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Design and Validation of an Individualizable Thermophysiological Model to Assess Thermal, Hydric and Cardiovascular Strain

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Abstract

As heatwaves become more frequent, severe and longer, populations, especially urban population, become more vulnerable in part because of aging. Understanding heat-related symptoms and identifying vulnerability factors are crucial for risk prevention. While thermophysiological simulations enable the study of effects of heat stress at an individual scale, existing models lack the ability to simulate thermal, hydric and cardiovascular strain considering vulnerability factors. Therefore, this paper proposes an adaptation of the JOS-3 multi-segment multi-node model. The adapted model, called aJOS-3, incorporates water balance, heart rate calculation, and the modeling of thermoregulatory limitations linked to dehydration and cardiovascular capacity. The adapted model is validated through comparison with simulation and experimental data from the literature. A sensitivity analysis is also carried out to address concerns regarding the individualization of the adapted model, aJOS-3. This model proves to be relevant for comparing different situations to assess heat strain responsible for heat-related symptoms, although estimating water intake and cardiac index

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remains a challenge for heat strain prediction.

Keywords:

Heat stress, heat strain, physical health, thermophysiological model, water balance, cardiovascular model

Nomenclature		
Symbol		
a	Age	[yr]
AG_{dilat}	Aging factor of vasodilation	[−]
AG_{sweat}	Aging factor of sweating	[−]
AG_{strict}	Aging factor of vasoconstriction	[−]
B	Heat exchange by blood flow	[W]
BF	Blood flow	[L/h]
BFB	Bassal blood flow	[L/h]
BMR	Basal metabolism rate	[W]
BSA	Body surface area	[m ²]
C	Heat exchange by convection	[W]
Cap	Heat capacity	[J/K]
Cdt	Heat exchange by conduction	[W]
CI	Cardiac index	[L/(min m ²)]
$CLDS$	Integrated error signal in the cold receptor	[°C]
CO	Cardiac output	[L/min]
de	Dehydration	[%]
E	Heat exchange by evaporation	[W]

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E_{max}	Maximum evaporative heat flux	[W]
Err	Error signal	[°C]
E_{sw}	Sweating evaporation flux	[W]
h	Height	[m]
HR	Heart rate	[bpm]
L	Latent heat of vaporization	[J/g]
$\dot{m}_{sw}$	Sweating rate (Deng's model)	[kg/h]
O_{ava}	Opening rate of arteriovenous anastomoses	[—]
Q	Heat production	[W]
Q_{all}	Total body heat production	[W]
R	Heat exchange by longwave radiation	[W]
RES	Heat exchange by respiration	[W]
RH	Relative humidity	[%]
R_{sw}	Heat exchange by shortwave radiation	[W]
Sig_{dilat}	Vasodilation signal	[W]
Sig_{stric}	Vasoconstriction signal	[—]
Sig_{sweat}	Sweating signal	[—]

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$SKINC$	Distribution coefficient of vaso- constriction	$[-]$
$SKIND$	Distribution coefficient of va- sodilation	$[-]$
$SKINS$	Distribution coefficient of sweating	$[-]$
SV	Stroke volume	$[L/beat]$
T	Body temperature	$[^{\circ}C]$
T_a	Air temperature	$[^{\circ}C]$
v_a	Air velocity	$[m/s]$
$Vasodilation$	Vasodilation signal (Deng's model)	$[-]$
$\dot{V}_{O_2,max}$	Maximal oxygen uptake	$[L/min]$
$\dot{V}_{O_2}$	Oxygen consumption	$[L/min]$
w	Weight	$[kg]$
$WRMS$	Integrated error signal in the warm receptor	$[^{\circ}C]$
α	Coefficient for effects of dehy- dration on sweating threshold	$[-]$
$\alpha_{\overline{T_{sk}}}$	Stroke volume reduction due to dehydration	$[-]$

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α_{de}	Stroke volume reduction due to high mean skin temperature	[-]
β	Coefficient for effects of dehydration on sweating thermosensitivity	[-]
γ	Coefficient for effects of dehydration on vasodilation threshold	[-]
Subscript		
i	Body tissue	
j	Body segment	
j'	Adjacent body tissue	
lh	Latent heat	
n	Normal value	
sk	Skin	
std	Standard individual	
Abbreviation		
aJOS-3	Adapted JOS-3	
HSCI	Heat Strain Classification Index	
HTR	Human Thermal Risk	

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JOS-3	JOint System thermoregulation model : Original model
TWI	Total Water Intake

1. Introduction

In Europe, heatwaves are more frequent, severe and longer and different record-breaking heatwaves occurred in recent decades [1]. In this context, overheating effects in urban areas are expected to increase also because of the urbanization effects and the increasing urban population [2, 3]. More specifically, cities are particularly vulnerable to heat, because of *i)* the concentrated population and *ii)* human activities, building structure, materials and urban density, which generally exacerbate heat stress and lead to higher temperatures in urban areas than in rural regions, also limiting possible nighttime temperature decrease [4].

The most vulnerable populations to heat stress are elderly people, infants, people with preexisting pathologies, and people with low socioeconomic status or with low educational level [2, 3]. Along with climate change and urbanization problems, global populations will become more vulnerable because of aging [5] which is a major issue in cities [6] and because of increasing in chronic diseases [7]. Worldwide, by the end of the century, heat-related mortality could increase by 2 to 7 times compared to current mortality [2]. Indeed, heat exposure accelerates death and increases the number of hospitalizations for respiratory, cardiovascular [8, 3], renal and mental diseases [2]. Heat exposure have also indirect effects on health: accidents, disease transmission via mosquitoes and ticks, health service overload, infrastructure degradation [2]. On a large scale, this results in an increase of both mortality and morbidity. For instance, in France

during the three heatwaves that occurred in 2022, more than 2,000 deaths were attributed to heat and emergency entrances doubled [9].

At the individual scale, heat is responsible for heat strain and then for a continuum of symptoms such as headaches, palpitations [10], nausea [11], dizziness, feebleness [10], sleep disorders [12, 13], decrease in attentional span [14], decrease in physical capacity [10], dry skin, etc. Moreover, heat causes illness, notably heat exhaustion and heat stroke. Heat exhaustion is a common illness in cases of heat exposure, generally without severe consequences. This illness is mainly caused by dehydration [15]. Heat stroke is characterized by a rise in core temperature and neurologic disorders. It can lead to several vital organ disorders and death [16, 17, 18]. Figure 1 shows the triggering mechanisms for heat-related illnesses, which are linked to three different types of heat strain: thermal strain (heat stroke, sleep disorders, etc.), hydric strain (heat exhaustion, cramps, etc.) and cardiovascular strain (syncope, edema, etc.). Therefore, studying the effects of heat stress on physical health requires the study of these three strains.

Evaluating heat exposure impacts on health, especially in urban context, is crucial to better adapt cities, prepare populations and prevent risk. The effects of heat on health are generally analyzed on the basis of epidemiological studies, at the city or regional scales. Some of these studies have contributed to the development of heat-health warning systems, helping reducing heat-related mortality [3]. At the same time, focusing on the individual scale enables to better understand heat strain and heat-related illness and to identify risky situations and vulnerability factors. In particular, contextualizing risk assessment — such as understanding the effects of individual characteristics, exposure history and displacement — has been identified as a key research perspective [22, 23, 24]. Moreover, the individual scale is necessary to study non-emergency or non-

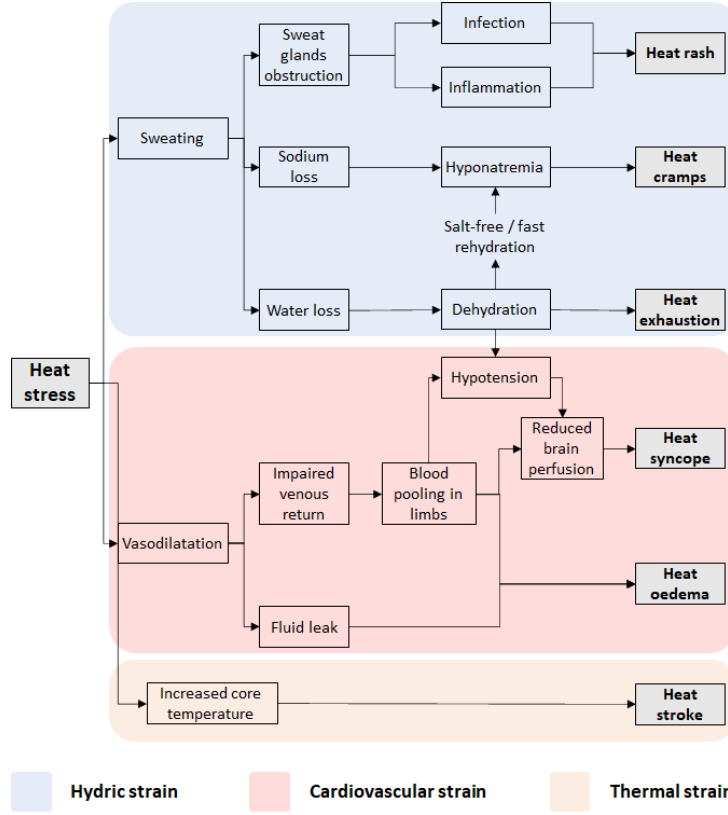


Figure 1: Triggering mechanism for heat-related illnesses [19, 11, 20, 21, 13]

pathological heat-related health issues, such as skin damage, digestive illnesses, irritability, and sleep disorders, which affect well-being and, in the long term, can contribute to more severe health conditions, including skin cancer, chronic stress, impaired focus leading to accidents, depression, or neurological disorders [25]. Thus, a better understanding of the effects of heat on human health at an individual scale, considering various vulnerability factors, is essential for adapting urban planning and building design to users' vulnerabilities and practices, implementing prevention measures, and ultimately making cities more sustainable and healthier.

Among the different existing methods to study heat strain at the individual

scale, the use of thermophysiological modeling appears very attractive as simulations enable the testing of different dynamic situations, comparing them and reproducing real-life conditions. Moreover, simulations are valuable to study risky situations for vulnerable people without putting in danger real people, and without the limitations and invasiveness associated with direct measurements. Therefore, this paper aims to develop a model to simulate thermal, hydric and cardiovascular strain responsible for heat illness, as highlighted in Figure 1, for various types of individuals.

Basically, thermophysiological models aim to simulate heat transfer within the human body and thermoregulatory responses. They calculate various body temperatures, including core temperatures and skin temperatures, variables related to thermoregulation (evaporation flow rate, skin wettedness, skin blood flow, etc.) and different heat fluxes. They are generally divided into two systems: the passive system, which deals with the heat exchanges in the body and with the environment, and the active system, which deals with the thermoregulation processes.

Figure 2 illustrates the historical development of thermophysiological models and synthesizes the key features of some of the most commonly used models. From 1970's, thermophysiological models have evolved from single-segment models to two-node models, then to more sophisticated multi-segment and multi-node models. More recently, 3D models constructed from MRI (Magnetic Resonance Imaging) data and multi-element models with fine divisions into segments, layers, and angular sectors have been developed. This evolution enables a more detailed representation of human body geometry and allows for the simulation of non-uniform environments. The development of thermophysiological models has expanded their applications, enabling simulations of different activity levels and unsteady environments. Improvements to active systems have en-

Model	Year	Body division			Outputs				Assumed Conditions
		Segments	Nodes per segment	Angular sectors	Active system	Inputs	Individualization		
Fanger	1970	✱	✱	✱	✱	✱ + Mechanical work	✱	✱	✱
Gagge	1971	1	2	✱	✱	✱ + work efficiency + HTC	✱	✱	✱
Stolwijk	1971	6	4	✱	✱	✱	✱	✱	✱
MEMI	1983	1	2	✱	✱	✱ + work efficiency + posture	✱	✱	✱
Kranling & Gonzalez	1997	1	6	✱	✱	✱ + type of work	✱	✱	✱
Fiala	1999	15	7 to 19	3	✱	✱	✱	✱	✱
65MN	2001	16	4	✱	✱	✱	✱	✱	✱
Berkeley comfort	2001	Unlimited	4 + clothes	✱	✱	✱ + skin color	✱	✱	✱
Hirata	2019	13	64 regions divided into 2x2x2 mm ³ voxels		✱	✱	✱	✱	✱
NHTM	2020	21	21 elements	12	✱	✱ + turbulence intensity	✱	✱	✱
Itani	2020	25	2	✱	✱	✱ + basal metabolism	✱	✱	✱
JOS-3	2021	17	2 or 4	✱	✱	✱ + Posture	✱	✱	✱

Active system
 Shivering
 NST Non Shivering thermogenesis
 Sweating
 Vasodilation / vasoconstriction
 AVA Arteriovenous Anastomoses

Inputs
 Air temperature
 Radiant temperature
 Clothing insulation
 Activity level
 Heat transfer coefficients
 Humidity
 Air velocity

Individualization
 Sex
 Height
 Weight
 Cardiac capacity
 Age
 Fat percentage
 Origin

Outputs
 $\dot{m}_{sweat}$ Sweating flow
 ΔW Weight loss
 $\dot{V}_b$ Blood Temperature
 $\dot{V}_b$ Blood flow
 Cardiac Output

Assumed conditions
 Uniform environment
 Non-uniform environment
 Steady
 Unsteady

Figure 2: Overview of widely used thermophysiological models

hanced model accuracy, extended thermoregulation mechanisms [26], and even replicated neurological control [27]. Moreover, the individualization of models is increasing, with a growing number of input parameters accounting for the specific characteristics of the modeled individual. These parameters modify the construction of passive systems and/or the regulation of active systems. In some models, parameters are optimized for specific populations through comparison with empirical data, as seen in COMFA-senior [28]. Adapting a model to elderly individuals generally consists of adjusting fat percentage, cardiac output, skin blood flow, body surface area, and basal metabolism [29, 30, 31]. Going further, Ji et al. [32] adapted a two-node model for older adults, addressing age-related thermoregulatory changes through attenuation coefficients, threshold temperature adjustments, and parameter optimization using experimental data.

Despite these evolutions, significant gaps remain in thermophysiological models for generally studying the heat effects on health, notably the inability to simulate the three types of strain and to relevantly address prolonged heat exposures, and the impossibility to validate thermophysiological models under a wide range of real-life conditions.

First, to the best of our knowledge, no existing model is capable of calculating all the relevant outputs required to study thermal, hydric, and cardiovascular strain. The suitable outputs are:

- the core temperature for thermal strain [33, 34, 35, 36],
- the dehydration rate (the ratio between weight loss and initial body weight) for hydric strain [37, 38],
- the heart rate [39, 17, 33] and the core-to-skin temperature gradient [40, 39] for cardiovascular strain.

Although all thermophysiological models simulate the core and skin temperatures, with varying levels of accuracy, the dehydration rate and the heart rate are more rarely calculated. To the best of our knowledge, only the one-segment, five-node model of Kraning and Gonzalez calculates both outputs [41]. However, this model is not suitable for studying heat strain and the effects of physiological and exposure-related vulnerability factors, as it simulates a standard healthy man without any possibility of individualization. It has only one segment, whereas cardiac output simulations are more accurate when variations in blood flow across the body are considered, especially when modeling elderly individuals [42]. Moreover, hydric and cardiovascular strain can limit thermoregulatory capacity, and thus increase thermal strain during severe or prolonged exposure. However, the effects of cardiovascular limitations and dehydration are as well rarely taken into account in current thermophysiological models.

Second, current thermophysiological models are unsuitable for multiday studies, even though thermal, hydric and cardiovascular strain must be studied over several days due to the duration of heatwaves, the cumulative effects of heat stress, and the possibility of recovery and of delayed effects of heat-related illnesses [43, 44]. Moreover, the thermoregulation responses differ depending on the exposure history. Two contrary behaviors could occur. On the one hand, prolonged heat stress (during several hours) can induce thermoregulation fatigue. For instance, a high rate of sweating for several hours causes fatigue of the sweat glands and therefore a reduction in the ability to sweat [45]. On the other hand, prolonged exposure (during several days) involves acclimatization which notably modify thermoregulation by increasing sweating, improving skin blood flow regulation and reducing body temperature [46, 47]. 75 % to 80 % of adaptation occurs between days 4 and 7 of exposure [46]. It is therefore essential

to account for acclimatization when studying the health impacts of heat exposure lasting more than four days. However, to the best of our knowledge, no existing thermophysiological model considers both acclimatization, thermoregulation fatigue, recovery and cumulative heat stress.

Finally, regarding the available experimental data, which are necessarily complementary to numerical developments, heat stress experiments are often performed for two to twelve hours of exposure in climatic chambers while in real exposure, exposure history and some non-thermal parameters can affect thermoregulation and sensitivity to heat strain such as light, mental state and time of day [48]. However, obtaining physiological measurements in real-life conditions also presents challenges [49]. For instance, measuring the core temperature typically necessitates the use of rectal or esophageal sensors or ingestible capsules, which can be uncomfortable, invasive, and only usable for a limited period [16]. Assessing the water loss can involve weighting the body before and after short-term exposure [50, 40], or measuring the local sweat rates using ventilated capsules [34]. These experimental methods are impractical outside controlled laboratory environments, limiting their use in daily life conditions or over extended periods. Furthermore, assessing physical activity levels, and by extension, metabolic rate, in real-life conditions can be particularly challenging, especially for vulnerable populations [51]. This lack of experimental data for validation also contributes to the limited applicability of thermophysiological models [52, 8]

Hence, to address these literature gaps and more specifically those related to the simulation of the three types of strain and the associated validation, the present paper suggests an adaptation and validation of a multi-segment multi-node model with individualization inputs to simulate thermal, hydric and cardiovascular strain and more precisely the dehydration and the heart rate. The

adapted model, called aJOS-3, is based on the original JOS-3 model (*JOint System thermoregulation model*) developed by Takahashi et al. [26]. JOS-3 has been chosen because of its applicability to non-uniform and unsteady environments, its open source code and its short computation time step. The original JOS-3 model can be individualized with the following parameters: age, sex, height, weight, fat percentage and cardiac index. The basal metabolism rate, tissue conductance, basal cardiac output and thermosensitivity of thermoregulation mechanisms are then adjusted. JOS-3 has been validated for young men and older people and it can be used to assess local thermal sensation [52].

This article describes the adaptation of the original JOS-3 model and discusses perspectives to improve the modeling process to better understand heat-related symptoms and to take into account individual vulnerability factors. Section 2 presents the original JOS-3 model and its adapted version, aJOS-3, which incorporates dehydration and heart rate modeling. In Section 3, aJOS-3 is validated by comparing its results with both simulated and experimental data from the literature. Then, Section 4 examines the strengths, limitations, and precautions to consider when using aJOS-3 to assess heat risk or heat effects on health. Finally, the conclusion (Section 5) synthesizes the main results and perspectives of this work.

2. From JOS-3 to aJOS-3

2.1. The original JOS-3 model

JOS-3 (*JOint System thermoregulation model*) [26] is the latest version in the family of thermophysiological models based on Stolwijk's model. JOS-3 [26] has been developed to improve the prediction of thermophysiological responses, considering personal characteristics, in transient and non-uniform environments.

Figure 3 represents the fundamentals of JOS-3 with the body division and

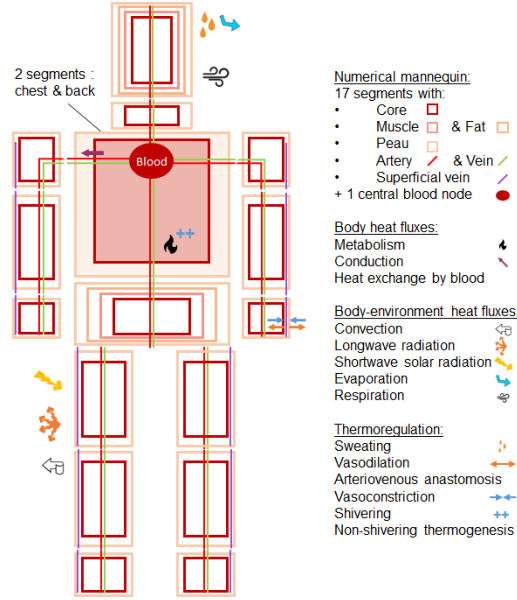


Figure 3: Body division, heat fluxes modeled and thermoregulation mechanisms included in JOS-3

the modeled phenomena. For the passive system, the body is divided into 17 cylindrical or spherical segments. Each segment includes one node for the core, one for the skin, one for the artery and one for the vein. In limb segments, there is an additional node for the superficial vein. To improve the accuracy of the core temperature calculations, intermediate nodes take place in head and pelvis. There is also a node for the central blood pool. A heat balance equation is expressed for each node as:

$$\text{Cap}_{j[i]} \frac{dT_{j[i]}}{dt} = Q_{j[i]} + B_{j[i]} + Cdt_{j-j'[i]} + Cdt_{b-j[i]} - (C_{[i]} + R_{[i]})_{sk} - E_{sk[i]} + R_{sw,sk[i]} - \text{RES}_{\text{Chest}} \quad (1)$$

where, Q is heat production [W], B is heat exchange by blood flow [W], $Cdt_{j-j'}$ heat exchange by conduction with adjacent tissue [W] and Cdt_{b-j} heat

exchange by conduction with blood vessels [W] especially in the limbs. C , R , R_{sw} and E are heat exchange [W] at skin (subscript sk) surface by convection, longwave radiation, shortwave radiation and evaporation, respectively. RES is respiration heat exchange, which is included only in the chest core node [W]. Cap and T , are the heat capacity [J/K] and temperature of the node [$^{\circ}\text{C}$], respectively. Subscript i represents the segment number, j represents the tissue node and j' represents the adjacent node.

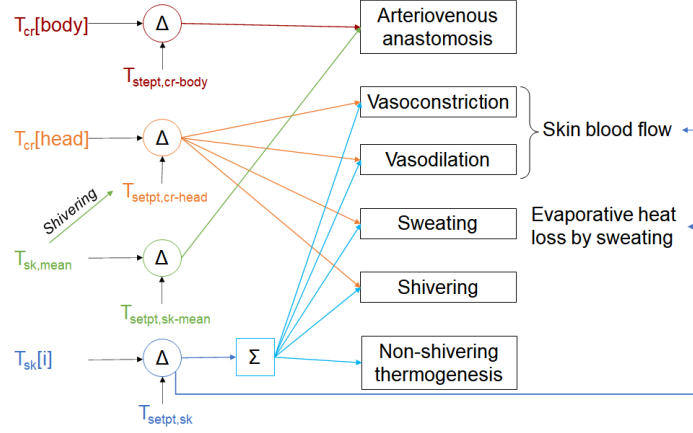


Figure 4: Signal errors involved in thermoregulation control in JOS-3

The active system, represented in Fig. 4, models the four main thermoregulation mechanisms plus the arteriovenous anastomoses and non-shivering thermogenesis. The arteriovenous anastomosis is a modification of the opening level of connection between artery and superficial vein in feet and hands. Non-shivering thermogenesis is an additional heat production in adipose tissues and organs. The modeling of these two mechanisms is expected to improve local skin temperature and blood distribution simulations in moderately cold or hot environments. All the thermoregulatory responses are governed by error signals (Fig. 4). Threshold temperatures are defined as equilibrium body temperatures without thermoregulation under neutral ambient conditions which are simulated

by $PMV = 0$. Since adaptations involve hydric and cardiovascular thermoregulation, the following paragraphs focus only on sweating and skin blood flow regulation.

The evaporation flux E [W] is calculated as the sum of perspiration, equal to 6% of the maximal evaporation E_{max} [W] possible in the environment, and of sweating evaporation flux $E_{sw[i]}$ [W], calculated as:

$$E_{sw[i]} = SKINS_{[i]} \cdot AG_{sweat} \cdot \frac{BSA}{BSA_{std}} \cdot Sig_{sweat} \cdot \frac{Err_{sk[i]}}{10} \quad (2)$$

where AG_{sweat} is the aging factor of sweating quantifying the decrease in sweat rate with age, $SKINS$ is the distribution coefficient of sweating according to the different body segments, $\frac{BSA}{BSA_{std}}$ is the ratio of body surface area to that of the standard man, Err_{sk} is the error signal on local skin temperature [$^{\circ}\text{C}$] and Sig_{sweat} is the sweating signal [W]:

$$Sig_{sweat} = 371,2 \cdot Err_{cr[0]} + 33,64 \cdot (WRMS - CLDS) \quad (3)$$

where $Err_{cr[0]}$ is the error signal on the head core temperature [$^{\circ}\text{C}$] and $WRMS$ and $CLDS$ are the integrated error signals in warm and cold skin thermoreceptors [$^{\circ}\text{C}$], respectively.

Furthermore, the skin blood flow is calculated according to the vasodilation and vasoconstriction signals as follows:

$$BF_{sk[i]} = \frac{1 + SKIND_{[i]} \cdot AG_{dilat} \cdot Sig_{dilat}}{1 + SKINC_{[i]} \cdot AG_{stric} \cdot Sig_{stric}} \cdot BFB_{sk[i]} \cdot 2^{\frac{Err_{sk[i]}}{6}} \quad (4)$$

where $SKIND$ and $SKINC$ are the distribution coefficients of vasodilation and vasoconstriction to the different body segments respectively, AG_{dilat} and AG_{stric} the aging factors for vasodilation and vasoconstriction, BFB_{sk}

the basal skin blood flow [L/h] and Sig_{dilat} and Sig_{stric} the vasodilation and vasoconstriction signal.

These thermoregulation terms modify heat balances which are solved for each of the 85 nodes via a quasi-implicit method. JOS-3 calculates the different node temperatures, skin blood flow, water loss by evaporation and different exchanges between the body and environment.

The present work is based on a modified version 3 0.3.2 of JOS-3 model available on <https://github.com/TanabeLab/JOS-3>. The version 3.0.3.2 is not the one validated by Takahashi et al. [26]. The modifications are as follows:

- Modification of the arteriovenous anastomosis equations (Eq. (5), Eq. (6) and Eq. (7) - modification from version 3 0.3.2 are bold) to ensure that initial conditions are thermoneutral and to match with Takemori et al. [53]

$$O_{ava,hand} = 0.265 \cdot \left[\overline{T_{sk}} - \left(\overline{T_{sk}^{setpt}} - \mathbf{0.48} \right) \right] + 0.953[T_{cr,tronc}(\overline{T_{cr,tronc}^{setpt}} + \mathbf{0.1045})] + 0.9126 \quad (5)$$

$$O_{ava,foot} = 0.265 \cdot \left[\overline{T_{sk}} - \left(\overline{T_{sk}^{setpt}} + 0.997 \right) \right] + 0.953[T_{cr,tronc}(\overline{T_{cr,tronc}^{setpt}} + \mathbf{0.2855})] + 0.9126 \quad (6)$$

$$O_{ava}(t = 0) = 0.9126 \quad (7)$$

- Modification of basal metabolism rate (BMR [W]) equation according to

Roza and Shizgal [54] (eq. 8) based on the height h , the weight w and the age a of the individual

$$BMR = \begin{cases} 88.362 + 13.397 \cdot w + \mathbf{479.99} \cdot h - 5.677 \cdot a & (male) \\ 47.593 + 9.247 \cdot w + \mathbf{309.8} \cdot h - 4.330 \cdot a & (female) \end{cases} \quad (8)$$

- Addition of condition as E_{max} positive

Calculations of dehydration rates and heart rates have been added as explained in the next sections.

2.2. Dehydration modeling

The acceptable limits for hydric strain are mainly expressed as the dehydration rate. The original JOS-3 model calculates the water loss through evaporation by summing evaporated sweat, perspiration, and respiratory latent heat exchange (RES_{lh}) [W].

However, the calculation of the dehydration rate requires implementing a more comprehensive water balance. Therefore, the adapted model, aJOS-3, considers the total amount of sweat produced represented by sweating signal Eq. (??), instead of evaporated sweating, as well as the Total Water Intake (TWI) and the non-thermal water loss, from urine and feces. The TWI encompasses water from drinking and eating [55]. In this work, the TWI and non-thermal water loss are combined into a single term referred to as "water intake" [g/s]. The dehydration rate [%] is then calculated as:

$$de = \frac{\int_0^t \left(\frac{E_{sw} + 0.06 \cdot E_{max} + RES_{lh}}{L} - water\ intake \right) \cdot dt}{w \cdot 1000} \cdot 100 \quad (9)$$

where L is the latent heat of vaporization [J/g] and w is the initial body weight [kg].

Moreover, dehydration affects thermoregulation. Kraning and Gonzalez [41] reported that heat exposure and exercise intensity have no influence on the core temperature or the stroke volume if an individual is properly rehydrated. Without regular rehydration, the stroke volume decreases during exposure, leading to an increase in heart rate and sweating threshold temperature, and thus a greater rise in core temperature. Therefore, in addition to the dehydration rate calculation, aJOS-3 models effects of dehydration on thermoregulation based on the work of Deng et al. [35]. Deng et al.'s model is designed for health applications, specifically to study heat stroke and is based on a model of the original JOS-3 family: 25 MN multi-segment, multi-node model [56]. Deng et al.'s model includes a decrease in the thermosensitivity of sweating and an increase in the threshold for activation of sweating (eq. 10) and vasodilation (eq. 11).

$$\begin{aligned} \dot{m}_{sw} = & (0,2898 - \beta \cdot de) \cdot (T_{cr} - (T_{cr}^{set} + \alpha \cdot de)) \\ & + 0,0336 \cdot (Warms - Colds) \end{aligned} \quad (10)$$

$$Vasodilation = 117 \cdot (T_{cr} - (T_{cr}^{set} + \gamma \cdot de)) + 7,5 \cdot (Warms - Colds) \quad (11)$$

Where, according to Deng et al., $\dot{m}_{sw}$ is the sweating rate [kg/h], $Vasodilation$ is the signal for vasodilation, T_{cr}^{set} is the threshold core temperature [°C] and $Warms$ and $Colds$ the error signals from skin thermoreceptors. The dehydration effect coefficients were set by Deng et al. as $\alpha = 0.06$ °C, $\beta = 0.068$ kg/(h °C) and $\gamma = 0.06$ °C.

The coefficients α , β and γ are integrated in the aJOS-3 model by identifying

the relationships between equations 3 and 10 and between equations 4 and 11. Both equations have similar structures. Therefore, α and γ , which represent the increase in the threshold core temperature, were kept equal to 0.06 °C. The thermosensitivity decrease coefficient β cannot have the same value in aJOS-3 as in Deng's model due to differences in units and thermosensitivity values. After comparing various solutions, the conservation of proportionality between β and thermosensitivity was selected: it provides the closest results to the simulations conducted by Deng et al. Finally, the sweating and vasodilation signal equations have been adapted in aJOS-3 as follows:

$$\begin{aligned} Sig_{sweat} = & (371,2 - \beta \cdot de) \cdot (Err_{cr[0]} - \alpha \cdot de) \\ & + 33,64 (WRMS - CLDS) \end{aligned} \quad (12)$$

$$Sig_{diat} = 100,5 \cdot (Err_{cr[0]} - \gamma \cdot de) + 6,4 (WRMS - CLDS) \quad (13)$$

with:

$$\alpha = \alpha_{Deng} = 0.06 \text{ °C}$$

$$\gamma = \gamma_{Deng} = 0.06 \text{ °C}$$

$$\begin{aligned} \beta &= \beta_{Deng} \cdot \frac{\text{thermosensitivity of JOS-3}}{\text{thermosensitivity of Deng}} \\ &= 0.068 \cdot \frac{371.2}{0.2898} = 87.1 \text{ W/°C} \end{aligned}$$

The implementation of Deng et al. model in aJOS-3 is validated in section

3.2. The water balance validation is addressed in section 3.3.

2.3. Heart rate modeling

Heart rate HR [bpm] is commonly used to monitor cardiovascular strain, with thresholds such as 95 % of the age-predicted maximum heart rate (calculated as 220 bpm minus age) sustained over several minutes [57], or a maximal increase of 30 bpm due to heat stress [33]. To avoid discomfort, the limit for the heart rate is between 94 and 100 bpm [10]. The heart rate can be determined by the ratio of the cardiac output CO [L/min] to the stroke volume SV [L/beat]. The original JOS-3 model calculates cardiac output as the sum of various blood flows.

Different models for simulating the heart rate have been described in the literature. Yermakova et al. [58] used metabolism and blood temperature to evaluate the heart rate during exercise in hot environments. Zakyntinaki et al. [59] proposed an empirical model of cardiac kinetics during exercise based on the heart rate at rest, cardiovascular condition and exercise intensity. Looney et al. [60] proposed an empirical model to link heart rate and core temperature during passive exposure. Itani et al. [42] compared the heart rates of young and older adults from literature results and proposed a cardiac model based on metabolism and core temperature. Among the models found in the literature, the Kraning and Gonzalez’s cardiac model [41] has been chosen because of its better performance in comparison with the literature results. Moreover, this is a rational model which simulates the cardiovascular limitations. Indeed, when cardiac effort is too important, which means that when the heart rate should be higher than its maximum value, vasodilation is reduced.

To integrate this cardiovascular limit to aJOS-3, a condition is added to cardiac output. If the sum of all blood flows is superior than the maximal cardiac output, all skin blood flows and arteriovenous anastomose blood flows

are considered equal to their previous values. The maximal cardiac output CO^{max} is based on the maximal heart rate HR^{max} as follows:

$$CO^{max} = HR^{max} \cdot SV = (220 - a) \cdot SV \quad (14)$$

Where a is the age and SV the stroke volume that means the volume of blood ejected per cardiac contraction. Its calculation has been added to aJOS-3. According to Kraning and Gonzalez, this volume is bounded by values dependent on the maximal oxygen uptake $\dot{V}_{O_2, max}$ [L/min] [61] and varies according to physical effort modeled by oxygen consumption $\dot{V}_{O_2}$ [L/min] (Eq. (15) and Eq. (16)). The stroke volume decreases when the mean skin temperature is high and because of dehydration, which causes a reduction in blood plasma volume (Eq. (17), Eq. (18) and Eq. (19)).

$$SV_n = \begin{cases} SV_n^{\min} & \text{If } \dot{V}_{O_2} < 0.5 \text{ L/min} \\ SV_n^{\max} & \text{If } \dot{V}_{O_2} \geq 2 \text{ L/min} \\ \left(\frac{SV_n^{\max} - SV_n^{\min}}{1.5} \right) \cdot (\dot{V}_{O_2} - 0.5) + SV_n^{\min} & \text{If } 0.5 \leq \dot{V}_{O_2} < 2 \text{ L/min} \end{cases} \quad (15)$$

$$\dot{V}_{O_2} = \frac{Q_{all}}{BSA} \cdot \frac{3.5}{58.15} \cdot \frac{w}{1000} \quad (16)$$

$$SV = \frac{SV_n - \alpha_{\overline{T_{sk}}} - \alpha_{de}}{1000} \quad (17)$$

$$\alpha_{\overline{T_{sk}}} = \begin{cases} 0 & \text{If } \overline{T_{sk}} < 33^\circ\text{C} \\ \frac{25 \cdot (SV_n - SV_n^{\min})}{SV_n^{\max} - SV_n^{\min}} & \text{If } \overline{T_{sk}} > 38^\circ\text{C} \\ \frac{5 \cdot (\overline{T_{sk}} - 33) \cdot (SV_n - SV_n^{\min})}{SV_n^{\max} - SV_n^{\min}} & \text{If } 33 \leq \overline{T_{sk}} \leq 38^\circ\text{C} \end{cases} \quad (18)$$

$$\alpha_{de} = 1,03 \cdot 4,37 \cdot de \quad (19)$$

where SV_n is the normal stroke volume [L/beat] ranging between SV_n^{min} and SV_n^{max} , Q_{all} is the total body heat production [W] (1 met = 58.15 W/m² = 3.5 mL O₂/(kg min) [62]). $\alpha_{\overline{T_{sk}}}$ and α_{de} are coefficients that model the reduction in the stroke volume respectively due to a high mean skin temperature and to dehydration.

Thus, Eq. (14) to Eq. (19) have been integrated into aJOS-3 to calculate the heart rate as the ratio between the cardiac output and the stroke volume at each time step. This implementation requires an additional individual parameter, the maximal oxygen uptake, as well as several model parameters for the calculation of normal stroke volume.

3. Validation

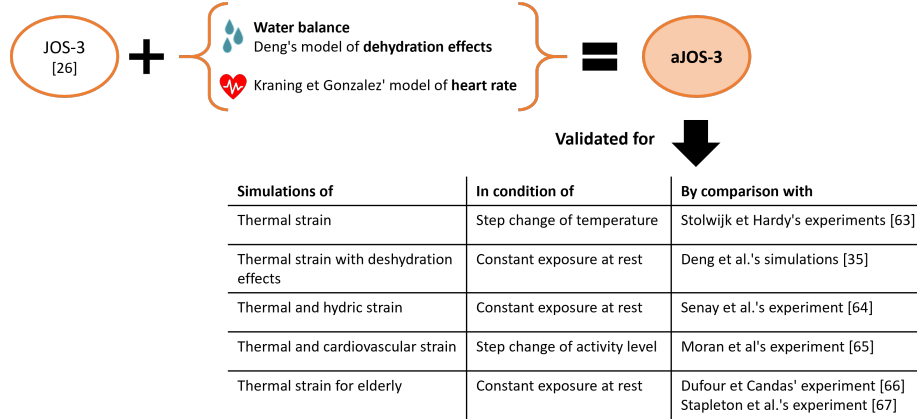


Figure 5: aJOS-3: adaptation of JOS-3 and validation process

Figure 5 summarizes the adaptations made to JOS-3 in developing aJOS-3 and presents its validation process. Indeed, human thermophysiological responses have been simulated with aJOS-3 based on five experimental setups

and one simulation process from the literature. For the comparisons with the corresponding reference data, raw data were extracted by digitizing graphs from publications. These validation studies are presented below.

3.1. Thermal strain: base case

To validate that the modifications and adaptations preserve the initial performance of the original JOS-3 model, the same validation process as that used Takahashi et al. is done. The comparison with the Stolwijk and Hardy's experiments [63] is therefore quickly discussed in this article.

During these experiments, three young men were exposed in climatic chambers to air temperature step changes. They sat two hours exposed to $T_a = 28^\circ\text{C}$ and $RH = 40\%$, stayed two hours exposed to hot conditions and returned in the first climatic chamber. The hot condition for the first experiment, called here "moderate heat stress" experiment, was $T_a = 37.5^\circ\text{C}$ and $RH = 33\%$. For the second experiment, called "extreme heat stress" experiment, it was $T_a = 47.8^\circ\text{C}$ and $RH = 40\%$. The air motion near participants was too slow to be measured. The participants remained seated throughout the experiments and wore only shorts.

Simulations are done with aJOS-3 taking into account the personal characteristics of each participant and the temperature and humidity described before. The air velocity is assumed to be equal to 0.1 m/s , the activity to 1 met and the clothing insulation 0.1 clo . In comparison with the simulation done by Takahashi et al. with the original JOS-3 model, the modifications and adaptations to JOS-3 done in this paper resulted in a deterioration of rectal temperature predictions for the extreme heat stress experiment: the RMSE increased from 0.12°C to 0.36°C . On the contrary, for the moderate heat stress experiment, the rectal temperature prediction improved: the RMSE decreased from 0.32°C to 0.2°C . These differences are mainly due to the modifications of core heat ca-

capacity (correction done between the version of Takahashi article and the version 3.0.3.2) and of arteriovenous anastomosis initialization.

3.2. Thermal strain and hydric strain: effects of dehydration on core temperature

The implementation of dehydration effects in the adapted model, aJOS-3, has been validated by comparison with the simulations of Deng et al. A standard man exposed to constant thermal conditions is simulated to study the modeled effect of dehydration on the head core temperature. The simulated individual is at rest with an air velocity v_a equal to 0.1 m/s, and different air temperatures and relative humidities.

For the aJOS-3 simulations, clothing insulation is assumed to be equal to 0 clo, activity level to 1.04 met and the characteristics of the standard man are matched to Deng et al.'s parameters: 74.1 kg and 1.75 m.

Figure 6 presents the head core temperature simulated with aJOS-3 compared to Deng et al.'s simulation results. The aJOS-3 results are close to those of Deng et al. The maximal RMSEs are around 0.28 °C for moderate (38 °C) and high air temperature (41 °C) and moderately high relative humidity (75 %). RMSEs are lower for lower air temperature. For moderate heat stress, the deviations are visible from the first hours which appear thus linked not to the dehydration modeling but rather to a lower sweating efficiency in aJOS-3. Moreover, compared to Deng's model, aJOS-3 anticipates the effects of dehydration at high temperatures or high relative humidity. At lower heat stress, the effects of dehydration seem less significant in the aJOS-3 model. The causes of these differences are difficult to determine due to potential differences in clothing, activity level and initialization and because the dehydration rate could not be compared with Deng et al.'s simulations.

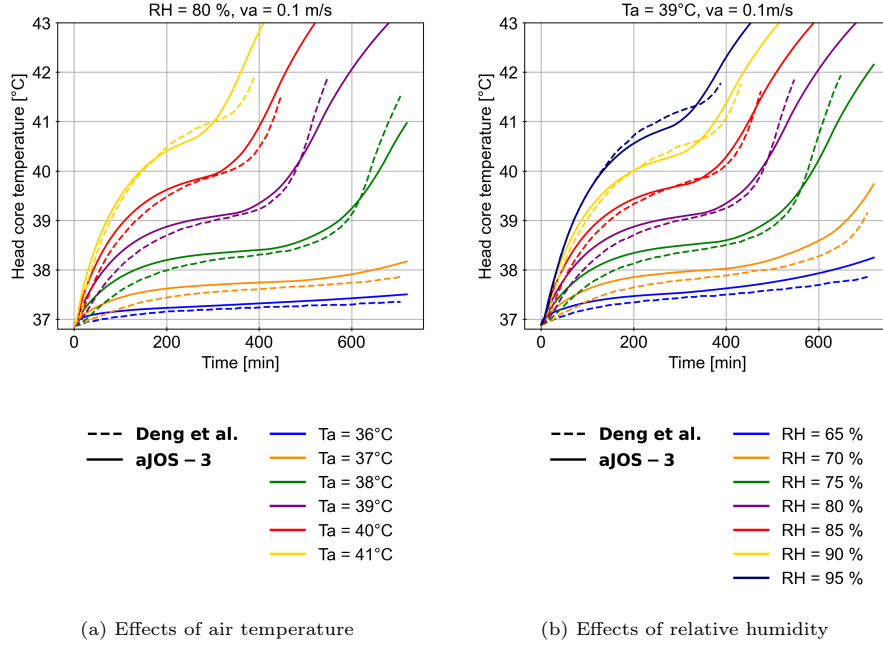


Figure 6: Comparison between Deng et al.'s [35] and aJOS-3 simulations: effects of dehydration on the head core temperature during constant heat exposure at different air temperatures or relative humidities

3.3. Thermal strain and hydic strain: water balance

To validate the consistency of the water balance in aJOS-3, simulations were carried out to reproduce the experiment of Senay et al. [64]. The goal of this experiment was to study the effects of acclimatization on water loss under heat stress. aJOS-3 does not model acclimatization, so the preacclimatization experiment was simulated.

Senay et al. exposed five South African male workers to environmental conditions of $T_a = 45^\circ\text{C}$ and $RH = 35\%$ with an air velocity of 1 m/s for 12 hours. Table 1 presents the characteristics of the participants. During the experiment, participants were seated and ingested 0.2 L of water every hour. Rectal temperature, body weight, and various blood parameters related to dehydration were measured hourly. Measurements and water ingestions were staggered ev-

ery 30 min.

Table 1: Characteristics of the participants used to simulate the experiments of Senay et al. [64]

Participant	Age [yr]	Weight [kg]	Height [m]	Estimated body water [kg]
A	39	60.8	1.666	36.48
R	24	55.15	1.59	33.09
J	21	65.55	1.666	39.33
Au	25	62.1	1.678	37.26
B	21	70.6	1.692	42.36
Mean	26.00	62.84	1.6584	37.70

The same thermal environment is implemented as input of aJOS-3 simulations, assuming an instantaneous water intake of 200 g every hour without any non-thermal water loss. The activity level is set to 1 met. Since Senay et al.’s article did not provide information on clothing, the clothing insulation is assumed to be 0.3 clo for standard light clothing. The effects of this assumption have been studied with a sensibility study, not presented in this paper, which concludes that an uncertainty of 0.4 clo leads to an uncertainty of 0.2°C on the head core temperature and of 0.05 % on dehydration. The effect of clothing insulation uncertainty on water balance is therefore negligible. Simulations were conducted for each participant using the personal parameters presented on table 1. Then, results are averaged for comparison with the mean experimental data (Fig. 7).

The rectal temperature, represented by the simulated pelvis core temperature, and the water loss are both within the range of the mean plus or minus the standard deviation among the different individuals. However, the rectal temperature is slightly underestimated by aJOS-3. The RMSE is equal to 0.2°C which is acceptable according to the measurement uncertainty and the hypothesis. Moreover, we ignore conditions before exposure and some individual parameters such as the cardiac index, fat percentage and maximal oxygen uptake. The

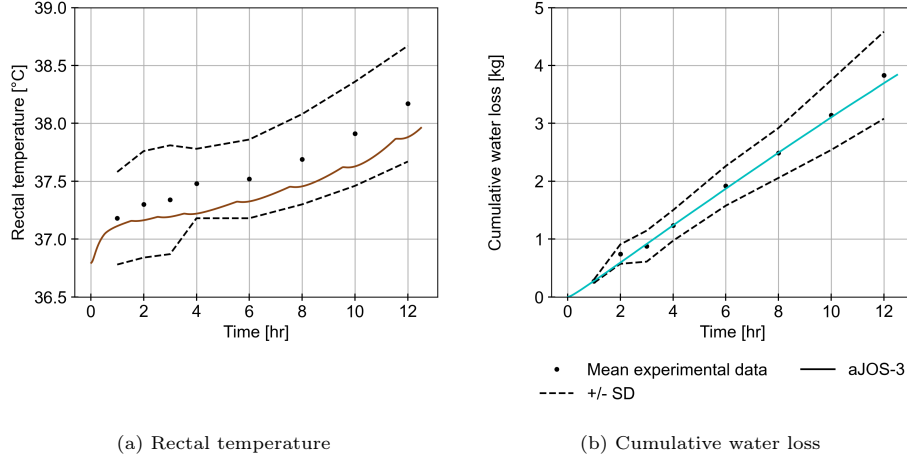


Figure 7: Comparison of the data from Senay et al. [64] and the predicted data with aJOS-3 model

rectal temperature difference is visible from the first measurement point, so the underestimation is probably in part due to the model initialization.

The water loss is well predicted: the RMSE is approximately 65 g which is not significant considering the uncertainty of body weight measurement (20 g) and calculating the dehydration rate (RMSE approximately equal 0.1 %). Therefore, according to this comparison, the water balance in aJOS-3 is validated in the case of severe steady heat stress [64].

3.4. Thermal strain and cardiovascular strain

To evaluate the performance of the heart rate model under step change activity levels, simulations were carried out to reproduce the experiment of Moran et al. [65]. The goal of this experiment was to develop a heat strain index, the Physiological Stress Index (PSI). This index is based on rectal temperature and heart rate.

During this experiment, one hundred young men exercised on a treadmill for two hours in a climatic chamber. The participants were exposed to $T_a = 40^\circ\text{C}$ and $RH = 40\%$. The participants wore shorts and sports shoes. After 10 min of

rest, the participants walked at a constant speed of 1.34 m/s for 120 min. Heart rate and rectal temperature were measured every minute.

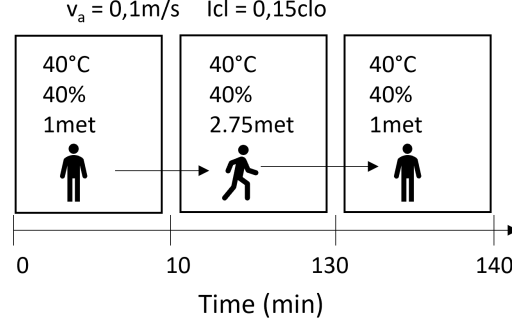


Figure 8: Input conditions to simulate the experiment of Moran et al. [65]

The same thermal configuration is implemented as input of aJOS-3 simulations. Figure 8 summarizes the input parameters. Consistent with the experimental conditions, the clothing insulation is set to 0.15 clo, and the activity level is set to 2.75 met for the walking session. As Moran et al.'s article did not provide information on ventilation, the air velocity is assumed to be 0.1 m/s to represent an unventilated chamber. The following mean characteristics of the experimental participants were used for the simulation: 20 years old, 1.78 m and 74.6 kg.

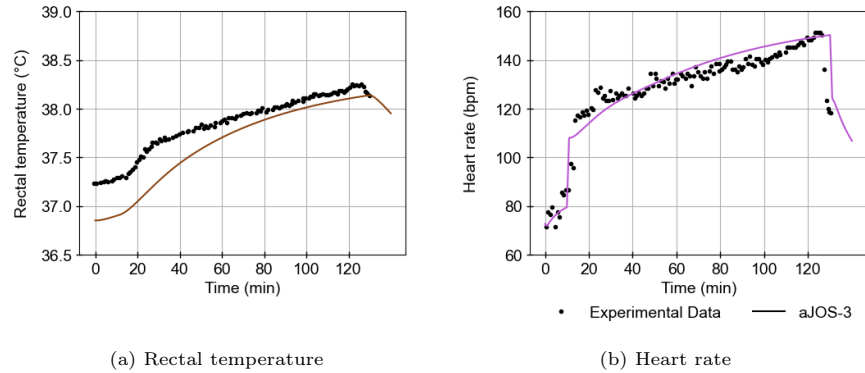


Figure 9: Comparison of the data from Moran et al. [65] and the predicted data with aJOS-3

Figure 9 shows the comparison between Moran’s data and aJOS-3 results. On the one hand, the rectal temperature is underestimated by aJOS-3 as discussed in section 3.1 and 3.3. However, for this comparison with Moran’s results, the RMSE for rectal temperature is approximately 0.29°C which is within the range of values found by Takahashi et al. [26] for the validation of JOS-3 and is acceptable in comparison to the conservative ISO 9886 heat strain limit: an increase of 1°C . The difference is notably visible before the beginning of exercise: around 25 % of this deviation can be explained by the initial difference which is then compensated by a higher increase during exercise. Therefore, dynamics of rectal temperature during step changes in activity has to be checked without the initial difference. On the other hand, the increase in the heart rate because of activity level and cumulative heat stress is correctly simulated. The RMSE is approximately 8 bpm, which is acceptable given that the heart rate strain threshold is defined as an increase of 30 bpm. Therefore, heart rate prediction with aJOS-3 is validated for young men during step change of activity levels.

3.5. Thermal strain for elderly people

To evaluate the validity of aJOS-3 for elderly people, simulations were carried out to reproduce the experiments of Dufour et Candas [66] and Stapleton et al. [67] (Fig. 10).

Ou et al. [31] used these experimental data to validate their thermophysiological model for elderly people. They modified Deng’s model to assess heat-stroke risk in healthy elderly men while accounting for dehydration. Figure 11 summarizes the effects of age in Ou’s model compared to aJOS-3. In Ou’s model, simulating an elderly person is an option that reduces and delays sweating and vasodilation. In contrast, aJOS-3 considers age as a continuous input parameter.

The assumptions made by Ou et al. are used to parametrize aJOS-3 simu-

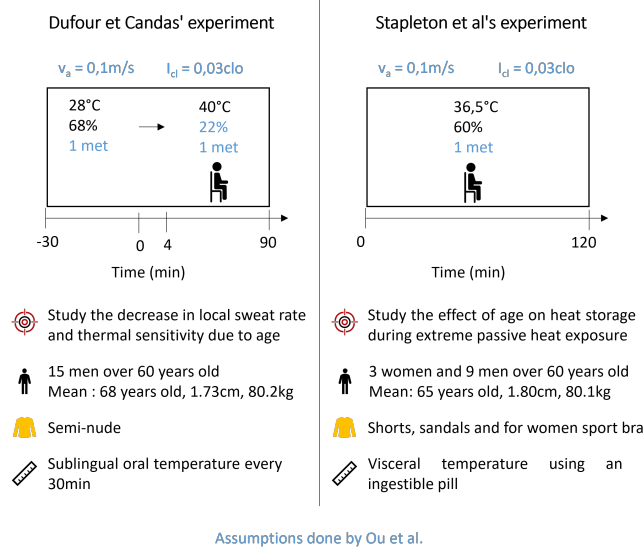


Figure 10: Experiments of Dufour et Candas [66] and Stapleton et al. [67]: objectives, experimental designs, participants and input conditions to simulate

lations that reproduce the experiments of Dufour and Candas and Stapleton et al. Figure 10 summarizes the input conditions for these simulations. In particular, the activity level is set to 1 met, and for Dufour and Candas' experiment, relative humidity is set at 22%. Only the characteristics of the simulated individuals are based on the mean characteristics of the participants from both experiments. As in Ou et al.'s study, this paper focuses only on core temperatures: the head core temperature compared to the sublingual temperature for Dufour and Candas, and the pelvis core temperature compared to the visceral temperature for Stapleton et al.

Figure 12 presents the core temperatures simulated with aJOS-3 and Ou's simulation results compared to measurements from Dufour and Candas' and Stapleton et al.'s experiments. In the Dufour and Candas case (Fig. 12a), the gap between aJOS-3 and the experimental data is nearly twice as large as the gap between Ou's simulation and the experimental data. The initial core

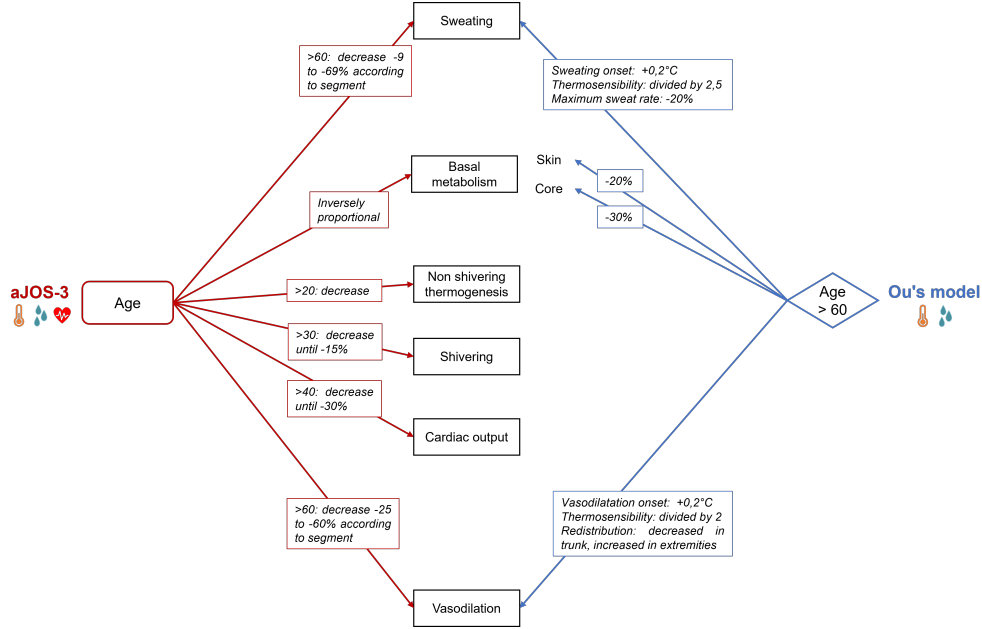


Figure 11: Effects of age modeled in aJOS-3 and Ou's model

temperature in aJOS-3 is higher than in the other datasets, and its temperature increase during the experiment is lower. This suggests that aJOS-3 has a stronger thermoregulation capacity than the participants, likely due to default input parameters corresponding to a young man, such as fat percentage, cardiac index, and maximal oxygen uptake. Moreover, Figure 12a compares two slightly different variables: the head core temperature and the sublingual temperature. The sublingual temperature is more sensitive to external conditions due to respiration. For Stapleton et al.'s experiment (Fig. 12b), aJOS-3 results align closely with those of Ou et al., though with a higher initial temperature. Both simulated temperatures match the experimental data well. However, at the end of the exposure, the simulated core temperature increase is lower than the measured one, suggesting that simulated dehydration is lower than in the experiment. In both experiments, the difference between aJOS-3 results and experimental data remains below 0.15°C , with RMSEs under 0.1°C . This in-

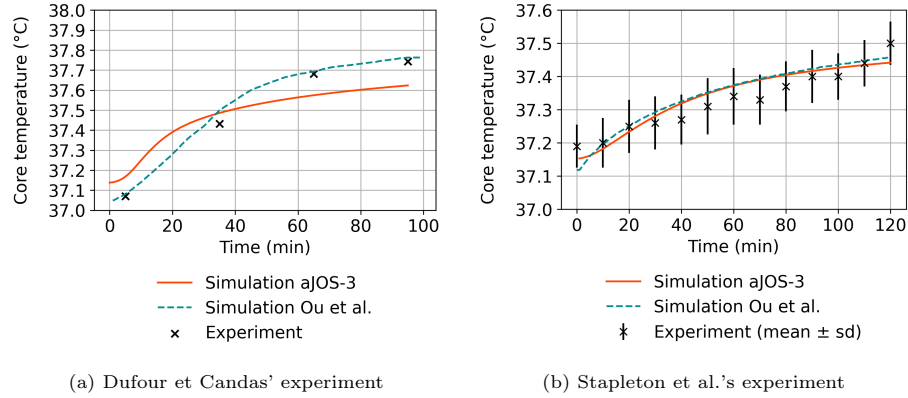


Figure 12: Comparison between Ou et al.'s [31], aJOS-3 simulations and experimental: effects of dehydration on the head core temperature during constant heat exposure for elderly men

indicates that aJOS-3 is validated for elderly people exposed to constant heat stress.

To conclude, comparisons with literature experimental data validate aJOS-3 for:

- young men during constant severe heat exposure;
- young men during a step change in activity levels;
- elderly during constant heat exposure.

However, extending the model's applicability to different conditions, identifying sources of inaccuracy, and improving the precision of core temperature predictions require further comparisons with experimental data.

4. Discussion

Beyond the aJOS-3 ability to simulate thermal, hydric, and cardiovascular strain, this section discusses its ability to assess heat risk and its effects on health. Simulations are carried out to calculate a thermal risk index and compare it with experimental data. Then, aJOS-3's sensitivity to individual

parameters to assess is analyzed the strengths and limitations of its individualization.

4.1. Thermal risk assessment

A hundred indices exist to assess heat risk, but most of them are based on heat stress, meaning they rely on environmental thermal data [68]. Indices based on thermal, hydric, and cardiovascular strain are rare. The Heat Strain Classification Index (HSCI) is one such index [69]. Its equation (Eq. 20) uses the core temperature, the dehydration and the heart rate to quantify heat strain on a scale from 0 to 10.

$$HSCI(t) = 3.1 \cdot \frac{T_{cr}(t) - T_{cr}(0)}{39.5 - T_{cr}(0)} + 3.3 \cdot \frac{HR(t) - HR(0)}{180 - HR(0)} + 3.6 \cdot \frac{de(t)}{0.05} \quad (20)$$

This score is then translated into Human Thermal Risk (HTR) index based on a five-level scale. Therefore, aJOS-3 can theoretically be used to calculate both HSCI and HTR, but the reliability of such an approach should be investigated.

For this purpose, aJOS-3 simulations were carried out to reproduce the experiments of Zhao et al. [69] which led to the development of HSCI and HTR. A total of 120 experiments with 56 young men under 24 different conditions were carried out. During each experiment, a participant spent 30 min at rest in a preparation room and then ran on a treadmill in a climatic chamber for 90 min, with 5 min for rest and measurements every 15 min. For the results, the labor time was used, that means without counting rest time. The air temperatures were set to 33, 35, 37 or 39 °C, and relative humidity to 60, 70, 80 or 90 %. Two levels of activity were tested: 3 km/h and 4.5 km/h. The clothing thermal insulation was 0.5 clo, and the air velocity was around 0.2 m/s. Oral tempera-

ture, heart rate, and body weight were measured at every break. Participants were free to drink water, and the amount of water consumed was included in the total water balance and thus in the dehydration calculation.

The same thermal configuration was implemented as input for the aJOS-3 simulations. The activity level was set to 2.4 met for experiments conducted at 3 km/h and to 3.1 met for those at 4.5 km/h. Since Zhao et al.'s article did not provide information on water consumption, water intake was disabled. The following mean characteristics of the experimental participants were used for the simulation: male, 21 years old, 1.75 m, and 68.7 kg. The HSCI was calculated using Eq. 20 with both aJOS-3 results and raw data from Zhao et al.'s article, and the HSCI values were then translated into HTR levels.

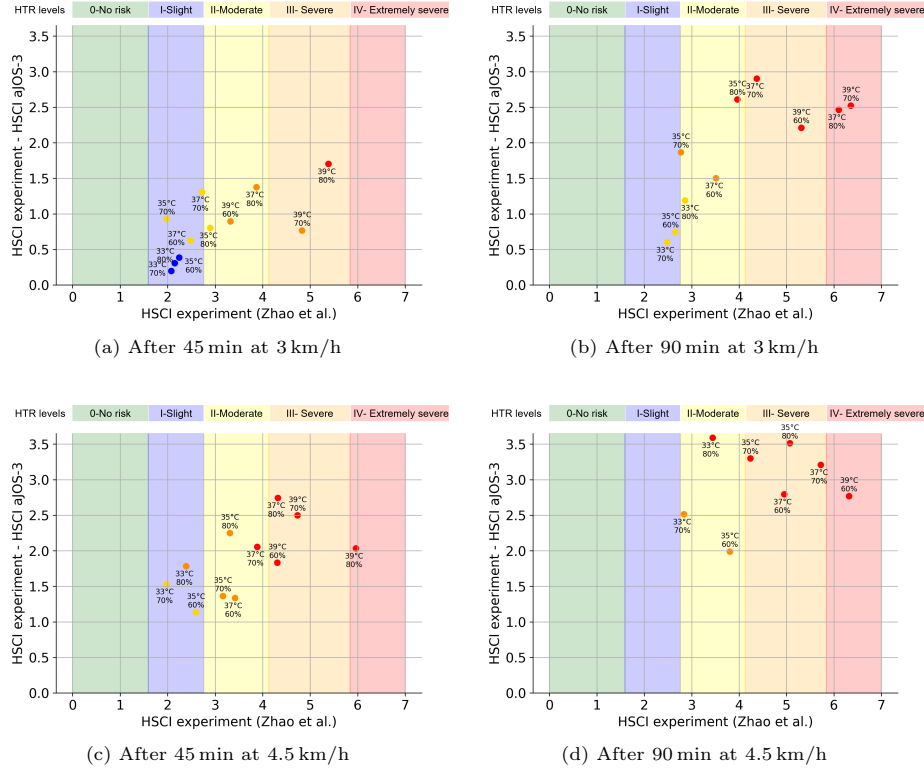


Figure 13: Comparison of HSCI and HTR levels calculated from Zhao et al.'s experimental data and aJOS-3 simulations: Point color represents HTR levels from aJOS-3 simulations.

Figure 13 shows the difference between the HSCI calculated from Zhao et al.’s data and from aJOS-3 simulations. The background color represents the HTR levels from Zhao et al., while the point color indicates the HTR levels from aJOS-3. The results highlight that aJOS-3 overestimates the HSCI for all configurations, with values reaching up to twice the experimental values and with a maximum absolute error of approximately 3.6. This difference from the experimental data translates into an overestimation of HTR levels by one or two levels. The differences are more significant for configurations with a running velocity of 4.5 km/h, particularly for severe exposure, and increase with labor time. The HSCI errors appear not mainly explained by the differences between simulated and measured water loss (less than 0.6 % of body weight), but rather by significant differences in core temperature (up to 1.42 °C) and heart rates (up to 64 bpm).

These errors in thermal and cardiovascular strain, and thus in HSCI and HTR, highlight key points for the use of aJOS-3 and, more generally, thermophysiological models to predict heat risk. Starting from the most specific to the most general aspects, these points are:

- Assessing heat risk requires properly parametrizing water intake. In this comparison, water intake was not simulated. The total dehydration had a direct impact on thermoregulation, increasing thermal and cardiovascular strain. In contrast, in the experimental case, part of the water loss was compensated by fluid intake, thus reducing strain.
- Exposure history, even over a short term, affects heat risk assessment. For the reproduction of Zhao et al.’s experiment, preparation time was not simulated due to a lack of information, leading to the significant differences in the heart rate even before the start of the activity (up to 34 bpm).
- Adapting model parameters to individuals is necessary for predicting per-

sonal heat risk. In this comparison, average participant characteristics were used as input for the simulations, even though not all participants were involved in every configuration. This assumption may explain a significant part of the difference between the experimental and simulated results, as the standard deviation of measurements can reach $\pm 0.7^{\circ}\text{C}$ for the oral temperature, ± 26 bpm for the heart rate, and leading a standard deviation of around ± 1.44 for the HSCI according to Zhao et al.'s measurements.

Therefore, heat risk assessment is sensitive to individual behavior (such as water intake), exposure history, and personal characteristics. Hence, next section discusses the sensitivity of aJOS-3 to these factors.

4.2. Sensitivity to individual parameters

The aJOS-3 model, as the original JOS-3 model, has several parameters that enable individualization. These parameters affect significantly the aJOS-3 outputs. To illustrate this, a hypothetical day was simulated with different individual parameters (Table 2). This day aims to represent a wide variety of possible cases: high or low temperatures, similar or different air and radiant temperatures, still air or high air velocity, humid or dry rooms, and different levels of activity.

Table 2: Typical day configuration for sensibility analysis

Time (hr)	0-9	9-10	10-13	13-15	15-17	17-20	20-21	21-24
Activity	Sleeping	Washing	Rest In AC room	Cooking	Sport	Rest In AC room	Cooking	Rest
Air temperature [$^{\circ}\text{C}$]	28	30	23	30	30	23	30	30
Radiant temperature [$^{\circ}\text{C}$]	28	30	23	35	30	23	35	30
Relative humidity [%]	50	80	30	50	50	30	50	50
Air velocity [m/s]	0.1	0.1	3	0.1	0.1	3	0.1	0.1
Physical Activity Ratio [met]	0.833	1.67	1	2.08	3.54	1	2.08	1
Clothing insulation [clo]	0.13	0	0.42	0.42	0.15	0.42	0.42	0.42

In order to compare only the effects of age and sex, figure 14 shows the results for young man and woman (20 years old) and for older man and woman (70 years old). All other input parameters are set up by default although it is known there are significant difference of morphology and cardiac capacity according to age and sex. Results show that aJOS-3 simulates a higher head core temperature for men than for women and for older people than for young. The change in core temperature, both increases and decreases, is higher in simulations for older individuals. This indicates that the regulation of core temperature is less efficient in this population, which is consistent with existing literature on elderly thermoregulation [70]. Simulated thermoregulation appears more effective in younger individuals, leading to larger variations in heart rate, higher cardiovascular strain, and higher dehydration. The three types of strain simulated are more severe for men than women. All other parameters being equal, the main difference between men and women is the higher metabolism for men, which leads to a higher heat load.

To study the sensitivity of aJOS-3 to body weight, Figure 15 presents the results for young men with different body weights: the default weight (74.43 kg), the 2.5th percentile of French men aged 18-29 (56.9 kg), and the 97.5th percentile of French men aged 18-29 (106 kg). Changing weight affects the basal metabolism and the tissue thermal capacity in aJOS-3. The simulation results show that the heavier the simulated individual, the higher the core temperature, without affecting thermal strain. However, the simulated hydric and cardiovascular strain are significantly lower for the 106 kg individual. The modeling choices explain this outcome: using maximal oxygen uptake per kilogram as an input simulates cardiovascular capacities that increase with weight, which may not be entirely realistic, and calculating hydric strain relatively to initial body weight.

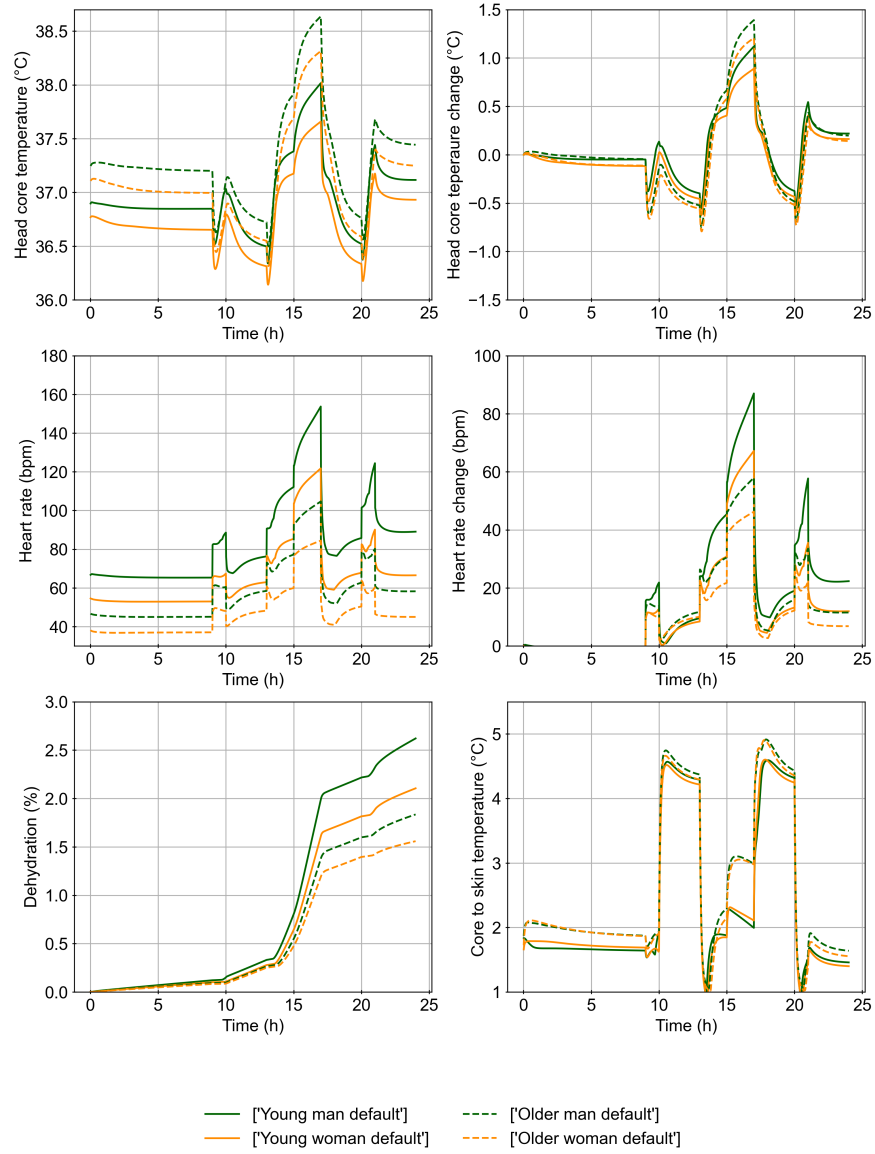


Figure 14: Results of simulations for an hypothetical day for young and old men and women (other parameters set up by default)

Therefore, sex, age and weight affect significantly the results of aJOS-3 simulations. However, other individual parameters can be set in aJOS-3 modeling, notably water intake and maximal oxygen uptake introduced with the addition

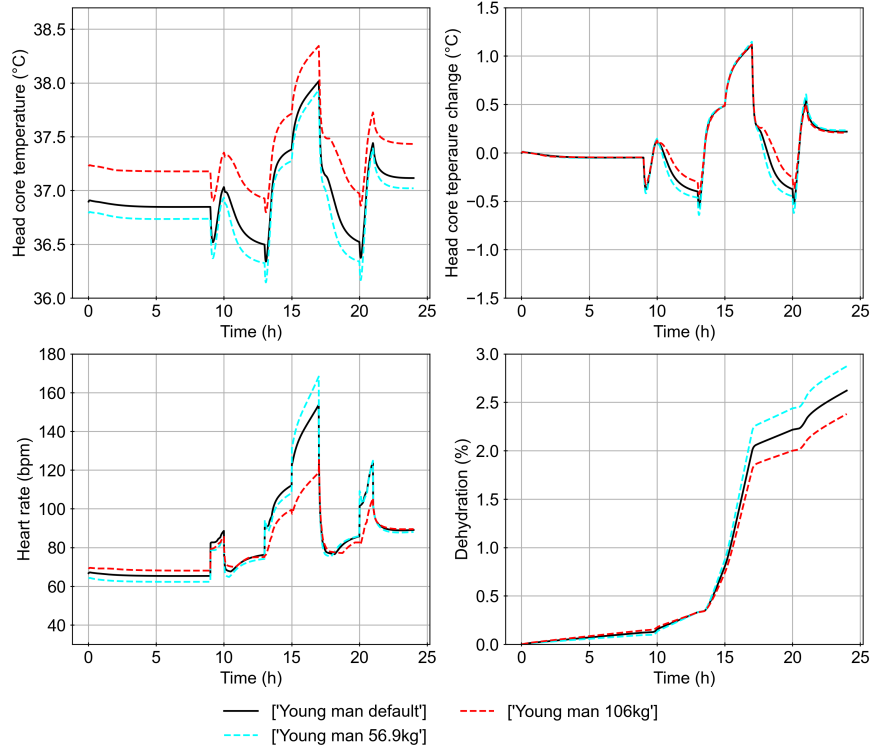


Figure 15: Results of simulations for a hypothetical day for young men with different weights (other parameters set up by default)

of hydric and cardiac models. These individual parameters are often difficult to evaluate for a given individual or population. The impact of these parameters on the output has to be evaluated, notably in order to estimate if using statistical mean values for a given population is a significant source of uncertainty or not. To address this issue, a sensitivity study to age, height, body weight, fat percentage, cardiac index, maximal oxygen uptake, and water intake was carried out. This sensitivity analysis is performed for both a standard young man and an elderly woman. The parameters vary within the ranges defined in Table 3. As previously, the hypothetical day (Table 2) is simulated. To highlight the effects of the adaptations made to JOS-3 in aJOS-3, the same sensitivity analysis has also been carried out for JOS-3 without maximal oxygen uptake

and water intake.

Table 3: Intervals of parameter variation for the sensitivity study

Parameters	Young man	Elderly woman	Unit	Type of interval	
Age	[18; 29]	[60; 80]	yr		
Height	[1.63; 1.95]	[1.47; 1.71]	m	Between 2.5 th and 97.5 th percentile	[71]
Weight	[56.9; 106]	[47.3; 98]	kg	Between 2.5 th and 97.5 th percentile	[71]
Fat percentage	[18.8; 32.8]	[26.7; 43.7]	%	Mean $\pm$ sd	[72]
Cardiac index	[2.2; 4.1]	[2.1; 3.2]	L/(min m ²)	Normal values	[73]
Maximal oxygen uptake	[46; 62.8]	[23.1; 33.5]	mL/(kg min)	Mean $\pm$ sd	[74]
Water Intake	[0; 2935.4]	[0; 2370.6]	g/d	TWI (95% confidence interval) - Urine volume (Mean $\pm$ sd)	[75, 76]

The Sobol method [77] is used. A total of 4608 simulations have been run for both types of individuals and both model. The temporal evolution of thermophysiological variables being important to assess heat strain, then the Sobol indices, the mean value and 95 % prediction interval of core temperature, dehydration, heart rate and core-to-skin temperature are calculated at each time step.

Figures 16, 17 and 18 present the corresponding results for a standard young man simulated with aJOS-3. The variability of each parameter independently explains more than 94 % of the variability of the outputs of interest. Therefore, only the first order Sobol indices are studied. Table 4 compares the maximal prediction interval width for a young man and an elderly woman and for both models.

For the core temperature (Fig. 16), the maximum width of the 95 % pre-

Table 4: Comparison of the maximum width of the 95 % prediction intervals for the standard man and elderly woman sensitivity analysis

	Maximum width of prediction interval				
	Young man		Elderly woman		Moment of widest interval
Outputs	aJOS-3	JOS-3	aJOS-3	JOS-3	
Head core temperature [°C]	1.37	1.45	1.43	1.42	During rest in AC room
Head core temp. change [°C]	0.40	0.28	0.58	0.47	During rest in AC room
Dehydration [%]	2.24	x	2.26	x	At last time step
Heart rate [bpm]	92.75	x	52	x	During sport
Heart rate change [bpm]	63.74	x	31.24	x	During sport
Core-to-skin temp. [°C]	1.53	1.56	1.83	1.87	End of rest in AC room

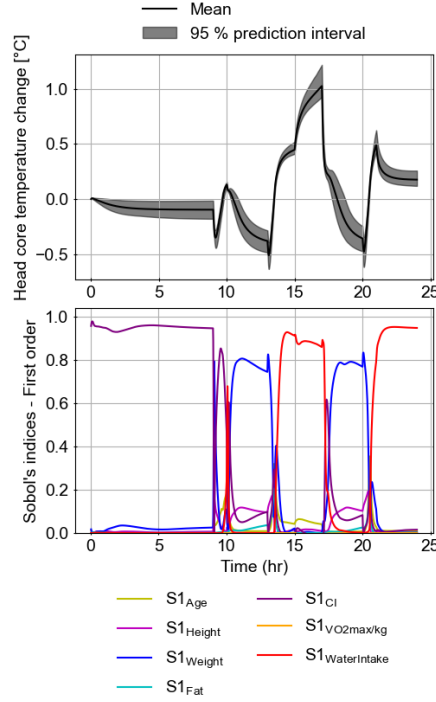


Figure 16: Results of the sensitivity analysis of individual parameters: simulated head core temperature change using aJOS-3 for a hypothetical day and a standard young man.

diction interval exceeds 1.3°C , which is significant for the study of thermal strain. Using the average parameters of a standard young man may thus lead to misinterpretation of the risk of heat stroke, as observed during the simulated exercise session between 15 and 17 h on the hypothetical day. The variability induced by cardiac index is the most significant, especially during exercise phases and during rest under heat stress. The core temperature is also sensitive to body weight variability during the fresh recovery phases: body weight affects the body inertia. Furthermore, the 95 % prediction interval is very wide from the first time step. Thus, the initial core temperature is very sensitive to the variability of individual parameters, and thermal strain is primarily represented by an increase in the core temperature. Therefore, studying the sensitivity of

changes in core temperature is better. The maximum width of the 95 % prediction interval is then divided by approximately 3. It appears that the increase in core temperature is still affected by body weight but also by water intake during physical exercise and at the end of the day. These are the periods when dehydration is significant or increases the most, and thus when the variability of dehydration is the greatest (Fig. 17). Dehydration is almost exclusively sensitive to water intake variability, which is logical according to the definition of dehydration. Hence, the sensitivity of the core temperature to water intake can be explained by the effects of dehydration on thermoregulation. For the head core temperature, the head core temperature change, and the core-to-skin temperature gradient, the sensitivity analysis results for aJOS-3 simulations are similar to those obtained with version 0.5.0 of the original JOS-3 model in terms of prediction interval width, the moment of the widest interval, and the most impactful parameters. The only notable difference is that, in JOS-3, the head core temperature change is also sensitive to age. In aJOS-3, this sensitivity appears less significant due to its predominant sensitivity to water intake. Both age and water intake influence thermoregulation capacity.

The 95 % prediction interval for heart rate (Fig. 18) is consistently wide. This variable is almost exclusively sensitive to the cardiac index, which is logical since this parameter directly calculates the resting heart rate. Notably, the discrepancies from initialization are significant, indicating that resting heart rate values, such as those of the core temperature, are highly impacted by individual parameters. The sensitivity of heart rate change has thus been studied. The corresponding width of the 95 % prediction interval is significantly reduced but remains important. The change in heart rate is mainly affected by the variability in water intake because of the effect of dehydration on vasodilation and by the body weight during recovery periods.

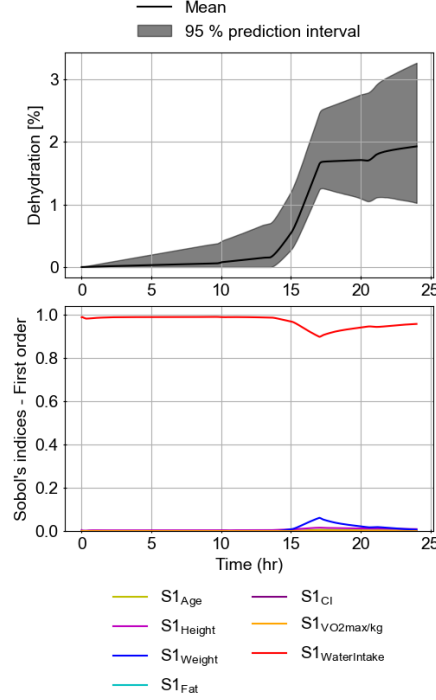


Figure 17: Results of the sensitivity analysis of individual parameters: simulated dehydration rate using aJOS-3 for a hypothetical day and a standard young man

Therefore, the individual parameters for which the use of average values can cause significant interpretation errors are water intake, body weight, and cardiac index. Body weight can be easily estimated with an accuracy of approximately one kilogram by measurement or by asking individuals. Cardiac index can be measured only through medical examinations (electrocardiography, Doppler, etc.) [78]. However, the cardiac index mainly affects resting values and model initialization, so with a sufficient initialization period, comparing situations using the average cardiac index is possible. Water intake needs to be studied in greater detail, notably to understand the interactions between drinking, eating, urinating and heat stress. The sensitivity analysis has shown that the adaptations of JOS-3 to develop aJOS-3 are a source of uncertainty

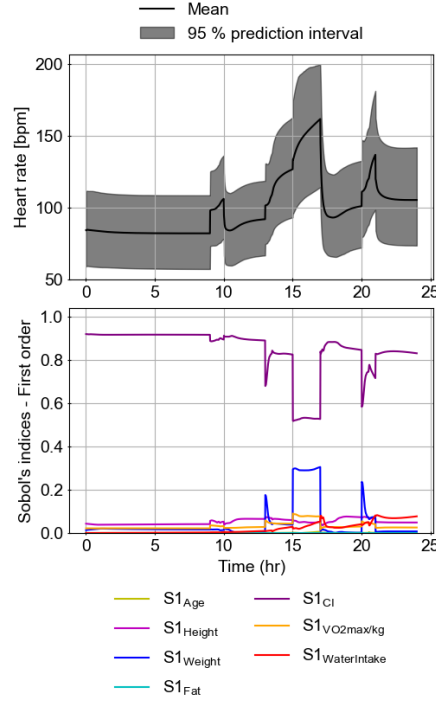


Figure 18: Results of the sensitivity analysis of individual parameters: simulated heart rate using aJOS-3 for a hypothetical day and a standard young man

due to water intake but not due to maximal oxygen uptake. Without further studies on this subject, this adapted model can be used to compare situations and identify at-risk hydration behaviors.

5. Conclusion

As a first step to improve the assessment of heat effects on health, this study suggests an adaptation, called aJOS-3, of a thermophysiological model in order to simulate thermal, hydric, and cardiovascular strain during heat exposure and to take into account some individual vulnerability factors. The original multi-segment multi-node JOS-3 model simulates core temperature, skin temperatures, water loss due to latent heat exchange with the environment and cardiac

output. The proposed adaptation extends these functionalities by incorporating a comprehensive water balance, stroke volume and heart rate calculation, and modeling thermoregulatory limitations due to hydric and cardiovascular strain. Thus, the adapted model, aJOS-3, is able to simulate hydric and cardiovascular strain in addition to thermal strain already simulated by the original JOS-3 model. Two additional input parameters, water intake, and maximal oxygen uptake, are necessary for this adaptation.

The validation of the adapted model is performed through comparison with simulation and experimental data from the literature. The water balance model is validated under conditions of constant severe heat stress, demonstrating a good agreement with Senay et al.'s data (RMSE = 65 grams). Similarly, the heart rate model was validated under severe heat stress with a step-change in activity levels, in agreement with Moran et al. (RMSE = 8 bpm). However, discrepancies are observed in the rectal temperature predictions, indicating a slight underestimation in all cases, partly due to initialization issues. For elderly men, aJOS-3 seems to accurately predict the core temperature under constant heat stress. To summarize, aJOS-3 is validated for simulating the core temperature, the heart rate, and the dehydration during constant heat exposure or following a step change in activity level.

The use of the adapted model, aJOS-3, for heat risk assessment faces limitations, particularly concerning individualization. The Sobol sensitivity analysis performed on one hypothetical day simulations highlights needs in accurately estimating, for input, the cardiac index and water intake. This result suggests perspectives for further research on easy-to-measure alternatives to the cardiac index and the evaluation of hydration behaviors according to heat stress and seasonality. However, the use of aJOS-3 as any other thermophysiological models, even the most individualizable ones, for vulnerable people (children, elderly

people or sick people) remains questionable because of a lack of validation on these populations. Indeed, 82 % of the participants included in heat strain experiment were men and almost 70 % of participants were younger than 30 years old (men and women) [79]. Thus, the validation process of thermophysiological models should be extended to vulnerable populations and to non-constant prolonged exposures in order to integrate the cumulative effects of heat exposure and the acclimatization effects into thermophysiological models. For this purpose, a reflection must be conducted to design a protocol for long-term real-life exposure experiments involving vulnerable individuals, ensuring their safety anticipating, and minimizing potential risks.

In conclusion, despite some precautions, the adapted model, aJOS-3, is shown to be a valuable tool for studying thermal, hydric and cardiovascular strain and further heat-related symptoms, by taking into account effects of physiological and environmental vulnerability factors under specific exposure condition and for healthy men, and comparing different risk scenarios.

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CRedit authorship contribution statement

Célia Sondaz: Conceptualization, Investigation, Writing - Original Draft, Visualization. **Lucie Merlier:** Writing - Review & Editing, Supervision, Project administration. **Claire Harpet:** Writing - Review & Editing, Supervision. **Frédéric Kuznik:** Writing - Review & Editing, Supervision.

Declaration of Interest statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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