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Modeling Thermoregulatory Responses to Cold Environments

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Abstract

The ability to model and simulate the rise and fall of core body temperature is of significant interest to a broad spectrum of organizations. These organizations include the military, as well as both public and private health and medical groups. To effectively use cold models, it is useful to understand the first principles of heat transfer within a given environment as well as have an understanding of the underlying physiology, including the thermoregulatory responses to various conditions and activities. The combination of both rational or first principles and empirical approaches to modeling allow for the development of practical models that can predict and simulate core body temperature changes for a given individual and ultimately provide protection from injury or death. The ability to predict these maximal potentials within complex and extreme environments is difficult. The present work outlines biomedical modeling techniques to simulate and predict cold-related injuries, and discusses current and legacy models and methods.

Keywords: hypothermia, cold injury, clothing, military, biophysics, survival

1. Introduction

Mitigating hot and cold injuries is a complex problem and has been shown to have significant links to a number of individualized factors, to include race, gender, job specialty, and geographic origin [1, 2]. There are many other individualized elements (e.g., fitness, body composition, and genetics) that are intuitively linked to these health outcomes; however, there is a lack of adequate data to scale that sufficiently addresses these issues.

The history of characterizing heat exchange and thermoregulatory functions in humans can be traced back to the late 1770s; where British military physiologist, Sir Charles Blagden conducted descriptive studies of man, dog, and beef steak responses in a hot room [3]. Mathematically describing heat exchange theory has roots in physics and with the development of the laws of thermodynamics and heat exchange, specifically as described in Fourier's law [4] a mathematical expression of the dynamics of heat balance in solids, simplified as:

$$\rho \cdot c \cdot \left(\frac{\partial T}{\partial t} \right) = \nabla k \cdot \nabla T + H \quad (1)$$

where ρ is density (g/m^3), c is specific heat [$(\text{kcal}/^\circ\text{K} \cdot \text{kg})$], k is heat conductance [$\text{kcal}/(\text{hr cm } ^\circ\text{K})$], T is temperature ($^\circ\text{K}$), t is time (hours), and H is the net flow rate of heat other than by diffusion.

Key work by Pennes in 1948 [5], reported measured temperatures of tissue and blood at the forearm and enabled the creation of the bioheat transfer equation. This equation has proven to be a key underlying basis of future models, seen as:

$$\nabla \cdot k \nabla T + q_p + q_m - W_{C_b}(T - T_a) = \rho c_p \left(\frac{\partial T}{\partial t} \right) \quad (2)$$

where k (w/m °C) is the tissue thermal conductivity, T is tissue temperature in °C, q_p (w/m³) is energy deposition rate, q_m (w/m³) is metabolism, W (kg/m³/s) is local tissue blood perfusion rate, C_b (J/kg/°C) is specific blood heat, T_a (°C) is arterial temperature, ρ (kg/m³) is the tissue density, and c_p (J/kg/°C) is the specific tissue heat.

Conceptually, heat exchange between the human and the environment was first described by Lefevre in 1911; where he characterized the human as a sphere with an internal core that exchanged heat through the shell into the environment [6]. In 1934, Burton applied Fourier's law, presenting this exchange mathematically and describing the human as one uniform cylinder in what is considered by many as the first visual conceptualization of human thermoregulatory modeling [7].

Representation of the human in a thermoregulatory model is most often done by sectioning the human into nodes, segments, and elements; typically using one of four different designs, (1) one-node, (2) two-node, (3) multi-node, or (4) multi-element [8]. An example of the difference between these designs is shown in **Figure 1**; while the multi-element approach is more realistic human shape (e.g., finite analysis distribution). Typically each node represents an independent layer with unique thermal properties, each segment represents a section or grouped section of an area of the body, and each element represents multiple thermal components that make up the whole body (often more geometrically accurate to the shape of the human).

One node models are essentially empirically derived and do not include elements within the thermoregulatory response system. There are several one node thermoregulatory models that have been used extensively over time to predict core body temperature and thermal discomfort within a given environment [9–12].

Simple two-node models describe specific thermodynamic responses of a single segment, typically separated into concentric core and shell nodes. They have often been used examine thermal discomfort and physiological responses, to include the work by Gagge and Nishi [13–15], and several others [16–19]. Two node model approaches have been used where the two node design was applied to multi-segments [20–23]. Multi-node models are essentially expanded versions of the two-node methods with additional shells or layers within them where the heat balance is

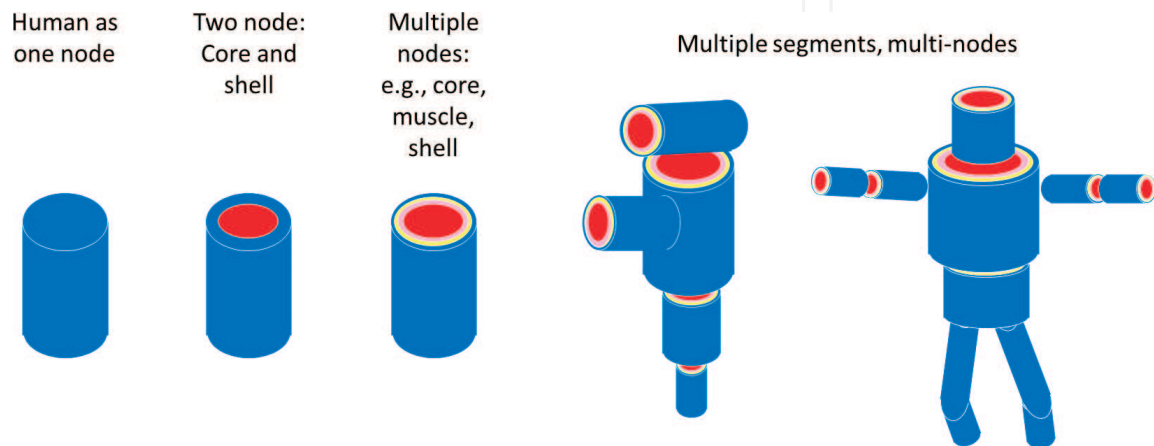


Figure 1.
Example of model designs.

calculated for each layer. Multi-node models, with both single- and multi-segment designs have become the more prevalent approach. The first multi-node model was developed by Crosbie et al. [24] and has been followed by many since [25–29]. Notable is the work of Solwijk and Hardy [30–33], where they first introduced the concepts of temperature set points and negative feedback in a controlled theory design. Their work has been built upon by many researchers over time [34–42]. The first multi-element model was originally published in 1961 by Wissler, and later improved upon [43–45]. Additional multi-element models include work by Smith [46], with the first three-dimensional (3D) transient multi-element model. As computation methods improved, a series of improvements has led to more realistic and complex models [8, 47–50].

While the majority of these models were developed with the intent of characterizing thermoregulation in various environments; several have been designed specifically to address cold environments or thermoregulatory events that specifically address cold issues (e.g., finger, hand, foot temperatures). With the intricacies of human response to cold, studies have focused on extremities, the specific areas most subject to cold injuries. One of the first attempts was by Molnar in 1957, used a heat balance approach to study hand temperature responses to cold [51]. This work was followed by work focused on finger freezing points [52–57] and whole hand modeling [58, 59]. Specific models have also been developed of the foot [60], toes [61], and facial tissues [62, 63]. Cold survival models have been developed over time to make predictions in both open air and submerged environments [64–68].

2. Clinical definitions of cold injuries

Characterizing cold related injuries is fairly complex, as the responses to cold have higher individual variability when compared to heat related injuries. From a clinical perspective, cold related injuries can be broadly divided into three categories: frostbite, nonfreezing cold injuries, and hypothermia. In addition, each of these has varying levels of severity and subcategories associated to them.

Frostbite is below the point at which skin tissue begins to freeze. While 0°C (32°F) is traditionally considered the freezing point of water, the freezing point of skin is understood to be marginally lower due to electrolytes [69]. Observed freezing points range from as low as –4.8°C to as high as –0.6°C [69, 70].

Nonfreezing cold injuries include an array of injury events where tissue freezing has not occurred but damage occurs. The level of severity of nonfreezing injuries is determined by the temperature, duration, and wetness of the exposure to the tissue. Four of the more common specific types of nonfreezing injuries include immersion (trench) foot, chilblain, cold urticaria, and cold-induced bronchoconstriction [71].

Immersion foot is a nonfreezing injury. The foot presents swollen, the skin is red initially but as severity increases the skin becomes lower in oxygen saturation and becomes cyanotic (purple, bluish discoloration) [69, 71]. Immersion foot is most often reported after tissue have been exposed for extended periods of time to non-freezing temperatures, between 0 and 15°C (32–60°F) [71]. The term ‘immersion’ itself refers to when the foot is actually immersed in water when the foot is wet within boots for sustained periods of time [69, 71].

Chilblain is a fairly common nonfreezing injury to the skin. It can occur during 1–5 hours of temperatures below 16°C (60°F) [69]. Cold urticaria is expressed as a quick onset of redness, swelling and itchiness of the skin in response to short-term exposure (i.e., minutes) to cold environments [71]. Cold-induced bronchoconstriction is a physiological response where an individual’s airways are narrowed during exercise in cold environments [69, 71–73].

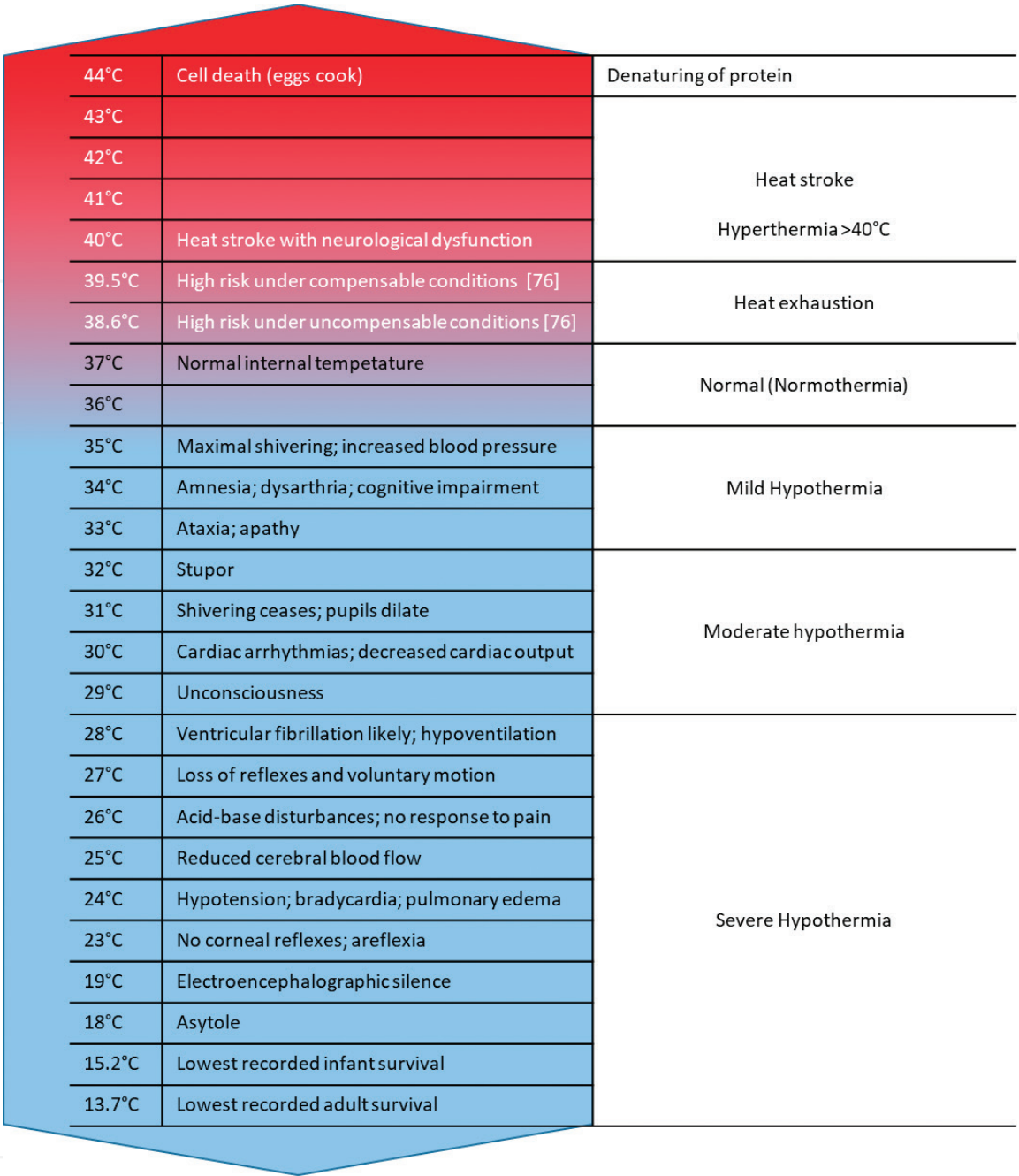


Figure 2.
The range of human core temperatures and associated physiological responses [76].

Hypothermia is a broad category of cold injury and is clinically described to be the point at which core body temperature has dropped below 35°C (95°F) [74]. However, hypothermia is more specifically defined with four levels of severity; where normothermia (normal temperature level) is approximately 37°C (98.6°F), mild hypothermia is between 33–35°C (91.4–95°F), moderate hypothermia being 29–32°C (85.2–89.6°F), and severe hypothermia being 13.7–28°F (56.7–82.4°F) [69, 71]. **Figure 2** outlines specific core temperature reference points associated with physiological responses using work by Castellani et al. [69] and Pozos and Danzl [74] and described in Army Guidance [75].

3. Basics of thermophysiology

The human body is capable of maintaining thermal balance while operating within a wide range of temperatures. The human system generally maintains an

internal core temperature (T_c) of approximately 37°C. Due to natural circadian rhythm, T_c fluctuates ~0.5°C daily. However, T_c can fluctuate based on physical activity or environmental conditions, and may range from 36.0–40.0°C. The microenvironment created between human skin and clothing typically must remain within 28–30°C to maintain thermal homeostasis at rest [45]. This microenvironment changes significantly with physical activity due to metabolic heat production and air movement.

Humans have an internal control system, primarily the preoptic area of the anterior hypothalamus, responsible for maintaining healthy body temperature. The hypothalamus uses feedback from two main sources, the skin and the blood. When temperature changes (hot or cold) are identified by either of these two sources, impulses are sent to the hypothalamus which in turn directs physiological changes to compensate for these temperatures. To protect from cold or heat injury, the human body attempts to either generate or dissipate heat to stay warm or cool off. Heat production is a natural process for humans and is a function of metabolism, oxidation of foods, and muscular activity. Heat transfer between the human and environment occurs via four pathways: conduction, convection, radiation, and evaporation. This heat exchange process is typically referred to as heat or thermal energy balance, and can be described in the heat balance equation:

$$S = M \pm W \pm R \pm C \pm K - E \text{ [W/m}^2\text{]} \quad (3)$$

where S is heat storage; M is metabolic rate; W is work rate; R is radiation; C is convection; K is conduction; and E is evaporation. Radiation is heat that is transferred via electromagnetic waves (e.g., solar radiation). Conduction is heat transfer due to the body's direct contact with a solid object (e.g., touching a cold surface). Convection is heat transfer between the body and a fluid such as air or water. Evaporation is heat loss to the environment due to the phase change from liquid to vapor, typically associated with evaporation of sweat and respiratory water.

Hyperthermia is when heat gain exceeds heat loss; while hypothermia occurs when body temperature drops below normal levels as heat production is inadequate to compensate for the rate of heat loss to the environment [77].

Vasoconstriction and vasodilation are the two key physiological responses of how heat transfer is regulated from the body to the periphery [78, 79]. Vasoconstriction is the constriction of blood vessels and occurs in response to cold environments to reduce the amount of blood flow to the skin. Vasoconstriction protects the internal organs from cold exposure but increases cold injury risk in the extremities due to lower blood flow and lower skin temperatures. Vasoconstriction in effect creates a two-layer distribution of body temperature; a cold outer shell surrounding a warmer core. The colder outer shell reduces heat loss to the environment by reducing the temperature gradient between the skin surface and the environment, and a colder surface radiates less heat.

Vasodilation is essentially the opposite of vasoconstriction; where blood vessels open to allow increased blood flow across the body and out to the extremities to enable increased heat dissipation [78, 79]. During these responses, there are other associated physiological responses that help compensate for the increased skin blood flow (e.g., increased heart rate and cardiac output).

The extremities are more affected by cold exposure than other parts of the body. When the human body cools, blood flow is reduced to the extremities (i.e., the hands and feet) decreasing the amount of warm blood flowing to these areas. It is a challenge to protect the hands and feet as they have lower metabolic heat production of the hands and feet due to their inherently small muscle mass and large surface area to mass ratio.

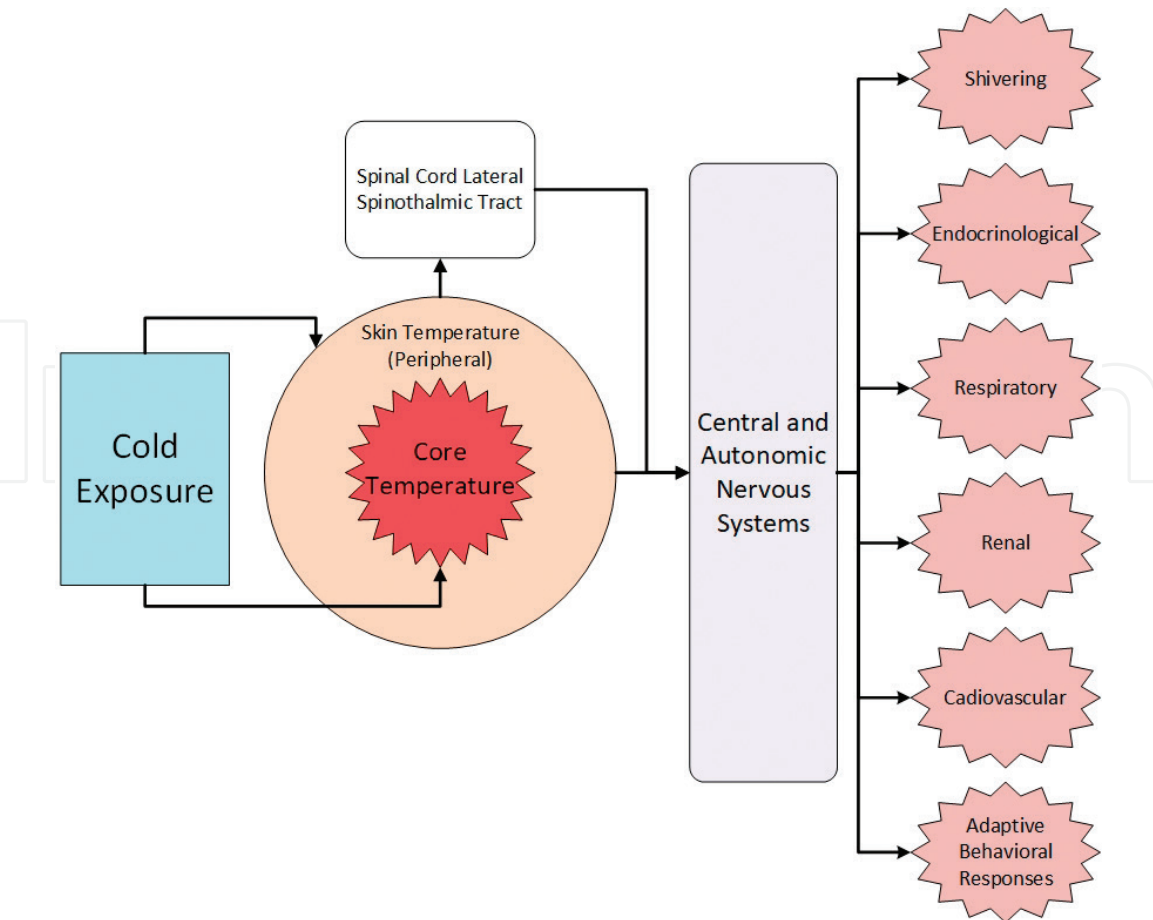


Figure 3.
Peripheral (skin) and core temperature influence on central nervous system (CNS) and physiological outcomes.

From a functional perspective, the balance of control within the human system depends on the response to cold exposure and interaction between peripheral (skin) and core body temperatures with the central nervous system (CNS) and the various physiological responses (**Figure 3**); [74].

4. Importance of clothing

Clothing has long been used to provide protection from environmental elements (heat, cold, etc.) or physical or biological hazards (e.g., rocks, thorns). Clothing properties and requirements vary widely among users and use cases. A single clothing ensemble cannot protect an individual from the extremes of the temperature spectrum of earth, being approximately -89°C at its coldest and 58°C at its warmest. However, clothing is a toll to protect each end of this spectrum of environmental extremes [80]. However, protections must be based on use cases to achieve the desired thermal comfort. For example, protective equipment for American football players (i.e., pads and helmet) is vastly different than protective equipment worn by soldiers (i.e., body armor, ballistic helmet). It should be noted that added protection may increase the thermal burden to wearers, and thus increases risk of heat injuries [81–83].

It is critical to understand the clothing option tradespace in order to predict and prepare for the impact clothing has on protecting or impairing human health. That is to say, the selection of the proper clothing, requires an understanding of how the human (physiology, anthropology, etc.), the anticipated activities (i.e., work rate, length of exposure and metabolic heat production), the work environments (temperature, humidity, etc.), and the biophysical properties of clothing worn (heat transfer performance) will interact in each workplace scenario.

4.1 Clothing biophysics

Clothing protects the wearer from environmental threats, but may impose a level of thermal burden. Both the biophysical resistances (thermal and evaporative) and spectrophotometric (reflectance, absorptivity, and transmittance) properties of clothing can have a significant influence on the impact of the environment on the wearer. Measurements of the biophysical properties of clothing can be used to model the impacts on thermal sensation (e.g., thermal comfort) and thermoregulatory responses (e.g., heat strain, cold protection). The thermal and evaporative resistances, wind effects, and spectrophotometric properties of the clothing are critical measurements for this purpose.

4.1.1 Thermal and evaporative resistance

Sweating thermal manikins have long been used to provide biophysical measures of clothing and equipment worn by the human [84]. While direct biophysical comparisons can be helpful, i.e., comparing one ensemble’s value to another [85], a more informative approach is to combine these measured values with thermoregulatory modeling. Models enable the prediction of thermoregulatory responses based on different individuals, as well as varied environments, clothing, or activity levels.

The current standard for thermal manikin testing calls for two fundamental measures: thermal resistance (R_t) [86] and evaporative resistance (R_{et}) [87]. These two measures represent the dry heat exchange (R_t : convection, conduction, and radiation) and wet heat exchange (R_{et} : evaporation). After converting both R_t and R_{et} into units of clo and i_m [88, 89], a ratio can be used to describe an ensemble’s evaporative potential (i_m/clo) [90].

Each ensemble should be tested using chamber conditions from the American Society for Testing and Materials (ASTM) standards for assessing R_t (ASTM F1291-16) and R_{et} (ASTM F2370-16) [86, 87] (Table 1).

Thermal resistance (R_t) is the dry heat transfer from the surface of the manikin through the clothing and into the environment, mainly from convection, and described as:

$$R_t = \frac{(T_s - T_a)}{Q/A} [m^2 K/W] \tag{4}$$

where T_s is surface temperature and T_a is the air temperature, both in °C or °K. Q is power input (W) to maintain the surface (skin) temperature (T_s) of the manikin at a given set point; A is the surface area of the measurement in m^2 . These measures of R_t can then be converted to units of clo:

$$1\ clo = 6.45(I_T) \tag{5}$$

| Variable (unit) | Skin/surface temperature (T_s , °C) | Ambient temperature (T_a , °C) | Relative humidity (RH, %) | Wind velocity (V , ms^{-1}) | Saturation (%) |
|---------------------------|--|---|---------------------------------|---|-------------------|
| R_t (m^2 K/W) | 35 | 20 | 50 | 0.4 | 0 |
| R_{et} (m^2 Pa/W) | 35 | 35 | 40 | 0.4 | 100 |

Table 1.
American Society for Testing and Materials standard chamber and manikin conditions for testing thermal (R_t) and evaporative (R_{et}) resistance.

where I_T is the total insulation including boundary air layers. Evaporative resistance (R_{et}) is heat loss from the body in isothermal conditions ($T_s \approx T_a$), described as:

$$R_{et} = \frac{(P_{sat} - P_a)}{Q/A} [\text{m}^2 \text{Pa/W}] \quad (6)$$

where P_{sat} is vapor pressure in Pascal at the surface of the manikin (assumed to be fully saturated), and P_a is ambient vapor pressure, in Pascal, of the chamber environment. Measures of R_{et} can then be converted to a vapor permeability index (i_m), a non-dimensional measure of water vapor resistance of materials defined as:

$$i_m = \frac{60.6515 \frac{Pa}{C} R_t}{R_{et}} \quad (7)$$

4.1.2 Wind effects on thermal and evaporative resistance

In order to use the biophysical measures, i.e., measures of R_t (clo) and R_{et} (i_m) for thermoregulatory modeling there is a need to first estimate the effects of wind velocity on the biophysical characteristics of the ensemble (i.e., to determine how wind affects clo and i_m values). These effects are typically referred to as wind velocity coefficients or gamma values (γ) [91]. Historically, these coefficients were determined by collecting measurements of both R_t and R_{et} at multiple wind velocities above the ASTM standard of 0.4 m/s. However, recent work suggests these coefficient values can be accurately estimated from single wind velocity tests [91, 92].

Clothing properties and wind coefficients are critical inputs to a number of predictive mathematical models [10, 11, 93, 94], as they use these values to describe wind-related effects, such as intrinsic insulation (I_{cl}) and intrinsic permeability index (i_{cl}) for either the whole body or segments of the body, as seen with:

$$I_{cl} = I_t - \left(\frac{I_a}{f_{cl}} \right) \quad (8)$$

where I_a is insulation measured on a nude thermal manikin, I_t is total insulation, and (f_{cl}) is clothing area factor, calculated by:

$$f_{cl} = \frac{A}{A_{cl}} \quad (9)$$

where A (m^2) is surface area of the nude manikin, and A_{cl} (m^2) is surface area of the clothed manikin.

True measures of A_{cl} require a three-dimensional scan. However, methods for estimating A_{cl} have been derived by McCullough et al. [95]. Simplified or estimated A_{cl} and f_{cl} is often used where a value of 1 is assumed for warm-weather or indoor clothing. For cold-weather clothing a value would be calculated from:

$$f_{cl} = 1.0 + 0.3 \cdot I_{cl} \quad (10)$$

While these estimation methods have been studied and produce acceptable variance between estimated and direct measured results [96], there are questions whether estimates remain acceptable for clothing insulation outside typical cold weather clothing insulation ranges, e.g., 0.2–1.7 clo [97].

Most clothing-based thermal models, by design, predict human thermoregulatory responses to various environmental conditions and therefore require quantitative insights into the change in clothing properties with changes in wind velocity. Furthermore, elements of wind can significantly influence physiological

responses and injury outcomes in cold environments due to wind chill effects [69, 98, 99]. There has been work to develop that relates exposure time to predicted injury (e.g., frostbite) likely to occur due to temperature and levels of wind speed exposure [98].

5. Modeling risk and predicting heat and cold related injuries

Mathematical models can predict the human thermal response (e.g., metabolic heat production, core body temperature (T_c), endurance time) resulting from activity, environment, and clothing. These mathematical models are typically binned into one of three categories, either as rational, empirical, or hybrid. Rational (mechanistic) models mathematically represent phenomena based on an understanding of physics and physiology (biology, chemistry, physics). Empirical models mathematically reflect the observed relationship among experimental data. While both methods, rational and empirical, are scientifically valid approaches, perhaps the most effective approach is the hybrid or mixed model method that uses a combination of the two.

5.1 Rational models

Rational modeling incorporates equations that describe heat balance and thermoregulatory processes [100]. Two fundamental equations are used to describe internal heat balance and for heat exchange between skin and environment. One equation outlines the temperature gradient change from core to skin and can be seen as:

$$\rho c \cdot \frac{\partial T}{\partial t} = q_m + \lambda \cdot \nabla^2 T + \omega_{bl} \cdot \rho_{bl} c_{bl} \cdot (T_{bl} - T) \quad [\text{W m}^{-3}] \quad (11)$$

where ρ is tissue mass (kg m^{-3}), c is the specific heat of the tissue ($\text{kJ kg}^{-1} \text{ }^\circ\text{C}^{-1}$), T is the tissue temperature ($^\circ\text{C}$), t is time (sec), q_m is metabolic heat production rate (W m^{-3}), λ is the tissue heat conductivity ($\text{W m}^{-1} \text{ }^\circ\text{C}^{-1}$), ∇^2 is a Laplace transform for heat conduction based on the tissue temperature gradient, ω_{bl} is blood flow rate ($\text{m}^3 \text{ s}^{-1} \text{ m}^{-3}$ tissue), ρ_{bl} is blood flow mass (kg m^{-3}), c_{bl} is the blood specific heat ($\text{kJ kg}^{-1} \text{ }^\circ\text{C}^{-1}$), and T_{bl} is the blood temperature ($^\circ\text{C}$).

The second equation describes heat exchange from the skin surface to the environment as:

$$-\lambda \cdot \frac{\partial T}{\partial n} = R + C + K + E \quad [\text{W m}^{-2}] \quad (12)$$

where λ is the tissue heat conductivity ($\text{W m}^{-1} \text{ }^\circ\text{C}^{-1}$), T is tissue temperature ($^\circ\text{C}$), n is the tissue coordinate normal to the skin surface; while the balance is the array of avenues of heat exchange (W m^{-2}): R is radiative, C is convective, K is conductive, and E is evaporative.

Rational models of thermoregulatory processes usually include equations for the controlling signals of the thermoregulation system and equations for thermoregulatory actions such as sweating, vasodilation, vasoconstriction, and shivering.

Understanding the interplay between each of the different layers of the human (grossly consisting of core, muscle, fat, and skin) along with clothing and air layers within clothing is only the first step to modeling the human's response in a given environment. **Figure 4** shows the rational basis behind the SCENARIO model where the human is mathematically represented as one multi-layer cylinder, based on the relationship of the layers of the human, their respective physiological responses, and clothing [93, 94].

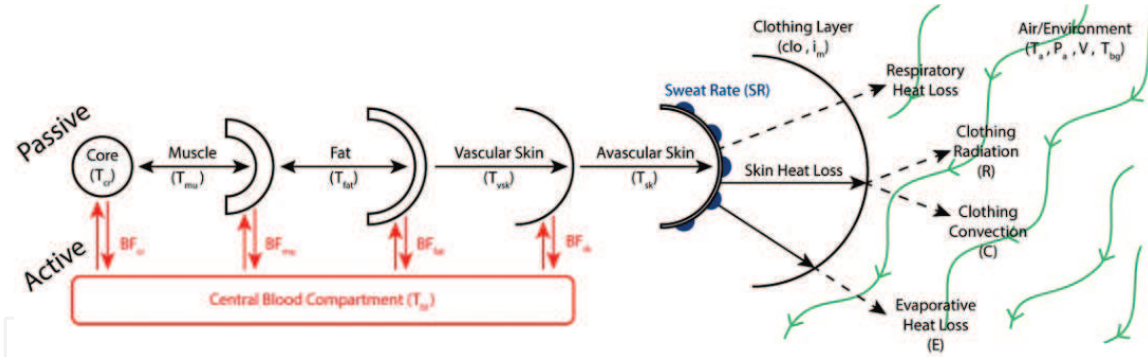


Figure 4. Fundamental rational basis (SCENARIO model) [93], reused with permission. Note: BF_{cr} is core blood flow, BF_{mu} is muscle blood flow, BF_{fat} is muscle blood flow, BF_{sk} is skin blood flow.

5.2 Empirical models

Empirical models are mathematical representations of data, often using statistical methods such as regression or correlational analysis. An example model is the Heat Strain Decision Aid (HSDA), empirically derived by the U.S. Army from an extensive database of human studies that incorporates the biophysics of heat exchange [10, 11, 101] and predicts core temperature, maximum work times, sustainable work-rest cycles, water requirements, and the estimated likelihood of heat casualties. This model has been used to derive guidance and doctrine for military [102] and fluid intake guidance for the public [103]. The basis of HSDA includes both principles of heat exchange along with empirical predictions of physiological responses. Collectively 16 inputs from four elements (individual characteristics, physical activity, clothing biophysics, and environmental conditions) are used to mathematically predict the rise in core body temperature during physical activity [10].

5.3 Simple models

Originally developed by Holmér [104], a simple calculation was adopted by the International Organization Standardization (ISO) technical report (ISO 11079) [105], as an evaluation metric of the insulation required (IREQ) for given environments and activities to compare ensemble performance. The IREQ method functionally describes the concept for balancing the heat exchange between the human and the environment, and simplified as:

$$M - W = E_{res} + C_{res} + E + K + R + C + S \quad (13)$$

where M is metabolic heat produced, W is effective mechanical work and collectively $M-W$ represents the heat produced within the human; while the opposite side of this balance, E_{res} and C_{res} represent the respiratory heat exchange (evaporative and convective), and E , K , R , and C represent the conventional heat exchange methods (evaporative, conductive, radiative, and convective) and S is heat storage.

The IREQ equation illustrates the rational balance between thermal insulation and heat transfer, seen as:

$$IREQ = \frac{\bar{t}_{sk} - t_{cl}}{R + C} \quad (14)$$

or more formally as:

$$IREQ = \frac{\bar{t}_{sk} - t_{cl}}{M - W - E_{res} - C_{res} - E} \tag{15}$$

where t_{sk} is mean skin temperature, t_{cl} clothing surface temperature, and $M - W - E_{res} - C_{res} - E = R + C$.

This method also determines the minimum and neutral IREQ (IREQ_{min} and IREQ_{neutral}), and describes amounts of insulation needed to maintain thermal balance (minimum) and to maintain an equilibrium balance (neutral). The ISO 11079 also outlines general scenarios for the minimum required insulation (IREQ_{min}) for multiple work intensities and environments. Collectively this method provides a simple method for evaluating the effectiveness of specific cold weather clothing at protecting from cold injuries [106].

5.4 Key elements for model development

When developing a cold-based thermal model there are a number of physiological, environmental, and biophysical parameters that can and should be considered. Particular attention should be paid to the extremity temperatures blood flow and metabolic heat production.

5.4.1 Blood flow

As blood flow is a major component to the overall movement of heat, it is important to be able to predict blood flow to the muscle, skin, and distribution of blood flow to these regions within the body. **Table 2** outlines some historical methods used in models for predicting each of these elements.

5.4.2. Shivering

Shivering is where, in response to cold exposure, muscles involuntarily contract rhythmically off and on in an attempt to increase body temperature [74]. During

| Prediction | Equation | Units | References |
|--------------------------------|---|--|---------------|
| Cutaneous blood flow (q_s) | $q_s = q_{s,r} \cdot AVD \cdot CVC_M \cdot CVCL \cdot CVCE$ | mL 100 mL tissue ⁻¹ min ⁻¹ | [79, 107–115] |
| Skin vasodilation (dilat) | $dilat = \beta_{dil,1} \cdot error r_1 + \beta_{dil,2} \cdot (warms - colds) + \beta_{dil,3} \cdot warm_1 \cdot warms$ | L h ⁻¹ | [33] |
| Skin vasoconstriction (stric) | $stric = \beta_{str,1} \cdot error r_1 + \beta_{str,2} \cdot (warms - colds) + \beta_{str,3} \cdot col d_1 \cdot colds$ | L h ⁻¹ | [33] |
| Skin blood flow (bf_s) | $bf_s = 0.53 \cdot bf_{forearm} - 0.83$ | mL min ⁻¹ | [116] |
| Local blood flow (lq_s) | $lq_s = \frac{q_{s,r} + \gamma_{dil} \cdot dilat}{1 + \gamma_{str} \cdot stric} \cdot Q_{10}^{\frac{T-T_0}{10}}$ | L h ⁻¹ | [33] |
| Muscle blood flow (q_m) | $q_m = q_{m,r} + c_m \cdot \Delta M_{W0}$ | L h ⁻¹ | [33] |
| Muscle blood flow (bf_m) | $bf_m = 0.47 \cdot bf_{forearm} + 0.83$ | mL min ⁻¹ | [116] |

Note: q_s and $q_{s,r}$ are skin blood flow and rate; AVD is active vasodilation; CVC is cutaneous vascular conductance—addition of M (mediated), L (locally), and E (effect of exercise); β_{dil} and β_{str} are control coefficients for vasodilation and vasoconstriction; *warms* and *colds* refer to calculated net warm and cold receptors; *bf forearm* is blood flow at the forearm; γ_{dil} and γ_{str} are distribution coefficients for vasodilation and vasoconstriction; c_m is a proportionality coefficient; and M_W is metabolic heat produced from exercise.

Table 2.
Methods for predicting skin blood flow in thermoregulatory models.

| Prediction | Equation | Units | References |
|--|--|---|------------|
| Total shivering (TOTM _{shiv}) | $= 300 \cdot (T_h - T_{h, set}) + 1.35 \cdot (\sum_{m=1}^{14} W_{a,m} \cdot (q'_{s,m} - q_{s, set,m}) + 75 \cdot (\sum_{m=1}^{14} W_{a,m} \cdot (T_{s,m} - T_{s, set,m}))$ | kcal h ⁻¹ | [37] |
| Maximal shivering (Shiv _{max}) | $= 30.5 + 0.348 \cdot VO_{2max} - 0.909 \cdot BMI - 0.233 \cdot age(yrs)$ | mLO ₂ kg ⁻¹ min ⁻¹ | [37] |
| Metabolic rate of shivering (M _{shiv}) | $= 60 \cdot (36.6 - T_{ty}) \cdot (34.1 - T_s)$ | kcal h ⁻¹ | [30] |
| Metabolic rate of shivering (M _{shiv}) | $= 36 \cdot (36.5 - T_{ty}) \cdot (32.2 - T_s) + 7 \cdot (32.2 - T_s)$ | kcal h ⁻¹ | [117] |
| Metabolic rate to open air (M1) | $= 41.31 - 57.77 \cdot \frac{dT_s}{dt} - 5.01 \cdot (T_s - 34)$ | W m ⁻² | [118] |
| Total metabolic rate (M2) | $= M1 + (894.15 - 23.79 \cdot T_{re})$ | W m ⁻² | [118] |
| Total metabolic rate (M) | $= 0.0314 \cdot (T_s - 42.4) \cdot (T_{re} - 41.4)$ | W kg ⁻¹ | [119] |
| Metabolic rate of shivering (M _{shiv}) | $= \frac{155.5 \cdot (37 - T_{es}) + 47 \cdot (33 - T_s) - 1.57 \cdot (33 - T_s)^2}{\sqrt{BF\%}}$ | W m ⁻² | [120] |

Note: *T* is temperature; *h* is head; *set* is set point of temperatures; *W_{a,m}* is a weighting coefficient; *q_s* is heat flux *s* is skin; *BMI* is body mass index; *ty* is Tympanic membrane; *re* is rectal; and *es* is esophageal; *BF%* is body fat percentage.

Table 3.
Methods for predicting shivering related model calculations.

| Prediction | Equation | Units | References |
|----------------|---|---|------------|
| Metabolic rate | $= 1.44 + 1.94 \cdot S^{0.43} + 0.24 \cdot S^4$ | W kg ⁻¹ | [124] |
| | $= 3.5 + 6 \cdot S + 1.08 \cdot S \cdot G$ | mLO ₂ kg ⁻¹ min ⁻¹ | [125] |
| | $= 17.7 - 18.138 \cdot S + 9.72 \cdot S^2$ | mLO ₂ kg ⁻¹ min ⁻¹ | [126] |
| | $= 1.4 + 0.42 \cdot G + 3.68 \cdot S - 0.01 \cdot M - 0.03 \cdot Age$ | W kg ⁻¹ | [127] |
| | $= 1.5 \cdot M + 2 \cdot (M + L) \cdot (L \cdot M^{-1})^2 + \eta(M + L) / (1.5 \cdot S^2 + 0.35 \cdot S \cdot G)$ | W | [128] |
| | $= Ht \cdot (0.0136 \cdot Ht - 0.375)^{-1} \cdot (1.92 \cdot S^{0.176} - 1.445) \cdot Wt \cdot 10^5 \cdot (0.82 \cdot S^2 - 3.94 \cdot S + 9.66)$ | l O ₂ min ⁻¹ | [129] |

Note: *G* is grade (° for Ref. [125], % for others); *Ht*, height (inches for Ref. [129]); *L*, external load (kg); *M*, mass (kg); *η*, terrain factor; *S*, speed (mph for Ref. [129], m s⁻¹ for others); *VO_{2-resb}* resting oxygen consumption (ml kg⁻¹ min⁻¹); *Wt*, weight (lbs).

Table 4.
Methods for predicting metabolic rates during walking or standing.

cold exposure the shivering response is a critical element to model, as the production of heat protects the body core temperature despite skin to the ambient heat loss. **Table 3** outlines some of the modeling approaches that have been used to predict the shivering response as they relate to the total metabolic rate (*M*) and the heat production from shivering (*M_{shiv}*).

5.4.3 Metabolic heat production

An individual's metabolic heat production can be estimated at rest and during activity using the assumed basal rate of 58.2 W/m^2 [121] and the estimated metabolic equivalents (METS) of activity; where 1 MET is resting. Ainsworth et al. [122] outlines a wide range of activities and their associated MET level for reference. However, there are metabolic rate estimation methods available based on energy costs of standing or walking (**Table 4**). Recently work has also been published that makes corrections to some of these prediction methods specific to traveling over snow terrain [123].

6. Summary and discussion

Mathematical models and decision aids are tools for inspiring advancements within the field of thermophysiology, and for providing solutions to help mitigate injury risk.

Scientifically based models have been used in the development of public [97, 98, 103, 104, 130–132] and military guidance [75, 131, 133], for forensic assessments [134–140], as well in the creation of operational tools for survival [141, 142]. Notably, the use of Xu and Werner's six cylinder model [41] was used to develop the Probability of Survival Decision Aid (PSDA), a computer model used to predict hypothermia and dehydration impact on functional time (i.e., duration of ability for useful work), and survival time while exposed to marine environments [67, 143, 144]. The PSDA model is underpinned by the rational principles described herein and the outputs are provided in a customized graphical user interface. This tool has been transitioned for use by Search and Rescue (SaR) personnel and continues to be refined and verified based on real-world feedback and data collected [144].

There is a need for continued advancement in the development of individualized modeling methods such as finite element models as well as providing models and decision aids that can be used in dynamic settings and for complex scenarios with prolonged durations. Additionally, inclusion of probabilistic and statistically based risk factors should be used as elements that help improve individualized predictions. The accessibility of the information from these tools continues to be a challenge for the scientific community. While providing usable information to the public, military, and other user communities should be the ultimate goal of these work efforts; feedback from these communities should be translated back to the scientists to ensure relevant improvements are made from real-world information.

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Conflict of interest

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