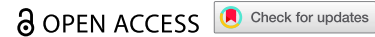


RESEARCH PAPER



Exercise-responsive skin blood flow modeling enhances thermophysiological predictions in occupational heat stress: The journal *Temperature* toolbox

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ABSTRACT

Thermophysiological models are widely used to predict human thermal responses during environmental and occupational heat stress. For predictions in the heat, a critical component is the representation of skin blood flow (SkBF), which transports heat from the body's core to the skin, where it can be lost to the environment. In many existing thermophysiological models, SkBF is represented primarily as a function of core and skin temperatures. However, they do not explicitly account for cardiovascular constraints that arise during exercise, when blood flow must be distributed between active skeletal muscles and thermoregulatory demands. In this study, we integrated the exercise-responsive SkBF model from Kraning and Gonzalez into a thermophysiological model (ThermoSEM) and compared predictions to experimental data. The experimental dataset consists of 83 trials from 25 participants performing 90 min of cycling under controlled environmental conditions (30–45°C) and metabolic heat production levels (200–500 W). Simulations using the original ThermoSEM SkBF formulation systematically overpredicted rectal temperature and underpredicted mean skin temperature, particularly at higher metabolic heat production levels. Incorporating the Kraning-based SkBF model substantially improved agreement with experimental data. Across all protocols, the root mean square error decreased by approximately 40% for rectal temperature and 26% for mean skin temperature, with the largest improvements observed during elevated metabolic heat production. The improved SkBF representation enhanced heat transport to the skin, increasing evaporative capacity and reducing excessive core heat storage. These results demonstrate that incorporating cardiovascular constraints into SkBF modeling can significantly improve thermophysiological predictions during exercise in hot environments.

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Introduction

When exposed to high levels of heat stress (arising from physical activity and/or a hot environment), the human body must regulate itself to prevent excessive increases in body temperature. The body's defense consists of both behavioral and physiological thermoregulatory mechanisms [1]. Behavioral responses include actions such as removing clothing or seeking shade, while sweating and cutaneous vasomotor responses represent two key physiological mechanisms. During physical exercise, heat is generated inside the body. Active vasodilation increases skin blood flow (SkBF), thereby enhancing convective heat transfer from the body's core to the skin [2]. At the skin surface, evaporative heat losses enabled by sweating dissipate heat to the environment and limit the rise in body temperature.

Thermophysiological models have evolved over several decades to mathematically describe heat transfer within the human body and the physiological mechanisms governing thermoregulation. Early models represented the body as a single, homogeneous compartment. A major advance was the two-node model of Gagge [3], which represented the body as a core surrounded by a skin layer connected through heat exchange and regulated by sweating, vasomotor, and shivering responses. Shortly thereafter, multi-node

models were created to capture the non-uniform distribution of temperature and thermoregulatory responses across the body. A major milestone was the 25-node model of Stolwijk [4], which divided the body into six segments, each having four tissue layers. This enabled regional predictions of tissue temperatures and thermoregulatory responses. Concurrently, Wissler developed a physiologically detailed multi-element model that did not assume uniform tissue properties within body segments. Instead, the model, published in 1985 [5], represented the human body as 15 anatomically distinct body parts connected through arteries, veins, and capillaries, with countercurrent heat exchange between arterial and venous blood. Together, the pioneering work of Wissler [6] and Stolwijk [4] established much of the physiological foundation upon which modern thermophysiological models have been developed. Building on these earlier developments, Fiala developed a detailed multi-node model [7,8] in which the thermoregulatory control mechanisms were obtained through regression on a wide range of experimental exposures. The Berkeley model [9,10] is a multi-node model that focuses on incorporating the individual's physiology and allows for flexible body segmentation. The joint system thermoregulation model (JOS) [11] further expanded the physiological representation of the human body by incorporating a detailed vascular network, including arteriovenous anastomoses, together with individualized physiological characteristics. ThermoSEM [12], based on the work of Fiala, incorporated a neurophysiological SkBF model [13]. Collectively, these successive developments have improved the physiological realism and predictive capabilities of thermophysiological models.

Today, thermophysiological models are widely used to predict human thermal responses under a broad range of environmental and exercise conditions. They are used in applications including sports, worker safety, thermal comfort, the built environment, and military operations. For example, they contributed to the development of the Portable Life Support System for the Apollo missions [14], the Universal Thermal Climate Index (UTCI) [15], which quantifies outdoor thermal stress on the human body, and, more recently, to the prediction of environmental heat and humidity limits beyond which human thermoregulatory capacity is exceeded [16]. For predictions during heat stress, the representation of SkBF is a critical component of these models because SkBF governs convective heat transfer from the core to the skin. Consequently, inaccuracies in the prediction of SkBF can propagate through the thermoregulatory system, affecting predicted skin temperatures, evaporative capacity, and ultimately core temperature.

The human cutaneous circulation is controlled by two sympathetic pathways, namely a sympathetic adrenergic vasoconstrictor system and a sympathetic cholinergic active vasodilator system recruited during heat stress [2,17]. The sympathetic cholinergic active vasodilator system is responsible for the majority of the large increases in SkBF that occur during whole-body heating, with SkBF capable of reaching $6\text{--}8\text{ L}\cdot\text{min}^{-1}$ during severe hyperthermia [18]. In addition to reflex neural control, local skin temperature also directly affects local SkBF [19]. Furthermore, SkBF regulation during exercise heat stress is influenced by factors including metabolic demand, hydration status, and interactions between thermoregulatory and cardiovascular responses.

During exercise, active skeletal muscles require additional blood flow to support increased metabolic demand, while thermoregulation simultaneously requires elevated SkBF to facilitate heat dissipation [20,21]. Consequently, SkBF regulation during exercise reflects the integration of thermal signals with cardiovascular constraints arising from competing blood flow demands. Most thermophysiological models, including those of Stolwijk [4], Gagge [3], Fiala [7,8], Kingma [12,13], and JOS-3 [11], represent SkBF primarily as functions of core and skin temperatures. However, some models explicitly incorporate the competition for blood flow between active skeletal muscle and the skin during exercise, including those of Xu and Werner [22], Wissler [6], and Kraning and Gonzalez [23]. For the present study, the exercise-response SkBF formulation developed by Kraning and Gonzalez [23] was selected because it incorporates exercise-induced modulation and can be readily integrated into our thermophysiological model ThermoSEM.

Thus, the aim of the present study was to evaluate whether incorporating the exercise-responsive SkBF formulation of Kraning and Gonzalez [23], which incorporates the influence of metabolic demand and dehydration in addition to thermal inputs, improves thermophysiological predictions during exercise in the heat. To this end, the formulation was integrated into the thermophysiological model called ThermoSEM and evaluated using experimental data from controlled exercise trials in dry heat. Predicted rectal temperature, mean skin temperature, and evaporative heat loss were compared with experimental observations to assess the influence of the SkBF formulation on model performance.

Materials and methods

ThermoSEM

ThermoSEM, which is based on the thermophysiological model of Fiala [7,8,24], is a multi-node model that predicts transient human thermal responses under varying environmental and activity conditions. The human body is represented as 18 connected concentric cylinders and a semi-spherical head, all coupled to a central blood pool. Each body element is divided into two or three sectors, and each concentric layer represents a body tissue (Figure 1a). For example, the hand consists of two sectors, dorsal and ventral, and the following tissue layers: bone (core), muscle, fat, inner skin, and outer skin. Each tissue layer is discretized into one or more computational nodes, resulting in a total of 262 nodes, including the blood pool. A detailed description of the model structure and governing equations has been provided elsewhere [12].

Thermoregulatory control includes shivering, non-shivering thermogenesis, vasomotor regulation (vasodilation and vasoconstriction), and sweating. In the original formulation, SkBF is determined by a neurophysiological control model based on weighted core and temperature signals [13]. The local sensitivity for non-thermal factors (γ_1), cold afferent pathways (γ_2), and warm afferent pathways (γ_3) obtained by Kingma et al. [12,13], and used by the neurophysiological model, have been updated in recent years. While differences are small, the updated values are given in Table 1.

This neurophysiological SkBF model predicts local SkBF responses, recognizing that thermoregulatory SkBF varies substantially between body regions, particularly between glabrous and non-glabrous skin [25–27]. During the development of the SkBF model, Kingma et al. [13] explicitly measured the local SkBF of the dorsal and ventral hand separately to capture these regional differences. While this formulation captures temperature-driven thermoregulatory responses, its performance during exercise heat stress and elevated metabolic heat production has not been extensively evaluated.

In this study, the overall ThermoSEM framework was retained without structural modifications. To isolate the effects of the SkBF formulation, sweat secretion was prescribed using experimentally measured

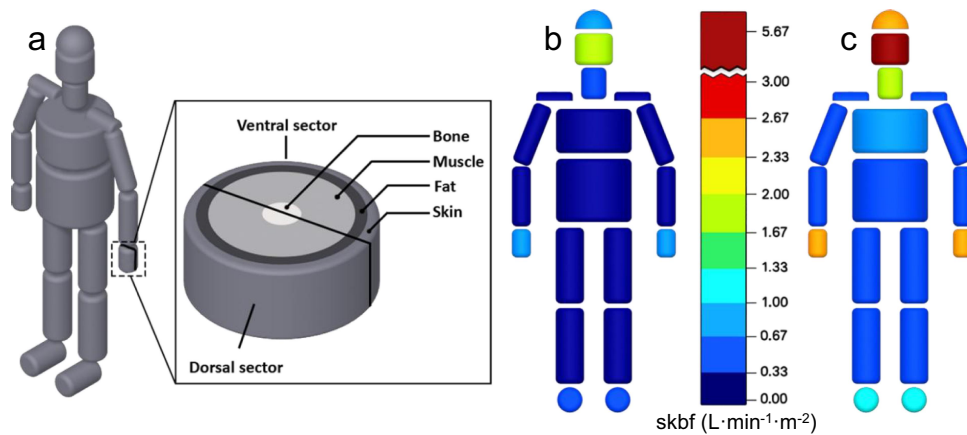


Figure 1. Overview of the ThermoSEM body representation and the implemented skin blood flow (SkBF) models. (a) ThermoSEM body geometry [24], showing the body segmentation and concentric tissue layers (hand shown as an example). (b) Predicted regional SkBF distribution using the original neurophysiological SkBF model. (c) Predicted regional SkBF distribution after application of the Kranning-based SkBF model. Both distributions correspond to the same simulation (a representative trial of protocol P7) and use the same color scale. To prevent a small number of extreme observations from dominating the color scale, values exceeding the 95th percentile were assigned to the highest color category (indicated by the discontinuity near the top of the scale).

Table 1. Neurophysiological model parameters.

Body element	γ_1	γ_2	γ_3
Head, face, neck, shoulders, upper arms, and legs	0.9763	−0.1919	−0.0577
Thorax and abdomen	2.3491	−0.1209	0.1571
Forearms	−0.4443	−0.0060	−0.2377
Hands and feet	0.6227	−0.2080	−0.0843

evaporated sweat data rather than being predicted by the model. This approach minimized potential confounding from sweat rate prediction errors, as further described in Experimental data and 2.4. Furthermore, the original SkBF formulation was retained for baseline simulations, and an alternative exercise-responsive SkBF model proposed by Kraning and Gonzalez [23], was implemented for comparison, as described in the following section.

Kraning and Gonzalez SkBF model and integration into ThermoSEM

To improve SkBF predictions during exercise-heat stress, the exercise-responsive SkBF model developed by Kraning and Gonzalez [23] was integrated into ThermoSEM. This model aims to capture the thermoregulatory control of the cardiovascular system, including predictions for heart rate, cardiac output, core blood flow, muscle blood flow, and skin blood flow [23]. However, in the present study, only the model's SkBF predictions were incorporated into ThermoSEM. Other cardiovascular variables predicted by the Kraning and Gonzalez model, including heart rate, cardiac output, and regional blood flows, were not integrated and continue to be governed by ThermoSEM's original physiological formulations.

The Kraning SkBF model requires oxygen consumption ($\dot{V}O_2$), which is estimated using the following expression:

$$\dot{V}O_2 = \frac{TMR \cdot w \cdot 3.5}{A_{sk} \cdot 58.15 \cdot 1000} \quad (1)$$

where $\dot{V}O_2$ [$L \cdot min^{-1}$] is the oxygen consumption, TMR [W] the total metabolic rate, w [kg] the body mass, and A_{sk} [m^2] the skin surface area. In equation 1 it is assumed that a metabolic heat production of 1 MET, defined as $58.15 W \cdot m^{-2}$, corresponds to an oxygen consumption of $3.5 m L_{O_2} \cdot kg^{-1} \cdot min^{-1}$ [28].

The maximum SkBF ($SkBF_{max}$) is based on the observations that exercise [29] and dehydration (water loss) [30] reduce the maximum attainable SkBF, and is given by [23]:

$$SkBF_{max} = -0.1 \cdot DL + \begin{cases} 7 & \dot{V}O_2 \leq 0.5 [L \cdot min^{-1}] \\ 7 - 1.33(\dot{V}O_2 - 0.5) & 0.5 [L \cdot min^{-1}] < \dot{V}O_2 < 2 [L \cdot min^{-1}] \\ 5 & \dot{V}O_2 \geq 2 [L \cdot min^{-1}] \end{cases} \quad (2)$$

where $SkBF_{max}$ [$L \cdot min^{-1}$] is the maximum SkBF and DL [%] is the percentage body mass loss (dehydration level). The dehydration level is defined as the net loss of body water relative to body mass, expressed as a percentage, and is given by [23]:

$$DL = \max\left(0, \frac{W_{loss} - W_{intake}}{1000 \cdot w}\right) \cdot 100 \quad (3)$$

where W_{loss} [g] and W_{intake} [g] are the cumulative water loss and intake, respectively.

The $SkBF_{\%}$ [%] is defined as a percentage of the maximum SkBF and is given by [23]:

$$SkBF_{\%} = \max(0, \min(\alpha_{SkBF} \cdot (T_{bl} - T_{set,SkBF}), 100)) \quad (4)$$

where $T_{set,SkBF}$ [$^{\circ}C$] is the threshold temperature for SkBF given by Equation 5, and α_{SkBF} [% $\cdot^{\circ}C^{-1}$] is a model coefficient given in Equation 6.

The threshold temperature for SkBF ($T_{set,SkBF}$) is modelled as a piecewise function of oxygen consumption and mean skin temperature, and is given by [23]:

$$T_{set,SkBF} = 0.06 \cdot DL + \begin{cases} 37.07 - 0.108 \cdot (T_{sk} - 30) & \dot{V}O_2 \leq 0.75 [L \cdot min^{-1}] \\ 37.32 - 0.093 \cdot (T_{sk} - 30) & \dot{V}O_2 > 0.75 [L \cdot min^{-1}] \wedge T_{sk} \leq 33 [^{\circ}C] \\ 37.04 - 0.030 \cdot (T_{sk} - 33) & \dot{V}O_2 \geq 0.75 [L \cdot min^{-1}] \wedge T_{sk} > 33 [^{\circ}C] \end{cases} \quad (5)$$

where T_{sk} [$^{\circ}C$] is the mean skin temperature.

The model coefficient α_{SkBF} is normally equal to 70.3 but decreases with increasing dehydration level, following [23]:

$$\alpha_{SkBF} = 70.3 \cdot (1 - 0.13 \cdot DL) \quad (6)$$

Finally, the whole-body SkBF is given by [23]:

$$\text{SkBF}_{\text{Kraning}} = \min \left(\text{SkBF}_{\text{min}}, \frac{\text{SkBF}_{\%} \cdot \text{SkBF}_{\text{max}}}{100} \right) \quad (7)$$

where SkBF_{min} [L/min] is the minimum SkBF, which is set at 0.03 L/min [23].

However, ThermoSEM requires local SkBF predictions for each body segment. Therefore, a factor is introduced to rescale the local SkBF predictions such that their sum matches the whole-body SkBF predicted by the Kraning model. This approach preserves the relative regional distribution of SkBF predicted by ThermoSEM while constraining total whole-body SkBF to match the Kraning-based formulation. This scaling factor is defined as the sum of all local SkBF values predicted by ThermoSEM's original SkBF model, divided by the total SkBF predicted by the Kraning model, and is expressed as:

$$f_{\text{scale}} = \frac{\sum \text{SkBF}_i}{\text{SkBF}_{\text{Kraning}}} \quad (8)$$

where SkBF_i [L/min] is the local SkBF predicted by ThermoSEM's original SkBF model.

The local SkBF for each body segment is then obtained by multiplying the original local SkBF by the scaling factor, ensuring consistency between segmental and whole-body SkBF predictions:

$$\text{SkBF}_{i,\text{Kraning}} = f_{\text{scale}} \cdot \text{SkBF}_i \quad (9)$$

where $\text{SkBF}_{i,\text{Kraning}}$ [$\text{L} \cdot \text{min}^{-1}$] is the rescaled local SkBF for body segment i .

The predicted local SkBF distributions for a representative simulation (protocol P7) are shown in Figure 1b–c. Figure 1b presents the regional skin blood flow distribution predicted when using the original neurophysiological SkBF model, and Figure 1c shows the distribution predicted when using the Kraning-based SkBF model. The colors represent the local SkBF normalized by the corresponding skin surface area. Figure 1b demonstrates that the neurophysiological model predicts physiologically realistic regional heterogeneous SkBF distributions, with glabrous body regions such as the hands, feet, and face exhibiting substantially higher flow densities than most other body regions, consistent with experimental observations reported in the literature [25–27]. Figure 1c demonstrates that the Kraning-based SkBF model preserves this regional distribution by applying the whole-body SkBF prediction only as a scaling factor to the local SkBF values as described by Equations 8 and 9, thereby modifying the overall magnitude of SkBF while retaining the regional distribution generated by ThermoSEM's original neurophysiological model.

Experimental data

Experimental data used in this study were obtained from work performed by Gagnon, Jay, and Kenny [31]. They examined the relationship between evaporative heat balance requirements and whole-body sweat rate during exercise in dry heat. Complete data were available for 25 male participants and 83 individual trials. Participant characteristics are summarized in Table 2.

In the experiments, participants performed 90 min of continuous cycling on an ergometer inside a modified Snellen air calorimeter, which enables precise measurement of whole-body heat exchange [33]. Trials were conducted under controlled environmental conditions and targeted metabolic heat production levels. Seven experimental protocols were defined, combining ambient temperatures of 30–45°C, water vapor pressures of 0.8–1.3 kPa, and metabolic heat production levels of 200–500 W (Table 3). Protocols P1–P3 varied metabolic heat production at a constant ambient temperature of 30°C, whereas protocols P4–P7

Table 2. Participant characteristics.

Characteristics	Dimension	Range	Participant average ($\pm$ SD)	Trial-weighted average ($\pm$ SD)
Age	y	19–50	31.4 ($\pm$ 11.0)	29.8 ($\pm$ 10.5)
Height	m	1.65–1.92	1.81 ($\pm$ 0.06)	1.81 ($\pm$ 0.07)
Mass	kg	70.3–99.8	83.3 ($\pm$ 8.7)	82.8 ($\pm$ 8.2)
Surface area ^a	m ²	1.82–2.25	2.04 ($\pm$ 0.12)	2.03 ($\pm$ 0.12)
Fat percentage	%	6.6–30.2	18.0 ($\pm$ 7.4)	—
VO _{2max}	mL·kg ^{−1} ·min ^{−1}	36.6–63.0	51.1 ($\pm$ 6.7)	—

^aMethod by Du Bois and Du Bois [32].

Table 3. Environmental conditions, including ambient temperature (T_{amb}), ambient water vapor pressure ($P_{\text{v,water}}$), and relative humidity (RH), together with total metabolic heat production (TMH), and the number of participants for each experimental protocol.

Protocol	T_{amb} [°C]	$P_{\text{v,water}}$ [kPa]	RH [%]	TMH [W]	Number of participants
P1	30	1.3	31	200	7
P2	30	1.3	31	350	6
P3	30	1.3	31	500	7
P4	30	0.8	19	290	21
P5	35	0.8	14	290	18
P6	40	0.8	11	290	19
P7	45	0.8	8	290	5

varied ambient temperature at a fixed metabolic heat production of 290 W. Participants completed multiple trials across different protocols, resulting in a total of 83 trials with complete measurement data.

Rectal temperature was measured using a thermocouple probe inserted 12 cm past the anal sphincter. Skin temperatures were measured using thermocouples at four sites (upper trapezius, chest, quadriceps, and calf). Mean skin temperature was calculated using the weighting factors proposed by Ramanathan [34]. Rectal and skin temperatures were recorded at 15 s intervals. Whole-body dry and evaporative heat exchange was measured using the modified Snellen air calorimeter, which determines evaporative and dry heat loss by measuring the difference in air temperature and humidity between the inlet and outlet [35]. These measurements were used to determine whole-body evaporative heat loss, which includes water evaporated in the respiratory tract. Metabolic rate was determined from indirect calorimetry based on measurements of oxygen consumption and carbon dioxide production, and metabolic heat production was calculated by subtracting external work performed on the ergometer. Continuous measurements were available during the 90-min exercise period and for the 15 min preceding the onset of exercise.

Simulations

Each experimental trial was simulated separately using the individual participant characteristics and the corresponding experimentally measured environmental and physiological inputs. Simulations were performed using three configurations: (i) the standard ThermoSEM model, (ii) ThermoSEM incorporating the Kraning-based SkBF model activated only during the exercise period, and (iii) ThermoSEM incorporating the Kraning-based SkBF model activated throughout the entire simulation duration. Although the experimental analysis presented in this study focused on the exercise period, the complete pre-exercise sequence of the experiment was included in the simulations to obtain physiologically realistic initial thermal conditions at exercise onset.

The total simulated duration was 210 min, consisting of two pre-exercise periods followed by the 90-min exercise period (Table 4). The prescribed input variables differed between periods depending on the availability of experimental measurements.

During the first pre-exercise period (0–60 min), participants were instrumented while seated outside the air calorimeter. No continuous physiological measurements were available for this phase; therefore,

Table 4. Overview of simulation periods and prescribed time-dependent input variables. Abbreviations: T_{amb} = ambient temperature; RH = relative humidity; v_{air} = air velocity.

Period description	Minutes	Environmental conditions (T_{amb} ; RH; v_{air})	Metabolic heat production	External Work	Sweat secretion rate
Participant sitting in a chair for the attachment of measurement equipment outside the air calorimeter	0 to 60	24°C [31]; 50%; 0.05 m/s	1 MET	0	0
Participant sitting inside the air calorimeter on the ergometer	61 to 120	Table 3; 0.1 m/s	Average of the 15 min of available measurement data prior to the onset of exercise		
Participant exercises on the ergometer inside the air calorimeter	121 to 210	Table 3; 0.3 m/s	90 min time average		Direct measurement data

standardized resting conditions representative of the experimental protocol were imposed, as summarized in Table 4.

The second pre-exercise period (61–120 min) corresponded to seated rest inside the air calorimeter. Although this period lasted 60 min, measurement data were available only for the final 15 min preceding exercise. For each individual trial, the time-averaged values of metabolic heat production, external work, and sweat rate over these 15 min were calculated and prescribed as constant inputs for the entire 60-min period. Note that the experimentally measured evaporative sweat rate includes water losses from the respiratory tract. Respiratory losses were calculated following the method described in ISO 7933 [36] and subtracted from the evaporative sweat measurement data. These corrected values were then prescribed as the sweat secretion rate in the simulations. Although the air calorimeter cannot directly distinguish between sweat evaporated at the skin surface and sweat that evaporated after dripping, the dry environmental conditions and airflow used in the experiments were specifically selected to promote complete evaporation of secreted sweat. Prescribing sweat secretion enabled isolation of the influence of the SkBF formulation on thermal predictions but prevented fully coupled physiological interactions between sweating and thermoregulatory control.

During the 90-min exercise period (121–210 min), metabolic heat production and external work were prescribed as the time-averaged values over the full 90 min of the corresponding individual trial and imposed as constant inputs for the entire exercise duration. In contrast, the sweat secretion rate was prescribed using the directly measured evaporated sweat rate (corrected for respiratory evaporative losses).

The simulation configurations were designed to distinguish between controlled and full-duration application of the Kraning-based SkBF model. In the standard configuration, ThermoSEM's default SkBF formulation was applied throughout the entire simulation. In the exercise-only configuration, the exercise-responsive Kraning-based SkBF model was activated exclusively during the exercise period (121–210 min), while the standard SkBF model was retained during pre-exercise (0–120 min). This approach ensured identical thermal states at exercise onset, thereby isolating the effect of the SkBF formulation during exercise. In contrast, in the continuous configuration, the Kraning-based SkBF model was applied over the entire simulation (0–210 min), allowing evaluation of the model as a general SkBF formulation. In this case, differences in thermophysiological responses during exercise reflected both the direct influence of the SkBF formulation and its impact on the development of the initial thermal state prior to exercise.

In the experiments, clothing insulation was standardized for all participants at 0.2–0.3 clo, consisting of cotton underwear, shorts, socks, and athletic shoes [37]. ThermoSEM, however, requires segment-specific thermal clothing properties. Therefore, local clothing insulation values representative of the experimental clothing ensemble were obtained from thermal manikin measurements reported by Veselá et al. [38]. Moving manikin data were selected to reflect the cycling motion of the participants. Additionally, the thermal effect of contact with the ergometer seat and back support was accounted for. Local insulation values for the upper legs and feet were obtained from Table A.17 (light clothing ensemble; moving), and corrections for the seated posture were applied using Tables 2.3 and 2.4 from reference [38]. Because clothing covered only a limited body surface area (only upper legs and feet) and the insulation values were low, uncertainties in the estimated local insulation properties were expected to have a minor influence on whole-body thermal responses.

Results and discussion

Figure 2 shows the measured and predicted rectal temperature (T_{re}) responses during the final 10 min preceding exercise and throughout the exercise period. Thin lines show individual runs, bold lines indicate the group mean, and shaded areas denote the 95% confidence interval. Experimental data are shown in gray, while model predictions are distinguished by SkBF configurations: blue represents ThermoSEM with the standard SkBF model as described by Kingma et al. [13], orange represents the Kraning-based SkBF model activated only during the exercise period, and yellow represents the Kraning-based SkBF model applied over the full simulation duration. Each subfigure corresponds to a single experimental protocol (P1–P7), as defined in Table 3. All subfigures use identical y-axis limits except P3, for which an adjusted scale is used to accommodate higher rectal temperatures and preserve readability across panels.

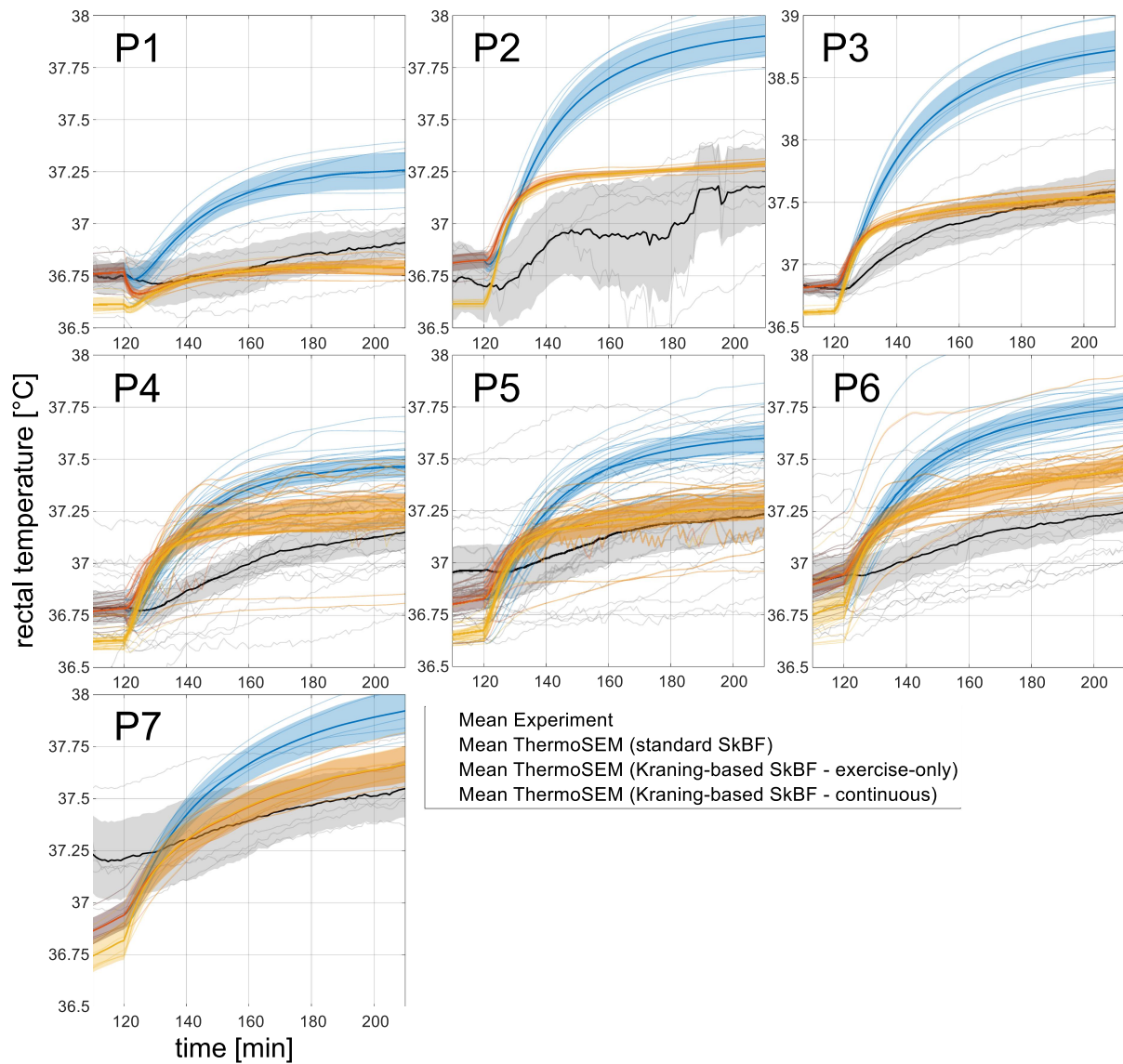


Figure 2. Comparison of experimental and simulated rectal temperature responses during the exercise period for protocols P1–P7 (see Table 3). The thin lines show individual runs, the bold lines the group mean, and shaded areas the 95% confidence interval.

Across all protocols, the onset of exercise (starting at minute 120) induces a progressive increase in rectal temperature, with both the rate of increase and the final rectal temperature depending on metabolic heat production and environmental conditions. Experimental data exhibit substantial inter-individual variability, as reflected by the spread of individual trajectories (thin gray lines) and the width of the 95% confidence intervals (gray shaded areas).

Protocols P1–P3 isolate the effect of increasing metabolic heat production (200 W in P1, 350 W in P2, and 500 W in P3) with identical ambient conditions ($T_{\text{amb}} = 30^{\circ}\text{C}$, $\text{RH} \approx 31\%$). The experimental data show that higher metabolic heat production leads to a larger and faster increase in rectal temperature. Across all three protocols, ThermoSEM, using the standard SkBF, systematically overestimates this increase, with the magnitude of the overprediction increasing as metabolic heat production increases. This behavior is most pronounced in protocol P3, where predicted rectal temperatures reach 38.7°C ($\Delta T_{\text{re}} = 1.9^{\circ}\text{C}$) at the end of the exercise period, whereas experimental data reach only 37.6°C ($\Delta T_{\text{re}} = 0.8^{\circ}\text{C}$). The Kraning-based SkBF substantially reduces these overpredictions in rectal temperature. In protocol P3, the ΔT_{re} is 0.7°C with exercise-only activation and 0.9°C with continuous activation. However, despite this overall improvement,

the Kraning-based model still tends to overpredict the initial rate of rectal temperature rise during the early stages of exercise.

Protocols P4–P7 vary ambient temperature (30°C in P4, 35°C in P5, 40°C in P6, and 45°C in P7) while maintaining a constant metabolic heat production of approximately 290 W. The experimental data of protocols P4–P6 show comparable rectal temperature progressions, despite increasing ambient temperature. In protocol P7, the rectal temperature is elevated throughout the exercise period; however, this difference primarily reflects a higher initial rectal temperature at the onset of exercise rather than a greater rate of increase. Model characteristics are similar to those found in protocols P1–P3; ThermoSEM with the standard SkBF systematically overpredicts rectal temperatures, whereas the Kraning-based SkBF reduces these overpredictions and improves agreement with experimental measurements across all environmental conditions.

Differences in the predicted rectal temperature responses of the exercise period when the Kraning-based SkBF is activated only during the exercise period (orange lines) or throughout the entire simulation (yellow lines) are small. While both models start at different temperatures at the onset of exercise (i.e. min 120), their predictions rapidly converge, becoming nearly indistinguishable within approximately 15–20 min. This initial offset is expected, as the exercise-only configuration follows the standard SkBF model during the pre-exercise period, whereas the continuous configuration applies the Kraning-based SkBF throughout the entire simulation, including the pre-exercise periods. Consequently, the continuous configuration starts at lower rectal temperatures at exercise onset compared to the exercise-only configuration. Notably, pre-exercise rectal temperatures are closer to the experimental data when the standard SkBF model is used, consistent with previous validation of ThermoSEM under mild conditions [12,13,38]. Once both models operate under the Kraning-based SkBF during exercise, the influence of the differing initial conditions decays quickly, resulting in near-identical rectal temperature responses for the remainder of the exercise period.

Figure 3 shows the measured and predicted mean skin temperature (T_{sk}) responses for all protocols, using the same graphical conventions as in Figure 2. Note that in protocol P7, an adjusted scale is used to accommodate the higher mean skin temperatures observed in this protocol due to the high ambient temperature (45°C). Experimental measurements show a small rise in mean skin temperature during the exercise period. In contrast to rectal temperature, absolute mean skin temperature depends primarily on ambient temperature rather than metabolic heat production. Experimental data also show considerable inter-individual variability, particularly in the warmest protocol (P7), where individual trajectories diverge substantially.

The experiments show that under identical ambient conditions ($T_{amb} = 30^\circ\text{C}$, $RH \approx 31\%$), increasing metabolic heat production from 200 W (P1) to 500 W (P3) results in similar overall mean skin temperature responses, with mean skin temperature consistently increasing from $\sim 33.9^\circ\text{C}$ at exercise onset to $\sim 34.3^\circ\text{C}$ at the end of exercise. Inter-individual variability decreases as metabolic heat production increases. In protocols P1 and P2, some individuals have relatively low mean skin temperatures, whereas others exhibit relatively high values. In protocol P3, low mean skin temperature responses are absent, while maximum mean skin temperatures remain comparable to those observed in protocols P1 and P2, thereby reducing inter-individual variability.

For protocols P1–P3, ThermoSEM with the standard SkBF predicts a pronounced decrease in mean skin temperature, and the magnitude of this decrease increases with increasing metabolic heat production. This behavior is consistent with insufficient convective heat transfer from the body core to the skin, as SkBF transports heat from the warmer body core to the periphery. If the simulated SkBF is insufficient to sustain heat delivery from the core to the skin, evaporative heat loss can exceed heat supplied to the skin, resulting in underprediction of mean skin temperature.

ThermoSEM using the Kraning-based SkBF predicts higher mean skin temperatures that more closely match experimental observations. However, at higher metabolic heat productions (P2 and P3), small underpredictions persist. For protocols P1–P3, the Kraning-based model predicts an average SkBF approximately 4 to 8 times higher than those predicted using the standard SkBF model, as shown in Table 5. This implies a greater convective heat transfer from the core to the skin, thereby increasing predicted skin temperatures.

Experimental data from protocols P4–P7 show a clear effect of ambient temperature on mean skin temperature. While absolute mean skin temperature increases at higher ambient temperatures, the rise in skin temperature during the exercise period remains comparable across all four protocols.

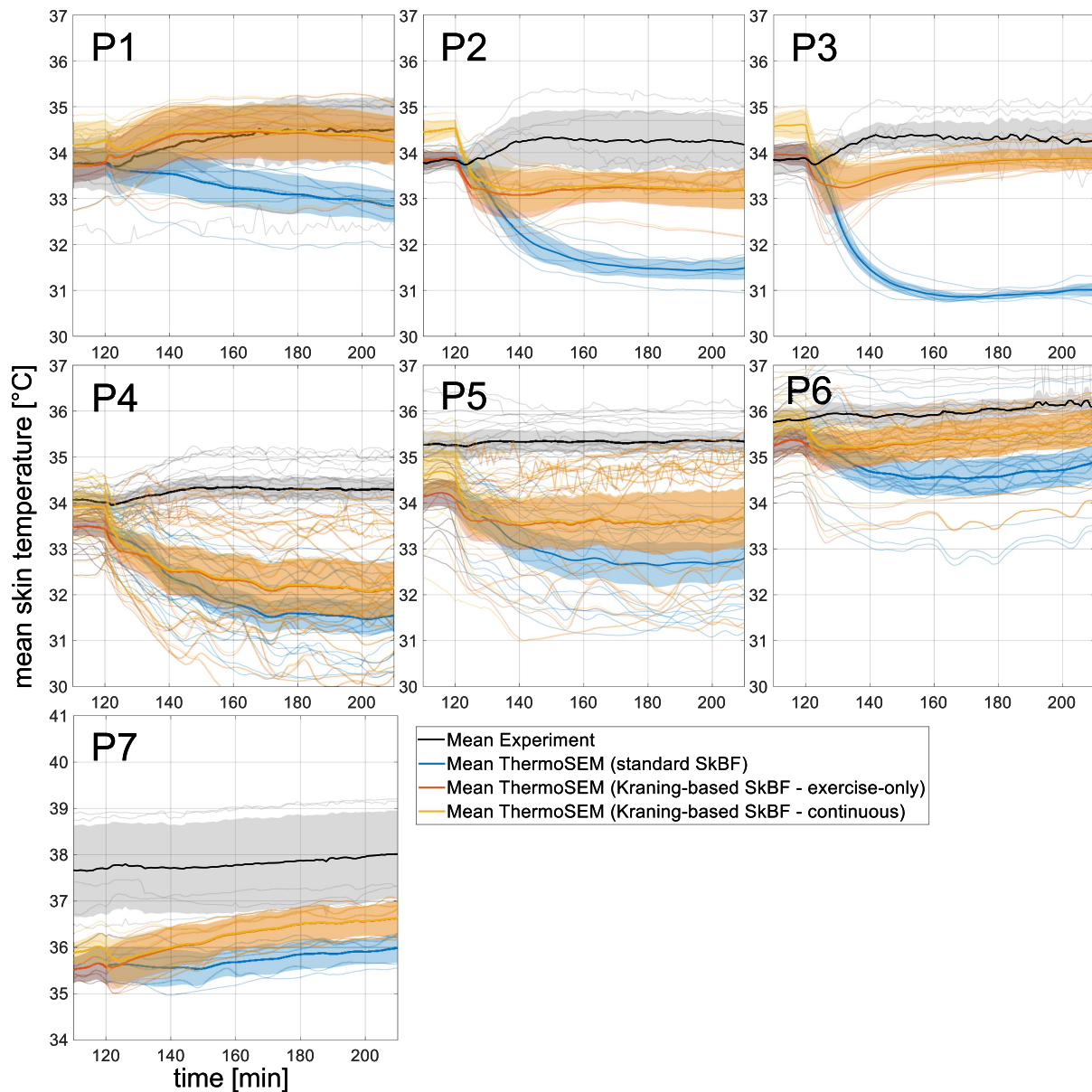


Figure 3. Comparison of experimental and simulated mean skin temperature responses during the exercise period for protocols P1–P7 (see Table 3). The thin lines show individual runs, the bold lines the group mean, and shaded areas the 95% confidence interval.

Table 5. Averaged skin blood flow (SkBF) predictions of the exercise period in [L/min].

Protocol	Standard SkBF	Kraning-based SkBF (exercise-only)	Kraning-based SkBF (continuous)
P1	0.29	1.26	1.29
P2	0.20	0.68	0.69
P3	0.16	1.22	1.23
P4	0.20	0.39	0.39
P5	0.26	0.68	0.69
P6	0.48	1.44	1.46
P7	0.66	1.99	2.01

For protocols P4–P7, ThermoSEM with the standard SkBF systematically underpredicts mean skin temperatures. However, these underpredictions tend to decrease slightly as ambient temperature increases. For example, the error in mean skin temperature at the end of exercise is approximately -2.8°C in P4, -2.6°C in P5, -1.3°C in P6, and -2.0°C in P7.

While the predicted mean skin temperature progressions with the Kraning-based SkBF model are similar to those predicted with the standard model, the absolute temperatures are higher. Consequently, the underpredictions in mean skin temperature are reduced, resulting in improved agreement with the measurement data.

For mean skin temperatures, a similar convergence between the two Kraning-based configurations is observed, with predictions becoming nearly identical within approximately 10–20 min after the onset of exercise. In contrast to rectal temperature, no consistent improvement in agreement with experimental data is observed at the onset of exercise; depending on the protocol, either configuration may more closely match the measurements. The continuous Kraning-based configuration consistently predicts higher mean skin temperatures at exercise onset than the exercise-only configuration, which is likely attributable to higher SkBF values predicted during the pre-exercise period.

The large underpredictions in mean skin temperature predicted by ThermoSEM using the standard SkBF model are likely related to the conditions under which this model was originally developed. The data used for the development of this SkBF model consist of experiments performed in mild conditions in which participants rested in a supine position and experienced minimal to no active sweating [13]. Under such conditions, thermophysiological predictions accurately represented experimental measurement data when using this SkBF model. However, experimental conditions of the University of Ottawa dataset differ substantially, as they involve sustained exercise and significant sweat evaporation. The standard SkBF model likely overemphasizes the influence of skin temperature while incompletely representing the effects of sweat evaporation and the additional exercise-related physiological drive on SkBF. For example, decreases in skin temperature due to evaporative cooling lead to a reduction in SkBF. This reduction in SkBF lowers convective heat transfer from the core to the skin, which further decreases skin temperature, thereby creating a reinforcing feedback loop.

In the Kraning-based SkBF model, core temperature plays a more predominant role than skin temperature. Moreover, this model also uses total metabolic rate (TMR) as an important input for predicting SkBF. Consequently, it is less sensitive to decreases in skin temperature due to sweat evaporation than the standard approach, thereby reducing the risk of excessive reductions in SkBF and the associated underprediction of mean skin temperature observed in the standard SkBF model.

Overall, in [Figures 2 and 3](#), it is seen that ThermoSEM using the standard SkBF model overpredicts rectal temperatures and underpredicts mean skin temperatures. The increased SkBF from the Kraning-based SkBF model attenuates core heat storage by enhancing convective heat transfer from the core to the skin and increasing evaporative heat dissipation, thereby limiting the rise in rectal temperature and elevating mean skin temperatures. Consequently, agreement with experimental measurements of both rectal and mean skin temperature is improved. However, thermal predictions of ThermoSEM using the standard SkBF model align more closely with experimental data at, and immediately before, the onset of exercise (i.e. min 110–120), particularly for rectal temperature. This is consistent with previous studies showing good performance of the standard SkBF formulation under resting and moderate environmental conditions [13,38]. This suggests that the standard SkBF formulation may be more appropriate under resting and moderate environmental conditions, whereas the Kraning-based formulation improves predictions during exercise heat stress.

A summary of the average SkBF predicted during exercise is shown in [Table 5](#). The Kraning-based model predicts substantially higher SkBF values than the standard SkBF model, with average SkBF values approximately 3.4 times greater across all protocols. For protocols P1–P3, the standard model predicts decreasing SkBF values with increasing metabolic heat production. This behavior is consistent with the previously discussed tendency of the standard model to overemphasize the influence of skin temperature, resulting in a reduction in SkBF under conditions of enhanced evaporative cooling at higher exercise intensities. In contrast, the SkBF predictions of the Kraning-based model for protocols P1–P3 reflect its ability to capture the competing vascular demands of active musculature and thermoregulatory heat dissipation. Although the heat stress and rectal temperature responses, and consequently the thermoregulatory drive, in protocol P1 are lowest, it also has the lowest metabolic demand. As a result, competition for blood flow between active skeletal muscle and the skin is minimal, allowing a relatively large proportion of cardiac output to be directed toward thermoregulatory SkBF, yielding a relatively high predicted SkBF ($1.26 \text{ L}\cdot\text{min}^{-1}$). In protocol P2, the higher metabolic demand increases the competing perfusion requirements of active skeletal muscle, thereby reducing the SkBF predicted by the model ($0.68 \text{ L}\cdot\text{min}^{-1}$). Despite the further increase in metabolic demand in protocol P3, the substantially greater thermoregulatory drive associated with the higher body temperatures produces a much stronger demand for cutaneous vasodilation, which outweighs the additional cardiovascular competition from exercising muscle. Consequently, predicted SkBF

increases to $1.22 \text{ L}\cdot\text{min}^{-1}$, illustrating that the model represents SkBF as the outcome of a dynamic balance between competing muscular blood flow requirements and thermoregulatory demand, rather than muscle blood flow competition alone. For protocols P4–P7, both models predict increasing SkBF with increasing ambient temperature; however, this increase is more pronounced in the Kraning-based model. These differences in SkBF are consistent with the previously observed thermal responses. Finally, differences between the two Kraning-based configurations (exercise-only vs. continuous) are negligible across all protocols, with deviations being smaller than $0.03 \text{ L}\cdot\text{min}^{-1}$.

Figure 4 shows the measured and predicted evaporated sweat rate [g/min] for all protocols, using the same graphical conventions as in Figures 2 and 3. As stated previously, the air calorimeter measures the rate of sweat evaporation. Although the calorimeter cannot directly distinguish between sweat evaporated at the skin surface and sweat evaporated after dripping, the authors of the experimental study [37] argue that the selected environmental conditions (e.g. high air temperature, low humidity, and sufficient airflow) promoted near-complete evaporation of secreted sweat. In the simulations, sweat secretion rate was prescribed using the

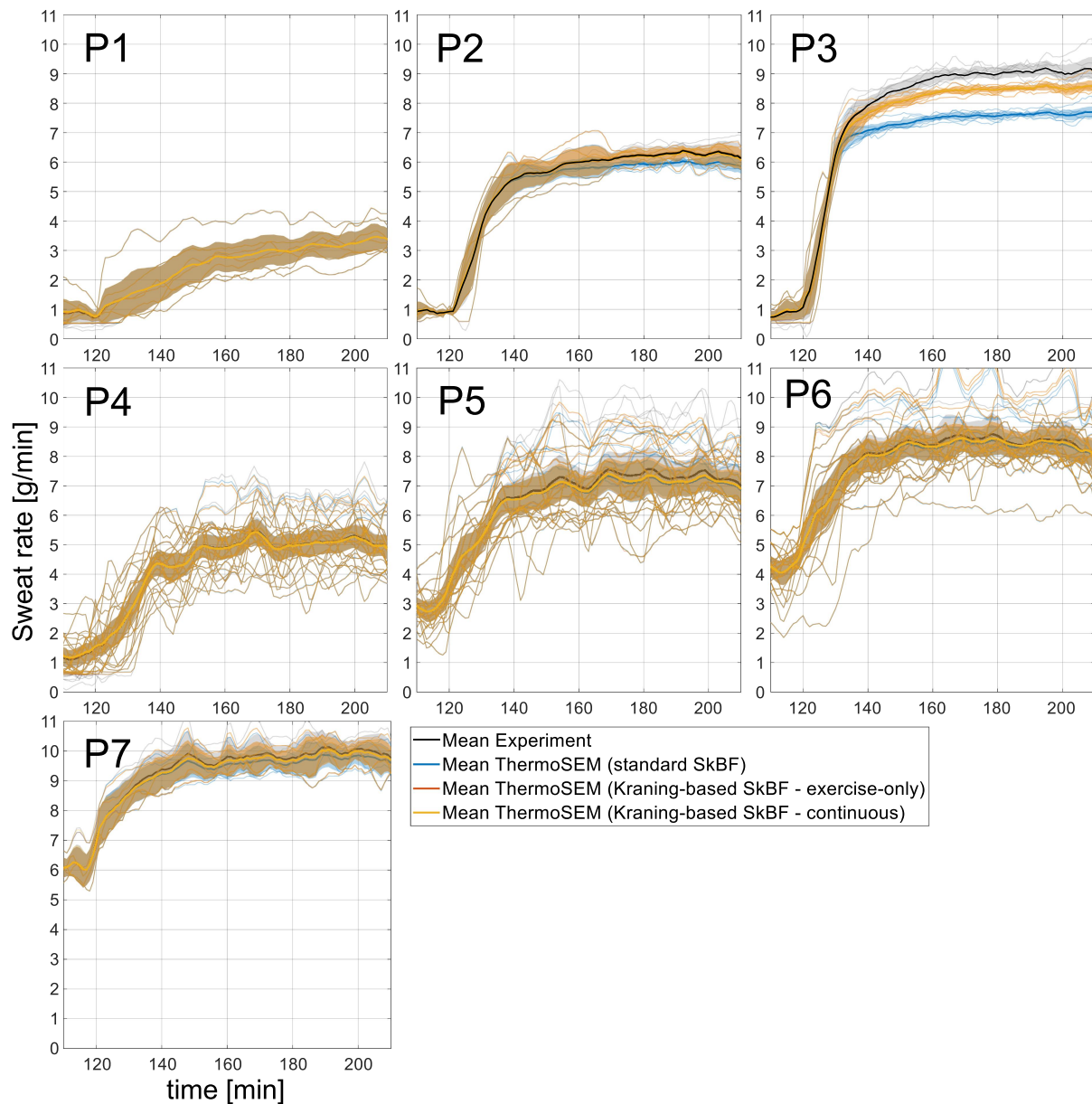


Figure 4. Comparison of experimental and simulated evaporated sweat rate during the exercise period for protocols P1–P7 (see Table 3). The thin lines show individual runs, the bold lines the group mean, and shaded areas the 95% confidence interval.

measured evaporation rates corrected for respiratory evaporative losses. The predicted evaporated sweat rate is determined by the model's representation of evaporative heat and mass transfer, which depends on local skin and environmental conditions, and therefore does not necessarily equal the secreted sweat rate.

Figure 4 shows that the predicted evaporated sweat rate closely matches the experimental measurements across most protocols. On average, ThermoSEM predicts that 96.7% of secreted sweat evaporates when using the standard SkBF model, and 98.7% when using either of the two Kraning-based SkBF model configurations. These simulation results are consistent with the experimental observation that the selected environmental conditions promoted near-complete evaporation of secreted sweat during the trials.

A notable exception occurs in protocol P3. In this case, ThermoSEM using the standard SkBF model predicts a substantially lower evaporated sweat rate than measured, with only 86.0% of the prescribed sweat secretion predicted to evaporate. However, this mismatch between measured and predicted evaporation rate arises from the large underprediction of mean skin temperature observed in Figure 3. The evaporative mass flux is governed by the water vapor pressure gradient between the skin surface and the surrounding air. Because the saturation vapor pressure at the skin surface increases strongly with skin temperature, lower predicted skin temperatures reduce the vapor pressure gradient and thereby limit the model's evaporative capacity. As a result, not all secreted sweat can evaporate in the simulation when using the standard SkBF model. This highlights the coupled interactions among SkBF, skin temperature, and evaporative heat loss, indicating that accurate thermal predictions require all three processes to be represented consistently. Furthermore, this explains why there is no observable difference in evaporated sweat rate between the two Kraning-based SkBF model configurations (exercise-only vs. continuous), as the predicted mean skin temperatures are nearly identical.

When the Kraning-based SkBF model is used, the predicted mean skin temperature approaches the level observed in the experimental data, leading to a higher skin surface vapor pressure and a corresponding increase in evaporative capacity. Consequently, the predicted evaporated sweat rate in P3 increases from 86.0% to 94.8%, resulting in substantially improved agreement with the experimental measurements.

Next, the root mean square error (RMSE) was calculated for each protocol to quantify agreement between model predictions and experimental measurements for both rectal temperature (Figure 5a) and mean skin temperature (Figure 5b).

Figure 5a shows that for ThermoSEM using the standard SkBF model, the RMSE values range from approximately 0.3°C (P1) to 1.0°C (P3). For this model, the RMSE quickly rises with increasing metabolic heat production (P1–P3). In contrast, protocols characterized by elevated ambient temperatures (P4–P7) do not demonstrate a commensurate rise in RMSE, suggesting that the model is more sensitive to metabolic load than to external thermal conditions. This reflects the fundamentally different origin of heat stress in these protocol groups. In P1–P3, heat stress is primarily driven by internal heat generation due to metabolic activity. Under these conditions, SkBF plays a dominant role in regulating core temperatures. Two distinct mechanisms are important for the rise in rectal temperature. First, low SkBF limits convective heat transfer from the core to the skin, thereby increasing core heat storage. Second, at elevated sweat rates, insufficient SkBF limits the delivery of warm blood to the periphery, resulting in lower skin temperatures, reduced evaporative capacity, reduced evaporative heat dissipation, and a further rise in core temperature. This latter mechanism is clearly observed in P3, where metabolic heat production was highest (500 W), and the measured evaporation rate was second-highest among all protocols. Here, low SkBF predictions result in a notable loss of evaporative capacity (see Figure 4), thereby increasing heat storage in the body and producing the largest overpredictions in rectal temperature (approximately 1.1°C, see Figure 2).

In contrast, in protocols P4–P7, heat stress arises primarily from elevated ambient temperatures rather than increased internal heat generation. Higher ambient temperatures directly elevate skin temperatures through environmental heat exchange, which partially compensates for reduced convective heat transfer from the core to the skin caused by low SkBF predictions, and avoids any significant decrease in evaporative capacity. As a result, RMSE values remain relatively stable across increasing ambient temperatures.

Figure 5a also shows that when using the Kraning-based SkBF model (both exercise-only or continuous configuration), rectal temperature prediction errors are substantially reduced across all protocols. RMSE values of the two Kraning configurations are nearly identical, as predicted thermal responses are similar throughout most of the exercise period (from approximately min 140 to 210). When using the Kraning-based SkBF model, RMSE values decrease to a range of approximately 0.1°C (P1) to 0.4°C (P2). On average, RMSE

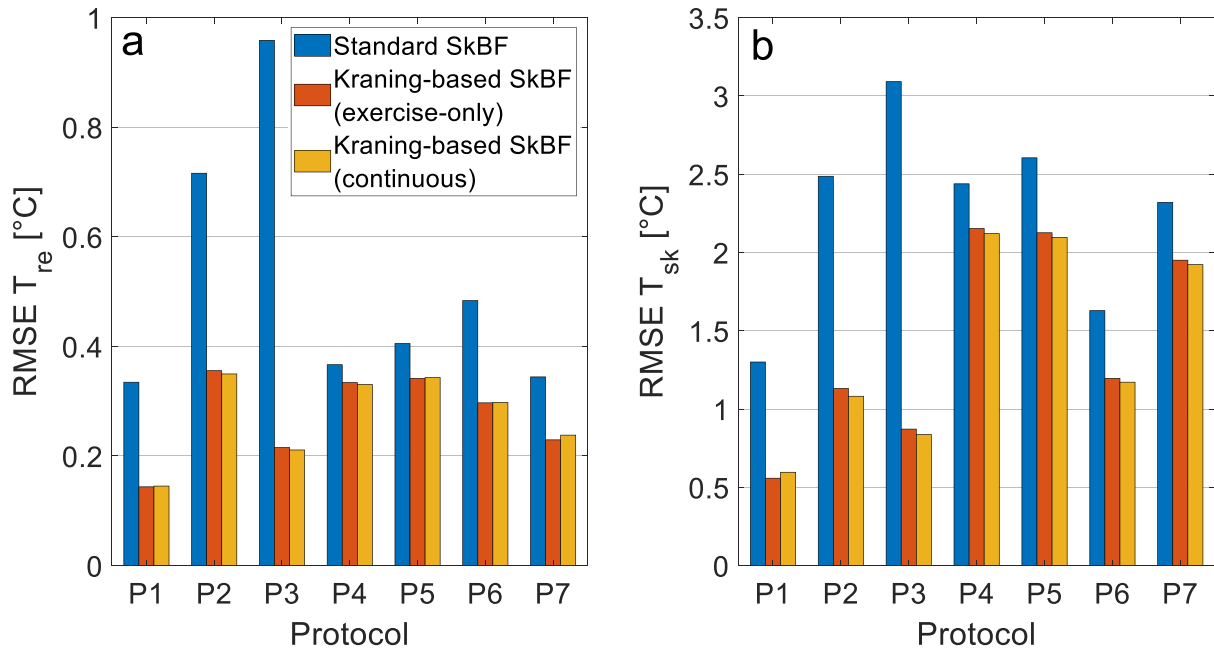


Figure 5. RMSE per protocol of ThermoSEM predictions using the standard SkBF model or Kraning-based SkBF model configurations of (a) rectal temperature and (b) mean skin temperature.

decreases by 40% or 0.2°C, with an average decrease of 65% or 0.5°C for P1–P3 and 23% or 0.1°C for P4–P7. As expected, the largest improvements are observed in protocols with elevated metabolic heat production, where SkBF plays a more important role in transferring heat from the core to the skin. In contrast to the standard SkBF model, RMSE values from ThermoSEM using the Kraning-based SkBF model remain low and relatively uniform across both metabolic and environmental heat stress conditions. This suggests improved model performance across varying metabolic and environmental heat stress conditions.

Figure 5b shows the RMSE values for mean skin temperature for both models. ThermoSEM using the standard SkBF model yields RMSE values ranging from 1.3°C to 3.1°C. When using the Kraning-based SkBF model, RMSE values are consistently reduced across all protocols, with values ranging from 0.6°C to 2.2°C. On average, the RMSE is reduced by 26% or 0.6°C, with an average decrease of 64% or 1.5°C for P1–P3 and 17% or 0.4°C for P4–P7. Again, the largest RMSE reductions are observed in protocols with elevated metabolic heat productions (i.e. P2 and P3).

Figure 6 summarizes the total increase in rectal temperature (ΔT_{re}) and mean skin temperature (ΔT_{sk}) during the exercise period for experimental measurements (gray) and model predictions (blue for ThermoSEM using the standard SkBF model, orange for ThermoSEM using the Kraning-based SkBF model activated only during the exercise period, and yellow for ThermoSEM using the Kraning-based SkBF model activated throughout the entire simulation period, including the pre-exercise periods). For ΔT_{re} (Figure 6a), ThermoSEM using the standard SkBF model systematically overpredicts the temperature rise across all protocols, and overpredictions are largest when metabolic heat production is elevated (P2 and P3). Both Kraning-based SkBF configurations reduce these overpredictions across all protocols, with the largest reductions realized in P2 and P3, and improve agreement with experimental data.

For ΔT_{sk} (Figure 6b), the standard SkBF model systematically underpredicts the temperature rise across all protocols except P7. While RMSE in P7 remains relatively large, this is primarily attributable to differences in mean skin temperature at exercise onset, as both models predict a similar rise in mean skin temperature during exercise. Underpredictions are significantly reduced in both Kraning-based SkBF configurations, resulting in improved agreement with experimental measurements. For all protocols except P7, the Kraning-based SkBF activated only during the exercise period shows slightly better agreement with the experimental data than the continuous configuration.

Note that the differences between the two Kraning-based SkBF configurations are more pronounced in Figure 6 than in the time-series results (i.e. Figures 2–5). This arises from the different SkBF models used

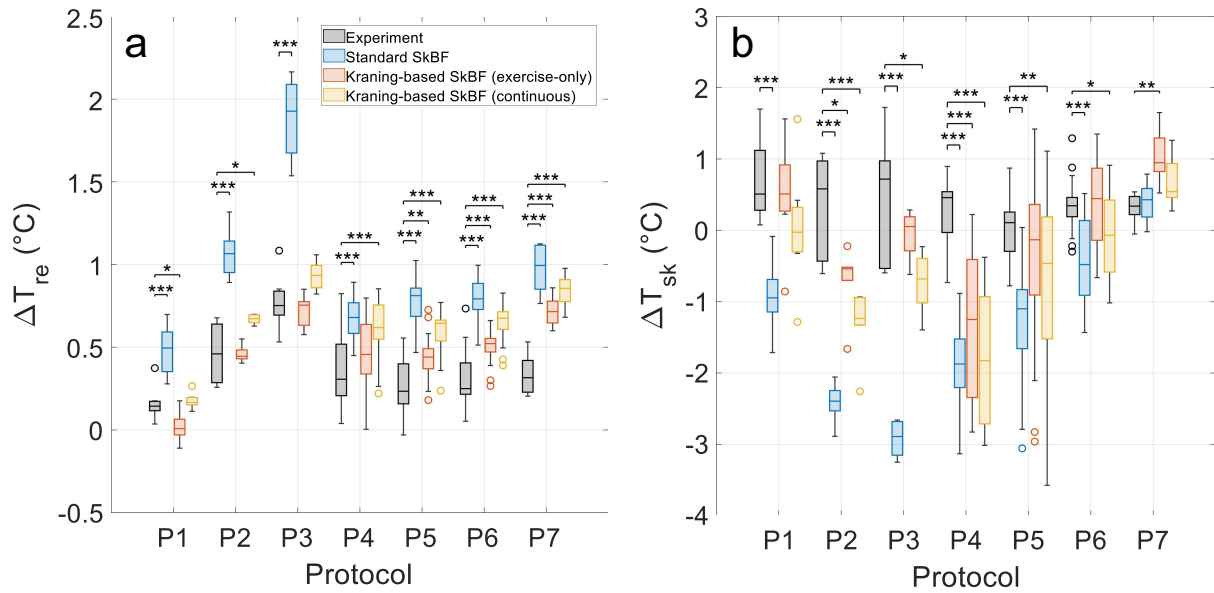


Figure 6. Boxplots of (a) the increase in rectal temperature (ΔT_{re}) and (b) mean skin temperature (ΔT_{sk}) during the exercise period for experimental data and model predictions with significance level between predictions and experimental data (*: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$).

during the pre-exercise period, which lead to different predicted thermal states at the onset of exercise. Although both configurations converge rapidly once the Kraning-based SkBF is applied during exercise, this initial offset is preserved in the ΔT , resulting in the larger observed differences.

These observations are further supported by statistical analysis. For ΔT_{re} , the standard SkBF model shows highly significant differences ($p < 0.001$) from the experimental measurements across all protocols. Fewer and smaller significant deviations are observed when using the Kraning-based SkBF model, indicating improved agreement with experimental observations. A similar pattern is observed for ΔT_{sk} , where the standard SkBF model shows significant deviations from experimental data in most protocols, while the Kraning-based SkBF model reduces both the magnitude and frequency of statistically significant differences. Collectively, these results indicate that the Kraning-based SkBF model provides a more accurate representation of both core and peripheral temperature responses during exercise in the dry heat.

Figure 6 also highlights differences in the representation of inter-individual variability between experimental and predicted temperature rises. For ΔT_{re} , both models generally underestimate the variability observed experimentally, as indicated by the narrower spread of predicted values (i.e. smaller interquartile range and whiskers). In contrast, agreement in ΔT_{sk} variability depends on the protocol. In protocols P1–P3, the predicted inter-individual variability is smaller than that observed experimentally. However, in protocols P4–P7, predicted variability is greater, particularly when using the Kraning-based SkBF model. This increased variability seen in the Kraning-based SkBF model is a consequence of the model formulation rather than improved representation of physiological variability. In the Kraning model, SkBF depends on the participant's VO_2 relative to a threshold of 0.75 L/min, resulting in two distinct regimes with substantially different SkBF. In protocols P4–P7, participant metabolic rates (~ 300 W) correspond to VO_2 values near this threshold, causing some individuals to be assigned substantially higher SkBF than others. This increases variability in predicted mean skin temperature. In contrast, in protocols P1–P3, participant metabolic rates place all individuals clearly within the same regime relative to this threshold. Consequently, the predicted variability is reduced and remains comparable to that of the standard SkBF model for these protocols.

Conclusion

This study evaluated whether integrating the SkBF model of Kraning and Gonzalez, which incorporates exercise-related physiological influences on SkBF, into ThermoSEM improves predictions of human thermal responses during exercise in dry heat. Simulations replicated 83 experimental exercise trials of 90 min under

various metabolic heat production levels (200–500 W) and ambient temperatures (30–45°C). Model predictions were compared with experimental measurements of rectal temperature, mean skin temperature, and evaporative heat loss.

The results indicate that ThermoSEM using the standard SkBF model, which is a temperature-driven model, underestimates SkBF during exercise, particularly at elevated metabolic heat production. This leads to excessive core heat storage, resulting in overpredictions of rectal temperature and underpredictions of mean skin temperature. At the highest metabolic heat productions, the underprediction of skin temperature also reduces skin surface vapor pressure and thereby the model's evaporative capacity below the measured evaporative heat loss. This further increases simulated heat storage and contributes to the overprediction of core temperature. Thermophysiological predictions were significantly improved when ThermoSEM used the Kraning-based SkBF model. Higher SkBF values were predicted, which reduced overpredictions in core temperature and underpredictions in mean skin temperature. The RMSE of the rectal temperature decreased by an average of 40% or 0.2°C, and the RMSE of the mean skin temperature by 26% or 0.6°C. The largest improvements were observed at higher metabolic heat productions, where SkBF plays a critical role in facilitating convective heat transfer from the core to the skin. Both Kraning-based configurations showed nearly identical RMSE values; despite starting from different initial thermal states at exercise onset, their thermal responses converged rapidly during exercise.

In conclusion, incorporating the Kraning-based SkBF model into ThermoSEM is shown to improve thermophysiological predictions during exercise in dry heat conditions. These results highlight the importance of modeling SkBF as a function of both thermal and exercise-related physiological drives and demonstrate the limitations of a purely temperature-driven SkBF formulation during exercise-heat stress. A more accurate representation of SkBF improved predictions of core temperature, skin temperature, and evaporative heat dissipation, thereby enhancing overall prediction of thermal strain. These findings may benefit applications involving occupational heat stress assessment, military operations, and sports performance modeling.

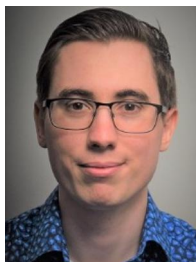
Disclosure statement

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ABBREVIATIONS

A_{sk}	Skin surface area
DL	Dehydration level – percentage body mass loss
f_{scale}	Scaling factor
$P_{v,water}$	Ambient water vapor pressure
RH	Relative humidity
RMSE	Root mean square error
SkBF	Skin blood flow
T_{amb}	Ambient temperature
T_{bl}	Blood pool temperature
TMH	Total metabolic heat production
TMR	Total metabolic rate
T_{re}	Rectal temperature
$T_{set,SkBF}$	Threshold temperature for SkBF
T_{sk}	Mean skin temperature
v_{air}	Air velocity
VO_2	Oxygen consumption
VO_{2max}	Maximal oxygen consumption
w	Body mass
W_{intake}	Mass of water intake
W_{loss}	Mass of water losses
α_{SkBF}	Skin blood flow gain
γ_1	Non-thermal skin blood flow regulation parameter
γ_2	Cold afferent pathway integration parameter
γ_3	Warm afferent pathway integration parameter

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