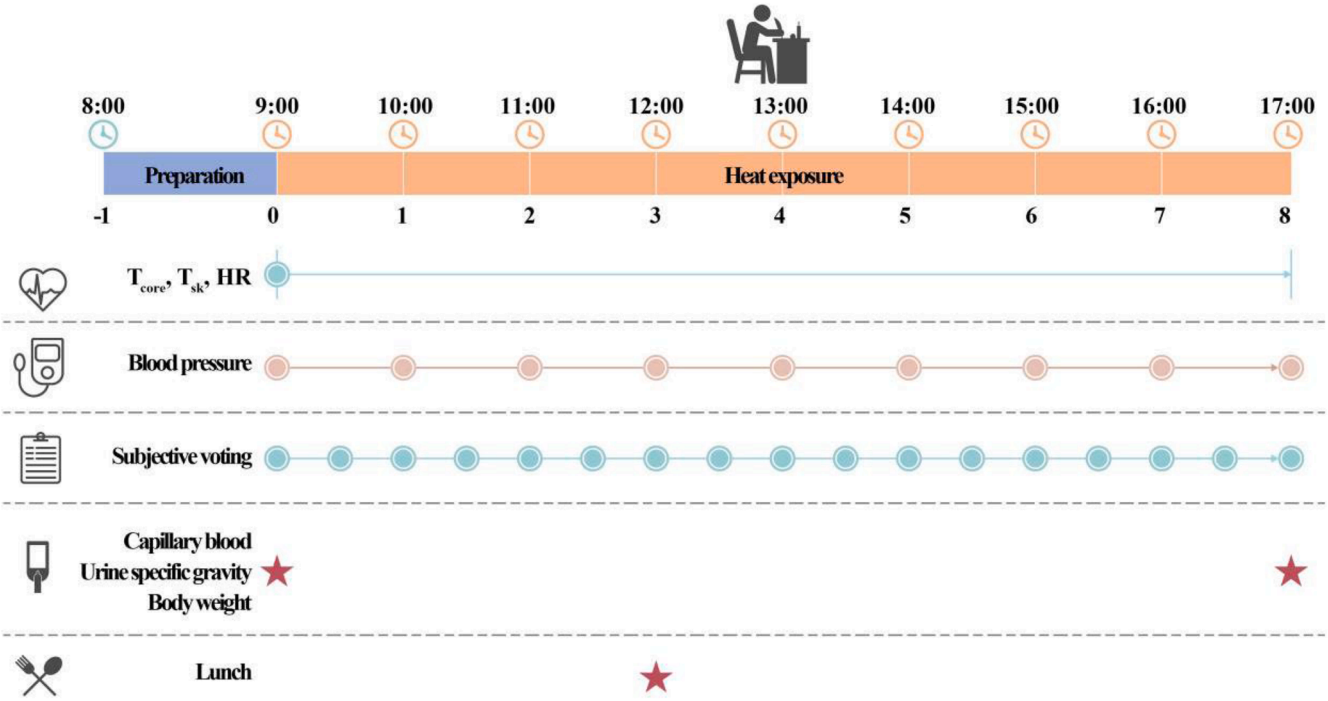

Fig. 1. Overview of the daylong humid heat exposure simulation.

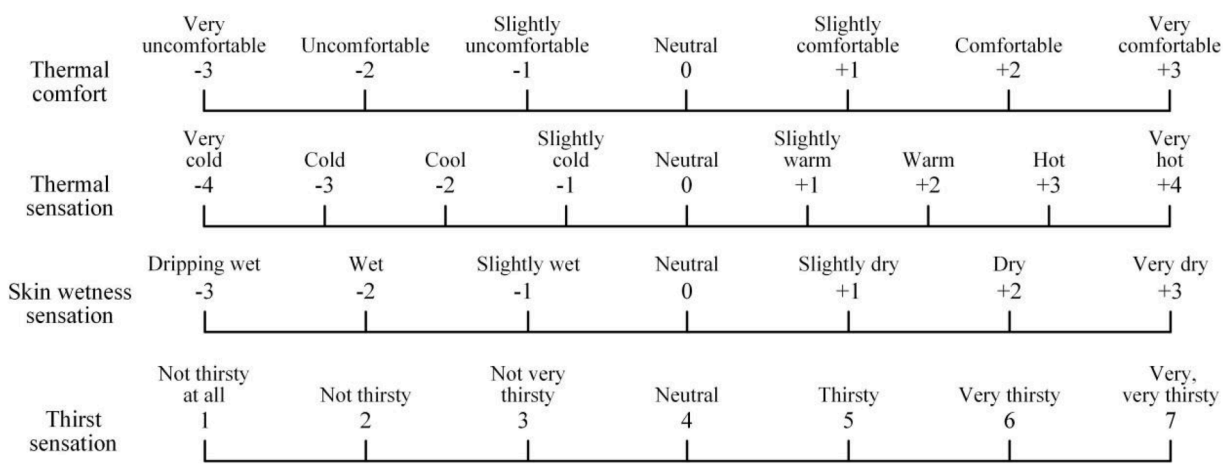

Fig. 2. Voting scales of perceptions.

2.5. Statistical analysis

Statistical analysis of all description data was processed by IBM SPSS Statistics version 27.0 (IBM Inc., Armonk, NY). Levene's test was performed to assess homogeneity of variance, and the Shapiro-Wilk test was employed to examine the normality of the data. For normally distributed data, a two-way (time × condition) repeated measures ANOVA was used to verify whether there are significant differences in heat strain responses among various environmental conditions. When the ANOVA results indicated a significant difference, post hoc analyses were performed using paired samples T-tests with Bonferroni correction to adjust for multiple comparisons. Non-normalized distributed data were subjected to analysis using the Friedman test and Wilcoxon signed-rank test. All data are presented as the mean±standard deviation. Statistical significance was set at $P < 0.05$.

3. Results

3.1. Body temperatures

Fig. 3 depicts participants' core temperature throughout the exposure under various environmental conditions. As shown in Fig. 3(a), T_{core} differed across different conditions ($P < 0.001$). Specifically, T_{core} at 29.0 °C increased progressively from 37.0 ± 0.1 °C to 37.7 ± 0.3 °C over the 8 h period, with a rising rate of 0.087 °C/h. When T_w=25.5 °C, subjects experienced a temporary decrease in T_{core} within the first 30 min after consuming the electrolyte beverage. Afterwards, T_{core} increased steadily over the first 5 h of exposure and then gradually stabilized. As shown in Fig. 3(b), T_{core} was significantly higher at the end of the exposure compared to pre-exposure for 25.5 °C and 29.0 °C (all $P < 0.001$). Post-exposure T_{core} at 29.0 °C were significantly higher compared to 25.5 °C and CON (all $P < 0.001$). Post-exposure T_{core} at 25.5 °C was significantly higher than that under CON condition ($P < 0.001$).

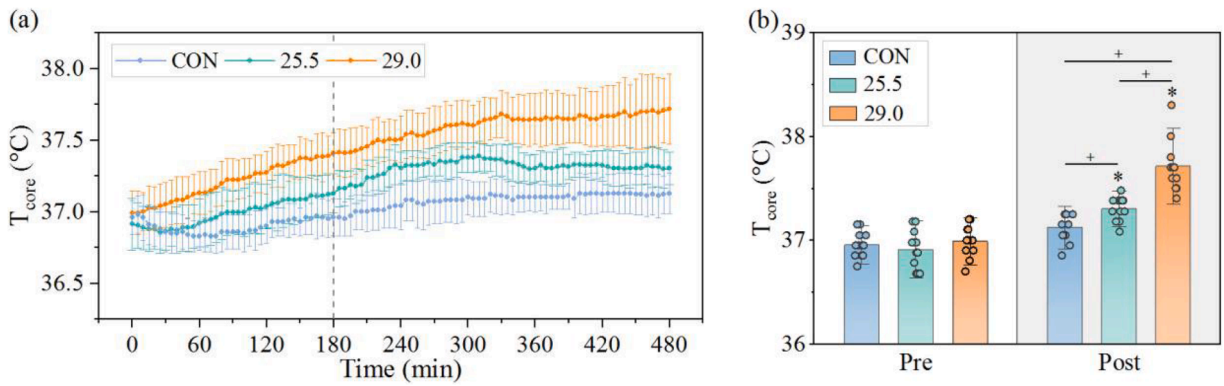

Fig. 3. Core temperature during the exposure across the three different trials. (a) Temporal variations of core temperature; (b) Core temperature before (Pre) and after (Post) the exposure.

Note: The dashed line at 180 min represents lunchtime. * indicates a significant difference when compared to Pre within condition; + indicates a significant difference between different conditions. (The same as below)

Fig. 4 illustrates the mean skin temperatures under different conditions. Throughout the 8 h exposure, T_{sk} at 29.0 °C was significantly higher compared to that under 25.5 °C ($P < 0.001$). When T_w=29.0 °C, T_{sk} rapidly increased during the first 30 min with a rising rate of 0.065 °C/min, followed by a steady increase until the end of the exposure. By contrast, T_{sk} at 25.5 °C initially increased from 33.5 ± 1.1 °C to 35.1 ± 0.3 °C over the first 30 min, and then tended to stabilize. Compared to pre-exposure, post-exposure T_{sk} was significantly higher for all trials (all $P < 0.01$), with increases of 2.8 °C at 29.0 °C, 1.9 °C at 25.5 °C, and 0.8 °C under CON, respectively. Moreover, post-exposure T_{sk} at 25.5 °C was 1.3 °C lower compared to 29.0 °C ($P < 0.001$), and was 1.6 °C higher compared to CON ($P < 0.001$).

3.2. Cardiovascular responses

Variations of HR during the exposure are presented in Fig. 5. It was observed that HR differed across conditions (Fig. 5(a), $P < 0.001$) and showed an interactional effect across conditions. Under the 29.0 °C condition, HR continued to rise with slight fluctuation, increasing from 84 ± 11 bpm initially to 102 ± 16 bpm after exposure. HR at 25.5 °C was significantly lower compared to 29.0 °C ($P < 0.001$) during the 8 h period. Notably, there was a slight increase in HR after lunch in all the three trials. In the 29.0 °C trial, HR was significantly higher at the end of the exposure compared to the pre-exposure level (Fig. 5(b), $P < 0.001$). However, no differences were observed between the pre-exposure and post-exposure HR in the 25.5 °C and CON trials (all $P > 0.05$).

Fig. 6 expresses the variations in MAP under different environments. MAP differed across different conditions throughout the 8 h period (all $P < 0.001$). At 29.0 °C, MAP significantly decreased from a baseline of 77 ± 8 mmHg to 67 ± 5 mmHg by the end of the 8 h exposure ($P < 0.01$), indicating that noticeable hypotension following extreme humid-heat exposure. By contrast, at 25.5 °C, MAP was significantly higher compared to 29.0 °C throughout the 8 h exposure ($P < 0.01$) and at the end of exposure ($P < 0.01$). There were no significant differences in MAP between pre- and post-exposure levels in the 25.5 °C and CON conditions (all $P > 0.05$).

3.3. Blood cell counts

Fig.7 depictes the blood cell counts before and after the exposure. Following 8 h of high-level hot humid exposure (29.0 °C), blood cell counts were significantly elevated compared to pre-exposure levels (all $P < 0.01$). Specifically, leukocyte counts increased by 71 % and neutrophil counts increased by 108 %. After 8 h of low-level exposure (25.5 °C), however, there were no significant differences in leukocytes ($P = 0.065$) or neutrophils ($P = 0.309$) compared to baseline levels.

3.4. Post-exposure hydration status

Table 2 shows the hydration status before and after the exposure. At the end of the extreme hot-humid exposure (T_w=29.0 °C), participants experienced significant body weight loss, a decrease in plasma volume, mild dehydration, and a notable increase in USG ($P < 0.001$), due to excessive sweating during the exposure. Following 8 h exposure to moderate hot-humid condition (T_w=25.5 °C), a higher USG ($P < 0.001$) and an increased plasma volume were found compared to pre-exposure

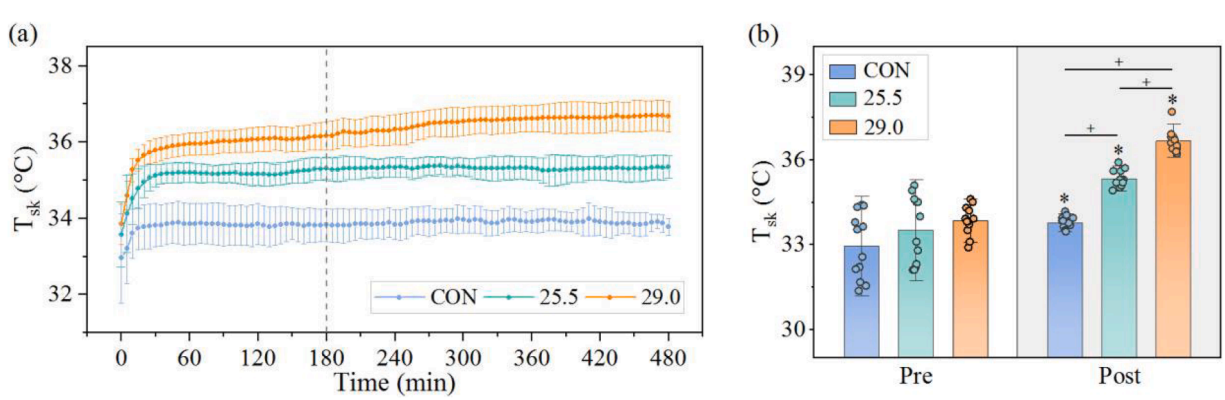

Fig. 4. Mean skin temperature during the 8 h heat exposure under different trials. (a) Temporal changes of mean skin temperature; (b) Mean skin temperature before and after the exposure.

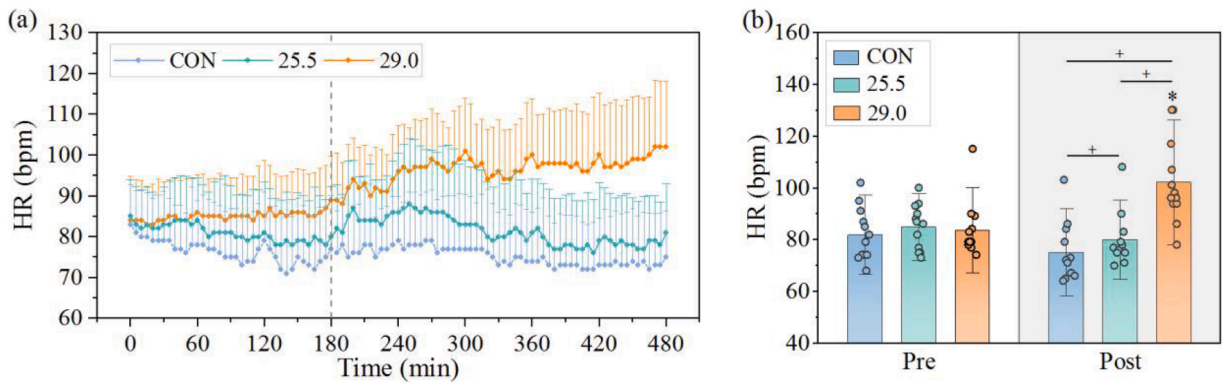

Fig. 5. Variations in heart rate during the exposure across different trials. (a) Temporal variations of heart rate; (b) Heart rate before and after the exposure.

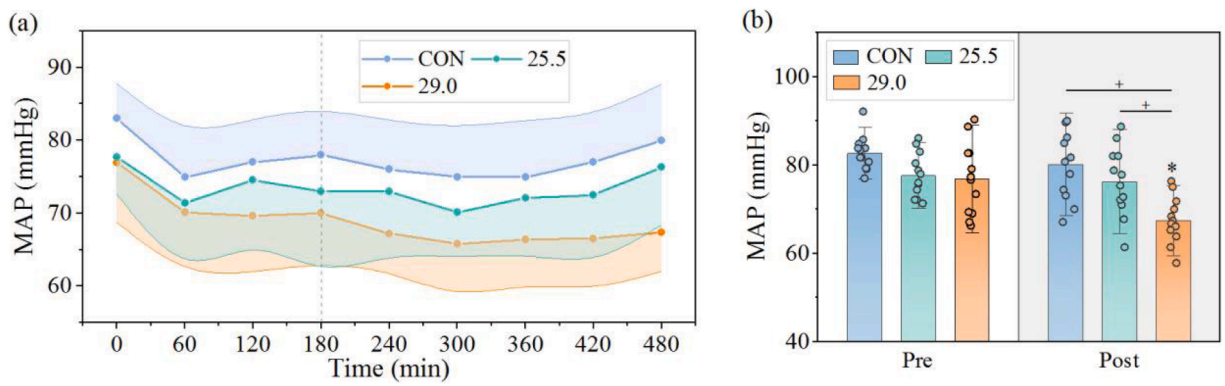

Fig. 6. Mean arterial pressure during heat exposure under different conditions. (a) Temporal variations of mean arterial pressure; (b) Mean arterial pressure before and after the exposure.

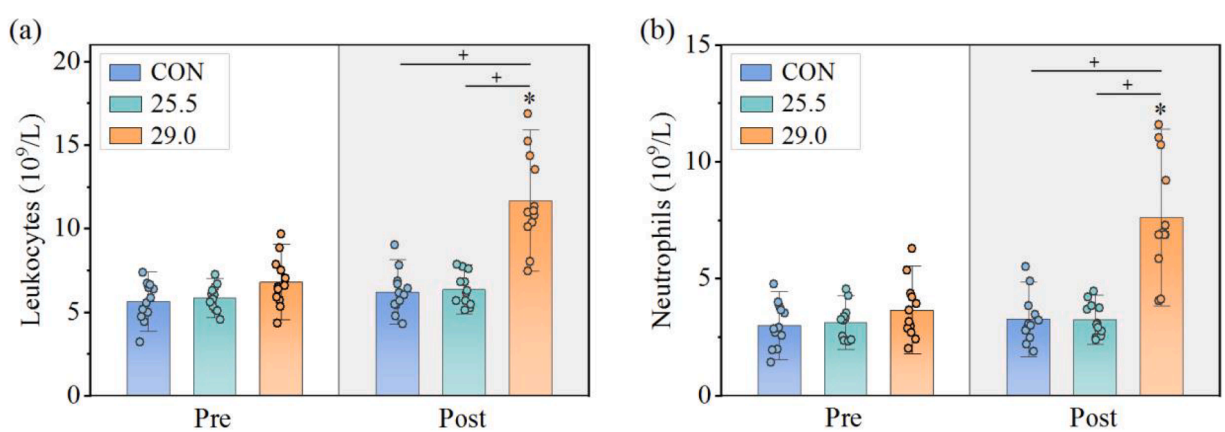

Fig. 7. Leukocyte (a) and neutrophil (b) counts pre- and post-exposure across the three different trials.

levels. Additionally, body weight, average sweating rate and dehydration rate at 25.5 °C were significantly lower compared to 29.0 °C ($P < 0.05$). However, no significant difference in USG ($P = 0.094$) was observed between 25.5 °C and 29.0 °C.

3.5. Perceptual responses

Fig.8 presents subjects' thermal perceptions throughout the exposure under three different conditions. Thermal comfort, thermal sensation, skin wetness sensation and thirst sensation all differed significantly across the three conditions (all $P < 0.01$). After 8 h of exposure to thermoneutral environment, participants reported similar subjective perceptions as pre-exposure levels (all $P > 0.05$). At the end of the 8 h exposure to 29.0 °C, participants reported significantly higher levels of thermal comfort (-2.33 ± 0.89), thermal sensation (2.83 ± 0.72), skin wetness sensation (2.83 ± 0.72) and thirst sensation (3.66 ± 1.23). Perceptions at the end of the exposure under 25.5 °C were significantly lower compared to those under 29.0 °C (all $P < 0.05$). Under the 25.5 °C condition, post-exposure thermal comfort and skin wetness sensation were higher than pre-exposure levels (all $P < 0.05$), but the thermal sensation ($P = 0.463$) and thirst sensation ($P = 0.189$) did not differ between pre- and post-exposure.

Table 2
Hydration status at baseline and at the end of the exposure.

Hydration	CON		T_w=25.5 °C		T_w=29.0 °C	
	Baseline	End	Baseline	End	Baseline	End
Body weight change (%)	–	-0.82 ± 0.81	–	-1.08 ± 0.60	–	-1.65 ± 0.67
Average sweating rate (g/h)	–	107.8 ± 26.7	–	168.9 ± 36.4	–	216.1 ± 54.8
Dehydration rate (%)	–	1.17 ± 0.32	–	1.40 ± 0.61	–	1.97 ± 0.66
Plasma volume change (%)	–	0.00 ± 0.07	–	0.90 ± 5.38	–	-1.51 ± 4.38
USG (g/mL)	1.006 ± 0.004	1.010 ± 0.004	1.007 ± 0.003	1.020 ± 0.006	1.008 ± 0.006	1.024 ± 0.008

4. Discussion

The principal findings of this investigation were as follows: (1) 8 h of extreme hot-humid exposure (T_w=29.0 °C) resulted in progressive increases in body temperatures and HR, noticeable hypotension and mild dehydration, elevated leukocyte and neutrophil counts, as well as high levels of thermal perceptions; (2) Exposure to a low-level humid heat environment (T_w=25.5 °C) for 8 h elicited significantly increases in body temperatures, accompanied by higher levels of thermal discomfort and skin wetness sensation compared to pre-exposure levels. These findings agree with our original hypothesis.

During the initial stage of both humid-heat trials (before 180 min), the body attempted to activate heat dissipation mechanisms [11], accompanied by heat accumulation and an increase in T_{core}. After lunch (at 180 min), increases in T_{core}, T_{sk} and HR were observed due to combined effects of heat exposure and the specific dynamic action of food [43]. Specifically, the digestion and absorption of food increased the metabolic rate [43], leading to a slight rise in T_{core} and T_{sk}. Additionally, more blood flowing to the gastrointestinal tract to aid digestion contributed to an increase in HR. Generally, the postprandial metabolic rate increases by 25 % on average and returns slowly to prefeeding level over several hours [43]. As exposure time increased (after 300 min), heat production and dissipation gradually reached equilibrium, maintaining T_{core} at a safe physiological level (T_w=25.5 °C) [11]. However, when exposed to extreme heat (T_w=29.0 °C), the body's thermoregulatory capacity was insufficient to counteract the environmental heat, resulting in a progressive rise in T_{core}.

In the present study, T_{core} increased by 0.7 °C after an 8 h exposure to high-level humid heat (36.5 °C, 57 %RH), indicating an excessive accumulation of heat load. This finding was inconsistent with previous research that Meade et al. [16,17] reported 0.3 °C increase in core temperature after a 9 h heat exposure, which was significantly lower than our observed increase. This discrepancy can likely be attributed to the dry hot condition (40.0 °C, 9 % RH) used in their study, which differs greatly from the humid heat environment in our study. The extremely low humidity (9 % RH) in those studies [16,17] facilitated sweat evaporation and heat loss from the body, thereby lowering core temperature [11,12]. By contrast, the impairment of evaporative heat loss capacity in humid heat contributed to a higher core temperature in this study. In another study, McKenna et al. [44] reported an increase of 0.5 °C in T_{core} after a 3 h heat exposure to similar conditions (41 °C, 40 % RH), which was slightly higher than our observed increase of 0.4 °C during the first 3 h of exposure. The difference in metabolic activity levels between previous study (3 Mets) [44] and our study (1.2 Mets) could provide an explanation for this variation.

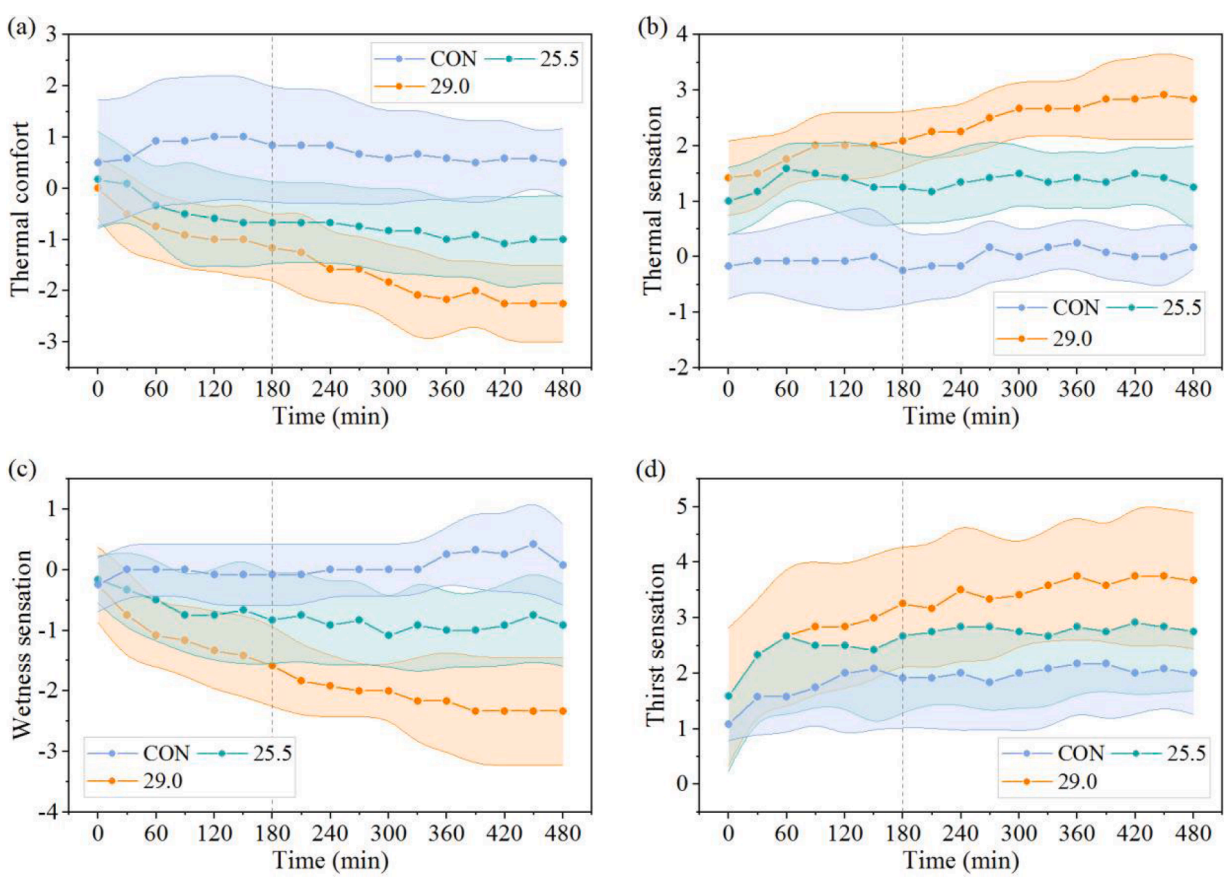

Fig. 8. Variations in thermal comfort (a), thermal sensation (b), skin wetness sensation (c) and thirst sensation (d) throughout the exposure under three different conditions.

Heat stress elicits sweating and associated fluid losses. After daylong exposure to high-level humid heat exposure (T_w=29.0 °C), participants exhibited mild dehydration responses, including significant body weight loss, decreased plasma volume and elevated USG. Although dehydration is not a direct cause of heat stroke, it can potentially impair heat transfer, reduce effective blood volume and cardiac output, thereby exacerbating cardiovascular failure [12]. In this study, participants were permitted to dry their sweat at any time during the trial. It is well-established that sweat evaporation efficiency decreases as skin wetness increases [45]. Therefore, keeping the skin surface dry promotes more effective sweat evaporation. Additionally, participants were required to drink at least 500 mL of water before the exposure and were provided with water intake of 2.5 mL/kg/h during the exposure, which may have alleviated fluid losses after exposure. If not replenished by fluid intake during this period, we speculate that subjects would likely experience more severe hypohydration and core temperature increases. In fact, chronic dehydration is common among the low-income population [27]. When dehydration is coupled with heat exposure, heat exhaustion probably occurs with smaller increases in core temperature [34].

In the presence of elevated temperature and dehydration, as expected, we observed noticeable hypotension and excessive increase in HR following the exposure. Our findings are supported by existing studies [11,12] which confirm that when exposed to extreme heat, peripheral vasodilation reduces vascular resistance, while fluid loss decreases blood volume and cardiac preload. This combination results in lower blood pressure and an increased HR necessary to compensate for the drop in blood pressure. Nevertheless, these findings differ from previous studies on daylong heat exposure. As reported by Meade et al. [16], there were no significant decreases in systolic and diastolic blood pressure after 9 h of heat exposure, with a post-exposure HR of only 86 ± 12 bpm. The slight increase in T_{core} (0.3 °C) [16], which was insufficient to induce cardiovascular stress, may be responsible for the observed differences between our study and Meade et al.'s findings. Additionally, water intake has been shown to attenuate the degree of blood pressure reduction in a hot environment [46], and the electrolyte water provided during the exposure in our study may have contributed to suppressing a greater drop in MAP.

Post-exposure blood sample measurements indicated that under the 29.0 °C condition, both leukocyte and neutrophil counts exceeded normal clinical reference ranges [47]. Specifically, leukocyte counts were 22.7 % above the maximum clinical value (9.5×10^9 L^{-1}), while neutrophil counts were 20.9 % above their clinical threshold (6.3×10^9 L^{-1}), suggesting a significant impact on the immune system following 8 h of extreme hot-humid exposure [13]. This finding contrasts with previous research [17], which reported no significant increase in pro-inflammatory cytokines after 9 h of hot dry exposure, as evidenced by decreased levels of tumor necrosis factor α (TNF-α) and interleukin-6 (IL-6) with a post-exposure T_{core} of 37.3 °C. This supports the possibility that elevated temperature may be the most dominant factor in inducing inflammatory responses [12,13].

Additionally, it was observed that under T_w of 29.0 °C, T_{core} continued to rise even in the final hour of exposure. Notably, our study primarily focused on the humid heat exposure during an 8 h daytime work period. Research on T_w diurnal variations during humid heatwave period indicates that T_w associated with uncompensable heat stress may also occur in the evening due to extremely high humidity [48]. Given this, it is possible that T_{core} will continue to increase overnight, posing a greater risk to human health. In fact, historically documented heatwave events, which were comparable to this study in terms of the humid heat environment levels, have caused mass mortality, such as the Western European heatwave in 2003 with more than 70,000 deaths [49], the Russian heatwave in 2010 with 55,000 deaths [32], and the Mecca, Saudi Arabia heatwave in 2024 which caused over 1300 fatalities during 6 days (between June 14 and 19) [33]. Although no heat-related deaths were reported in the Shanghai heatwave owing to the use of air conditioners, the findings of this study provide ecologically valid evidence regarding the impact of heatwaves on human health for heat-vulnerable regions and populations.

Following an 8 h exposure to low-level humid heat environment (T_w=25.5 °C), we observed significant increases in core temperature (37.3 ± 0.1 °C), mean skin temperature (35.4 ± 0.3 °C) and USG (1.020 ± 0.006). Additionally, participants reported higher levels of thermal discomfort and skin wetness sensation. The 0.4 °C increase in core temperature before and after exposure can be partly attributed to the circadian rhythm [50]. The daily mean T_w of 25.5 °C (33.5 °C, 52 % RH) in this study was essentially consistent with the minimum threshold for sauna-like conditions [23]. Based on these results, it can be tentatively concluded that 8 h exposure to such moderate conditions during the daytime would not cause severe hyperthermia, cardiovascular strain, or inflammatory responses. Considering that we only conducted an 8 h daytime exposure trial, there may be consecutive sauna weather conditions lasting for several weeks in heat-vulnerable regions [1,4,29]. For instance, from August 5th to September 5th, 2024, Shanghai experienced a continuous period of sauna weather for 32 days [28]. In such cases, the impact of repetitive prolonged hot-humid exposure on human health warrants further investigation.

There are still limitations in the current study. First, only male subjects were examined, leaving the effects of sex differences on thermophysiological responses during prolonged humid heat exposure unexplored. Second, the blood cell counts were only measured before the exposure and immediately after the exposure, with no measurements taken during the exposure. The temporal changes in blood cell counts in the context of daylong heat exposure remains unclear. Continuous sampling of blood every hour may help elucidate the underlying correlation between inflammatory response and core temperature. Third, body weight was measured pre- and post-exposure, with no weighing conducted during the 8 h exposure. The variation in sweating rate over time remains unknown, for instance, whether the sweating rate will decline due to sweat fatigue, which warrants further investigation.

5. Conclusion

The present study quantified changes in thermoregulation, cardiovascular function, blood inflammatory cell counts and subjective perceptions during 8 h of sedentary office work in different hot-humid environments. The findings revealed that following 8 h of high-level humid heat exposure (T_w=29.0 °C), participants endured significantly higher body temperature and HR, notable hypotension, mild dehydration, and markedly elevated blood inflammatory cell counts exceeding clinical thresholds. Compared to hot-dry conditions with similar ambient temperatures, the extreme hot-humid environments severely impair the evaporative cooling mechanism, resulting in greater heat accumulation and a higher physiological burden on the human body. Combined effects of dehydration and elevated temperature are probably responsible for the inflammatory responses observed during chronic heat exposure. Moreover, the results indicated that 8 h of exposure in a low-level humid heat environment (T_w=25.5 °C) which is more common and can last longer in real climates, did not affect cardiovascular function and immune system. Nonetheless, further research is needed to determine whether repetitive daylong exposures to such environments over consecutive days will have different impacts on human health. In addition, this study highlights the critical need for effective and easily accessible cooling strategies, such as electric fan use, liquid intake or cold-water immersion, to alleviate heat strain among heat-vulnerable populations during hot weather.

CRediT authorship contribution statement

Huijuan Xu: Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Conceptualization. **Lei Zhang:** Visualization, Validation, Investigation, Formal analysis. **Zhao Jin:** Visualization, Validation, Investigation, Formal analysis. **Aobing**

Wang: Visualization, Investigation, Funding acquisition, Formal analysis. **Zhijian Liu:** Supervision, Resources, Methodology, Conceptualization. **Faming Wang:** Writing – review & editing, Visualization, Methodology, Investigation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A

Table A1
Observed half-hourly weather data of Shanghai from July 20th to July 22nd, 2024 [18,28].

	July 20th			July 21st			July 22nd		
Time	T_a (°C)	RH (%)	T_w (°C)	T_a (°C)	RH (%)	T_w (°C)	T_a (°C)	RH (%)	T_w (°C)
0:00	32	59	25.5	31	75	27.3	30	75	26.4
0:30	32	59	25.5	31	75	27.3	30	75	26.4
1:00	31	66	25.8	30	79	27.0	30	75	26.4
1:30	31	62	25.1	30	79	27.0	30	75	26.4
2:00	31	66	25.8	30	79	27.0	30	70	25.6
2:30	31	62	25.1	31	79	27.3	30	70	25.6
3:00	30	66	24.9	31	79	27.3	30	70	25.6
3:30	30	66	24.9	31	75	27.3	30	70	25.6
4:00	30	70	25.6	31	75	27.3	30	70	25.6
4:30	30	70	25.6	31	75	27.3	29	74	25.3
5:00	30	75	26.4	31	70	26.5	30	70	25.6
5:30	30	75	26.4	31	70	26.5	30	70	25.6
6:00	31	70	26.5	31	75	27.3	30	70	25.6
6:30	31	70	26.5	31	70	26.5	31	66	25.8
7:00	32	67	26.9	31	70	26.5	32	63	26.2
7:30	32	67	26.9	31	70	26.5	32	63	26.2
8:00	33	63	27.1	32	71	27.6	33	59	26.3
8:30	33	59	26.3	33	67	27.8	33	59	26.3
9:00	34	59	27.2	32	67	26.9	34	56	26.6
9:30	35	56	27.5	32	67	26.9	34	56	26.6
10:00	35	56	27.5	33	67	27.8	35	53	26.9
10:30	36	50	27.1	33	63	27.1	35	53	26.9
11:00	36	53	27.7	35	60	28.3	36	50	27.1
11:30	36	53	27.7	35	60	28.3	37	50	27.9
12:00	37	50	27.9	36	60	29.1	36	53	27.7
12:30	38	47	28.1	36	60	29.1	37	50	27.9
13:00	38	47	28.1	36	56	28.3	37	50	27.9
13:30	39	45	28.4	37	53	28.6	37	50	27.9
14:00	39	45	28.4	38	53	29.4	38	47	28.1
14:30	39	48	29.1	39	50	29.6	38	52	29.2
15:00	38	50	28.8	39	51	29.8	38	47	28.1
15:30	38	50	28.8	39	51	29.8	38	47	28.1
16:00	37	50	27.9	38	50	28.8	37	50	27.9
16:30	37	50	27.9	38	50	28.8	37	50	27.9
17:00	36	53	27.7	37	57	29.4	36	56	28.3
17:30	36	53	27.7	36	60	29.1	36	56	28.3
18:00	36	56	28.3	35	60	28.3	35	60	28.3
18:30	35	60	28.3	35	59	28.0	34	63	28.0
19:00	34	63	28.0	33	67	27.8	33	67	27.8
19:30	34	67	28.7	33	67	27.8	32	71	27.6
20:00	33	71	28.5	32	71	27.6	32	67	26.9
20:30	33	67	27.8	32	71	27.6	31	70	26.5
21:00	32	72	27.7	32	71	27.6	31	75	27.3
21:30	32	75	28.2	31	75	27.3	31	75	27.3
22:00	32	75	28.2	31	75	27.3	31	70	26.5
22:30	32	75	28.2	31	75	27.3	31	70	26.5
23:00	31	79	27.9	31	75	27.3	30	75	26.4
23:30	31	75	27.3	30	75	26.4	30	75	26.4

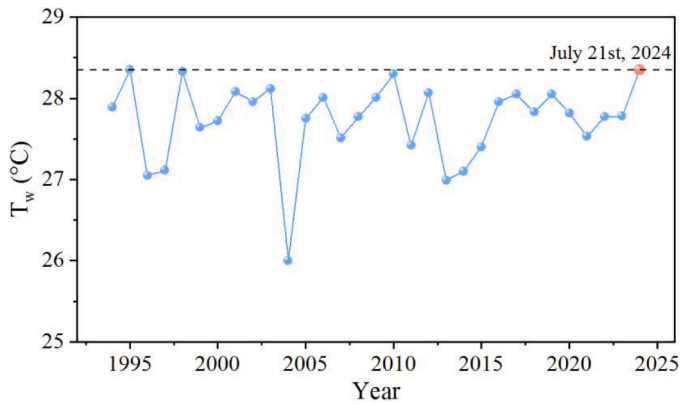

Fig. A1. Maximum daily 24 h average T_w of each year from 1994 to 2024 [18,28,30].

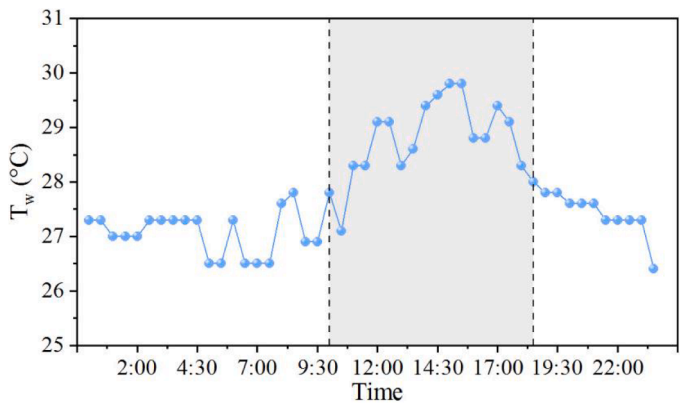

Fig. A2. Diurnal variation of T_w on July 21st, 2024 [18,28].

Table A2
Daily weather data of sauna weather days for Shanghai from June to September 2024 [18,23,28].

Date	Daily maximum T_a (°C)	Daily mean RH (%)	Mean T_a during 10 a.m. to 6 p.m. (°C)	Mean RH during 10 a.m. to 6 p.m. (%)	Mean T_w during 10 a.m. to 6 p.m. (°C)
July 2nd	35	72.3	33.2	60.9	26.8
July 3rd	36	72.2	34.9	55.5	27.3
July 4th	36	72.2	34.7	56.1	27.3
July 6th	39	60.9	37.5	48.2	28.0
July 7th	39	60.2	38.4	41.8	27.2
July 10th	34	71.2	31.7	68.1	26.8
July 15th	34	75.0	32.7	63.4	26.9
July 16th	36	68.7	35.1	53.0	27.0
July 17th	36	64.8	34.8	57.0	27.5
July 18th	38	63.5	36.1	48.9	27.0
July 20th	39	60.2	37.2	50.1	28.1
July 21st	39	66.5	36.5	57.0	29.0
July 22nd	38	62.8	36.4	51.8	27.8
July 23rd	37	64.5	35.9	51.9	27.4
July 24th	36	68.3	34.7	56.7	27.4
July 25th	34	74.7	32.9	63.8	27.1
July 28th	36	67.7	34.6	55.7	27.0
July 29th	36	66.0	34.8	55.8	27.3
July 30th	36	67.5	34.9	56.7	27.5
July 31st	39	63.3	37.2	49.8	28.0
August 1st	40	59.3	38.3	43.4	27.4
August 5th	38	65.2	36.8	53.2	28.4
August 6th	39	62.2	36.9	46.3	27.0
August 7th	38	63.2	36.7	47.9	27.2
August 8th	39	70.7	34.1	60.1	27.5
August 9th	38	69.0	36.3	52.3	27.8
August 10th	38	67.4	36.4	54.2	28.3
August 11th	38	69.7	34.6	60.5	28.0
August 12nd	36	71.4	32.3	65.1	26.8
August 13rd	36	68.5	33.8	61.0	27.4

(*continued on next page*)

Table A2 (*continued*)

Date	Daily maximum T_a (°C)	Daily mean RH (%)	Mean T_a during 10 a.m. to 6 p.m. (°C)	Mean RH during 10 a.m. to 6 p.m. (%)	Mean T_w during 10 a.m. to 6 p.m. (°C)
August 14th	35	65.1	33.9	53.6	26.1
August 15th	34	70.9	32.7	57.5	25.8
August 16th	35	68.1	33.6	55.2	26.1
August 17th	35	63.8	34.2	52.1	26.0
August 18th	34	68.6	32.9	59.2	26.3
August 19th	36	68.9	34.4	55.6	26.9
August 20th	36	67.3	34.7	55.9	27.2
August 21st	35	75.8	32.8	64.8	27.2
August 22nd	35	70.1	33.9	55.5	26.4
August 23rd	37	62.0	34.9	51.9	26.6
August 24th	37	68.4	35.0	52.4	26.7
August 25th	35	69.6	33.8	58.4	26.9
August 26th	36	69.5	34.3	56.8	27.1
August 27th	36	77.6	32.6	67.5	27.6
August 28th	34	77.9	32.8	62.0	26.7
August 29th	35	70.8	33.4	56.8	26.3
August 30th	35	68.0	34.1	53.5	26.2
August 31st	34	64.5	33.1	55.1	25.7
September 1st	36	63.1	34.7	56.2	27.3
September 2nd	36	66.3	34.6	58.1	27.5
September 4th	34	70.7	32.6	58.8	25.9
September 5th	36	67.1	33.4	55.2	26.0
September 7th	35	65.9	33.5	52.2	25.5
September 8th	35	66.7	33.4	55.4	26.0
September 9th	35	67.8	33.7	55.4	26.2
September 12th	34	74.0	32.8	61.6	26.6
September 13th	35	70.3	33.4	54.8	25.9
September 14th	34	68.0	32.4	57.8	25.6
September 17th	34	76.9	32.5	60.9	26.2

Data availability

Data will be made available on request.

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