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Control of sweating during the human menstrual cycle

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Summary. Thermoregulatory responses were studied in seven women during two separate experimental protocols in the follicular (F, days 4–7) phase and during the luteal (L, days 19–22) phase of the menstrual cycle. Continuous measurements of esophageal temperature (T_{es}), mean skin temperature ($\bar{T}_{sk}$), oxygen uptake and forearm sweating ($\dot{m}_s$) were made during all experiments. Protocol I involved both passive heat exposure (3 h) and cycle exercise at ~80% $\dot{V}_{O_2}$ peak during which the environmental chamber was controlled at $T_a = 50.0°$ C, rh = 14% ($P_w = 1.7$ kPa). In protocol II subjects were tested during thirty-five minutes of exercise at ~85% $\dot{V}_{O_2}$ peak at $T_a = 35°$ C and rh = 25% ($P_w = 1.4$ kPa). The normal L increase in resting T_{es} ($\approx 0.3°$ C) occurred in all seven subjects. $\bar{T}_{sk}$ was higher during L than F in all experiments conducted at 50° C. During exercise and passive heat exposure, the T_{es} threshold for sweating was higher in L, with no change in the thermosensitivity (slope) of $\dot{m}_s$ to T_{es} between menstrual cycle phases. This rightward or upward shift in T_{es} threshold for initiation of sweating averaged 0.5° C for all experiments. The data indicate the luteal phase modulation in the control of sweating in healthy women is also apparent during severe exercise and/or heat stress.

Key words: Exercise — Females — Menstrual cycle — Sweating — Thermoregulation

Introduction

The elevation in resting core temperature during the luteal phase of the human menstrual cycle is believed to be a consequence of altered control of heat loss mechanisms, affected by altered levels of the reproductive hormones (Rothchild 1952), or increased interleukin-1 levels (Cannon and Dinarello 1985). We previously reported an elevated esophageal temperature threshold for onset of both forearm cutaneous vasodilation and forearm sweating in the luteal phase of the menstrual cycle during moderate exercise in a warm environment in the early morning and late afternoon (Stephenson and Kolka 1985). A similar luteal phase elevation in the mean body temperature threshold for heat loss mechanisms and decreased mean body temperature threshold for heat production mechanisms at rest and exercise during the nighttime hours has been reported (Hessemer and Bruck 1985a and 1985b).

The purpose of the present investigation was two-fold; first to demonstrate an alteration in the esophageal temperature threshold for sweating onset during high intensity exercise during the luteal phase experiments, and secondly to evaluate this characteristic threshold shift during passive whole body heating in a severe environment. Two secondary areas of study involved the influence of skin temperature and circulating catecholamines on this response.

Materials and methods

Seven healthy women (Table 1) volunteered to serve as subjects for the experiments following approval of the protocol by the local human use review board. Each subject had a normal menstrual cycle as defined by a regular periodicity. Daily basal body temperature (BBT) was taken during the course of the study to verify that a normal luteal elevation in BBT had occurred (Hessemer and Bruck 1985a; Kleitman and Ramsaroop 1948; Rothchild 1952). Serum levels of progesterone and estradiol were not measured and deemed unnecessary, as the critical factor for luteal phase experiments was an elevated

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Table 1. Individual subject characteristics

Subject	Age (y)	Height (m)	Mass (kg)	DuBois surface area (m^2)	$\dot{V}_{O_2}$ peak ($l \cdot min^{-1}$)
1	32	1.73	64.2	1.77	2.70
2	26	1.62	60.9	1.65	2.65
3	27	1.65	64.0	1.71	2.55
4	30	1.70	59.0	1.68	2.55
5	21	1.63	68.0	1.73	2.64
6	26	1.73	77.8	1.91	3.83
7	24	1.63	56.0	1.57	2.73
$\bar{x}$	26.6	1.67	64.3	1.72	2.81
S.D.	3.6	0.05	7.1	0.11	0.46

resting core temperature. Furthermore, the luteal phase increase in core temperature has been consistently related to elevated circulating progesterone levels (Rothchild 1952).

Each subject recorded her early morning rectal temperature for at least one menstrual cycle before any testing began. Experiments were then conducted in a subsequent menstrual cycle during the follicular phase (days 4–7) or during the luteal phase when basal body temperature was elevated (days 19–22). Follicular and luteal phase experiments were done in a controlled order such that four subjects were tested first in their follicular phase and the remaining three subjects were tested first in their luteal phase. All experiments on a single subject within a specific protocol were completed within approximately 20 days. The subjects were not naturally acclimatized or artifically acclimated to heat, and all were participating in daily running or bicycling activity.

Physiological variables. Two separate experimental protocols were conducted. All subjects were familiarized with the experimental techniques before each study. Peak oxygen uptake was determined for each subject in the week before her first experiment. During this test, the subject was seated in a contour chair positioned behind the cycle ergometer such that when pedalling, her legs were parallel to the floor. The subject performed 5 min of warm-up exercise at 60 or 90 W, depending on her preference. The peak aerobic power test which followed was continuous in nature with incremental increases in workload (~30 W) each two minutes until voluntary termination. The peak aerobic power was determined as a stable oxygen uptake (< 150 ml difference in sequential measurements) with an increase in work load. In general, this peak value is 15 to 20% below the maximal aerobic power measured during a running treadmill test, as the muscle mass involved is significantly less.

Subjects wore cotton shorts, tee-shirt, shoes and socks for all exposures, and had not eaten during the 8-hour period before an experiment. All were well-hydrated. Activity and food intake during the 24 h before any experiment were standardized. In all experiments esophageal (T_{es}) and mean skin ($\bar{T}_{sk}$) temperature (8 thermocouple sites: Nishi and Gagge 1970), oxygen uptake, local sweating from the arm and whole body sweating rate from weight changes (pre-post exercise) were measured. All experiments were done at the same time of day to minimize the circadian effect(s) on heat loss (Stephenson et al. 1984).

Protocol I. Four experiments (0800 h) were conducted on subjects # 1, 2, 3, 4, 5. A separate room from the environmental chamber was used for equilibration and instrumentation. The ambient temperature of this room was adjusted so that each subject felt comfortable and averaged 28.8° C, rh = 17% (ambient water vapor pressure, $P_w = 0.7$ kPa). Two experiments were conducted in which the subject was passively heated ($T_a = 50.4$° C, rh = 14%, $P_w = 1.6$ kPa), one during the follicular phase (days 4–7) and one during the luteal phase (days 19–22) of the menstrual cycle. In the other two experiments, the subjects exercised at approximately 80% $\dot{V}_{O_2}$ peak during both the follicular and luteal phase in the same environment as described above. T_{es}, $\bar{T}_{sk}$ and local skin temperature ($T_{s,i}$) adjacent to the dew point sensor on the forearm were continuously recorded (Hewlett Packard 9816 computer and 3497 A data acquisition system). The dew point sensor was ventilated (500 $ml \cdot min^{-1}$) with ambient air from the chamber. The air flow was calibrated in situ at the end of the experiment. Sweating rates were calculated as described previously (Graichen et al. 1982; Kolka et al. 1987; Stephenson and Kolka 1985) and measured with an accuracy of 0.05 $mg \cdot cm^{-2} \cdot min^{-1}$. Metabolic heat production was estimated from oxygen uptake measurements (open circuit spirometry) taken periodically during the passive experiments and continuously during the exercise experiments. Plasma norepinephrine and epinephrine concentrations were determined by a radioenzymatic technique (Upjohn, Cat-a-Kit) in blood samples drawn at rest in a thermoneutral environment, and after T_{es} had increased ~0.8° C during both passive and exercise experiments. These sampling times were matched for T_{es} during passive and active experiments for each subject during a given cycle phase so that the influence of T_{es} on catecholamine secretion was standardized between active and passive experiments. The experiment was terminated after T_{es} had increased 0.8–1.0° C above initial values or due to undue discomfort. The passive heating experiments lasted approximately 3 h.

In the exercise experiments, the subject began to exercise at approximately 80% $\dot{V}_{O_2}$ peak within 2 min after entering the environmental chamber. Exercise continued until the esophageal temperature had increased by 0.8–1.0° C. The average time of the exercise experiments was 9 min. Blood samples were drawn at the same T_{es} as during the passive experiment for either the follicular or luteal phase.

Protocol II. Subjects 1, 2, 4, 5, 6 and 7 participated in exercise experiments at approximately 85% $\dot{V}_{O_2}$ peak in a $T_a = 35$° C, rh = 25% ($P_w = 1.4$ kPa) environment in both the follicular (days 4–7) and luteal (days 19–22) phase of their menstrual cycle. Core (T_{es}) and skin temperature ($\bar{T}_{sk}$) and arm sweating were measured as described above. After thermal equilibration to 35° C (determined by a constancy of esophageal and skin temperatures), a 20-min rest period was initiated followed by 35 min of exercise. Oxygen uptake was measured during the rest period and frequently during exercise to maintain the desired work intensity. All six subjects were able to complete the thirty-five minutes of exercise. Plasma norepinephrine and epinephrine concentrations were not measured in this protocol. All experiments were started at 0800 h.

Statistical analyses. In all experiments a regression equation was calculated during increasing $\dot{m}_s$ and T_{es} for each subject. The data collected after the subject reached steady state during exercise were not included in the regression equation. The T_{es} threshold (defined as T_{es} intercept) for initiation of thermal sweating was calculated from the regression equation at $\dot{m}_s = 0.06$ $mg \cdot cm^2 \cdot min^{-1}$ (Buettner 1959). An analysis of variance was performed using the individual slopes and thresholds of the $\dot{m}_s$ to T_{es} regression equations. Analysis of variance routines were used to compare all variables at rest, during steady-state exercise or at the time of the blood samples where

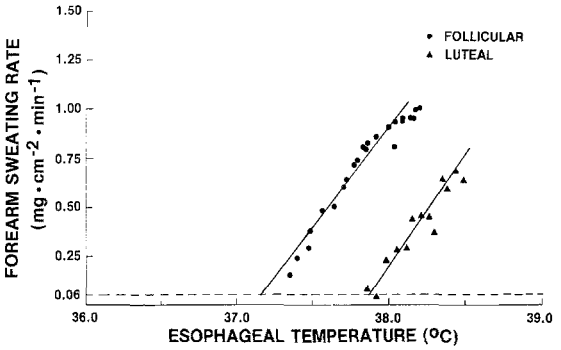

Fig. 1. Forearm sweating versus esophageal temperature during intense exercise in 50° C at 0800 h. The data presented are from a single subject in both the follicular (●) and luteal phases (▲)

appropriate. Tukey's test of critical difference was used when necessary. All data are reported as mean ± SD.

Results

Protocol I exercise

The T_{es} threshold for onset of sweating averaged 36.91 (±0.23)° C in follicular phase experiments and 37.45 (±0.31)° C during luteal phase experiments ($p < 0.05$). Figure 1 illustrates these data in a single subject during the intense exercise. There was no change in the thermosensitivity (gain or slope) of sweating to esophageal temperature in these exercise experiments which averaged 1.10 (±0.12) and 0.95 (±0.24) $mg \cdot cm^{-2} \cdot min^{-1} \cdot °C^{-1}$ for the follicular and luteal phase experiments, respectively. The mean (±SD) thermoregulatory, cardiovascular and hormonal data measured during severe exercise at 50° C are shown in Table 2. The average increase in T_{es} during exercise was 0.8° C which was not different between follicular and luteal experiments. $\bar{T}_{sk}$ was higher in luteal phase experiments during exercise compared to follicular experiments. Plasma norepinephrine concentration increased significantly during exercise, but was not different between follicular and luteal phase exercise experiments.

Protocol I rest

During passive heating, the esophageal temperature threshold for arm sweating averaged 36.60

Table 2. Mean (±SD) thermoregulatory and hormonal data before ($T_a = 28.8°$ C) and during exercise at 80% $\dot{V}_{O_2}$ peak or passive heating ($T_a = 50°$ C)

Time (min)	T_{es} (° C)	$\bar{T}_{sk}$ (° C)	M ($W \cdot m^{-2}$)	$\dot{m}_s$ ($mg \cdot cm^{-2} \cdot min^{-1}$)	HR ($bt \cdot min^{-1}$)	NE	E ($ng \cdot l^{-1}$)	Weight loss ($g \cdot min^{-1}$)
EXERCISE								
Follicular								
Pre	36.96	—[a]	—	—	—	225	92	
	(0.22)					(87)	(31)	
8.6	37.78	37.32	395	1.1	166	1350*	278	16.9
(min)[b]	(0.28)	(0.35)	(44)	(0.4)	(15)	(796)	(126)	(3.6)
Luteal								
Pre	37.21*	—	—	—	—	380*	177	
	(0.27)					(128)	(157)	
8.4	37.98*	37.83*	397	1.1	165	919*	246	17.2
(min)	(0.25)	(0.38)	(26)	(0.4)	(11)	(308)	(123)	(2.8)
PASSIVE HEATING								
Follicular								
Pre	36.97	—[a]	—	—	—	189	60	—
	(0.23)					(105)	(12)	
165	37.69	37.44	39	1.1	83	338	59	7.4
(min)[b]	(0.19)	(0.22)	(7)	(0.4)	(8)	(155)	(32)	(1.3)
Luteal								
Pre	37.28*	—	—	—	—	342*	98	—
	(0.22)					(109)	(43)	
169	37.84*	37.75*	43	1.0	92	545*	183	6.5
(min)	(0.16)	(0.38)	(5)	(0.2)	(8)	(114)	(76)	(1.2)

* Different from follicular $p < 0.05$; ** different from passive $p < 0.05$

T_{es} esophageal temperature; $\bar{T}_{sk}$ mean weighted skin temperature; M metabolic rate calculated from oxygen uptake; $\dot{m}_s$ arm sweating; NE plasma norepinephrine concentration; E plasma epinephrine concentration

[a] These data were not collected in the pre-exercise period

[b] Time listed is when blood samples were taken

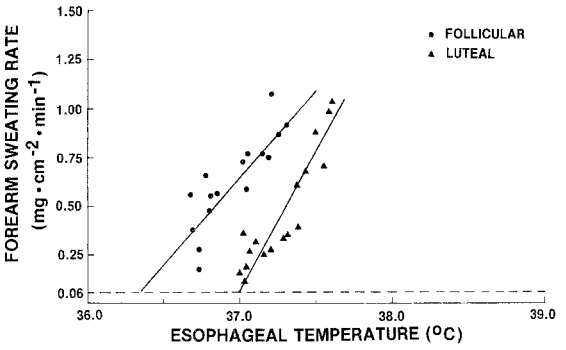

Fig. 2. Forearm sweating versus esophageal temperature during intense exercise in 35° C at 0800 h. The data presented are from a single subject in both the follicular (●) and luteal phases (▲)

(±0.54)° C for follicular phase experiments and 37.08 (±0.32)° C for the luteal phase experiments ($p<0.05$). There was no change in thermosensitivity (gain or slope) between the two menstrual cycle phases which averaged 0.91 (±0.16) and 1.49 (±0.18) $mg \cdot cm^{-2} \cdot min^{-1} \cdot °C^{-1}$ for the follicular and luteal phases, respectively, which were not significantly different from the exercise experiments. The mean (±SD) measured variables for the five subjects are presented in Table 2. Norepinephrine concentration was significantly (74%) higher at rest and during passive heating in the luteal phase compared to follicular phase experiments. $\bar{T}_{sk}$ was significantly higher during luteal phase experiments ($p<0.05$). None of the 5 subjects could remain in the hot environment until T_{es} increased 0.8–1.0° C during the luteal phase. All were removed from the heat complaining of dizziness, headache or nausea.

There were no statistically significant differences in $\bar{T}_{sk}$ within a given cycle phase between passive and exercise experiments. Plasma norepinephrine concentration was higher during exercise than during passive heat exposure in both phases of the menstrual cycle.

Protocol II

The T_{es} threshold for onset of sweating averaged 36.66 (±0.23) in the follicular experiments and 37.08 (±0.20) in the luteal experiments ($p<0.05$). There was no change in the thermosensitivity (gain or slope) of sweating to esophageal temperature which averaged 1.09 (±0.28) and 1.10 (±0.14) $mg \cdot cm^{-2} \cdot min^{-1} \cdot °C^{-1}$ in the follicular and luteal phase experiments, respectively. Rest and exercise (30 min) steady-state temperature and cardiovascular data are shown in Table 3 for the 6 subjects. $\bar{T}_{sk}$ was not different between follicular and luteal experiments ($p=0.18$).

Discussion

Delayed onset time and a higher esophageal temperature for onset of thermoregulatory sweating have been consistently reported in eumenorrheic women during the luteal phase of the menstrual cycle (Bittel and Henane 1975; Haslag and Hertzman 1965; Hessemer and Bruck 1985a and 1985b; Stephenson and Kolka 1985) a finding corroborated during intense exercise and passive heating in hot environments in the present study. The increased mean skin temperature associated with the luteal phase was not an expected observation. Haslag, in an earlier report (1965), demonstrated this same response in two of her three subjects. Kenshalo (1966) has reported that female subjects

Table 3. Mean (±SD) ($n=6$) rest and steady-state (30 min) exercise (85% $\dot{V}_{O_2}$ peak) at $T_a=35°C$

	T_{es} (° C)	$\bar{T}_{sk}$ (° C)	M ($W \cdot m^{-2}$)	$\dot{m}_s$ ($mg \cdot cm^{-2} \cdot min^{-1}$)	HR ($bt \cdot min^{-1}$)	Weight loss ($g \cdot min^{-1}$)
Rest						
Follicular	36.86	35.81	46	0.11	67	—
	(0.17)	(0.26)	(9)	(0.03)	(3)	
Luteal	37.13*	36.09	46	0.07	70	—
	(0.17)	(0.34)	(15)	(0.01)	(4)	
Exercise						
Follicular	38.14	35.14	429	1.05	156	17.0
	(0.29)	(0.62)	(45)	(0.17)	(10)	(4.8)
Luteal	38.39*	35.39	445	0.95	159	17.2
	(0.31)	(0.63)	(46)	(0.24)	(7)	(5.4)

* different from follicular, $p<0.05$

T_{es} esophageal temperature; $\bar{T}_{sk}$ mean weighted skin temperature; M metabolic heat production; $\dot{m}_s$ arm sweating; HR heart rate

"perceived cold at" a warmer skin temperature in the luteal phase; and in a third study, female subjects perceived thermal comfort at a higher core temperature during the luteal phase, when given a cutaneous thermal challenge (Cunningham and Cabanac 1971). Others (Hessemer and Bruck 1985a and 1985b) have not been able to demonstrate differences in skin temperature during the luteal phase of the menstrual cycle, in fact during exposure to 35° C in the current study (Protocol II), the increased skin temperature was not statistically significant.

The slope (gain or thermosensitivity) of sweating to core temperature was not affected at either environmental temperature in the present study during severe exercise or during passive heating which is in agreement with earlier reports (Bittel and Henane 1975; Haslag and Hertzmann 1965; Stephenson and Kolka 1985). However, an increased thermosensitivity of sweating and/or vasodilation during the luteal phase has been reported (Hessemer and Bruck 1985a and 1985b; Hirata et al. 1986). In general, the slope of a thermoregulatory response is influenced by peripheral events, such as the reduction in gain which occurs during hypobaric hypoxic exposure (Kolka et al. 1987), or the increase in gain which occurs as a result of exercise training (Gisolfi and Wenger 1984). The disparate findings between the present study and those of other investigators may be explained by the variable thermal input from the skin. Hirata (1986) used two levels of exercise (40 and 70% $\dot{V}_{O_{2max}}$) at an ambient temperature of 20° C in his experiments, while Hessemer and Bruck (1985b) used exercise (70% $\dot{V}_{O_{2max}}$) at 18° C or passive heating or cooling in their studies, thus, the skin temperature in these earlier studies in general was lower, which has a greater influence on the sweating to esophageal temperature relationship (Elizondo and Bullard 1971; Gisolfi and Wenger 1984).

We imposed stress on the thermoregulatory system by means of intense exercise and by high ambient temperature. In both experiments, the esophageal temperature for sweating onset was higher in the luteal phase. Recent reports of thermoregulatory effector function associated with the human menstrual cycle (Hessemer and Bruck 1985a and 1985b; Hirata et al. 1986; Stephenson and Kolka 1985) over a range of ambient temperatures, hence skin temperatures, point to a luteal phase rightward or upward shift in core or mean body temperature threshold for effector onset. These findings suggest the involvement of a central mechanism (Boulant 1980; Hammel 1968) which may be hormonal or hormone-like such as progesterone (Nakayama et al. 1975; interleukin 1 (Cannon and Dinarello 1985) or some other factor. The thresholds of both autonomic effector organs (sweat gland and cutaneous vasculature) change in a similar manner suggesting a change in central thermoreceptor activity or associated integrative neuronal elements. The increase in core temperature during the circadian cycle is also associated with elevated esophageal temperature thresholds for both forearm cutaneous vasodilation and forearm sweating (Stephenson et al. 1984). A similar effect has been observed during the elevated core temperatures associated with fever and dehydration. During fever, this elevated core temperature threshold appears to be related to the actions of prostaglandins in the pre-optic anterior hypothalamus, but the precise mechanism involved in the modulation of thermoregulation during the circadian cycle, menstrual cycle or dehydration has not been identified.

The effect of the menstrual cycle on physiological responses of women to exercise and heat stress has been summarized recently by Drinkwater (1984) who suggested exercise performance in the heat was unaffected by the menstrual cycle. Performance of an exercise task however, should not be confused with thermoregulation. The data from the present study and from several other investigations (Bittel and Henane 1975; Haslag and Hertzman 1965; Hessemer and Bruck 1985a and 1985b; Stephenson and Kolka 1985) indicate that the regulation of body temperature is affected by the menstrual cycle as the esophageal temperature thresholds for heat loss and heat production are shifted upward to maintain core temperature at an elevated level in the luteal phase of the menstrual cycle.

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