



***Peripheral Sweat Gland Function,
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Increases in Women Following
Humid Heat Acclimation***

***M. J. Buono
S. L. Martha
J. H. Heaney***



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***Naval Health Research Center
140 Sylvester Road
San Diego, California 92106***



Peripheral sweat gland function, but not whole-body sweat rate, increases in women following humid heat acclimation

Michael J. Buono^{a,*}, Sandra Leichter Martha^a, Jay H. Heaney^b

^a San Diego State University, MC-7251, San Diego, CA 92182, USA

^b Naval Health Research Center, 141 Sylvester Rd., San Diego, CA 92106, USA

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ABSTRACT

The purpose of this study was to compare both the whole-body and pharmacological-induced sweat rates of men and women following humid heat acclimation.

Whole-body sweat rate was significantly ($P < 0.05$) increased 20% in men following heat acclimation; however, it was essentially unchanged in women. The most important new finding was that humid heat acclimation produced a significant ($P < 0.05$) 60–70% increase in pilocarpine-induced sweat rate in both men and women.

These results suggest that humid heat acclimation significantly improves peripheral sweat gland function equally in both men and women. However, during exercise in humid heat, the increased peripheral sweat capacity in women is suppressed via either pre- or post-glandular mechanisms, thus limiting wasteful sweat production.

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1. Introduction

Numerous studies (Eichna et al., 1950; Garden et al., 1996; Hertig et al., 1963; Moran et al., 1999; Sawka et al., 1983) have shown that both men and women can successfully acclimate to heat. Specifically, following heat acclimation (HA) both men and women demonstrate similar reductions in traditional indicators of physiological stress, such as heart rate, core temperature, and skin temperature (Frye and Kamon, 1983; Horstman and Christensen, 1982). However, one area of gender difference is that whole-body sweat rate is increased to a much greater extent following humid heat acclimation in men than in women (Avellini et al., 1980; Frye and Kamon, 1981; Weinman et al., 1967; Wyndham et al., 1965). For example, Avellini et al. (1980) reported that following 10 days of humid heat acclimation whole-body sweat rate (WBSR) increased significantly more for men than women (35% vs. 15%). Since the high ambient vapor pressure associated with humid environments restricts evaporation, the prolific sweating seen in men following humid heat acclimation results in the wasteful dripping of unevaporated sweat (Frye and Kamon, 1981; Wyndham et al., 1965).

The physiological mechanism by which women prevent the wasteful increase in sweat production following humid heat acclimation is currently unknown. It has been suggested that women have a more sensitive feedback system from the wetted

skin surface, thus preventing excessive dripping of unevaporated sweat (Avellini et al., 1980; Shapiro et al., 1980), or that they respond to hydromeiosis sooner than men (Weinman et al., 1967; Wyndham et al., 1965). Both of these hypotheses imply that peripheral sweat gland function in women is increased following humid heat acclimation, but it is somehow suppressed via either a pre- or post-glandular mechanism, thus limiting wasteful sweat production. However, a review of the literature shows that no study has previously been published that examines both the whole-body and pharmacological-induced sweat rates of women and men before and after humid, heat acclimation. Thus, it is currently not known if peripheral sweat gland function increases to a similar extent in men and women following humid heat acclimation.

Therefore, the purpose of this study was to compare both the whole-body and pharmacological-induced sweat rates of men and women following humid heat acclimation.

2. Methods

The subjects for this study were 13 male and 20 female volunteers. The men had a mean $\pm$ SE age, height, and weight of 22.5 ± 0.9 y, 175.4 ± 2.1 cm, and 78.20 ± 2.01 kg, respectively. The women had a mean $\pm$ SE age, height, and weight of 23.5 ± 1.2 y, 162.2 ± 1.9 cm, and 67.20 ± 2.23 kg, respectively. Nine of the women were currently using some form of hormonal birth control. There were no differences in the responses seen in women who were or were not using hormonal birth control, thus

* Corresponding author. Tel.: +1 619 594 6823.

E-mail address: mbuono@mail.sdsu.edu (M.J. Buono).

the data were combined. Furthermore, no attempt was made to control for menstrual phase since it appears to have minimal effects on the responses seen during heat acclimation (Frye and Kamon, 1983; Kenney, 1985). The study was approved by the Naval Health Research Center IRB and prior to testing and data collection subjects were briefed on the purpose and procedures of the study and signed informed consent was obtained. Each subject reported to the laboratory at approximately 7 a.m. for 8 consecutive days, excluding Sundays. A urine sample was immediately collected and measured for specific gravity and to ensure that none of the female subjects was pregnant. If need be, subjects consumed 200 ml of water every 15 min during seated rest until their urine specific gravity was ≤ 1.025 . Next, on days 1 and 8, prior to performing that day's HA trial, peripheral sweat production was induced via pilocarpine iontophoresis on the proximal half of the flexor surface of both forearms. The subject was seated during the data collection and the room temperature was between 22 and 24 °C. The reported value was the mean of both forearms. The iontophoresis current was fixed at 1.5 mA for 10 min, and was applied using a Wescor sweat inducer (model 3700, Logan, UT). Pilocarpine, an acetylcholine agonist, was delivered via reagent-impregnated (0.5%) solid agar gel discs (Pilogel, Wescor). Sweat was collected for 15 min immediately after iontophoresis using a Wescor Macroduct sweat collection system using the procedures outlined by Webster (1983). A different forearm site was used on each of the measurement days to avoid any potential effects of desensitization of the sweat glands from repeated pharmacological stimulation (Chen and Elizondo, 1974). The test-retest correlation and intrasubject coefficient of variability for the pilocarpine-induced sweat rate procedure has been reported to be $r=0.85$ and 15%, respectively (Buono et al., 1991).

Each day during HA the subjects completed 2 h of exercise in an environmental chamber. The ambient temperature and relative humidity were controlled at 35 °C and 75%, respectively. The exercise consisted of four 25 min intervals with 5 min of seated rest in the environmental chamber between each work interval. Water was allowed ad libitum during the 2 h heat exposures.

The four exercise intervals included two bouts of walking on a motorized treadmill (3 mph, 3% grade) and two bouts of cycle ergometry at a power output of 60 W. Each day the order of the first three exercise intervals was randomized; however, the fourth and final exercise interval for all subjects, during which time the end-exercise HR, rectal temperature and skin temperature data were collected, was always walking on the treadmill. The alternating treadmill walking and cycle ergometry protocol was well tolerated by all the subjects and allowed for multiple subjects to be tested simultaneously. Oxygen uptake measured by open-circuit spirometry during the exercise intervals was approximately 1 L min⁻¹. Core body temperature was measured each minute during the exercise bouts using a thermistor (YSI 400 series) inserted 15 cm past the anal sphincter. Skin temperatures were measured on the right chest, shoulder, thigh and calf using thermistors (YSI 400 series) taped to the skin. Mean skin temperature was calculated as the unweighted mean of the four sites. Heart rate was measured every minute using a Polar monitor. Whole-body sweat rate, expressed in L m⁻² h⁻¹, was calculated by measuring nude, dry pre- and post-exercise body weight, corrected for fluid intake and urine production, on a scale accurate to ± 0.01 kg. DuBois (1916) body surface area was determined based on height and weight measurements.

Dependent *t* tests, using the Bonferroni correction, were used to analyze pre- versus post-acclimation end-exercise HR, core temperature, mean skin temperature, WBSR, and pilocarpine-induced sweat rate in both men and women. Significance was set at $P < 0.05$.

3. Results

The mean $\pm$ SE rectal temperature during exercise was significantly reduced during the eight days of HA in both men and women. As shown in Fig. 1, end-exercise rectal temperature in men fell from 38.9 ± 0.2 °C on day 1 to 38.4 ± 0.1 °C on day 8, while in women it decreased from 39.0 ± 0.1 °C on day 1 to 38.6 ± 0.1 °C on day 8. In addition, as shown in Fig. 2, ending HR during exercise was also significantly reduced in both men and women following HA. In the men it fell from 154 ± 6 bpm on day 1 to 137 ± 6 bpm on day 8, while in the women it decreased from 154 ± 5 bpm on day 1 to 146 ± 4 bpm on day 8 of HA. The magnitude of the decrease in rectal temperature and HR is consistent with the results of previous studies (Avellini et al., 1980; Pandolf et al., 1988; Strydom et al., 1966). Such reductions strongly suggest that the 8-day protocol used in the current study was successful in conferring HA in both groups of subjects. The

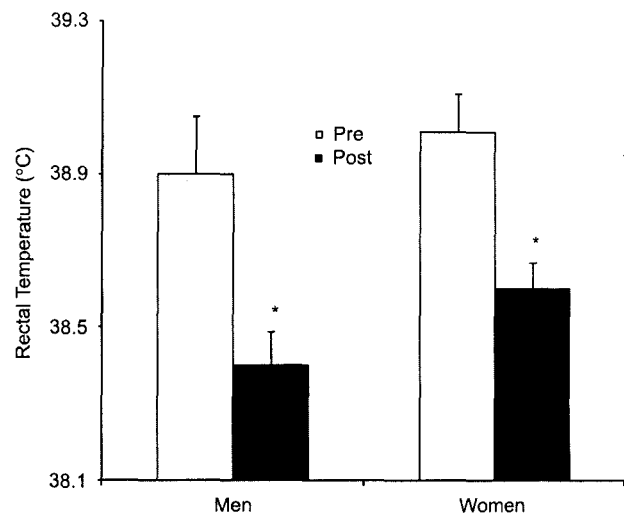


Fig. 1. The values are mean $\pm$ SE end-exercise rectal temperature for men and women measured pre- and post-acclimation. *Significantly ($P < 0.05$) different from the preacclimation value.

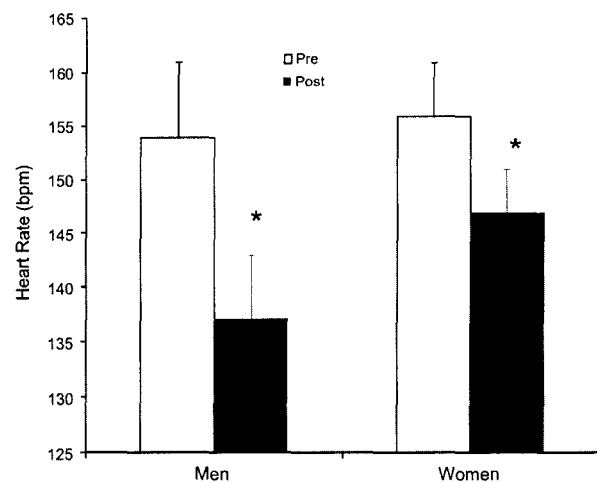


Fig. 2. The values are mean $\pm$ SE end-exercise heart rate for men and women measured pre- and post-acclimation. *Significantly ($P < 0.05$) different from the preacclimation value.

mean $\pm$ SE pre- and post-acclimation WBSR and pilocarpine-induced sweat rate for both groups are presented in Figs. 3 and 4, respectively. In men the WBSR significantly increased from 0.55 ± 0.06 to $0.66 \pm 0.05 \text{ L m}^{-2} \text{ h}^{-1}$, or approximately 20%, as a result of HA. Women, on the other hand, did not have a significant increase in WBSR during the 8 days of HA. Their mean $\pm$ SE WBSR was 0.41 ± 0.02 and $0.42 \pm 0.02 \text{ L m}^{-2} \text{ h}^{-1}$ on days 1 and 8, respectively. The most important new finding of the current study was that HA produced a significant 60–70% increase in pilocarpine-induced sweat rate in both men and women (Fig. 4). Specifically, it increased from $0.64 \pm 0.06 \text{ mg cm}^{-2} \text{ min}^{-1}$ on day 1 to $1.04 \pm 0.11 \text{ mg cm}^{-2} \text{ min}^{-1}$ on day 8 in the men, while it increased from 0.34 ± 0.03 to $0.57 \pm 0.04 \text{ mg cm}^{-2} \text{ min}^{-1}$ during the same time period in women. Thus, the mean increase in both groups was essentially identical (63% for males, 68% for females). The end-exercise mean skin temperature, on days 1 and 8, for men was 37.3 ± 0.2 and 37.0 ± 0.2 °C, respectively. In women it fell from 37.6 ± 0.1 on day 1 to 37.3 ± 0.1 °C on day 8. Thus, both the men and women had a 0.3 °C reduction in mean skin

temperature following HA. However, the change was not significant in the men while it was significant in the women.

4. Discussion

Four previous studies have measured the change in sweating that occurs in men and women following humid heat acclimation (Avellini et al., 1980; Frye and Kamon, 1981; Weinman et al., 1967; Wyndham et al., 1965). Wyndham et al. (1965) exercised 10 men and 4 women for approximately 10 days in humid heat conditions. Although both groups increased their WBSR during the acclimation period, the increase was much greater in the male versus the female subjects. Specifically, during the second and third hours of exercise, the men's WBSR increased by 92% following acclimation, while the women's WBSR increased only 60%. These findings led the authors to conclude, "the male in hot, humid conditions is a prolific, wasteful sweater whereas the female adjusts her sweat rate better to the required heat loss."

Weinman et al. (1967) acclimated 5 men and 5 women for 8 days in humid heat (34 °C, 88% RH). Following heat acclimation, the women had a nonsignificant 9% increase in WBSR during a 4 h exercise bout while the men showed a significant 42% increase in WBSR. Frye and Kamon (1983) reported that following dry heat acclimation the WBSR and chest sweat rate of men and women were identical. However, following humid heat acclimation the men had a 25% greater chest sweat rate than the women.

The results of the aforementioned studies (Avellini et al., 1980; Frye and Kamon, 1981; Weinman et al., 1967; Wyndham et al., 1965) clearly suggest that after humid heat acclimation WBSR increases to a much greater extent in men compared with women. The results of the current study agree with these previous findings. Fig. 3 shows that the mean WBSR in men increased 20% as a result of 8 days of humid heat acclimation, while the women's value was essentially unchanged during the same time period.

The physiological mechanism that prevents the wasteful increase in WBSR in females following humid heat acclimation, thus limiting the excessive dripping of nonevaporated sweat, is unknown. It has been suggested that women have a more sensitive feedback system from the wetted skin surface (Avellini et al., 1980). However, the skin sensors responsible for such a negative feedback mechanism have yet to be identified (Candas et al., 1983). It has also been suggested that women respond to hydromeiosis sooner than men (Shapiro et al., 1980; Wyndham et al., 1965). It is generally believed that hydromeiosis is the result of wetted skin causing the stratum corneum to swell, thus causing mechanical obstruction of the sweat duct (Peiss et al., 1956). This is supported by Brown and Sargent (1965) who found that during prolonged heat exposure the decrease in sweat rate was reversed if the subject was moved from a humid to a dry environment. Furthermore, they reported that small areas of the skin that were stripped of the stratum corneum maintained greater sweat outputs compared with intact areas of the skin. Such findings may help to explain the differences in sweat rate seen in men and women following humid heat acclimation. If ductal occlusion is the cause for hydromeiosis, then it does not seem unreasonable to hypothesize that the larger the sweat duct orifice, the less sensitive the gland would be to closing due to swelling of the stratum corneum (i.e., less hydromeiosis). Since men generally have larger sweat glands than women (Sato, 1993) and heat acclimation has been shown to result in glandular hypertrophy (Sato et al., 1990), it may be that men are less susceptible to hydromeiosis, especially after heat acclimation, due to the physical size of their eccrine glands (i.e., larger diameter lumen). Such a hypothesis is constant with previous results

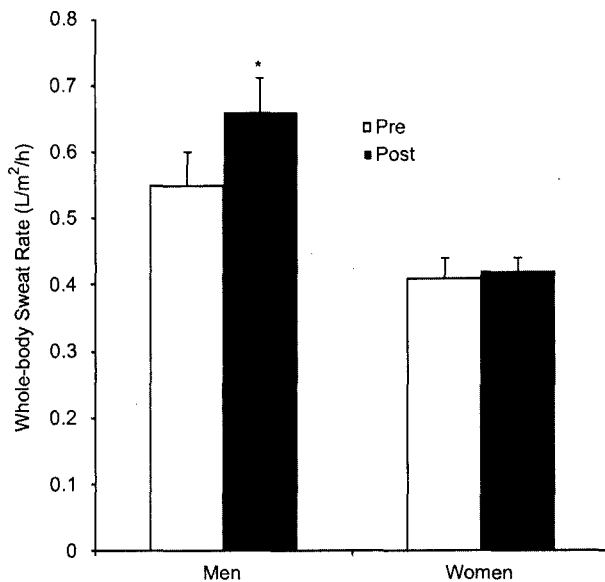


Fig. 3. The values are mean $\pm$ SE whole-body sweat rate for men and women measured pre- and post-acclimation. *Significantly ($P < 0.05$) different from the preacclimation value.

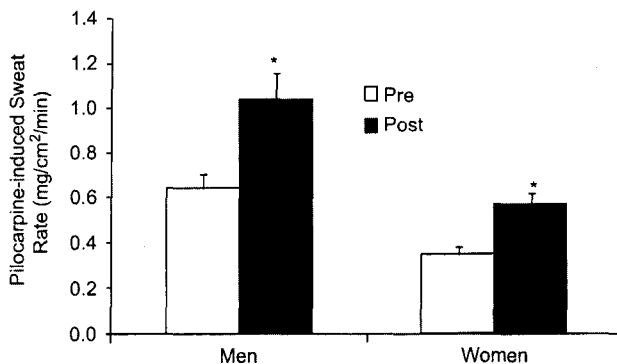


Fig. 4. The values are mean $\pm$ SE pilocarpine-induced sweat rate for men and women measured pre- and post-acclimation. *Significantly ($P < 0.05$) different from the preacclimation value.

(Shapiro et al., 1980) that showed that women reduced sweat loss during humid heat exposure primarily through a reduction in the number of actively secreting sweat glands.

The most important new finding of the current study was that heat acclimation significantly increased the pilocarpine-induced sweat rate 60–70% in both male and female subjects (Fig. 4). To our knowledge this is the first study to compare pharmacological-induced and whole-body sweat rates in men and women before and after humid, heat acclimation. The importance of such findings is twofold. First, they strongly suggest that the relatively smaller WBSR previously seen in women versus men after humid heat acclimation (Avellini et al., 1980; Frye and Kamon, 1981; Weinman et al., 1967; Wyndham et al., 1965) is not due to a lack of improvement in eccrine sweat gland function. In fact, humid heat acclimation increased the sweating capacity of both men and women by approximately the same magnitude (Fig. 4). However, the increased peripheral sweat capacity in women is somehow suppressed during exercise in humid conditions, thus limiting wasteful sweat production. Second, the increased peripheral sweating capacity seen in women following heat acclimation, although suppressed during humid conditions, should be able to be realized for evaporative cooling during dry heat exposure. This helps to explain the findings of three previous studies (Frye and Kamon, 1981, 1983; Shapiro et al., 1980), all of which reported that following heat acclimation, women have a significantly smaller WBSR than men in humid environments, but a similar WBSR under dry conditions. For example, Frye and Kamon (1981) reported that following 9 days of HA, the WBSR of men and women during exercise in a dry environment were nearly identical, while during exercise in a humid environment the women had a value that was approximately 20–25% less than the men.

It is interesting to note that the women demonstrated a significant decrease in rectal temperature following HA (Fig. 1) even though they did not have a significant increase in WBSR (Fig. 3). The physiological mechanisms responsible for this are not known, but probably include some combination of a lower resting core temperature (Lee et al., 2009), a lower threshold for the onset of sweating (Roberts et al., 1977), a more uniform sweating response between the trunk and limbs (Hofler, 1968) and greater non-evaporative heat loss following HA.

In conclusion, the results of the current study suggest that humid heat acclimation improves peripheral sweat gland function, as measured by pilocarpine iontophoresis, equally in both men and women. However, during exercise in humid heat, the increased peripheral sweat capacity of women is suppressed via either a pre- or post-glandular mechanism, thus limiting wasteful sweat production.

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The views expressed in this article are those of the authors and do not reflect the official policy or position of the Department of the Navy, Department of Defense, or the U.S. Government. This research has been conducted in compliance with all applicable federal regulations governing the protection of human subjects in research.

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