Sustained increases in skin blood flow are not a pre-requisite to initiate sweating during passive heat exposure

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ABSTRACT

Some studies have observed a functional relationship between sweating and skin blood flow. However, the implications of this relationship during physiologically-relevant conditions remain unclear. We manipulated sudomotor activity through changes in sweating efficiency to determine if parallel changes in vasomotor activity are observed. Eight young males completed two trials at 36°C and two trials at 42°C. During these trials, air temperature remained constant while ambient vapor pressure increased from 1.6 to 5.6 kPa over 2 hours. Forced airflow across the skin was used to create conditions of high (HiS_{eff}) or low (LoS_{eff}) sweating efficiency. Local sweat rate (LSR), local skin blood flow (SkBF), as well as mean skin and esophageal temperatures were measured continuously. It took longer for LSR to increase during HiS_{eff} at 36°C (HiS_{eff}: 99 ± 11 vs. LoS_{eff}: 77 ± 11 min, $P<0.01$) and 42°C (HiS_{eff}: 72 ± 16 vs. LoS_{eff}: 51 ± 15 min, $P<0.01$). In general, an increase in LSR preceded the increase in SkBF when expressed as ambient vapor pressure and time for all conditions ($P<0.05$). However, both responses were activated at a similar change in mean body temperature (average across all trials, LSR: 0.26 ± 0.15 vs. SkBF: 0.30 ± 0.18°C, $P=0.26$). These results demonstrate that altering the point at which local sweat rate is initiated during heat exposure is paralleled by similar shifts for the increase in SkBF. However, local sweat production occurs before an increase in SkBF, suggesting that SkBF is not necessarily a pre-requisite for sweating.

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INTRODUCTION

The regulation of internal body temperature during heat stress relies upon sweat production and cutaneous vasodilation. Although both responses promote the exchange of heat between the body and the surrounding environment, evaporation of sweat provides by far the greatest potential for heat loss (1, 12, 23, 47). In contrast, cutaneous vasodilation and subsequent increases in skin blood flow are generally thought to transfer heat from core tissues to the periphery, therefore providing the heat to be removed by sweat evaporation (6, 14).

A functional relationship between sweating and skin blood flow has long been hypothesized, beginning with the theory that bradykinin released by activated sweat glands leads to cutaneous vasodilation (11). Although this hypothesis was later refuted (20), a number of studies provide evidence for a functional relationship between sweating and skin blood flow. Most notably, Brengelmann et al. (4) reported a lack of active cutaneous vasodilation during passive heating in individuals with a congenital absence of sweat glands (i.e. anhidrotic ectodermal dysplasia). In addition, manipulations of local skin temperature (and probably skin blood flow) affect the onset threshold and thermal sensitivity of the sweating response during heat stress (2, 5, 31, 32). Finally, the sweating response to heat stress is attenuated when skin blood flow is prevented from increasing using arterial occlusion (7, 25) or pharmacological blockade (49). Regardless of the potential mechanism, such observations clearly highlight a functional relationship between skin blood flow and sweating, in that one response becomes compromised when the other is absent or prevented from increasing.

The implications of a functional relationship between skin blood flow and sweating during physiologically-relevant conditions remain unclear. This question has been examined by comparing the onset threshold of both responses when they are allowed to increase normally during heat exposure.

Most recently, Smith et al. (42) reported that the onset threshold for skin blood flow precedes the one for sweat rate and suggested that an increase in skin blood flow may be required to initiate sweat production during whole-body passive heat stress. Other studies have provided conflicting evidence that skin blood flow increases before (40, 41), after (24), or simultaneously (45) with sweat production. However, these studies did not manipulate one heat loss response to observe if parallel changes are observed in the other. Recently, we demonstrated that fan use delays the critical humidity at which elevations in heart rate are observed during step-wise increases in humidity in warm and hot conditions (36, 37). The delayed increase in heart rate was paralleled by improved sweating efficiency and lower levels of local sweat rate during fan use relative to a still airflow condition (36). These observations suggest that increased air velocity delays the point at which sweating is activated, which provided us with a unique opportunity to examine the relationship between skin blood flow and sweating when sudomotor activity is manipulated though changes in sweating efficiency. If, as previously suggested (42), increased skin blood flow is a pre-requisite for sweating during heat exposure, one might expect that: i) skin blood flow consistently increases prior the initiation of sweating, and; ii) manipulating the activation of sweating would lead to parallel changes in the activation of skin blood flow. Furthermore, the step-wise increase in humidity protocol elicits a slow and gradual heating stimulus, which we hypothesized would allow more resolution to detect subtle differences between the point at which sustained increases in sweating and skin blood flow occur. We hypothesized that increases in skin blood flow would precede increases in sweat production implying a functional dependency of sweating on changes in local skin blood flow.

METHODS

The data presented in this manuscript were collected as part of a larger study examining the effectiveness of fan use during exposure to extreme heat and humidity, the results of which have been

published previously (36, 37). The study participants included 8 healthy normotensive and non-smoking males, with no pre-existing cardiovascular, metabolic, or neurological diseases (age: 24 ± 3 y; body mass: 80.69 ± 11.68 kg; height: 1.77 ± 0.05 m; body surface area: 1.98 ± 0.14 m^2). None of the subjects were taking medications. All participants provided written informed consent. The experimental protocol was approved by the University of Ottawa Research Ethics Committee and conformed to the guidelines set forth in the Declaration of Helsinki. Participants were instructed to avoid vigorous physical activity 24 hours prior, refrain from alcohol 12 hours prior, eat a light meal, and avoid any caffeinated beverages at least 6 hours prior to testing.

Instrumentation

Esophageal temperature was measured using a pediatric thermistor probe (Mon-a-therm®, Mallinckrodt Medical, St. Louis, MO) inserted through the nasal cavity into the esophagus, to a depth estimated to be nearest the left ventricle (27). Skin temperature was measured using four thermistors (Concept Engineering, Old Saybrook, CT, USA) which were secured to the skin using surgical tape (Transpore®, 3M, London, ON). Mean skin temperature was calculated as the weighted average of four sites (35): chest 30%, triceps 30%, thigh 20%, and calf 20%. Temperature measurements were sampled every 5 s (NI cDAQ-91722 module, National Instruments, Austin, TX) and recorded to a desktop computer using customized LabView software (v7.0, National Instruments, Austin, TX). Local sweat rates were measured on the chest and forearm using ventilated sweat capsules. Anhydrous air was supplied through 4.1 cm^2 capsules at a rate of 1.2-1.4 L/min. Capsules were secured to the skin using surgical tape. The temperature and humidity of the air leaving both capsules were measured with factory calibrated capacitance hygrometers (HMT333, Vaisala, Vantaa, Finland) every 5 seconds. Local sweat rates were calculated as the product of absolute humidity and flow rate, and expressed relative to the amount of skin surface covered by the capsule. Skin blood flow (SkBF) was measured

using laser-Doppler flowmetry probes (Small Angled Thermostatic Probe #457, Perimed, Järfälla, Sweden) placed on the chest and forearm in close proximity to the ventilated sweat capsules. Skin blood flow was sampled at a rate of 60 Hz by the Transcutaneous Oxygen Monitoring System (Periflux System 5000, Perimed, Järfälla, Sweden) and recorded in real-time (Perisoft for Windows Version 2.5.5, Perimed, Järfälla, Sweden) before being exported to an Excel Spreadsheet (Excel 2000, Microsoft, USA) at a sampling rate of 5 seconds. Blood pressure was measured using an automated cuff (E-Sphyg II 9002, American Diagnostics Corporation, Hauppauge, NY, USA). Urine specific gravity was measured using a refractometer (Reichert TS 400, Depew, NY). Ambient air velocity was measured using a hot wire anemometer (VelociCalc 9535, TSI Inc, Shoreview MN, USA).

Experimental protocol

The participants volunteered for 4 experimental trials performed in a climatic chamber that precisely regulated air temperature and humidity. For all trials, participants provided a urine sample upon arrival before entering the climatic chamber regulated at a temperature of either 36°C or 42°C and a standardized ambient vapor pressure of 1.6 kPa where they were fully instrumented. Following a 45 min baseline period, ambient vapor pressure within the chamber was increased in a step-wise fashion by ~0.3 kPa every 7.5 minutes until reaching 5.6 kPa; a comparable protocol to previous studies (3, 19, 22). The step-wise increases in ambient vapor pressure therefore lasted a total of 120 min. For both air temperatures, the participants performed the trial without and with forced air flow across the skin to manipulate sweating efficiency (S_{eff}) and thus the physiological requirement for sweat production to attain heat balance (36). During the trials with a low sweating efficiency (i.e. $36LoS_{eff}$ and $42LoS_{eff}$), participants sat behind a 122 cm barrier made of foamcore boards (122 cm x 250 cm) placed in a 'V' shape against a wall. The barrier minimized airflow across the skin to less than 0.1 m/s, confirmed by a hot-wire anemometer placed approximately 30 cm anterior to the participant's torso. During the trials

with a high sweating efficiency (i.e. 36HiS$_{eff}$ and 42HiS$_{eff}$), an 18" diameter mechanical fan (High velocity orbital air circulator, Whirlpool, Benton Harbor, MI, USA) set at full speed (generating a free space air velocity of ~4.0 m/s) was placed 1 m in front of the participant and turned on following the baseline period. The trials were performed in a balanced order and were separated by a minimum of 48 h. During the trials, participants wore a standardized t-shirt and shorts and sat on a plastic chair, which covered their back and upper rear thighs.

Data analysis

Mean body temperature was calculated as: esophageal temperature × 0.9 + mean skin temperature × 0.1 (32). Mean arterial pressure was calculated as the weighted sum of systolic (1/3) and diastolic (2/3) blood pressures. Cutaneous vascular conductance was calculated as skin blood flow divided by mean arterial pressure. Step-wise increases in ambient vapor pressure elicited relatively stable values of mean body temperature, skin blood flow, cutaneous vascular conductance, and sweating until a critical ambient vapor pressure was attained where an upward inflection in each variable occurred (3). The critical ambient vapor pressure at which the inflection point occurred was determined separately for skin blood flow and sweating using segmented regression analysis of the minute-averaged data (21, 22, 37). For cutaneous vascular conductance values, only one data point per ambient vapor pressure stage was used for segmented regression as blood pressure was measured at the end of each stepwise increment. Figure 1 depicts the segmented regression analysis for one participant using one data point per ambient vapor pressure stage. The inflection point was also characterized based on corresponding time (in minutes) and change in mean body temperature from baseline. No statistical differences were observed between the forearm and chest for skin blood flow and local sweat rate. Therefore, values from both sites were averaged to yield one value for each variable.

Statistical Analysis

Mean skin, esophageal, and mean body temperatures, as well as mean arterial pressure were analyzed within each air temperature (i.e. 36°C and 42°C) using 2 way repeated measures ANOVA with the repeated factors of time (9 levels: baseline, 15, 30, 45, 60, 75, 90, 105, and 120 min) and condition (LoS_{eff} vs. HiS_{eff}). To determine if sweating efficiency affected the inflection point for local sweat rate, skin blood flow, and cutaneous vascular conductance, differences between conditions (at a given air temperature) were compared using paired samples t-tests. The alpha was set to 0.05 and corrected for multiple comparisons using the Holm-Bonferroni approach. All data are presented as mean ± standard deviation.

RESULTS

Baseline urine specific gravity was similar between conditions ($36HiS_{eff}$: 1.012 ± 0.006 vs. $36LoS_{eff}$: 1.012 ± 0.006; $42HiS_{eff}$: 1.013 ± 0.005 vs. $42LoS_{eff}$: 1.016 ± 0.002, $P>0.24$). Baseline mean skin, esophageal, and mean body temperatures also did not differ between conditions at 36°C and 42°C ($P>0.15$, Fig. 2). Manipulations of sweating efficiency did not affect esophageal and mean body temperatures during the conditions performed at 36°C ($P>0.20$). In contrast, time × condition interactions for esophageal and mean body temperatures were observed at 42°C ($p<0.01$, Fig. 2). However, none of the post-hoc comparisons reached statistical significance (all $P>0.12$). A time × condition interaction for was also observed for mean skin temperature at 36°C ($P<0.05$), but not at 42°C ($P=0.24$). Again, no post-hoc comparisons at 36°C reached statistical significance (all $P>0.12$). Overall, the change in mean skin temperature did not exceed 1.5°C during any condition ($36HiS_{eff}$: 0.74 ± 0.32°C vs. $36LoS_{eff}$: 1.16 ± 0.41°C; $42HiS_{eff}$: 1.01 ± 0.33°C vs. $42LoS_{eff}$: 1.30 ± 0.36°C). Mean arterial pressure remained unchanged throughout the protocols at 36°C and 42°C (main effects of time,

163 P>0.44). Furthermore, mean arterial pressure was similar between high and low sweating efficiency
164 conditions at 36°C and 42°C (main effects of condition, P>0.20).
165
166 Tables 1 and 2 present the ambient vapor pressure (kPa), time (min), and change in mean body
167 temperature corresponding with increases in local sweat rate, skin blood flow, and cutaneous vascular
168 conductance at 36°C and 42°C, respectively. Individual responses for the time corresponding with the
169 upward rise in local sweat rate and skin blood flow for all four conditions are presented in Figure 3.
170 Figures 4 and 5 illustrate skin blood flow and sweat rate as a function of kPa, time, and mean body
171 temperature for both conditions and both air temperatures. Manipulations of sweating efficiency
172 resulted in different ambient vapor pressure inflection points for local sweat rate at 36°C (P<0.01) and
173 42°C (P<0.01). The inflection point for local sweat rate also differed when expressed as time at both air
174 temperatures (P<0.01). When expressed as a change in mean body temperature, different onset
175 thresholds for local sweat rate were observed at 42°C (P=0.03), but not 36°C (P=0.20). In general,
176 increases in local sweat rate occurred later when sweating efficiency was high, regardless of how it was
177 expressed (see Tables 1 and 2).
178
179 Manipulating sweating efficiency also resulted in different inflection points for skin blood flow
180 and cutaneous vascular conductance. When expressed as ambient vapor pressure (SkBF, P=0.04; CVC,
181 P=0.01) and time (SkBF, P=0.02; CVC, P<0.01), the inflection points for skin blood flow and
182 cutaneous vascular conductance differed between conditions at 36°C. A similar trend was observed at
183 42°C, although differences did not reach statistical significance for skin blood flow (ambient vapor
184 pressure: P=0.09; time: P=0.07) whereas a statistical difference was observed for cutaneous vascular
185 conductance (ambient vapor pressure: P<0.01; time: P<0.01). When expressed as a change in mean
186 body temperature, the onset threshold for skin blood flow and cutaneous vascular conductance did not

differ between conditions at 36°C (SkBF, P=0.80, CVC, P=0.10), while it was different at 42°C (SkBF, P=0.05; CVC, P=0.03). Increases in skin blood flow and cutaneous vascular conductance generally occurred later with high sweating efficiency (see Tables 1 and 2).

When expressed in terms of ambient vapor pressure and time, the inflection point for local sweat rate differed from the one for skin blood flow (P<0.01), but not cutaneous vascular conductance (P=0.86), at $36HiS_{eff}$ (Table 1). During $36LoS_{eff}$, the time and ambient vapor pressure associated with the inflection point for local sweat rate differed from skin blood flow (P<0.001) and cutaneous vascular conductance (P<0.01). During $42HiS_{eff}$, the inflection points for local sweat rate (expressed as time and ambient vapor pressure) did not differ statistically from the ones for skin blood flow (P>0.06), but were different than the ones for cutaneous vascular conductance (P<0.03; Table 2). Similarly, the time and ambient vapor pressure inflection points for local sweat rate were not statistically different from the ones for skin blood flow (P>0.12), but were different than the ones for cutaneous vascular conductance (P<0.02) during $42LoS_{eff}$ (Table 2). In general, increases in local sweat rate preceded increases in skin blood flow and cutaneous vascular conductance. During the conditions performed at 36°C, all subjects exhibited an earlier inflection point in local sweat rate compared to the one for skin blood flow (Fig. 3). At an air temperature of 42°C, the inflection point for sweating preceded the one for skin blood flow in 75% of the trials (Fig. 3). When expressed as a change in mean body temperature, the onset threshold for local sweat rate occurred at a lower change in mean body temperature than the one for skin blood flow during 36LoSeff (P=0.04, see Table 1). However, the mean body temperature onset thresholds for local sweat rate, skin blood flow, and cutaneous vascular conductance were similar for all other conditions (P>0.10).

DISCUSSION

This study examined the relationship between skin blood flow and local sweat rate during physiologically-relevant conditions when sweat production is manipulated through changes in sweating efficiency. Furthermore, changes in mean skin and esophageal temperatures occurred gradually over an extended period of time. Although the inflection points of both responses changed in parallel, the increase in local sweat rate preceded an increase in skin blood flow. These results suggest that increases in skin blood flow are not necessarily a pre-requisite to initiate sweat production.

Based on early reports of a close temporal relationship between sweating and cutaneous vasodilation, it has been hypothesized that a functional relationship exists between vasomotor and sudomotor activities (50). Fox and Hilton (11) initially suggested that bradykinin released from activated sweat glands mediates cutaneous vasodilation, thus providing a physiological basis for the functional relationship between sweating and skin blood flow. However, a role for bradykinin was later refuted (20), and it remains unclear whether sudomotor and vasomotor activity are controlled by common or independent nerves (17, 18, 44, 50). Regardless of the potential underlying mechanism, a functional relationship between skin blood flow and sweating is observed under certain conditions. Brengelmann et al. (4) observed a lack of active cutaneous vasodilation in individuals with a congenital absence of sweat glands. In that study, neurological abnormalities were ruled out and the cutaneous vasculature retained the ability to vasodilate in response to local heating and vasoconstrict in response to lower-body negative pressure (4). Furthermore, occluding limb blood flow attenuates the increase in sweat rate during heat exposure (7, 25) and Wingo et al. (49) observed that the sensitivity of the sweating response to changes in body temperature is substantially reduced when the increase in skin blood flow is blocked pharmacologically. Taken together, these observations demonstrate that full expression of either heat loss response is not achieved when the other response is absent (4) or

prevented from increasing (7, 25, 49). However, the nature of a functional relationship between sweating and skin blood flow during more common and physiologically-relevant conditions remains less clear. For example, we recently reported that an ~20% reduction in skin blood flow, such as those associated with healthy aging (15, 42, 43), does not affect sweat rate or the critical environmental limit for heat balance (8). It is therefore debatable whether the functional relationship between skin blood flow and sweating is relevant when both responses operate within a physiological range (as opposed to a complete absence of one heat loss response).

A few studies have considered the relationship between skin blood flow and sweating during physiologically-relevant conditions by examining the onset threshold of both responses when they are allowed to increase normally during heat exposure. Using this approach, early studies provide conflicting evidence with some reporting that skin blood flow increases before (40, 41), after (24) or simultaneously (45) with the increase in sweat production. Most recently, Smith et al. (42) examined whether differences in local skin blood flow could explain regional differences in local sweat rate. Although regional differences in sweat rate could not be explained by regional differences in skin blood flow, they suggested that an increase in skin blood flow is necessary for the initiation of sweating since the onset threshold for skin blood flow occurred prior to the one for sweat rate. However, these studies did not manipulate sweating or skin blood flow to determine if and/or how the other response is affected. Furthermore, an important consideration of the study by Smith et al. (42) is the use of the water-perfused suit model of heat stress that elicits large (~4-5°C) and very rapid (~10-15 min) changes in mean skin temperature. Such large and rapid changes in skin temperature may have provided a potent local temperature-dependent increase in skin blood flow, which could explain the apparent dependency of sweating upon an increase in skin blood flow. In contrast, the current protocol resulted in gradual and moderate changes in mean skin temperature. Importantly, sudomotor activity was

manipulated through the use of forced airflow across the skin surface which affects sweating efficiency. Using this approach, we observed a delayed inflection point for local sweat rate during conditions of high sweating efficiency, which was paralleled by a delayed inflection point for skin blood flow. This occurred whether air temperature was warm or hot and was most evident when the inflection point was expressed in terms of ambient vapor pressure and time. These findings support, at the very least, a temporal relationship between sweating and skin blood flow (24, 45). However, the main finding of the current study is that an increase in local sweat production generally preceded the increase in skin blood flow. This was also most evident when the inflection point was expressed in terms of ambient vapor pressure and time, and it was evident at both air temperatures. In contrast to our hypothesis, these results suggest that an increase in skin blood flow is not a pre-requisite for increases in sweat production under the conditions examined.

An interesting observation of the current study is the substantial difference in the inflection points for local sweat rate and skin blood flow when expressed as ambient vapor pressure and time. For example, the increase in sweat production occurred 15 ± 9 min prior to an increase in skin blood flow when averaged across all conditions. This observation cannot be attributed to differences in mean skin and esophageal temperatures between conditions (Fig. 2). In fact, the mean body temperature onset thresholds for local sweat rate and skin blood flow were generally similar. Due to the nature of the experimental protocol, the increase in mean body temperature occurred very gradually. As such, mean body temperature remained at stable levels for prolonged periods, despite substantial increases in ambient vapor pressure and time. This resulted in a decoupling between the mean body temperature onset threshold and the inflection point when expressed as time and ambient vapor pressure. Had we solely examined onset thresholds, we would have concluded that local sweat rate and skin blood flow increase simultaneously. As such, any potential relationship between skin blood flow and sweating may

not be fully characterized if only examined through mean body temperature onset thresholds. It is well established that sweating and skin blood flow are stimulated by changes in skin and deep body temperatures (26, 29, 30, 34, 46, 51). Therefore, an earlier inflection point for local sweat rate compared to skin blood flow, despite similar changes in mean body temperature, may suggest that increases in skin blood flow occur secondary to an increase in sweating to support sudomotor activity (rather than being a pre-requisite). However, future studies are needed to evaluate this possibility.

As expected from the experimental design, sweating efficiency markedly affected the point at which local sweat rate was initiated. For example, the inflection point for local sweat rate during the low efficiency condition occurred 29 ± 9 min earlier compared to when sweating efficiency was high at an air temperature of 36°C. Increased airflow across the skin surface promotes sweat evaporation, thereby reducing skin wettedness. The results of the current study suggest that the accumulation of sweat, and therefore increased skin wettedness, is associated with an earlier ambient vapor pressure/time inflection for sweat production. This was documented by Mole (28), who reported that sweat rate increases in proportion with the required skin wettedness to maintain heat balance. Alternatively, when sweat is removed from the skin surface by wiping the skin dry, sweat rates increase to counterbalance the absence of potential heat loss via evaporation (33). Placed within the context of the current study, the step-wise increases in ambient vapor pressure progressively reduced the ambient vapor pressure gradient between the skin surface and the surrounding air, resulting in a greater required skin wettedness to maintain heat balance. This necessity for a greater skin wettedness resulted in a greater drive for sweating at a given mean body temperature, as evidenced by the earlier inflection point for local sweat rate during the low sweating efficiency conditions.

Considerations

During heat exposure, skin blood flow initially increases due to withdrawal of vasoconstrictor tone and sustained increases are subsequently mediated by active cutaneous vasodilation (10, 17, 38). It is possible that withdrawal of vasoconstrictor tone explains why some studies have observed an earlier increase in skin blood flow relative to sweating. In the current study, subjects were exposed to a 36°C or 42°C environment for ~60 min prior to the step-wise increases in ambient vapor pressure. It is assumed that all vasoconstrictor tone was withdrawn with this approach and that the inflection points for skin blood flow/cutaneous vascular conductance represent active cutaneous vasodilation. It should therefore be considered that the current results suggest that sustained increases in skin blood flow (i.e. active cutaneous vasodilation) are not required to initiate sweat production. It is also likely that the temporal relationship between skin blood flow and sweating is dependent upon the experimental model of heat stress employed and the environmental conditions. For example, skin blood flow may increase prior to sweating when heat stress is induced using the water-perfused suit model (42). An increase in skin blood flow was also observed prior to any evidence of sweating upon the decision to move from a warm (~40°C) to a cool (~17°C) environment (39). The results of the current study therefore suggest that sustained increases in skin blood flow do not *always* precede the initiation of sweating during heat exposure. It should also be considered that sweating and skin blood flow were not measured from the exact same location. Although we maintained close proximity (~1 cm) between both measures, it is unknown if this distance would alter our observations. Finally, we only examined sweating and skin blood flow on the forearm and chest. Although similar patterns were observed when each area was examined separately, we cannot determine if our findings would be similar or different in other areas not measured (e.g. back, lower limbs, forehead).

Perspectives

Determining if the functional relationship between sweating and skin blood flow has implications during physiologically-relevant conditions is important for our understanding of thermoregulatory control in various scenarios and/or populations. For example, a number of conditions (primary aging, heart failure, diabetes) limit the ability of the skin to vasodilate during heat stress resulting in attenuated skin blood flow (9, 13, 16, 48). If the functional relationship between skin blood flow and sweating is relevant under physiological conditions, reduced skin blood flow in these populations may affect sweat production and ultimately the control of internal body temperature since sweat evaporation provides by far the greatest potential for heat loss during heat exposure (1, 12, 23, 47). The findings of the current study suggest that sustained increases in skin blood flow are not a pre-requisite to initiate local sweat production within the conditions tested. Alternatively, reduced skin blood flow could compromise sweating if increases in skin blood flow support the increase in sweat rate. However, we recently reported that an ~20% reduction in skin blood flow does not affect sweat production or the critical environmental limits for heat balance (8). Combined, the findings of our current and previous (8) studies suggest that the functional relationship between skin blood flow and sweating may not have implications during physiologically-relevant conditions. That said, we have only examined young healthy adults exposed to extreme heat and humidity. These findings may not be applicable to other populations and/or conditions. Future studies are therefore required to determine whether physiological reductions in skin blood flow associated with primary aging or various health conditions has an impact, if any, on sweat production in environments that may be encountered by these populations.

Conclusion

The current study examined if a sustained increase in skin blood flow occurs prior to the initiation of local sweating during step-wise increases in ambient vapor pressure in warm (36°C) and hot (42°C) environments. By changing sweating efficiency, the point at which local sweat rate was activated was manipulated to determine if parallel changes in skin blood flow would be observed. Although directional changes in the inflection point for local sweat rate and skin blood flow were similar, sweat production preceded any sustained increase in skin blood flow. This was particularly noticeable when inflection points were quantified as ambient vapor pressure and time; while the mean body temperature onset thresholds for local sweat rate and skin blood flow were generally similar. These results suggest that a sustained increase in skin blood flow is not always a pre-requisite for sweating to be initiated during passive heat exposure.

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DISCLOSURES

The authors have no competing interests.

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FIGURE CAPTIONS

Figure 1. Individual tracings used to determine the critical vapor pressure for the upward rise in local sweat rate (**LSR**, **A**), skin blood flow (**B**), and cutaneous vascular conductance (**CVC**, **C**). The data are from a participant resting at 36°C during conditions permitting a high (**HiS_{eff}**) or low (**LoS_{eff}**) sweating efficiency. The inflection point was determined using segmented linear regression.

Figure 2. Mean skin temperature (**A:** 36°C, **D:** 42°C), esophageal temperature (**B:** 36°C, **E:** 42°C), and mean body temperature (**C:** 36°C, **F:** 42°C), during passive exposure to an air temperature of 36°C or 42°C with conditions permitting a high (**HiS_{eff}**) or low (**LoS_{eff}**) sweating efficiency. Values are mean ± standard error.

Figure 3. Individual inflection points (open symbols) for local sweat rate (**LSR**) and skin blood flow (**SkBF**) expressed as a function of time. The inflection points were derived using segmented regression analysis during 4 experimental conditions: 36**°**C and high sweating efficiency (**A**); 36**°**C and low sweating efficiency (**B**); 42**°**C and high sweating efficiency (**C**); 42**°**C and low sweating efficiency (**D**). The filled symbols represent mean values. **P*<0.05 vs. LSR for the mean value.

Figure 4. Local sweat rate (**LSR**) and skin blood flow (**SkBF**) expressed as a function of ambient vapor pressure (kPa), time (min), and mean body temperature (°C) during passive exposure to 36°C with conditions permitting a high (**A**, **B**, and **C**) or low (**D**, **E**, **F**) sweating efficiency. The vertical lines indicate the mean inflection points for LSR and SkBF. Values are mean ± standard error. **P*<0.05 vs. LSR.

Figure 5. Local sweat rate (**LSR**) and skin blood flow (**SkBF**) expressed as a function of ambient vapor pressure (kPa), time (min), and mean body temperature (°C) during passive exposure to 42°C with conditions permitting a high (**A**, **B**, and **C**) or low (**D**, **E**, **F**) sweating efficiency. Values are mean ± standard error. The vertical lines indicate the mean inflection points for LSR and SkBF.

TABLES

Table 1. The inflection points for sweating, skin blood flow (SkBF), and cutaneous vascular conductance (CVC) expressed as ambient vapor pressure (kPa), change in mean body temperature (ΔT_b), and time (min) during conditions of high (**HiS_{eff}**) and low (**LoS_{eff}**) sweating efficiency at an air temperature of 36°C.

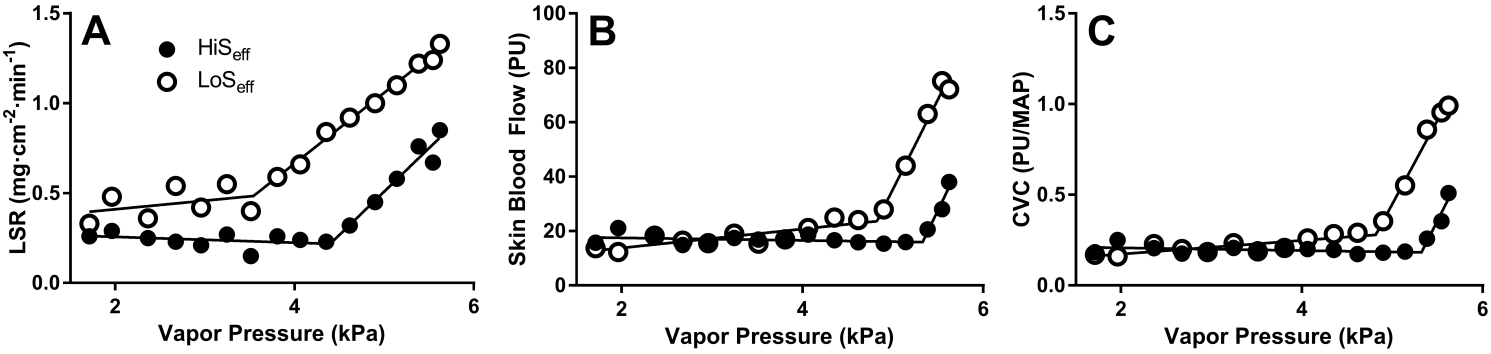
	36HiSeff			**36LoSeff**		
	Sweating	**SkBF**	**CVC**	**Sweating**	**SkBF**	**CVC**
Vapor Pressure (kPa)	4.96 ± 0.36[a,b]	5.34 ± 0.22[b]	5.02 ± 0.50[b]	3.96 ± 0.42[c,d]	4.68 ± 0.37	4.50 ± 0.36
ΔT_b (°C)	0.27 ± 0.15	0.27 ± 0.14	0.26 ± 0.14	0.20 ± 0.13[c]	0.26 ± 0.17	0.23 ± 0.13
Time (min)	99 ± 11[a,b]	111 ± 6[b]	102 ± 15[b]	70 ± 11[c,d]	91 ± 11	85 ± 9

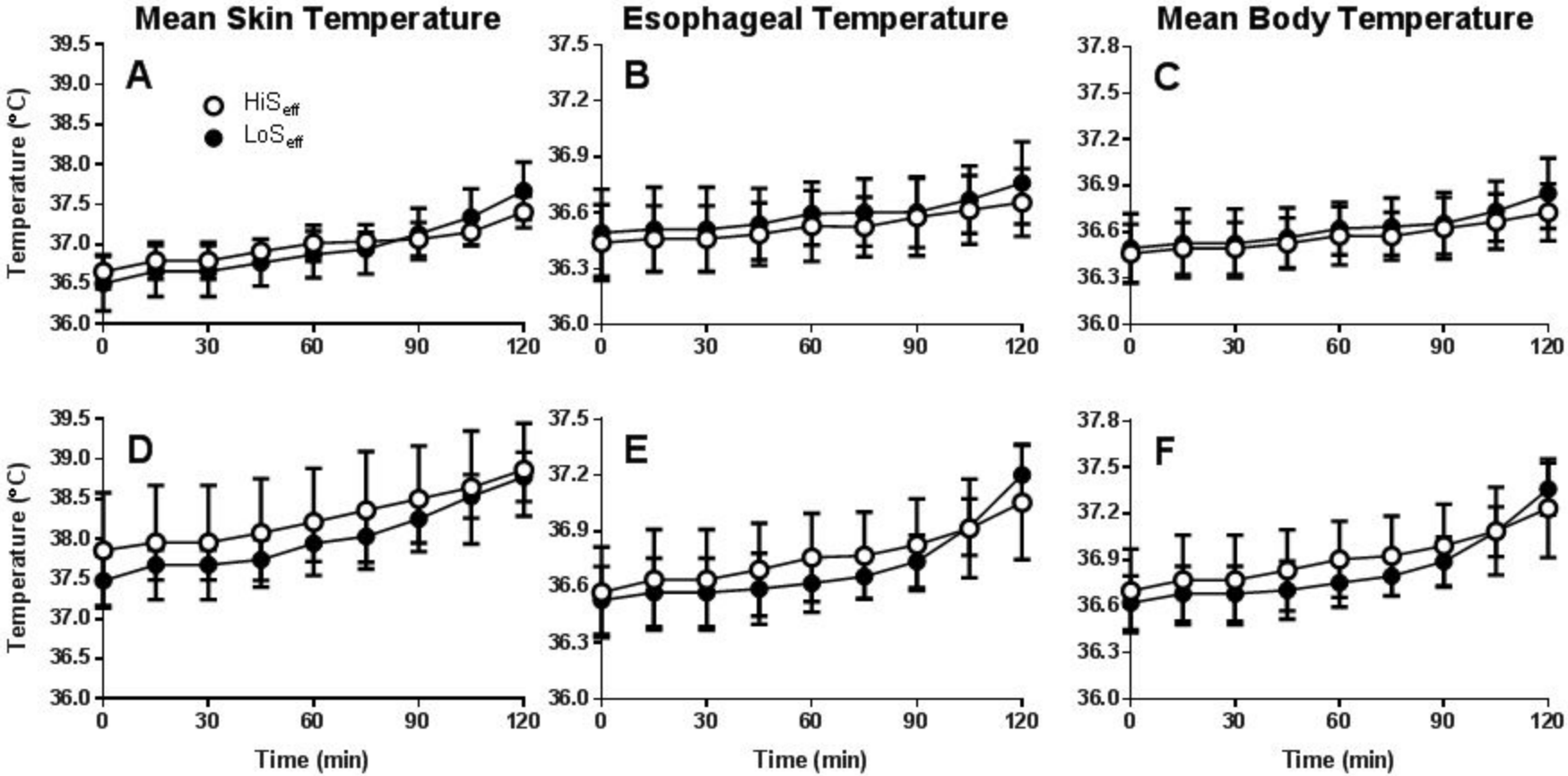
Values are mean ± standard deviation. [a]$P<0.05$ vs. SkBF during $36HiS_{eff}$; [b]$P<0.05$ vs. 36LoSeff; [c]$P<0.05$ vs. SkBF during $36LoS_{eff}$; [d]$P<0.05$ vs. CVC during $36LoS_{eff}$.

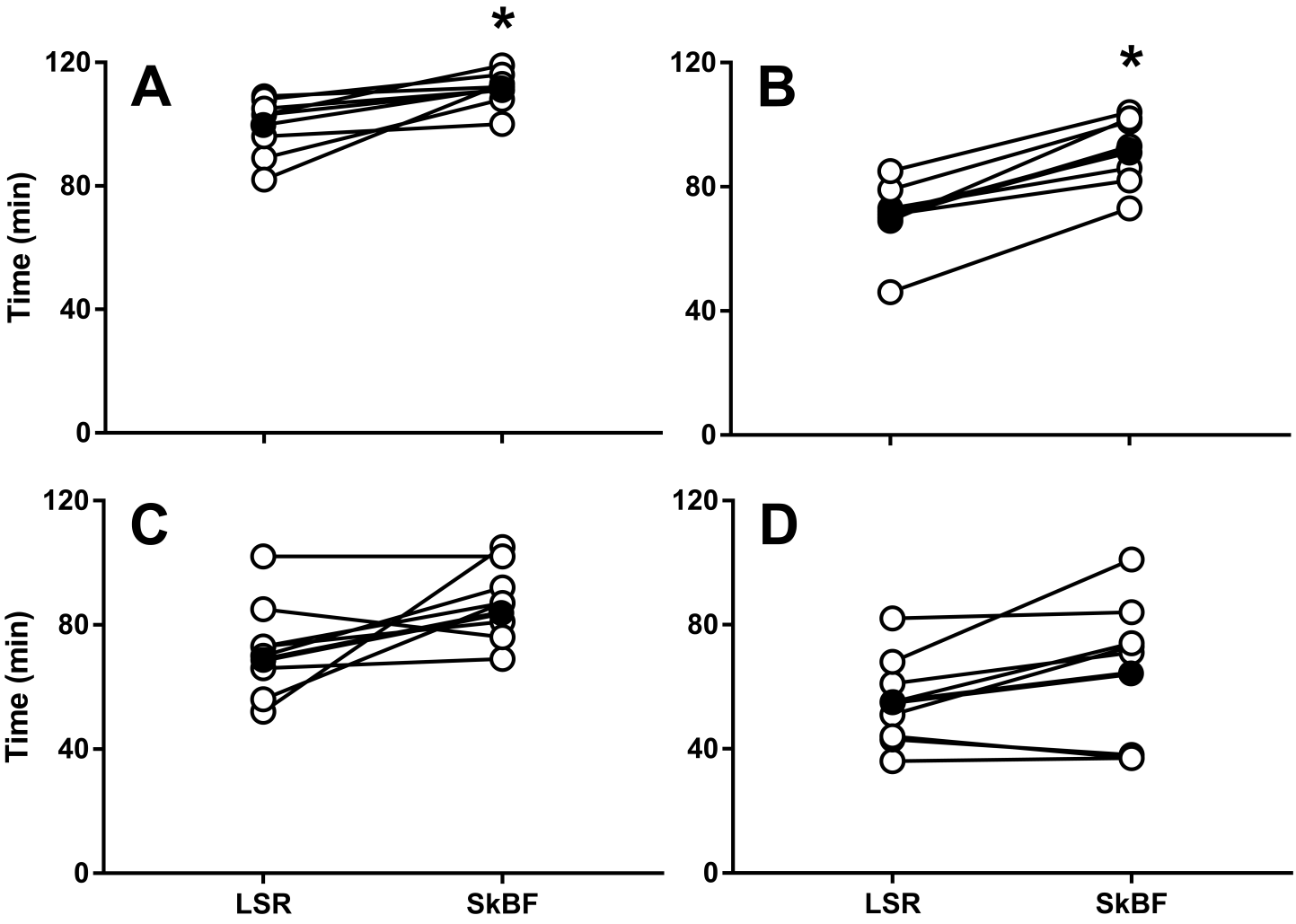
Table 2. The inflection points for sweating, skin blood flow (SkBF), and cutaneous vascular conductance (CVC) expressed as ambient vapor pressure (kPa), change in mean body temperature (ΔT_b), and time (min) during conditions of high (**42HiS$_{eff}$**) and low (**42LoS$_{eff}$**) sweating efficiency at an air temperature of 42°C.

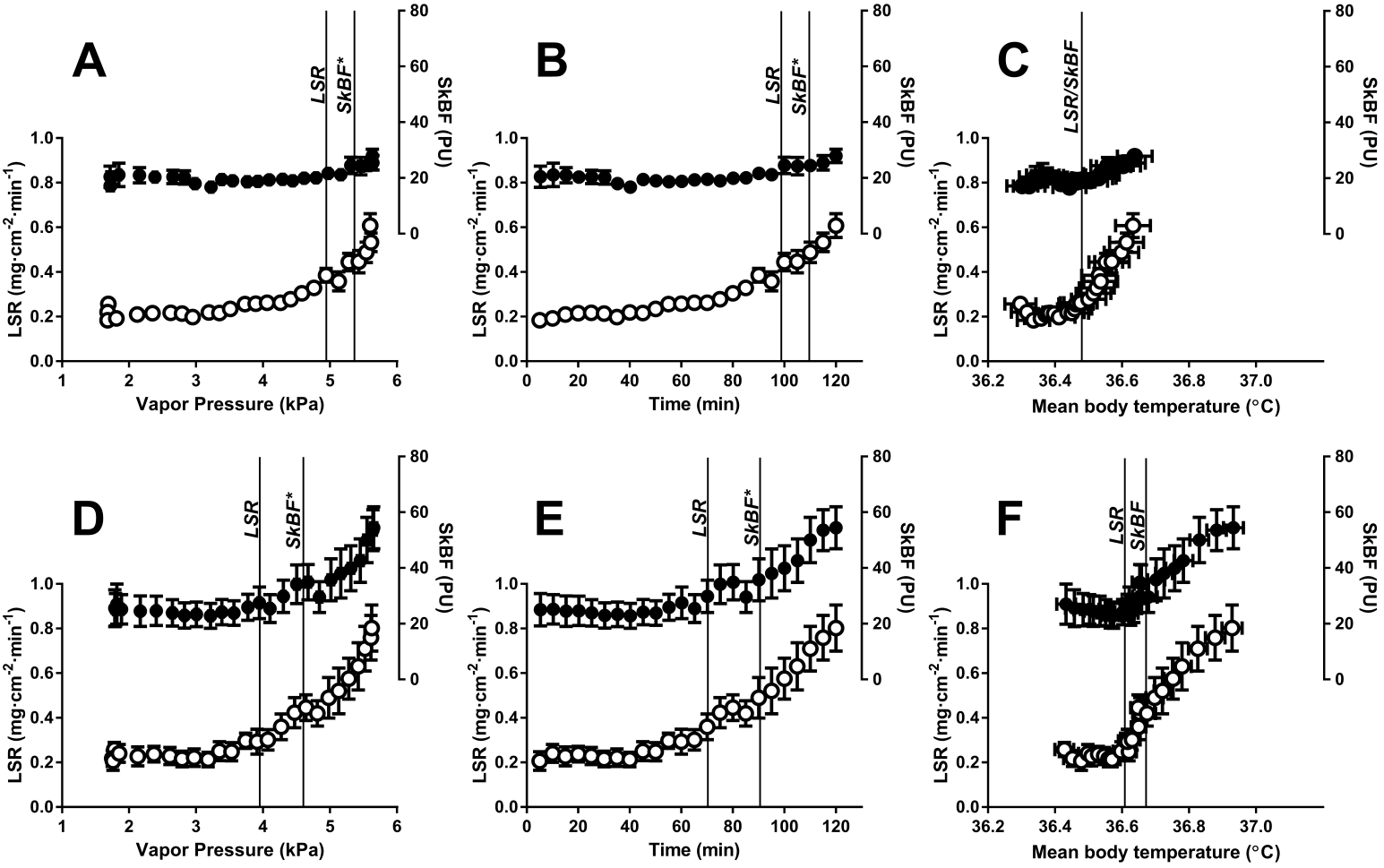
	42HiS$_{eff}$			**42LoS$_{eff}$**		
	Sweating	**SkBF**	**CVC**	**Sweating**	**SkBF**	**CVC**
Vapor Pressure (kPa)	3.83 ± 0.57[a,b]	4.29 ± 0.45	4.38 ± 0.26[a]	3.00 ± 0.65[c]	3.54 ± 0.78	3.63 ± 0.43
ΔT_b (°C)	0.36 ± 0.16[a]	0.44 ± 0.18[a]	0.45 ± 0.17[a]	0.19 ± 0.12	0.25 ± 0.20	0.27± 0.18
Time (min)	72 ± 16[a,b]	87 ± 12	92 ± 8[a]	51 ± 15[c]	64 ± 24	66 ± 13

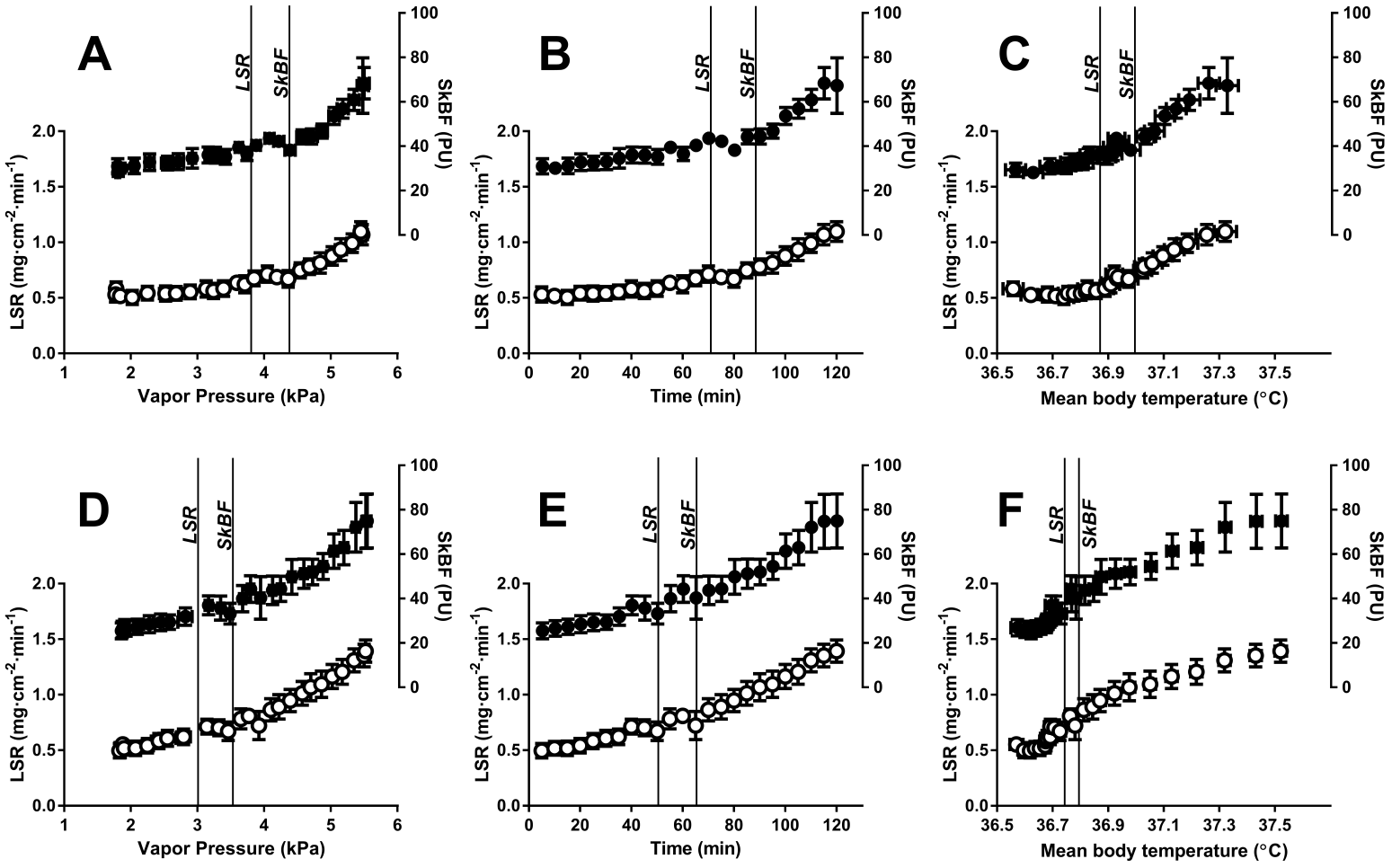
Values are mean ± standard deviation. [a]$P<0.05$ vs. 42LoS$_{eff}$; [b]$P<0.05$ vs. CVC during 42HiS$_{eff}$; [c]$P<0.05$ vs. CVC during 42LoS$_{eff}$.


A
HiS$_{eff}$
LoS$_{eff}$
LSR (mg·cm^{-2}·min^{-1})
1.5
1.0
0.5
0.0
2
4
6
Vapor Pressure (kPa)
B
Skin Blood Flow (PU)
100
80
60
40
20
0
2
4
6
Vapor Pressure (kPa)
C
CVC (PU/MAP)
1.5
1.0
0.5
0.0
2
4
6
Vapor Pressure (kPa)


Mean Skin Temperature
Esophageal Temperature
Mean Body Temperature
A
B
C
D
E
F
HiS$_{eff}$
LoS$_{eff}$
Temperature (°C)
Time (min)
39.5
39.0
38.5
38.0
37.5
37.0
36.5
36.0
37.5
37.2
36.9
36.6
36.3
36.0
37.8
37.5
37.2
36.9
36.6
36.3
36.0
0
30
60
90
120


A
B
C
D
*
*
120
80
40
0
Time (min)
LSR
SkBF


A
B
C
D
E
F
LSR
SkBF*
LSR/SkBF
SkBF
LSR (mg·cm-2·min-1)
SkBF (PU)
Vapor Pressure (kPa)
Time (min)
Mean body temperature (°C)


A
B
C
D
E
F
LSR
SkBF
LSR (mg·cm-2·min-1)
SkBF (PU)
Vapor Pressure (kPa)
Time (min)
Mean body temperature (°C)