

Function of human eccrine sweat glands during dynamic exercise and passive heat stress

NARIIHIKO KONDO,¹ MANABU SHIBASAKI,² KEN AOKI,¹ SHUNSAKU KOGA,³ YOSHIMITSU INOUE,⁴ AND CRAIG G. CRANDALL⁵

¹Laboratory for Applied Human Physiology, Faculty of Human Development, Kobe University, Kobe 657-8501; ²Nara Women's University, Nara 630-8506;

³Kobe Design University, Kobe 651-2196; ⁴Osaka International University for Women, Osaka 570-8555, Japan; and ⁵Institute for Exercise and Environmental Medicine, Presbyterian Hospital of Dallas, Texas 75231

Received 6 March 2000; accepted in final form 20 November 2000

Kondo, Narihiko, Manabu Shibasaki, Ken Aoki, Shunsaku Koga, Yoshimitsu Inoue, and Craig G. Crandall. Function of human eccrine sweat glands during dynamic exercise and passive heat stress. *J Appl Physiol* 90: 1877–1881, 2001.—The purpose of this study was to identify the pattern of change in the density of activated sweat glands (ASG) and sweat output per gland (SGO) during dynamic constant-workload exercise and passive heat stress. Eight male subjects (22.8 ± 0.9 yr) exercised at a constant workload (117.5 ± 4.8 W) and were also passively heated by lower-leg immersion into hot water of 42°C under an ambient temperature of 25°C and relative humidity of 50%. Esophageal temperature, mean skin temperature, sweating rate (SR), and heart rate were measured continuously during both trials. The number of ASG was determined every 4 min after the onset of sweating, whereas SGO was calculated by dividing SR by ASG. During both exercise and passive heating, SR increased abruptly during the first 8 min after onset of sweating, followed by a slower increase. Similarly for both protocols, the number of ASG increased rapidly during the first 8 min after the onset of sweating and then ceased to increase further ($P > 0.05$). Conversely, SGO increased linearly throughout both perturbations. Our results suggest that changes in forearm sweating rate rely on both ASG and SGO during the initial period of exercise and passive heating, whereas further increases in SR are dependent on increases in SGO.

density of activated sweat glands; sweat output per gland; thermoregulatory sweating

EVAPORATION OF SWEAT is an important heat-loss process in the control of internal body temperature in a hot environment, in which ambient temperature is higher than skin temperature. Increases in sweating could be due to an increase in the density of activated sweat glands (ASG), an increase in the sweat output per gland (SGO), or a combination of both factors (1, 8, 11, 16). Remaining unclear is the contribution of ASG and SGO in elevating sweating rate (SR) during dynamic constant-workload exercise and passive heating.

After the onset of sweating during dynamic constant-workload exercise in normothermic conditions, SR increases abruptly within ~ 10 min, with a slower increase thereafter (19). However, remaining unknown is the exact pattern of change in ASG and SGO during this perturbation and how these parameters affect changes in SR. In normothermic humans, local administration of methylcholine rapidly increases ASG during the initial 2 min after the onset of sweating, followed by no further change in ASG for the next ~ 4 min (7). Previously our laboratory showed that the increase in SR during graded dynamic exercise [i.e., 35–50% maximal oxygen consumption ($\dot{V}_{\text{O}_2 \text{max}}$)] was initially due to a combination of increases in ASG and SGO (8). Further increase in SR during changes in exercise intensity from 50 to 65% $\dot{V}_{\text{O}_2 \text{max}}$ was due solely to increases in SGO. The contribution of ASG and SGO in mediating SR during constant-workload dynamic exercise remains unknown. Based on prior findings from our laboratory (8), we hypothesize that, during constant-workload exercise, the initial increase in SR will be mediated by increases in both ASG and SGO; however, as the duration of exercise continues, further increase in SR will be primarily mediated by increases in SGO.

Sweating response during exercise not only involves changes in internal and skin temperatures (thermal factors) but also nonthermal factors (i.e., central command, mechanoreceptors, metaboreceptors, baroreflex, and so on) (2, 9, 10, 14, 20–22). This is in contrast to passive heating, in which the primary stimuli for sweating are thermal factors. Thus it is possible that the contribution of ASG and SGO in mediating sweating responses during constant-workload exercise may be different than during passive heat stress.

The purpose of this study was to investigate the contribution of ASG and SGO in elevating SR during dynamic constant-workload exercise. In addition, we tested the hypothesis that differences in the contribu-

Address for reprint requests and other correspondence: N. Kondo, Laboratory for Applied Human Physiology, Faculty of Human Development, Kobe Univ., 3-11 Tsurukabuto, Nada-ku, Kobe 657-8501, Japan (E-mail: kondo@kobe-u.ac.jp).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

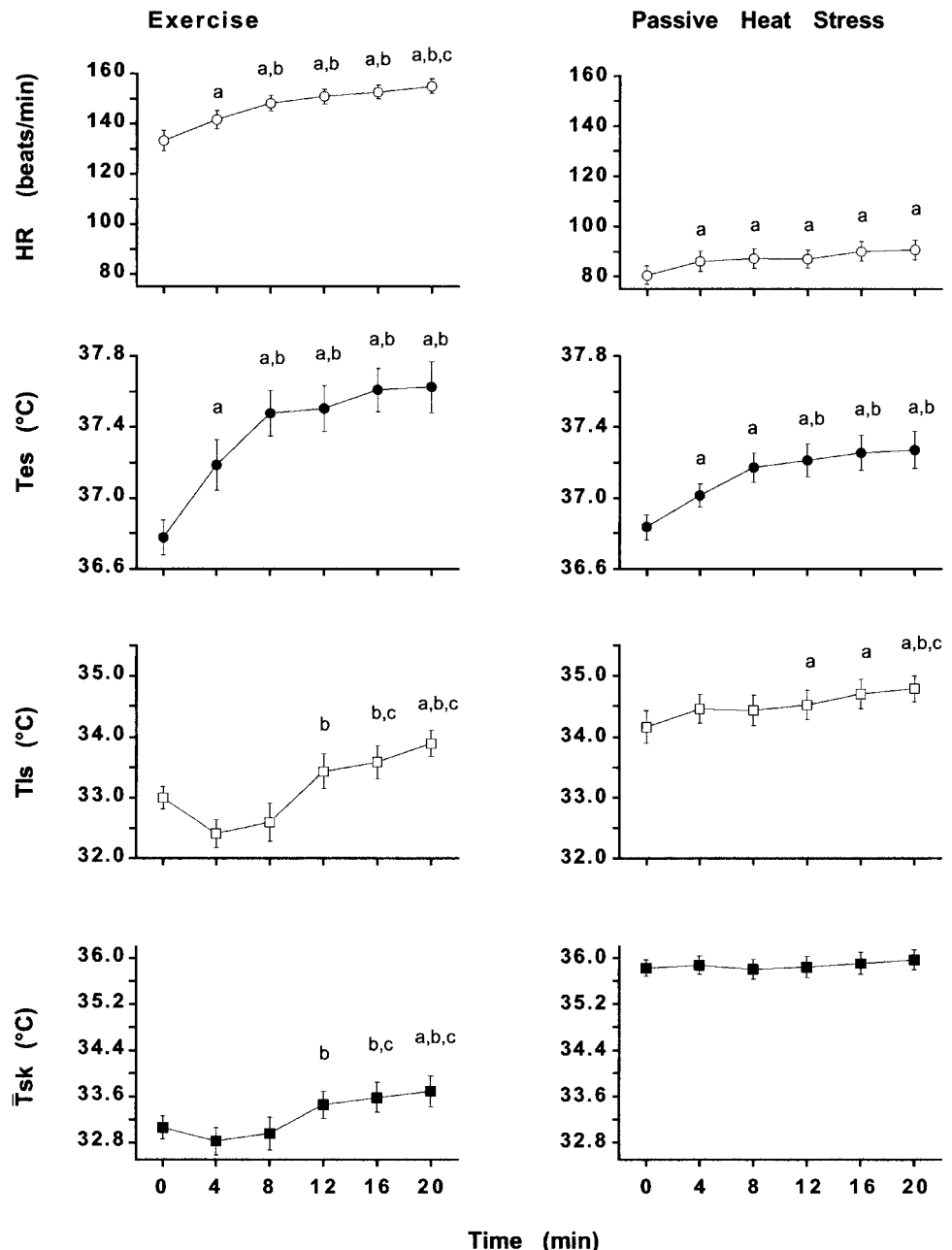


Fig. 1. Changes in heart rate (HR), esophageal temperature (T_{es}), local forearm skin temperature (T_{ls}), and mean skin temperature (T_{sk}) after the onset of sweating during constant-workload exercise and passive heat stress. Values are means \pm SE ($n = 8$ subjects). ^aSignificantly different from 0 min (the onset of sweating) ($P < 0.05$); ^bsignificantly different from 4 min ($P < 0.05$); ^csignificantly different from 8 min ($P < 0.05$).

tion of ASG and SGO in the control of SR may exist between dynamic exercise and passive heating. These objectives were accomplished by measuring SR, ASG, and SGO during dynamic exercise and during passive heat stress following the onset of sweating.

METHODS

Subjects. Studies were performed in eight healthy male subjects (age: 22.8 ± 0.9 yr; height: 170.0 ± 0.1 cm; weight: 64.5 ± 9.8 kg). None of the subjects were taking medications or were smokers. Each subject was informed in advance about the purpose of the study and procedures, and a written consent was obtained. Experimental protocols were approved by the institutional committee on human investigation.

Study protocol. Subjects were requested to refrain from excessive eating for 12 h before the experiment and were

prohibited from taking food at least 2 h before each test. Subjects, wearing only shorts, rested in a sitting position on a cycle ergometer in a climatic chamber (SR3000, Nagano Science, Osaka, Japan) for 40 min at an ambient temperature of 25°C and relative humidity of 50% with minimum air movement. This temperature and humidity was maintained throughout both experimental procedures. After this period of time, baseline data were obtained for 3 min. This was followed by either 30 min of cycle ergometry exercise at 117.5 ± 4.8 W and a pedaling frequency of 60 rpm or immersion of the subjects' lower legs in a hot water bath (42°C) for 60 min. The order of these experiments was randomized, and each experiment was conducted on a different day. All subjects performed both experiments, and each test was performed at the same time of day to avoid potential effects of circadian variations. During exercise, subjects rested their

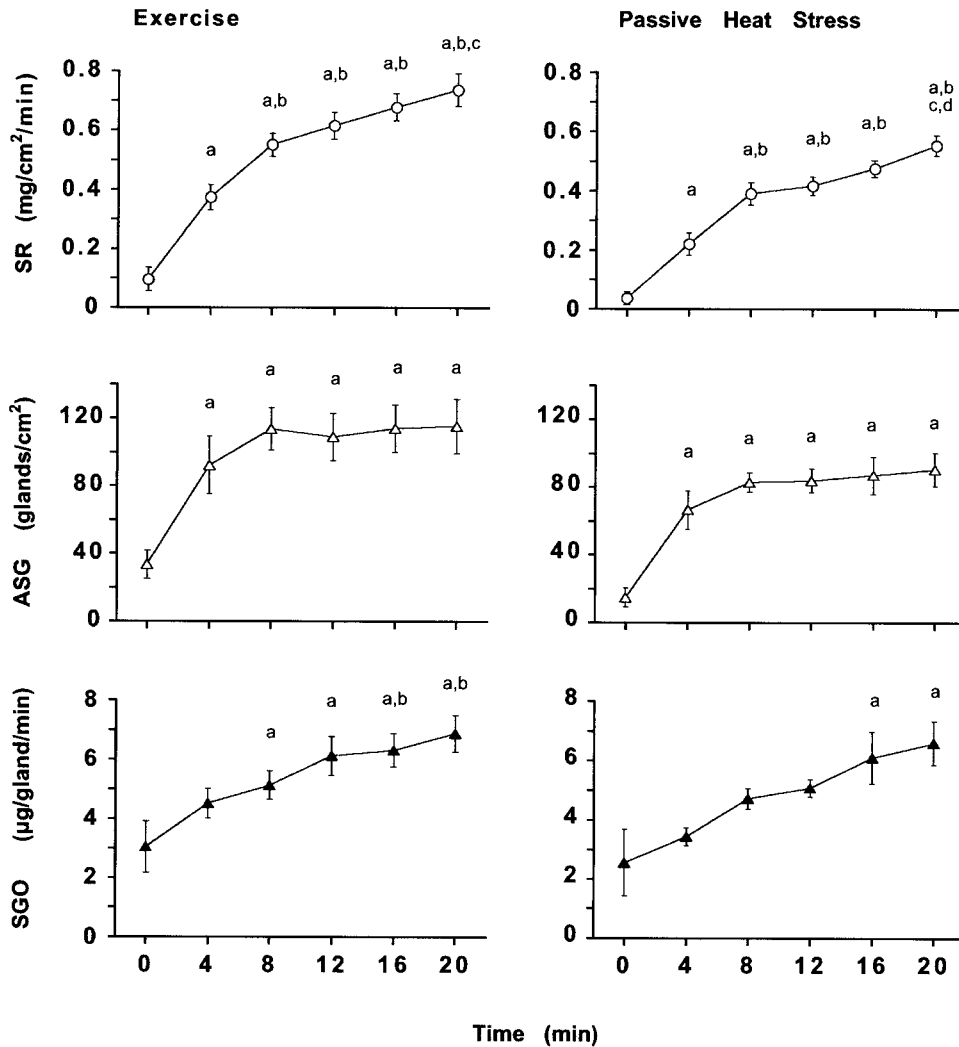


Fig. 2. Forearm sweating rate (SR), density of activated sweat glands (ASG), and sweat output per gland (SGO) after the onset of sweating during constant-workload exercise and passive heat stress. Values are means \pm SE ($n = 8$ subjects). ^aSignificantly different from 0 min (the onset of sweating) ($P < 0.05$); ^bsignificantly different from 4 min ($P < 0.05$); ^csignificantly different from 8 min ($P < 0.05$); ^dsignificantly different from 12 min ($P < 0.05$).

forearms and hands on a board mounted to the handlebars of the cycle ergometer to keep their upper body and hands relaxed.

Measurements. In all studies, esophageal temperature (T_{es}), local skin temperature at eight different sites (chest, forearm, palm, forehead, abdomen, thigh, lower leg, and foot), SR on the left forearm, ASG on the same forearm, and heart rate (HR) were measured. T_{es} and local skin temperatures were measured with copper-constantan thermocouples. The tip of the thermocouple used for measuring T_{es} was coated with silicone and inserted through the nose to a distance equal to one-quarter of the subjects' heights. Mean skin temperature (\bar{T}_{sk}) was calculated according to the method of Hardy and DuBois (3). SR was measured continuously by the ventilated-capsule method. Dry nitrogen gas was supplied to the capsule (5.31 cm²) at a rate of 2.0 l/min, and the humidity of the nitrogen gas flowing out of the capsule was measured with a capacitance hygrometer (HMP 133Y, Vaisala, Helsinki, Finland). For both procedures, ASG and SGO were identified during a 10-s window and measured every 4 min after the onset of sweating. With the use of the starch-iodide technique (4, 18), the number of ASG was measured at a site adjacent to the sweat capsule. SGO was calculated by dividing SR over the same period of time by the number of ASG. A data logger was used to record temperatures and SR at 1 Hz on a personal computer. These values were used to calculate the average response for each minute.

Statistical analysis. Because body temperatures and sweating parameters plateaued 20 min after the onset of sweating during the passive heat stress protocol, variables were statistically analyzed during only the first 20 min after the onset of sweating for both the exercise and passive heat stress protocols. One-way analysis of variance with repeated measures was used, followed by Scheffé's test when F values were significant. A Student's t -test was used to assess differences in the internal temperature threshold between protocols for the onset of sweating and for the regression lines of the T_{es} -SR, T_{es} -ASG, and T_{es} -SGO relationships. T_{es} threshold for sweating was calculated by determining the T_{es} at which SR increased above resting values. Thresholds for T_{es} -SR and sensitivities for T_{es} -SR, T_{es} -ASG, and T_{es} -SGO relationships were calculated for each subject and followed by statistical analysis. P value for significance was set at 0.05. All data are expressed as means \pm SE.

RESULTS

Baseline HR and T_{es} were similar before dynamic exercise relative to before passive heat stress (73.5 ± 2.5 beats/min, $36.66 \pm 0.09^\circ\text{C}$ for dynamic exercise; 69.9 ± 2.2 beats/min, $36.64 \pm 0.05^\circ\text{C}$ for passive heat stress). Figure 1 shows changes in HR, T_{es} , local forearm skin temperature (T_{ls}), and \bar{T}_{sk} during exercise

and passive heat stress after the onset of sweating (0 min). Exercise significantly increased HR, T_{es} , T_{ls} , and T_{sk} . Similar responses were observed during passive heat stress with the exception of a progressive increase in T_{sk} . Moreover, after the onset of sweating, HR, T_{es} , T_{ls} , and T_{sk} were significantly different between protocols.

During exercise, SR increased abruptly up to 8 min after the onset of sweating and then at a less abrupt rate to the end of the test (Fig. 2). ASG also increased abruptly within the first 8 min after the onset of sweating and then did not increase further through the end of exercise. In contrast, SGO increased linearly throughout the exercise protocol and was significantly different from baseline at 8, 12, 16, and 20 min. During the first 20 min of passive heating, SR, ASG, and SGO responses were virtually identical to those observed during exercise (Fig. 2). However, SR and ASG at each time interval were significantly higher during exercise than during passive heat stress. In contrast, SGO during exercise was significantly higher than during passive heat stress at 4 and 12 min. The highest value of SGO did not differ between the conditions.

T_{es} threshold for the onset of sweating was not markedly different between exercise and passive heat stress protocols (exercise: $36.75 \pm 0.10^\circ\text{C}$; passive heating: $36.81 \pm 0.07^\circ\text{C}$). The relationships between T_{es} and sweating parameters (SR, ASG, and SGO) during exercise and passive heat stress are illustrated in Fig. 3. Sweating parameters during both exercise and passive heat stress increased linearly with a rise in T_{es} . The slopes of the linear regression equation between T_{es} and SR, T_{es} and ASG, and T_{es} and SGO were not significantly different between exercise and passive heat stress trials, although SGO during passive heat stress tended to be greater than during exercise ($P = 0.056$).

DISCUSSION

In this study, we investigated the contribution of ASG and SGO in elevating SR during dynamic constant-workload exercise and during passive heating. SR during exercise increased abruptly for 8 min after the onset of sweating and then continued increasing at a much lower rate. Similarly, ASG during exercise increased steeply 8 min after onset of sweating and then plateaued (Fig. 2). In contrast, during the same time interval, SGO increased linearly throughout the 20-min exercise protocol (Fig. 2). These results suggest that, after this initial period, whereas changes in both ASG and SGO contributed to the initial rise in SR during constant-workload dynamic exercise, further changes in SR are primarily dependent on increases in SGO. To our knowledge, the present study is the first to report serial changes in ASG and SGO during constant-workload dynamic exercise.

During the first 8 min of exercise, T_{ls} decreased and T_{es} increased rapidly (Fig. 1). Given the aforementioned rise in ASG and SGO during this period of time, it is likely that these increases are primarily mediated by increases in sudomotor drive secondary to increases in internal

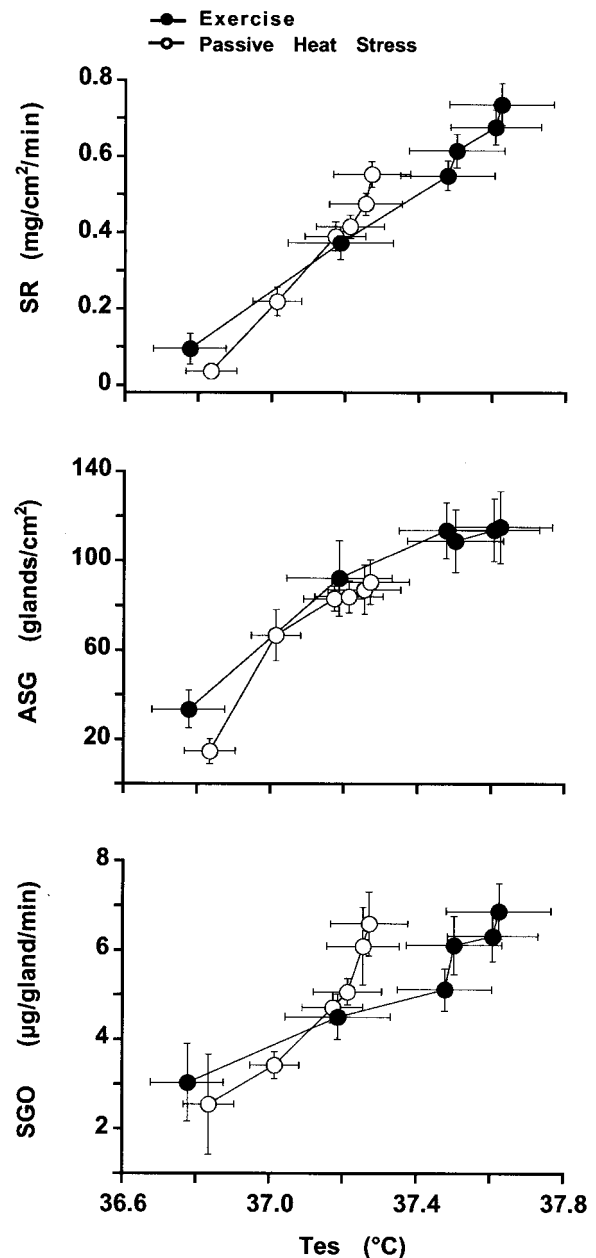


Fig. 3. Changes in SR, ASG, and SGO relative to changes in T_{es} during constant-workload exercise and passive heat stress. Values are means \pm SE ($n = 8$ subjects).

temperature but not by effects of local skin temperature on increasing sweat gland activities. After the first 8 min following the onset of sweating, ASG responses plateaued, whereas SR and SGO continued to increase (Fig. 2). During this period of time, both T_{es} and T_{ls} increased. Thus the increase in SGO after 8 min of exercise may not only be due to changes in internal temperature but may also be modulated by increases in local skin temperature. This latter point is confirmed by Ogawa and Asayama (15), in which local skin temperature modulated neurotransmitter release leading to both sweating and glandular responses.

ASG on the forearm was 115 ± 16 glands/cm² after 20 min of exercise (HR: 155.0 beats/min). These values are

consistent with prior findings using the starch-iodide technique, in which ASG was reported to be 100 and 90 glands/cm² during dynamic exercise at 65% $\dot{V}O_{2\max}$ (HR: 152 beats/min) (8) and 43% $\dot{V}O_{2\max}$ (HR: 114 beats/min) (18), respectively. Thus it is apparent that the number of ASG is consistent between these exercise studies.

In this study, we compared the contribution of ASG and SGO on changing SR during exercise with that observed during passive heat stress. During the first 20-min period, changes in SR, ASG, and SGO were virtually identical between protocols (Fig. 2). It is interesting to note that the pattern of change of these variables was similar despite differing HR, T_{es} , \bar{T}_{sk} , and absolute levels of SR and ASG between protocols. Moreover, the larger SR during exercise, when compared with passive heating, is due primarily to increases in ASG because maximal SGO was not markedly different between protocols.

The control of sweating is often evaluated by examining the relationship between body temperature and SR (5, 8, 12) as shown in Fig. 3. Our results show that SR and ASG at a given T_{es} did not differ between exercise and passive heat stress. Skin temperature is an important factor in determining the internal temperature threshold for the onset of sweating (13). In the present study, \bar{T}_{sk} after the onset of sweating was significantly higher during passive heat stress than during exercise (Fig. 1). Thus one would expect the internal temperature threshold for the onset of sweating to be elevated during the exercise bout when skin temperature is lower, relative to this response during passive heating. Because this was not the case, it is likely that the differences in \bar{T}_{sk} were insufficient to cause such a shift. This observation is similar to the findings of others who reported that dynamic exercise does not influence the internal temperature threshold for the onset of sweating relative to passive heating (5, 6, 8, 12). On the basis of the lack of difference in the T_{es} -ASG and T_{es} -SGO relationships between protocols (Fig. 3), ASG and SGO may not be influenced by the addition of nonthermal factors associated with dynamic exercise. However, to more accurately compare the responses of ASG and SGO per change in T_{es} between protocols, skin temperature should be similar between these conditions.

In this study, SR and ASG were measured on the forearm. The density and capacity of sweat glands to produce sweat differ from one region to another (8, 17, 18). In addition, the pattern of change in ASG and SGO with a rise in exercise intensity differed from one region of the body to another (8). Thus a detailed study should be undertaken to examine regional differences in the contribution of ASG and SGO to changes in SR during both constant-workload dynamic exercise and passive heat stress.

In conclusion, findings from the present study demonstrate that increases in forearm sweating during the initial period of constant-workload exercise are associated with increases in the number of ASG and SGO. As the exercise bout continues, the number of ASG plateaus, whereas SGO continues to increase. Similar findings were observed during passive heat stress. These results

suggest that changes in forearm SR during constant-workload exercise and during passive heating initially depend on ASG and SGO and then mainly on SGO as the exercise bout or heat stress continues.

We sincerely thank our volunteer subjects.

This work was supported in part by a Grant from Ono Sports Science (1998).

REFERENCES

1. **Buono MJ and Connolly KP.** Increases in sweat rate during exercise: gland recruitment versus output per gland. *J Therm Biol* 17: 267-270, 1992.
2. **Gisolfi C and Robinson S.** Central and peripheral stimuli regulating sweating during intermittent work in men. *J Appl Physiol* 29: 761-768, 1970.
3. **Hardy JD and DuBois EF.** The technique of measuring radiation and convection. *J Nutr* 15: 461-475, 1938.
4. **Inoue Y.** Longitudinal effects of age on heat-activated sweat gland density and output in healthy active older men. *Eur J Appl Physiol* 74: 72-77, 1996.
5. **Johnson JM and Park MK.** Effect of upright exercise on threshold for cutaneous vasodilation and sweating. *J Appl Physiol* 50: 814-818, 1981.
6. **Kellogg DL Jr, Johnson JM, and Kosiba WA.** Control of internal temperature threshold for active cutaneous vasodilation by dynamic exercise. *J Appl Physiol* 71: 2476-2482, 1991.
7. **Kenney WL and Fowler SR.** Methylcholine-activated eccrine sweat gland density and output as a function of age. *J Appl Physiol* 65: 1082-1086, 1988.
8. **Kondo N, Takano S, Aoki K, Shibasaki M, Tominaga H, and Inoue Y.** Regional differences in the effect of exercise intensity on thermoregulatory sweating and cutaneous vasodilation. *Acta Physiol Scand* 164: 71-78, 1998.
9. **Kondo N, Tominaga H, Shibasaki M, Aoki K, Koga S, and Nishiyasu T.** Modulation of the thermoregulatory sweating response to mild hyperthermia during activation of the muscle metaboreflex in humans. *J Physiol (Lond)* 515: 591-598, 1999.
10. **Kondo N, Tominaga H, Shiojiri T, Aoki K, Takano S, Shibasaki M, and Koga S.** Sweating responses to passive and active limb movements. *J Therm Biol* 22: 351-356, 1997.
11. **Kuno Y.** *Human Perspiration*. Springfield, IL: Thomas, 1956.
12. **Montain SJ, Lutzka WA, and Sawka MN.** Control of thermoregulatory sweating is altered by hydration level and exercise intensity. *J Appl Physiol* 79: 1434-1439, 1995.
13. **Nadel ER, Mitchell JM, Saltin B, and Stolwijk JAJ.** Peripheral modifications to the central drive for sweating. *J Appl Physiol* 31: 828-833, 1971.
14. **Nielsen B.** Thermoregulation in rest and exercise. *Acta Physiol Scand Suppl* 323: 1-74, 1969.
15. **Ogawa T and Asayama M.** Quantitative analysis of the local effect of skin temperature on sweating. *Jpn J Physiol* 36: 417-422, 1986.
16. **Sato K and Dobson RL.** Regional and individual variations in the function of the human eccrine sweat gland. *J Invest Dermatol* 54: 443-449, 1970.
17. **Sato K and Sato F.** Individual variations in structure and function of human eccrine sweat gland. *Am J Physiol Regulatory Integrative Comp Physiol* 245: R203-R208, 1983.
18. **Shibasaki M, Inoue Y, Kondo N, and Iwata A.** Thermoregulatory responses of prepubertal boys and young men during moderate exercise. *Eur J Appl Physiol* 75: 212-218, 1997.
19. **Takano S, Kondo N, Shibasaki M, Aoki K, Inoue Y, and Iwata A.** The influence of work loads on regional differences in sweating rates. *Jpn J Physiol* 46: 183-186, 1996.
20. **Van Beaumont W and Bullard RW.** Sweating: exercise stimulation during circulatory arrest. *Science* 152: 1521-1523, 1966.
21. **Van Beaumont W and Bullard RW.** Sweating: its rapid response to muscular work. *Science* 141: 643-646, 1963.
22. **Yamazaki F, Sone R, and Ikagami H.** Responses of sweating and body temperature to sinusoidal exercise. *J Appl Physiol* 76: 2541-2545, 1994.