Wilderness and Environmental Medicine, **19,** 238–244 (2008)

ORIGINAL RESEARCH

The Influence of Ethnicity on Thermoregulation After Acute Cold Exposure

Gregory S. Farnell, PhD; Katie E. Pierce, MA; Tiffany A. Collinsworth, MEd; Leigh K. Murray, PhD; Rob N. Demes, MA; Judi A. Juvancic-Heltzel, MA; Ellen L. Glickman, PhD, FACSM

From the Department of Kinesiology and Health Studies, University of Central Oklahoma, Edmond, OK (Dr Farnell); the Department of Exercise, Leisure, and Sport, Kent State University, Kent, OH (Mses Pierce, Collinsworth, and Juvancic-Heltzel, Mr Demes, and Dr Glickman); and the Department of Physical Therapy, Walsh University, North Canton, OH (Dr Murray).

Objective.—It is well established that a combination of factors, including ethnicity, may influence an individual's response to cold stress. Previous work from our laboratory has demonstrated that when faced with a cold challenge, there is a similar response in heat production between Caucasian (CAU) and African American (AA) individuals that is accompanied by a differential response in core temperature. The objective of this study was to evaluate the influence of ethnicity (CAU vs AA) on the thermoregulatory response after acute cold exposure (ACE-REC, 25°C air).

Methods.—Five AA males (20.8 ± 0.5 years) and 10 CAU males (25.6±4.9 years) underwent pre-experimental testing to determine Vo_{2max} (AA = 37.2 ± 0.1 $mL \cdot kg^{-1} \cdot min^{-1}$, CAU = 44.3 ± 8.7 $mL \cdot kg^{-1} \cdot min^{-1}$) and body composition (AA = 14.6 ± 5.4%, CAU = 19.2 ± 5.0%). Participants underwent acute cold exposure that consisted of 120 minutes of exposure to 10°C air (ACE) followed by 120 minutes of recovery in 25°C air (ACE-REC). Rectal temperature (T_{re}) was measured via a rectal thermistor. Mean skin temperature (T_{sk}) was assessed with thermistors. Oxygen consumption (Vo_2) was assessed via indirect open circuit spirometry. Rectal temperature and T_{sk} were measured continuously, and if $T_{re} \leq 35°C$, testing was terminated.

Results.—Analysis of variance for ACE-REC revealed a significant main effect for T_{sk} across time ($P < .001$), T_{re} across time ($P < .001$), and Vo_2 across time ($P < .001$). In addition, a significant time × ethnicity interaction was revealed for T_{re} ($P = .008$), T_{sk} ($P = .042$), and Vo_2 ($P = .019$) during ACE-REC.

Conclusions.—Based on these data, there is a differential response between CAU and AA across time for Vo_2, T_{re}, and T_{sk} ACE-REC.

Key words: African American, Caucasian, ethnicity, thermal, metabolic, cold

Introduction

When an individual is exposed to environmental temperature extremes, the challenge is to maintain thermoregulatory homeostasis. Humans attempt to maintain thermoregulation through the integration of different mechanisms, acting in concert with a variety of contributing factors. The primary physiological responses include an increase in metabolism (shivering thermogenesis), an alteration in the vasomotor response (peripheral vasoconstriction/vasodilation),[1] and a circulatory response (countercurrent heat mechanism). Additionally, such factors as fitness level, body composition, age, gender, and race may also influence the individual's ability to thermoregulate.[2–7]

An understanding of how different individuals thermoregulate is valuable to those who engage in outdoor activities for prolonged periods. The majority of current literature in the field focuses on the cold exposure portion, whereas little research has examined the recovery phase. Acute cold exposure (ACE) has been shown to increase the clotting response during the recovery period, making individuals more susceptible to negative cardiovascular events,[8–10] such as heart attacks and strokes. Research in the area of cold exposure has also focused primarily on the Caucasian (CAU) male with very little

Corresponding author: Gregory S. Farnell, PhD, University of Central Oklahoma, 100 N. University Dr #189, Edmond, OK 73034 (e-mail: gfarnell@ucok.edu).

Table. Study group participant characteristics*

	CAU (n = 10)	*AA (n = 5)*
Age (y)	25.6 ± 4.9	20.8 ± 0.5
Weight (kg)	85.5 ± 17.8	85.5 ± 12.6
Height (cm)	175.7 ± 8.7	180.0 ± 8.8
Body fat (%)	19.2 ± 5.0	14.6 ± 5.4
Lean body mass (kg)	68.5 ± 11.2	70.9 ± 7.8
Body surface area (m^2)	2.0 ± 0.2	2.1 ± 0.2

*Values are expressed as mean ± SD. CAU indicates Caucasian; AA, African American.

research available concerning different ethnicities and how they may thermoregulate to the stressor of cold.

It has been well documented that African Americans (AA) suffer a disproportionately higher rate of cardiovascular disease.[11] Cardiovascular reactivity has also been shown to be greater among AA after a variety of stressors.[11] Further, it is unclear whether morphological or ethnic differences cause potential alterations in cold responsiveness between groups or whether the alterations are due to differences in thermosensitivity. McArdle et al suggests that the differences in thermoregulation at rest during cold stress may be due in part to the sensitivity of the thermogenic response.[12] Previous work from our laboratory has demonstrated that CAU individuals thermoregulate to cold stress by maintaining a higher metabolic rate than their AA counterparts.[1] Although numerous studies have demonstrated that certain physiological changes may ensue with cold stress, research in the area of ethnicity and how the individual may respond to this stressor is limited.[6,13–18] Therefore, the impact of ethnicity (eg, AA vs CAU) on thermoregulation warrants investigation. In an effort to establish possible differences that are involved in thermoregulation, the recovery phase of acute cold exposure (ACE-REC) was explored to discern if individuals who varied on the basis of ethnicity would demonstrate a differential response after ACE. In this differential response, the return of cooled peripheral blood after ACE for 120 minutes may elicit differences between individuals based on ethnicity.

In an effort to establish possible differences involved in thermoregulation between AA and CAU males, we studied ACE-REC to evaluate whether there is a differential thermoregulatory and metabolic response after ACE in individuals of these races.

Methods

Fifteen males (5 AA, 10 CAU) participated in this study. Participant characteristics are presented in Table 1.

Each participant experienced 120 minutes of exposure to 10°C air (ACE) and recovery (ACE-REC) wearing only a swimsuit for 120 minutes or until rectal temperature (T_{re}) was ≤35°C. The complete air exposure trial consisted of 3 periods: a 15-minute baseline (BASE) period followed by a 120-minute ACE, followed by a 120-minute recovery (ACE-REC) in 25°C air. Throughout the ACE and ACE-REC, T_{re}, skin temperature (T_{sk}), expired air, and heart rate (HR) were continuously monitored and metabolic rate ($\dot{V}O_2$) was measured at 15-minute increments. The protocol was reviewed and approved by the Kent State University Institutional Review Board. Participants completed a health history questionnaire and signed an informed consent. All trials were completed at the same time of the day and within 12 weeks.

BASELINE PERIOD

Upon arrival to the Applied Physiology Laboratory, participants had their body weight measured. They were then instructed to insert a rectal probe (ER400-12, Respiratory Diagnostic Products Inc, Irvine, CA) 13 cm beyond the anal sphincter for the purpose of monitoring T_{re}. Participants were also outfitted with skin thermistors (Model #409B, Yellow Springs Instruments Inc, Yellow Springs, OH) to measure mean skin temperature. Temperature-sensing thermistors were placed on the tricep, chest, thigh, calf, and forearm and secured with Hy-tape waterproof tape (Hy-tape International, Patterson, NY) for the purpose of collecting T_{sk} data. Rectal temperature and T_{sk} thermistors were then interfaced with a data acquisition system (iNet-100HC, Omega Engineering Inc, Stamford, CT), which was interfaced with a desktop computer. Mean skin temperature was calculated using the weighted formula used by Ramanathan.[19] Rectal temperature and T_{sk} data were monitored continuously and recorded every minute.

After instrumentation, the participant sat quietly, with arms and legs extended and separated, in a semirecumbent position on a plastic lounge chair outside of, and next to, the environmental chamber in a 24–26°C air environment for 15 minutes. During the BASE period, expired air samples, T_{re}, T_{sk}, $\dot{V}O_2$, and HR were monitored. Oxygen consumption was assessed via indirect open circuit spirometry with a Parvo metabolic cart (TrueOne 2400, Parvomedics, Sandy, UT) that was calibrated before and after data collection.

COLD AIR EXPOSURE/RECOVERY PERIOD

After completing the BASE period, participants were rolled into the environmental chamber, where they were required to remain seated and quiet in the chair with