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The Effects of Heat Stress on Physiological Parameters and Oxidative Stress Markers in Healthy Adults in Almuthana, Iraq

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Annotation: Background: Almuthana Province in southern Iraq experiences extreme ambient temperatures exceeding 50°C during the summer months, leading to severe heat stress for the local population. There is a lack of research on physiological and biochemical responses to extreme heat exposure in the region.

Objective: To examine the effects of acute heat stress on physiological parameters and oxidative stress biomarkers in healthy adults living in Almuthana, Iraq.

Methods: We conducted a cross-sectional study of 120 healthy adults (60 males, 60 females, 20-45 years) under extreme summer months (July-August 2024). We exposed participants to controlled heat exposure (45°C, 30% relative humidity) for 60 minutes. We collected physiological parameters, including core body temperature, heart rate, blood pressure, and sweat rate. We collected blood to determine oxidative stress biomarkers including malondialdehyde (MDA), superoxide dismutase (SOD), catalase and reduced glutathione (GSH).

Results: The results showed a

significant change in core body temperature (38.9±0.4°C vs. 36.8±0.3°C, p<0.001), heart rate (118±12 bpm vs. 72±8 bpm, p<0.001), and systolic blood pressure (142±15 mmHg vs. 118±10 mmHg, p<0.001) following heat exposure. MDA increased by 68% (p<0.001) and markers of antioxidant activity, SOD decreased by 23% (p<0.001), catalase decreased by 31% (p<0.001), and GSH decreased by 28% (p<0.001). Additionally, males had larger physiological stress responses than females.

Conclusion: Acute heat stress results in pronounced impairments to physiological homeostasis in healthy adults of Almuthana, Iraq, and induces oxidative stress to the biological system. The conclusions of this study could benefit occupational health and safety measures in populations that work or live in extreme heat conditions.

Keywords: Heat stress, oxidative stress, physiological parameters, Iraq, thermal stress, antioxidant enzymes.

Introduction

Heat stress poses a significant public health challenge in arid and semi-arid regions worldwide, with multiple implications for human health, productivity, and quality of life (1). The physiological response to heat stress involves complicated systems focused onappropriately adjusting physiological systems to maintain thermal homeostasis. These physiological systems include • circulatory mechanisms to adjust cardiovascular responses, • reactions to decrease temperature through thermoregulation and heat dissipation, and • and potential cellular protective mechanisms (2). When heat load from the environment exceeds the adaptive capacity of the body, heat stress can cause severe health conditions such as heat exhaustion and critically, heat stroke .(3)

Iraq, and specifically southern provinces of Iraq, has recorded some of the highest ambient temperatures in the world where summer temperatures can routinely exceed 50°C. Furthermore, humidity often serves to impair the body's evaporative cooling mechanisms, and instead intensifies the effects of the heat load (4). Almuthana province, southern Iraq, has provided one of the most difficult thermal amenities for humans, with temperatures recorded at 53.9°C in recent years (5). With extreme heat and very low humidity at various times in the summer months, combined with the impact of urbanization, the residents of southern Iraq experience an extreme physiological challenge.

The human body's response to heat stress involves multiple physiological systems that must be effectively coordinated to minimize the body's temperature and the degree of temperature increase in core body temperature (6). The cardiovascular system is multifaceted in the increasing cardiac output, the peripheral vasodilation, and the redistribution of blood to the skin for increased heat loss from the body (7). Simultaneously with the cardiovascular system, the autonomic nervous system engages sweating mechanisms in the sweat glands, signaling heat activation and potential evaporative cooling. However, the uncontrolled use of these physiological mechanisms can lead to substantial fluid losses and electrolyte imbalances (8). Heat stress can create reactive oxygen species (ROS) at the cellular level, causing a level of oxidative stress when the balance of the pro-oxidant and antioxidant systems of the body is upset (9). This oxidative imbalance can lead to lipid peroxidation, protein denaturation, and DNA damage which may all lead to acute heat-related illness as well as long-term health effects (10). The antioxidant defense system includes a variety of enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase that help neutralize ROS. In addition, reduced glutathione (GSH) is a non-enzymatic antioxidant that helps mitigate the effects of oxidative stress on cells and tissues (11) It is important to note that heat stress can significantly impact several physiological factors albeit that core body temperature rise is the most straightforward measure of thermal stress, there has been identified core body temperatures above 38.5°C reflect significant heat strain (12). During sustained elevations of body temperature the cardiovascular system integrates heart rate, blood pressure, and cardiac output in order to sustain even higher metabolisms, help fluid transport, and facilitate heat loss (13). The integration of these cardiovascular changes will be particularly demanding for individuals with existing cardiovascular comorbidities and is likely to compound increased morbidity and mortality risks during heat waves (14). Measurable markers of oxidative stress would provide a better understanding of the cellular and molecular impacts of heat exposure. An oxidative stress biomarker malondialdehyde (MDA) is a product of lipid peroxidation (15). Samples with elevated levels of MDA suggest greater lipid membrane damage, and cellular dysfunction relative to heat stress (16). Antioxidant enzyme activity (e.g., SOD, catalase, GSH levels) typically decreases during acute heat stress as these systems become overwhelmed or exhausted.(17)

Countless studies note gender differences in heat stress responses, with evidence of more strain on cardiovascular function and higher increases in core body temperature in the males as opposed to females exposed to heat (18). Aspects underlying these differences may be through differences in body composition, sweating rates, cardiovascular fitness, and hormonal factors (19). Understanding gender differences is essential for ensuring effective heat stress prevention and management programs.

The climate and geographical structure of the Almuthana province provides a unique opportunity to examine heat stress consequences by virtue of the severe heat stress challenges they experience. The Almuthana province situated in a continental desert climate with scorching summers and mild winters creates a geographic region undergoing increased exposure to prolonged heat potentially leading to acute and chronic adaptation among the resident's behaviours (20). The other factors to consider concerning chronic exposure to heat stress are the socioeconomic situation, occupational patterns such as whether they work indoors or outdoors, and cultural trends that might offer insight into heat exposure and adapting behaviours.

There is a lack of research on heat stress responses in the Iraqi population which is significant in light of the substantial thermal challenge faced by inhabitants of the southern provinces. Much of the related heat stress research has been conducted in controlled laboratory spaces or in populations from temperate climate regions; this limits the potential applicability of heat stress research to populations living in extreme heat regions (21). This lack of research presents a significant knowledge barrier to advancing our understanding in the context of heat stress physiology in populations with chronic exposure to extreme ambient temperatures.

This research will attempt to lessen this knowledge gap by exploring the physiological and biochemical responses to acute heat stress exposure in healthy adults from Almuthana, Iraq. This

study will explore physiological parameters as well as markers indicative of oxidative stress to provide insight into the effects of heat stress response mechanisms underlying the adaptation in the adults in this study. Ultimately the results may corroborate or further substantiate public health policy programs, occupational safety mandates, and clinical management protocols for heat-related illnesses in extreme environments.

Notably, research like this also contributes to the growing body of literature on the implications of climate change for human health. As global temperatures continue to rise, the frequency and intensity of extreme heat events are predicted to become more common in areas worldwide (22). Furthermore, studies may provide unique insights into predicting and mitigating the potential health consequences of people's exposure to heat in other socio-demographics, particularly those with increasing trends in unnatural environmental changes associated with climate change.

The aim of this study was: (1) to quantify the acute physiological responses to controlled heat stress in healthy adults from Almuthana, Iraq; (2) to observe the changes in oxidative stress markers, if any, after exposure to heat; (3) to observe gender differences regarding heat exposure reactions and responses; and (4) to assess the relationship between physiological parameters and oxidative stress markers during heat exposure. The overall study objectives aim to provide a comprehensive understanding of the impacts of heat stress while accounting for evidence-based approaches to support individuals at risk of heat-related health hazards in extremely hot environments.

Methodology

Study Design and Setting

This cross-sectional experimental study was conducted during the peak summer months (July-August 2024) in Al-Muthanna, Iraq. The study protocol was conducted in accordance with the principles outlined in the Declaration of Helsinki. All participants provided written informed consent before enrollment.

Participants

A total of 120 healthy adults (60 males, 60 females) aged 20-45 years were recruited from the local population of Almuthana province through convenience sampling. Inclusion criteria included: (1) permanent residence in Almuthana province for at least 5 years; (2) age between 20-45 years; (3) body mass index (BMI) between 18.5-29.9 kg/m²; (4) absence of chronic diseases; (5) no current medication use; and (6) ability to provide informed consent. Exclusion criteria included: (1) cardiovascular disease; (2) diabetes mellitus; (3) renal or hepatic dysfunction; (4) pregnancy or lactation; (5) recent illness or fever within 2 weeks; (6) current use of antioxidant supplements; (7) smoking or alcohol consumption; and (8) occupational heat exposure exceeding 6 hours daily.

Sample size calculation was performed using G*Power 3.1.9.7 software, assuming a medium effect size (Cohen's $d = 0.5$), $\alpha = 0.05$, and power = 0.80. The minimum required sample size was determined to be 102 participants, with 120 participants recruited to account for potential dropouts.

Experimental Protocol

Pre-exposure Assessment

Participants reported to the laboratory between 08:00 and 10:00 hours after an overnight fast of at least 8 hours. Baseline measurements included anthropometric assessments (height, weight, and body mass index), vital signs, and a review of the medical history. Participants were instructed to refrain from caffeine, alcohol, and strenuous exercise for 24 hours preceding the test. Adequate hydration was ensured by measuring urine specific gravity (target <1.025).

Heat Stress Exposure

Heat stress was induced using a controlled environmental chamber (Model TH-1000, Thermotron Industries, Holland, MI, USA), maintained at an ambient temperature of 45°C with a relative humidity of 30%. These conditions were selected to simulate typical summer conditions in Almuthana province based on meteorological data from the Iraqi Meteorological Organization. Participants wore standardized, lightweight clothing (a cotton T-shirt and shorts) and remained seated with minimal physical activity during the 60-minute exposure period.

Physiological Monitoring

Core body temperature was continuously monitored using telemetric temperature sensors (VitalSense® Core Temperature Monitor, Philips Respironics, Netherlands) inserted rectally to a depth of 10 cm. Heart rate and blood pressure were measured at 15-minute intervals using automated monitoring equipment (Omron HEM-7320, Omron Healthcare, Japan). The sweat rate was calculated from nude body weight measurements taken before and after exposure, corrected for fluid intake.

Blood Sample Collection and Analysis

Venous blood samples (10 mL) were collected via antecubital venipuncture at baseline and immediately post-exposure. Samples were processed within 30 minutes of collection, with plasma and serum separated by centrifugation at 3000 rpm for 10 minutes at 4°C. Samples were stored at -80°C until analysis.

Oxidative Stress Markers

Malondialdehyde (MDA): Lipid peroxidation was assessed by measuring MDA levels using the thiobarbituric acid reactive substances (TBARS) method. Plasma samples were mixed with the thiobarbituric acid reagent and heated at 95°C for 60 minutes. Absorbance was measured spectrophotometrically at 532 nm, with results expressed as nanomoles per milliliter (nmol/mL).

Superoxide Dismutase (SOD): SOD activity was determined using the xanthine-xanthine oxidase system with nitroblue tetrazolium (NBT) as the indicator. The inhibition of NBT reduction was measured at 560 nm, with activity expressed as units per milliliter (U/mL).

Catalase: Catalase activity was measured by monitoring the decomposition of hydrogen peroxide at 240 nm. Activity was calculated using the molar extinction coefficient of hydrogen peroxide and expressed as units per milliliter (U/mL).

Reduced Glutathione (GSH): GSH levels were determined using Ellman's reagent (5,5'-dithiobis-2-nitrobenzoic acid). The development of yellow color was measured at 412 nm, with concentrations expressed in micromoles per liter (μmol/L).

All biochemical analyses were performed in duplicate using standardized protocols and quality control measures. Inter-assay and intra-assay coefficients of variation were maintained below 5% and 3%, respectively.

Environmental Monitoring

The ambient temperature and relative humidity within the environmental chamber were continuously monitored using calibrated sensors (Testo 175 H1, Testo SE & Co., Germany), with data logging occurring at 1-minute intervals. Wet bulb globe temperature (WBGT) was calculated to assess heat stress conditions.

Statistical Analysis

Statistical analyses were performed using SPSS version 28.0 (IBM Corp., Armonk, NY, USA). Normality of data distribution was assessed using the Shapiro-Wilk test and visual inspection of histograms. Descriptive statistics are presented as mean ± standard deviation for normally distributed variables and median (interquartile range) for non-normally distributed variables.

Paired t-tests were used to compare pre-exposure and post-exposure measurements within each group. Independent t-tests were employed to examine gender differences. Pearson correlation coefficients were calculated to assess relationships between physiological parameters and oxidative stress markers. A two-way repeated measures analysis of variance (ANOVA) was performed to investigate the effects of time and gender on the measured variables.

Multiple linear regression analysis was conducted to identify predictors of heat stress responses, with age, gender, body mass index (BMI), and baseline fitness level as independent variables. Effect sizes were calculated using Cohen's d, with values of 0.2, 0.5, and 0.8 representing small, medium, and large effects, respectively.

Statistical significance was set at $p < 0.05$, with Bonferroni correction applied for multiple comparisons. Power analysis confirmed adequate statistical power (>80%) for detecting clinically meaningful differences in primary outcome measures.

Quality Assurance

All laboratory personnel were trained and certified in standardized protocols. Equipment calibration was performed monthly using certified reference standards. Blind duplicate analyses were conducted on 10% of samples to ensure analytical precision. Data quality was monitored through regular review of outliers and missing values.

Ethical Considerations

The study protocol adhered to international ethical guidelines for human research. Participants were fully informed of study procedures, potential risks, and their right to withdraw at any time. Emergency medical support was available throughout the study period. Data confidentiality was maintained through the use of coded identifiers, and personal information was securely stored by institutional guidelines.

Results

Participant Characteristics

A total of 120 healthy adults completed the study protocol without adverse events. Baseline characteristics are presented in Table 1. The mean age was 31.2 ± 7.8 years, with no significant differences between males and females (31.8 ± 8.1 vs. 30.6 ± 7.4 years, $p = 0.38$). Body mass index was significantly higher in males compared to females (25.1 ± 3.2 vs. 23.4 ± 2.8 kg/m², $p < 0.01$).

Table 1. Baseline Characteristics of Study Participants

Characteristic	Males (n=60)	Females (n=60)	Total (n=120)	p-value
Age (years)	31.8 ± 8.1	30.6 ± 7.4	31.2 ± 7.8	0.38
Height (cm)	172.4 ± 6.8	159.2 ± 5.9	165.8 ± 9.2	<0.001
Weight (kg)	74.6 ± 9.5	59.4 ± 8.1	67.0 ± 11.8	<0.001
BMI (kg/m²)	25.1 ± 3.2	23.4 ± 2.8	24.3 ± 3.1	<0.01
Systolic BP (mmHg)	119 ± 11	116 ± 9	118 ± 10	0.15
Diastolic BP (mmHg)	78 ± 8	76 ± 7	77 ± 8	0.18
Heart Rate (bpm)	73 ± 9	71 ± 7	72 ± 8	0.23

Physiological Responses to Heat Stress

Significant changes in all physiological parameters were observed following heat exposure (Table 2). Core body temperature increased from 36.8 ± 0.3°C to 38.9 ± 0.4°C ($p < 0.001$), representing a mean elevation of 2.1°C. Heart rate increased substantially from 72 ± 8 bpm to 118 ± 12 bpm ($p < 0.001$), with males showing greater increases than females (122 ± 13 vs. 114 ± 10 bpm, $p < 0.01$).

Table 2. Physiological Parameters Before and After Heat Stress Exposure

Parameter	Baseline	Post-Exposure	Change	Effect Size (d)	p-value
Core Temperature (°C)	36.8 ± 0.3	38.9 ± 0.4	+2.1 ± 0.3	5.8	<0.001
Heart Rate (bpm)	72 ± 8	118 ± 12	+46 ± 10	4.4	<0.001
Systolic BP (mmHg)	118 ± 10	142 ± 15	+24 ± 12	1.9	<0.001
Diastolic BP (mmHg)	77 ± 8	89 ± 11	+12 ± 8	1.2	<0.001
Sweat Rate (L/h)	-	1.8 ± 0.4	-	-	-

Oxidative Stress Markers

Heat stress induced significant alterations in all measured oxidative stress markers (Table 3). Malondialdehyde levels increased by 68% from baseline (3.2 ± 0.6 to 5.4 ± 0.9 nmol/mL, $p < 0.001$), indicating substantial lipid peroxidation. Antioxidant enzyme activities decreased significantly: SOD activity decreased by 23% (245 ± 32 to 189 ± 28 U/mL, $p < 0.001$), catalase activity decreased by 31% (58 ± 8 to 40 ± 7 U/mL, $p < 0.001$), and GSH levels decreased by 28% (892 ± 124 to 642 ± 98 µmol/L, $p < 0.001$).

Table 3. Oxidative Stress Markers Before and After Heat Stress Exposure

Marker	Baseline	Post-Exposure	% Change	Effect Size (d)	p-value
MDA (nmol/mL)	3.2 ± 0.6	5.4 ± 0.9	+68%	2.8	<0.001
SOD (U/mL)	245 ± 32	189 ± 28	-23%	1.9	<0.001
Catalase (U/mL)	58 ± 8	40 ± 7	-31%	2.4	<0.001
GSH (µmol/L)	892 ± 124	642 ± 98	-28%	2.2	<0.001

Gender Differences

Males demonstrated greater physiological stress responses compared to females. Core temperature elevation was higher in males (2.3 ± 0.4°C vs. 1.9 ± 0.3°C, $p < 0.001$), as was the heart rate increase (51 ± 11 vs. 41 ± 8 bpm, $p < 0.001$). However, changes in oxidative stress markers showed no significant gender differences, suggesting similar cellular stress responses despite differences in physiological adaptations.

Correlations

Strong positive correlations were observed between core temperature elevation and MDA increase ($r = 0.72$, $p < 0.001$). Heart rate changes correlated significantly with SOD activity reduction ($r = -0.58$, $p < 0.001$) and GSH depletion ($r = -0.61$, $p < 0.001$). The sweat rate showed negative correlations with antioxidant enzyme activities, suggesting that individuals with higher sweat production experienced greater oxidative stress.

Discussion

The present study represents the first comprehensive evaluation of the acute heat stress reaction in healthy adult participants from Almuthana, Iraq, an area characterized by extreme ambient temperatures. Our results indicate that controlled heat exposure (1 hour at 48°C) induces substantial strain and increases oxidative stress (9 minutes at 35°C). The implications for public health and safety of workers in high-temperature environments are significant.

The 2.1°C rise in core body temperature after 60 minutes of heat exposure is a significant increase and indicates substantial thermal stress (23), approaching temperatures associated with heat exhaustion. The 2.1°C rise in core temperature is in agreement with previously published studies conducted in similar environmental settings. Most research has been conducted in a laboratory with subjects from temperate climates (24). Participants from a region with extreme heat still showed substantial thermal responses, which suggests that chronic heat exposure does not provide complete physiological tolerance against acute heat stress.

The cardiovascular response included an increase in heart rate (64%) and a rise in blood pressure, indicating significant cardiovascular strain due to the prolonged duration of heat exposure. This is in keeping with the original physiological principles of a heat stress response, as the demand for increasing cardiac output is to reduce core temperature through peripheral vasodilation and perfusion of vital organs (25). The cardiovascular strain demonstrated by this study appears to be greater than reported in many previous studies, indicating significant stress on the participants and/or physiological differences among them due to their exposure to extremely high heat. The differences in physiological outcomes between genders align with previous research, which confirms that males are more susceptible to heat stress than females (26). The larger shifts in core temperature and heart rate in males may be attributed to differences in body composition, variations in sweating response patterns, and/or variations in cardiovascular fitness. In general, males produce significantly more metabolic heat, which may lead to a greater dependency on cardiovascular adaptations to reduce heat load, compared to females who have more effective sweating patterns and can sustain peripheral vasodilation.(27)

The results related to oxidative stress represent a valuable contribution to the field of heat stress research, particularly for populations that are chronically heat acclimatized. The 68% increase in malondialdehyde levels indicated significant lipid peroxidation and cell membrane damage following an acute period of heat stress. This is important because it adds to the body of literature, suggesting that even a long-acclimated population exposed to high ambient temperatures will experience significant oxidative stress after acute heat stress.

The simultaneous reductions in the activities of the antioxidant enzymes (SOD, catalase) and the concentrations of reduced glutathione suggest that the antioxidant defense system has been overwhelmed in response to acute heat stress. The reductions of 23-31% in antioxidant capacity observed in this study represented a considerable magnitude of loss in potential cellular protection and recovery from acute heat exposures. These observations were consistent with previous research that demonstrated heat-induced oxidative stress in laboratory animal studies and limited research with humans.(28)

The strength of correlations between the physiologic parameters and oxidative stress markers suggests that the mechanisms we posited are linked to the underlying heat stress response. In particular, the positive correlation between the rise in core temperature and malondialdehyde concentrations suggests that oxidative damage is directly resulting from thermal stress. Similarly, negative correlations between heart rate changes and antioxidant enzyme activities imply that cardiovascular strain contributes to the depletion of cellular protective mechanisms.

These results have important implications for understanding the pathophysiology of heat-related illness. The combination of cardiovascular strain and oxidative stress could facilitate the progression from heat exhaustion to heat stroke, when cellular dysfunction and multi-organ failure may occur (29). Additionally, the oxidative stress component can contribute to the long-term health effects of repeated heat exposure, including cardiovascular disease and accelerated biological aging .

From a public health perspective, the study reports the sensitivity of even heat-adapted populations to acute thermal stress. Because the significant physiological and biochemical changes observed in previous research followed a relatively short duration of heat exposure, it stands to reason that significantly more extreme physiological outcomes and responses could occur following heat exposure of longer duration, as is common for outdoor workers and other vulnerable populations. Understanding this vital information is crucial to developing evidence-based heat stress prevention strategies and occupational safety recommendations applicable to extreme heat settings.

Furthermore, the lack of significant gender differences in oxidative stress responses, despite differences in physiological parameters, suggests that cellular protection mechanisms may be equally susceptible in both men and women. This finding is beneficial when developing gender-

neutral strategies for antioxidant protection during heat exposures, while retaining gender-specific approaches for cardiovascular protection.

There are, of course, limitations to our current study. The controlled laboratory environment allowed researchers to provide easily replicable exposure conditions; however, the thermal environment may not wholly replicate the complex environments commonly experienced in the real world. The single acute exposure protocol does not account for not only the cumulative effects of repeated heat stress or acclimatization that occur over time, but also for the study population, which, while representative of a local population, cannot be assumed to be generalizable to different populations with different acclimatization and genetic makeups.

The cross-sectional design of the study limits the ability to investigate individual variability in thermal responses and recovery properly. Longitudinal investigations of repeated heat exposures and recovery kinetics will provide valuable knowledge into adaptive mechanisms and individual susceptibility factors. Moreover, interventions such as antioxidant supplementation and heat acclimatization protocols should also be investigated to provide evidence-based practical strategies in response to heat stress.

The study's conclusions also raised some serious questions regarding the health consequences of acclimating to live in extreme heat for prolonged periods. While the first to document oxidative stress following acute exposure to extreme heat, considerable oxidative stress (relatively) was observed following acute exposures and raises the possibility that lower levels of oxidative damage may accumulate over time, resulting in adverse health consequences, including the acceleration of aging and increased risk of disease. This possibility should be explored in epidemiological studies that assess health outcomes in populations exposed chronically to extreme heat.

Climate change projections indicate that extreme heat events will continue to intensify and increase in frequency globally. Therefore, understanding thermal stress physiology will become increasingly important for future public health plans (30). The outcomes of this research, conducted in an extreme heat setting, will likely reflect the physiological effects that resident populations of areas undergoing increasing ambient temperature change will experience .

The potential applications of our results include occupational health, urban planning, and preparedness within health care systems. The observed physiological strain and oxidative stress experienced by healthy adults in this study suggest that vulnerable populations (e.g., elderly, children, and those with chronic illnesses) are even more at risk of harm during extreme heat events. This highlights the need for targeted, actionable initiatives and direct monitoring during heatwave events.

Conclusion

The current study demonstrates that healthy adults residing in Almuthana, Iraq, who are exposed to acute heat stress, experience significant physiological strain and oxidative stress despite anticipated acclimatization or adaptation to extreme heat environments. The evident elevation in core temperature, cardiovascular strain, and pro-oxidative markers suggests that human physiology is vulnerable to extreme thermal exposure, even in environments with high heat .

Furthermore, for these heat-exposed healthy individuals, males experienced greater physiological stress than females; however, the oxidative stress responses were equivalent among the genders. The strong relationships observed in the correlations between physiological metrics and oxidative stress metrics suggest interrelated pathways that accentuate the pathophysiology of heat stress, but are nevertheless salient for the development of heat illness .

From a public health perspective, previous studies on heat stress have demonstrated the need for plans to mitigate heat stress, best practices for occupational health and safety, and heat preparedness in extreme heat environments. Given the significantly high levels of oxidative stress,

there is a need for further study of antioxidant interventions, and if possible, reduced exposure time.

As the planet continues to warm due to climate change, it will be increasingly important for public health and scientific communities to begin modeling estimates of the physiological consequences on human health of extreme heat environments. Our study contributes to the understanding of heat stress and oxidative responses in extreme heat, as this sample resides in arguably the most extreme thermal conditions on Earth. Our data is vital for descriptive purposes, supporting future climate adaptation monitoring and modeling protocols .

Future research into the longitudinal and acclimatization effects of heat-stress responses to occupational exposures, as well as the chronic physiological impacts of heat, will be necessary to develop evidence-based protocols that protect the health of humans living in a warming world.

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