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COMPREHENSIVE REVIEW

Physiology of sweat gland function: The roles of sweating and sweat composition in human health

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ABSTRACT

The purpose of this comprehensive review is to: 1) review the physiology of sweat gland function and mechanisms determining the amount and composition of sweat excreted onto the skin surface; 2) provide an overview of the well-established thermoregulatory functions and adaptive responses of the sweat gland; and 3) discuss the state of evidence for potential non-thermoregulatory roles of sweat in the maintenance and/or perturbation of human health. The role of sweating to eliminate waste products and toxicants seems to be minor compared with other avenues of excretion via the kidneys and gastrointestinal tract; as eccrine glands do not adapt to increase excretion rates either via concentrating sweat or increasing overall sweating rate. Studies suggesting a larger role of sweat glands in clearing waste products or toxicants from the body may be an artifact of methodological issues rather than evidence for selective transport. Furthermore, unlike the renal system, it seems that sweat glands do not conserve water loss or concentrate sweat fluid through vasopressin-mediated water reabsorption. Individuals with high NaCl concentrations in sweat (e.g. cystic fibrosis) have an increased risk of NaCl imbalances during prolonged periods of heavy sweating; however, sweat-induced deficiencies appear to be of minimal risk for trace minerals and vitamins. Additional research is needed to elucidate the potential role of eccrine sweating in skin hydration and microbial defense. Finally, the utility of sweat composition as a biomarker for human physiology is currently limited; as more research is needed to determine potential relations between sweat and blood solute concentrations.

ARTICLE HISTORY
Received 30 April 2019
Revised 6 June 2019
Accepted 8 June 2019

KEYWORDS
Chloride; potassium; sauna; sodium; sweat biomarkers; thermoregulation

Introduction

Sweat evaporation from the skin surface plays a critical role in human thermoregulation and this is most apparent when the ability to sweat is compromised during periods of strenuous physical labor and/or exposure to hot environments [1]. For example, in anhidrotic patients [2,3] or individuals wearing encapsulating protective clothing/equipment [4], body core temperature rises sharply with exercise-heat stress, which can lead to heat exhaustion or heat stroke if other means of cooling are not provided. Despite the well-accepted thermoregulatory role of sweating, it is common perception that sweating has a variety of other critical homeostatic functions unrelated to thermoregulation. For instance, sweat glands are perceived to play an important excretory function, similar to that of the renal system, responsible for clearing excess micronutrients, metabolic waste, and toxicants from the body. This belief can lead individuals to engage in practices (e.g. prolonged sauna exposure, exercise in uncompensable conditions) designed to induce heavy sweat losses for their perceived health benefits. However, the effectiveness of sweat glands as an excretory organ for homeostatic purposes is currently unclear as there are no comprehensive reviews on this topic. Another common perception is that excretion of certain constituents in sweat may lead to perturbations in health, such as micronutrient imbalances. A few studies have investigated this notion but a thorough review of the literature has not been published to date. Therefore, the first aim of this paper is to providea comprehensive review of the physiology of sweat gland function, including the types of sweat glands, their structure, and mechanisms that determine the amount and the composition of sweat excreted onto the skin surface. This will provide the background necessary to then discuss the physiological roles of sweat in the maintenance and/or perturbation of human health. In particular, this paper will provide the state of the evidence for the non-thermoregulatory as well as the thermoregulatory

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roles of sweating, consider the methodological challenges of studies in this area, and make suggestions where future research is needed.

Types of sweat glands

The purpose of this section is to compare and contrast the three main types of sweat glands: eccrine, apocrine, and apoeccrine [5,6], which are illustrated in Figure 1. Eccrine sweat glands are the most numerous, distributed across nearly the entire body surface area, and responsible for the highest volume of sweat excretion [5]. By contrast, apocrine and apoeccrine glands play a lesser role in overall sweat production as they are limited to specific regions of the body [7–10]. However, it is important to briefly discuss the apocrine and apoeccrine glands since their secretions can also impact the composition of sweat collected at the skin surface.

Eccrine sweat glands

Eccrine glands were the first type of sweat gland discovered; as they were initially described in 1833 by Purkinje and Wendt and in 1834 by Breschet and Roussel de Vouzzeme, but were not named eccrine glands until almost 100 years later by Schiefferdecker [11]. Eccrine glands are often referred to as the small gland variety, but are by far the most ubiquitous type of sweat gland [12]. Humans have ~2–4 million eccrine sweat glands in total and are found on both glabrous (palms, soles) and non-glabrous (hairy) skin [13–15]. Gland density is not uniform across the body surface area. The highest gland densities are on the palms and soles (~250–550 glands/cm^2) [16] and respond to emotional as well as thermal stimuli. The density of eccrine glands on non-glabrous skin, such as the face, trunk, and limbs are ~2–5-fold lower than that of glabrous skin [16], but distributed over a much larger surface area and are primarily responsible for thermoregulation.

The eccrine glands are functional early in life and, starting at ~2–3 years of age, the total number of eccrine glands is fixed throughout life [12–14]. Therefore, overall sweat gland density decreases with skin expansion during growth from infancy and is generally inversely proportional to body surface area. As a result, children have higher sweat gland densities than adults [11], and larger or more obese individuals have lower sweat gland densities than their smaller or leaner counterparts [13,17]. However, higher sweat gland density does not necessarily translate to higher sweating rate. In fact, most of the variability in regional and whole-body sweating rate within and between individuals is due to differences in sweat secretion rate per gland, rather than the total number of active sweat glands [18,19]. Eccrine sweat is mostly water and NaCl, but also contains a mixture of many other chemicals originating from the interstitial fluid and the eccrine gland itself. The structure and function of eccrine glands and the composition of eccrine sweat will be discussed in more detail in subsequent sections of this paper.

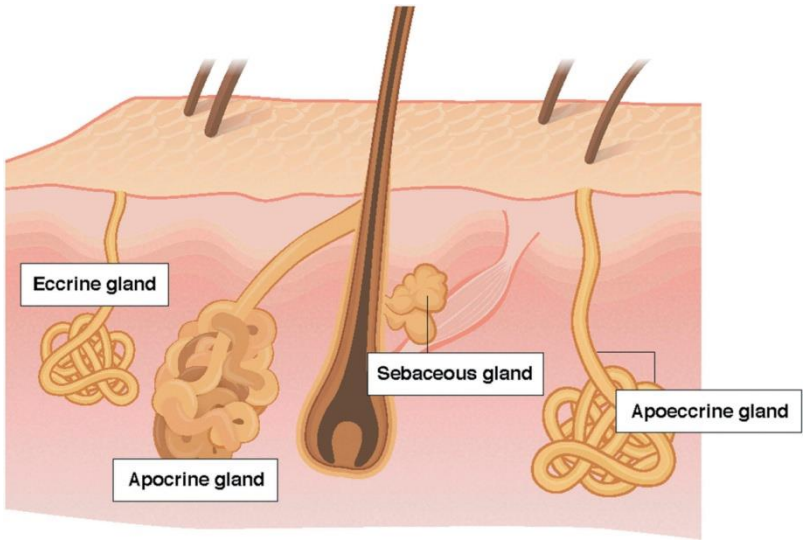

Figure 1. Comparison of the apocrine, eccrine, and apoeccrine glands in the axilla.

Apocrine sweat glands

The apocrine gland is a second type of sweat gland, which was first recognized by Krause in 1844 and later named by Schiefferdecker in 1922 [20,21]. Apocrine sweat glands are located primarily in the axilla, breasts, face, scalp, and the perineum [21,22]. As shown in Figure 1, these glands differ from eccrine glands in that they are larger and open into hair follicles instead of onto the skin surface [12]. In addition, although present from birth, the secretory function of apocrine glands does not begin until puberty [23]. Apocrine glands produce viscous, lipid-rich sweat, which is also comprised of proteins, sugars, and ammonia [21,23]. The function of apocrine glands in many species is generally regarded as scent glands involved in production of pheromones (body odor), although this social/sexual function is rudimentary in humans. Apocrine gland innervation is poorly understood, but isolated sweat glands have been found to respond equally to adrenergic and cholinergic stimuli [23].

Apoeccrine sweat glands

A third type of sweat gland, only recently described by Sato et al. in 1987 [23,24] is the apoeccrine gland. Apoeccrine glands develop from eccrine sweat glands between the ages of ~8 to 14 years and increase to as high as 45% of the total axillary glands by age 16−18 [23]. They are intermediate in size, but as the name suggests, apoeccrine glands share properties with both eccrine and apocrine glands. Like apoeccrine glands, apoeccrine glands are limited in distribution, as they are contained to only the axillary region. Apoeccrine glands are more similar to eccrine glands in that the distal duct connects to and empties sweat directly onto skin surface [23]. In addition, the apoeccrine gland produces copious salt water secretions similar to eccrine sweat [23]. The function of this secretion is unknown, but unlikely to play a significant role in thermoregulation since evaporation is inefficient in the axilla region. The innervation of the apocrine gland is still poorly understood, but in vitro models suggest the apocrine gland is more sensitive to cholinergic than adrenergic stimuli [23,24].

Sebaceous glands

Sebaceous glands are not a type of sweat gland but worth mentioning here since their secretions can impact the composition of sweat collected at the skin surface [25]. Sebaceous glands, first described by Eichorn in 1826 [26], are associated with hair follicles and present over much of the body surface but particularly the scalp, forehead, face, and anogenital area [26,27]. They are absent on the palms of hands and soles of the feet [26]. Sebaceous glands are holocrine glands that secrete a viscous, lipid-rich fluid consisting of triglycerides, wax esters, squalene, cholesterol, and cholesterol esters [25−27]. The rate of sebum production is related to the number and size of glands which is under hormonal (androgen) control [26]. The importance of sebaceous gland secretions is uncertain but sebum is thought to have antibacterial and antifungal properties and function as a pheromone [28].

Eccrine glands will be the focus of this review; therefore, unless otherwise specified, sweating rate and sweat composition will hereafter refer to that of the eccrine glands. The reader is referred to other papers for more details on apocrine and apoeccrine glands [12,20−24,27,29,30] as well as sebaceous glands [26−28].

Structure and function of eccrine sweat glands

Anatomy

The anatomical structure of the eccrine sweat gland, illustrated in Figure 2, consists of a secretory coil and duct made up of a simple tubular epithelium. The secretory tubule is continuous with and tightly coiled with the proximal duct. The distal segment of the duct is relatively straight and connects with the acrosyringium in the epidermis [5]. The secretory coil has three types of cells: clear, dark, and myoepithelial. As shown in Figure 2(c), clear cells are responsible for the secretion of primary sweat, which is nearly isotonic with blood plasma [6−8]. The clear cells contain a system of intercellular canaliculi, glycogen, and a large amount of mitochondria and Na-K-ATPase activity [5]. The dark cells are distinguishable by the abundance of dark cell granules in the

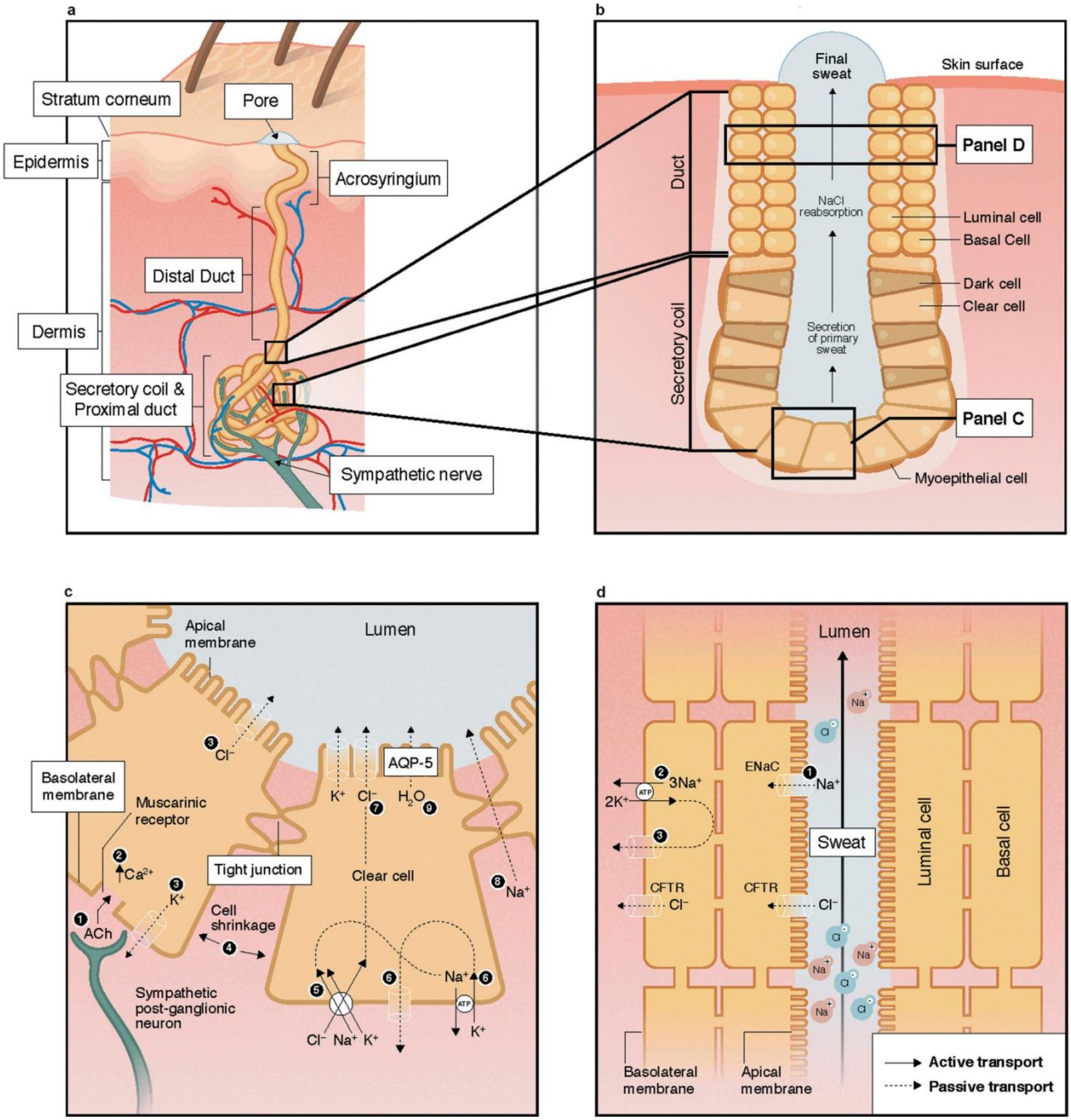

Figure 2. Structure of the eccrine sweat gland (panels A-B) and mechanisms of sweat secretion in the secretory coil (panel C) and Na and Cl reabsorption in the proximal duct (panel D). ACh; acetylcholine; AQP-5, aquaporin-5; CFTR, cystic fibrosis membrane channel; ENaC, epithelial Na channel; NaCl, sodium chloride.

cytoplasm. Their function is poorly understood, but thought to potentially act as a repository for various bioactive materials involved in regulation of clear cell and duct cell function [9,10]. The function of the myoepithelial cells is provision of structural support for the gland against the hydrostatic pressure generated during sweat production [5]. The duct has two cell layers: basal and luminal cells. Its primary function is reabsorption of Na and Cl ions as sweat flows through the duct, as shown in Figure 2(d). Most of the NaCl reabsorption occurs in the proximal duct, as these cells contain more mitochondria and Na-K-ATPase activity than that of the distal segment of the eccrine duct [5]. The result is a hypotonic final sweat excreted onto the skin surface [6,9].

Mechanisms of secretion and reabsorption

Secretion

The basic mechanism by which secretion of primary sweat occurs in the clear cells, according to the Na-K-2Cl cotransport model, is illustrated in Figure 2(c). First, binding of acetylcholine to muscarinic receptors on the basolateral membrane of the clear cell triggers a release of intracellular Ca stores and an influx of extracellular Ca into cytoplasm. This is followed by an efflux of KCl through Cl channels in the apical membrane and K channels in the basolateral membrane. This leads to cell shrinkage, which triggers an influx of Na, K, and Cl via Na-K-2Cl cotransporters on the basolateral membrane and subsequently Na and K efflux via Na-K-ATPase and K channels on basolateral membrane as well as Cl efflux via Cl channels on apical membrane. Increased Cl concentration in the lumen creates an electrochemical gradient for Na movement across the cell junction [9,10]. In turn, the net KCl efflux from the cell creates an osmotic gradient for water movement into the lumen via aquaporin-5 channels [31–33].

Ion reabsorption

Figure 2(d) shows the mechanism of ion reabsorption according to the modified Ussing leak-pump model. On the apical membrane of the luminal cells passive influx of Na occurs through amiloride-sensitive epithelial Na channels. Active transport of Na across the basolateral membrane of the basal cells occurs via Na-K-ATPase, which is accompanied by passive efflux of K through K channels on the basolateral membrane. The movement of Cl is largely passive via cystic fibrosis membrane channels (CFTR) on both the apical and basolateral membranes [9,34,35]. The two cell layers are thought to be coupled and behave like a syncytium. The sweat duct also reabsorbs bicarbonate, either directly or through hydrogen ion secretion, but the specific mechanism is unknown [5,8,36,37]. The activity of Na-K-ATPase is influenced by the hormonal control of aldosterone [38]. Overall the rate of Na, Cl, and bicarbonate reabsorption is also flow-dependent, such that higher sweating rates are associated with proportionally lower reabsorption rates resulting in higher final sweat electrolyte concentrations [39,40]. This concept will be covered in more detail in the *Effect of sweat flow rate* section below.

Sweat gland metabolism

Transport of Na across cellular membranes is an active process, thus sweat secretion in the clear cells and Na reabsorption in the duct require ATP. The main route of energy production for sweat gland activity is oxidative phosphorylation of plasma glucose [6,41]. Cellular glycogen is also mobilized in the eccrine sweat gland during sweat secretion, but its absolute amount is too limited to sustain sweat secretion. Thus, the sweat gland depends almost exclusively on exogenous substrates, especially glucose, as its fuel sources [6,42]. Although the sweat gland is capable of utilizing lactate and pyruvate as energy sources, these intermediates are less efficient than glucose [6,9]. Indeed, studies have shown that arterial occlusion of forearms [43,44] and removal of glucose and oxygen from the bathing medium of isolated sweat glands [6,45] inhibits sweat production. Consequently, lactate (as an end product of glycolysis) and NaCl concentrations in sweat rise sharply. Taken together, these results indicate that oxygen supply to the sweat gland is important for maintaining sweat secretion and ion reabsorption [45].

Control of eccrine sweating

Eccrine sweat glands primarily respond to thermal stimuli; particularly increased body core temperature [40], but skin temperature and associated increases in skin blood flow also play a role [9,46–49]. An increase in body temperature is sensed by central and skin thermoreceptors and this information is processed by the preoptic area of the hypothalamus to trigger the sudomotor response. Recent studies suggest that thermorecep- tors in the abdominal region [50,51] and muscles [52] also play a role in the control of sweating. Thermal sweating is mediated predominately by sympathetic cholinergic stimulation. Sweat production is stimulated through the release of acetylcholine from nonmyelinated class C sympathetic postganglionic fibers, which binds to muscarinic (subtype 3) receptors on the sweat gland (see

Figure 2(c)) [9]. Eccrine glands also secrete sweat in response to adrenergic stimulation, but to a much lesser extent than that of cholinergic stimulation [6,53]. Catecholamines, as well as other neuromodulators, such as vasoactive intestinal peptide, calcitonin gene-related peptide, and nitric oxide, have also been found to play minor roles in the neural stimulation of eccrine sweating [9,54,55]. In addition, eccrine sweat glands respond to non-thermal stimuli related to exercise and are thought to be mediated by feed-forward mechanisms related to central command, the exercise pressor reflex (muscle metabo- and mechanoreceptors), osmoreceptors, and possibly baroreceptors [55,56].

Sweating rate over the whole body is a product of the density of active sweat glands and the secretion rate per gland. Upon stimulation of sweating, the initial response is a rapid increase in sweat gland recruitment, followed by a more gradual increase in sweat secretion per gland [13,57–59]. Two important aspects of thermoregulatory sweating, depicted in Figure 3, are the onset (i.e. body core temperature threshold) and sensitivity (i.e. slope of the relation between sweating rate and the change in body core temperature) of the sweating response to hyperthermia [60]. Shifts in the sweating temperature threshold are thought to be central (hypothalamic) in origin, whereas changes in sensitivity are peripheral (at the level of sweat glands) [61].

Modifiers of eccrine sweating

Several intra- and interindividual factors can modify the control of sweating [60], some of which are shown in Figure 3. For example, the enhancement of sweating with heat acclimation [62–65] and aerobic training [66–69] has been associated with both an earlier onset and greater responsiveness of sweating in relation to body core temperature [64,70–75]. By contrast, dehydration has been shown to delay the sweating response [76,77], as hyperosmolality increases the body temperature threshold for sweating onset [78–81]. Hypovolemia may reduce sweating sensitivity [82], but this finding has not been consistent [79,83].

Other examples of host and external factors that modify regional and/or whole-body sweating are provided in Table 1. For example, older adults exhibit a lower sweat output per activated gland in response to a given pharmacological stimulus or passive heating compared with younger adults [84–86]. This decline in sweating occurs gradually throughout adulthood [85,87] and there are regional differences in the age-related decrement in sweat gland function [88–91]. However, the decline in sweating rate with aging has been primarily attributed to mechanisms related to a decline in aerobic fitness and heat acclimation (possibly due to decreased sensitivity of sweat glands to cholinergic stimulation [67,92]), rather than age per se [67,68,85,93–96]. In addition, lifetime ultraviolet

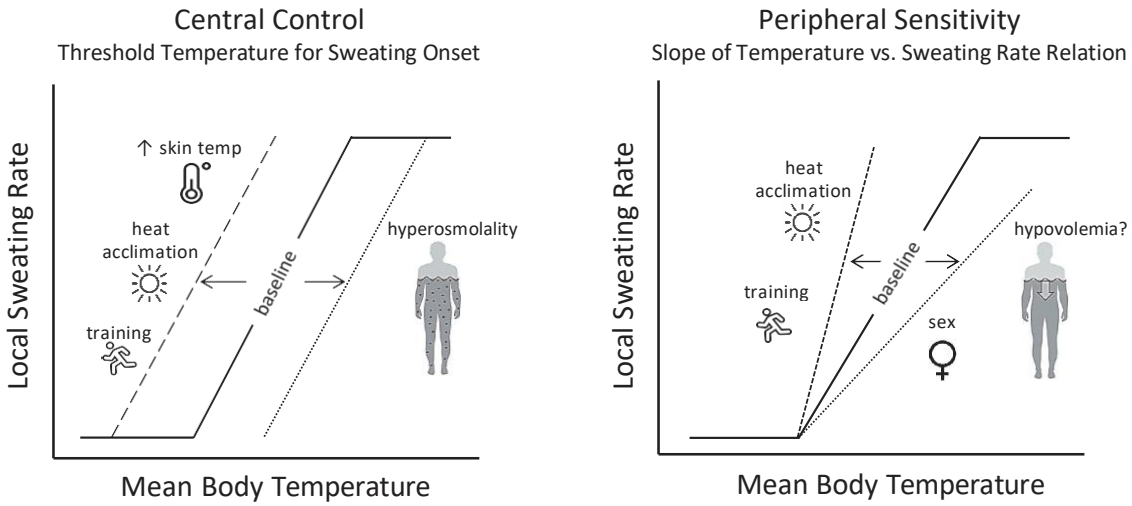

Figure 3. An illustration of central and peripheral control of sweating and the factors that modify the sweating response to hyperthermia. Shifts in the onset (threshold) and sensitivity (slope) of the sweating response to hyperthermia are depicted by the dashed lines. Other potential factors that may directly or indirectly modify sweating (altitude/hypoxia, microgravity, menstrual cycle, maturation, aging) are discussed in the text.

exposure and other environmental factors may have an interactive effect with chronological age in deter- mining sweat gland responsiveness [84]. Nonetheless, it is important to note that most studies have reported no significant difference in sweating between older and younger adults during exercise in the heat; with the exception of peak sweating rates associated with hot dry climates [94,97,98]. Therefore the ability of older adults to maintain body core temperature dur- ing heat stress is usually not compromised. When the effects of concurrent factors, such as fitness level, body composition, and chronic disease are removed, ther- mal tolerance appears to be minimally compromised by age [93].

It is often reported that men exhibit higher sweating rates than women; and several factors, some of which are independent effects of sex and others due to confounding physical characteristics, seem to contribute depending upon the study design. Men have a greater cholinergic responsive- ness (see Figure 3) and maximal sweating rate than women [83,99–101]. However, studies in which subjects were matched for body mass, surface area, and metabolic heat production, have shown that sex differences in whole-body sweat produc- tion are only evident above a certain combination of environmental conditions (e.g. 35–40°C, 12% rh) and rate of metabolic heat production (e.g. 300–500 W/m^2) leading to high evaporative requirements for heat balance [83,100–102]. Sweat gland density is generally higher in women than men (due in part to lower body surface area) [17,69,103]. Accordingly, the lower sweating rates by women reported in some studies were a result of lower output per gland [99,101,103]. Otherwise, higher whole-body sweating rates observed in men than women (e.g. in cross-sectional studies) can usually be attributed to higher body mass and metabolic heat production (higher absolute exer- cise intensities), rather than sex per se [104–109]. Taken together it seems that women are not at a thermoregulatory disadvantage compared with men for most activities and environmental condi- tions typically encountered [110,111]. As discussed in more detail elsewhere [109,110,112], other fac- tors such as body size, surface area-to-mass ratio, heat acclimation status, aerobic capacity, exercise intensity, and environmental conditions (all of which directly or indirectly impact the evaporative

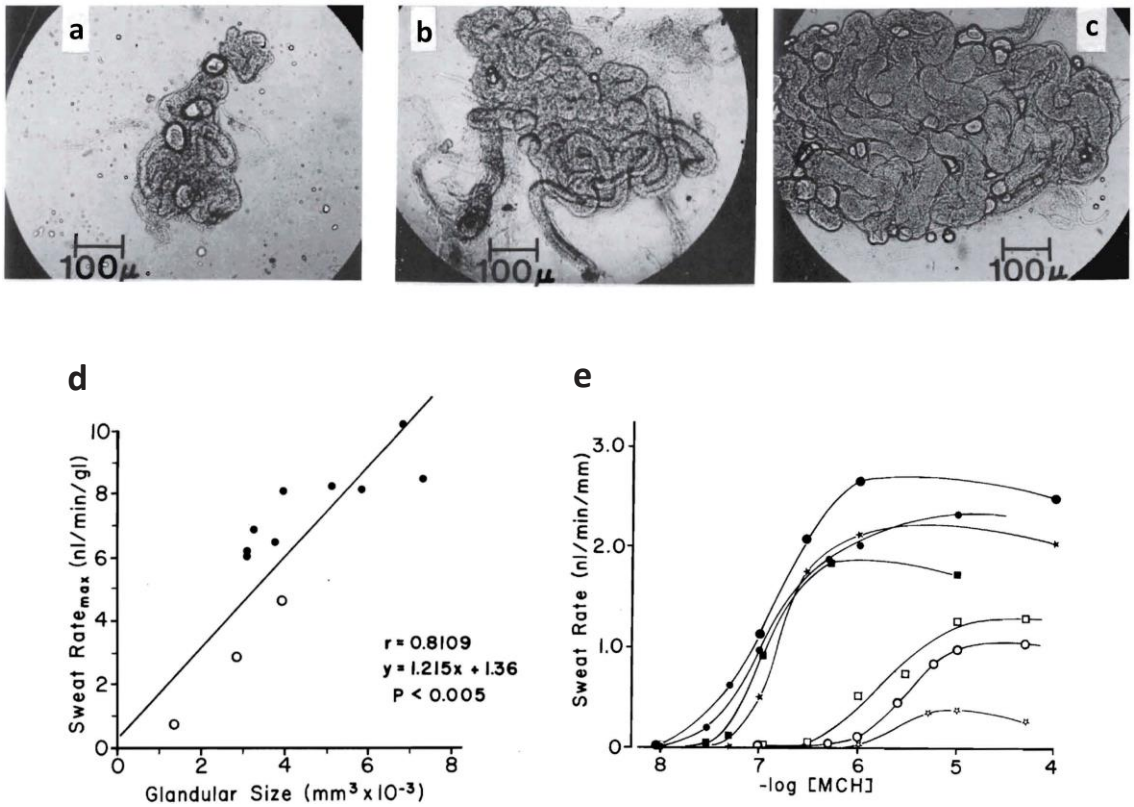

Figure 4. Top row (panels A-C): Variation in the size of human eccrine sweat glands taken from the backs of three different men who were described as poor (A), moderate (B), and heavy sweaters (C). Bottom row: Correlation between size of sweat gland and sweat rate$_{max}$ per gland (panel D). Dose-response curves (expressed per unit length of tubule) of sweat rates of 7 men to methacholine. Closed symbols show moderate to heavy sweaters. Open symbols show poor sweaters. Reprinted from Sato and Sato 1983 [131]with permission.

requirement for heat balance) are more important than sex in determining sudomotor responses to exercise-heat stress. The reader is referred to published reviews for more comprehensive discussions on the effects of sex and sex hormones on thermoregulation [110,113,114].

Additional factors, such as maturation [94,115–117], altitude/hypoxia [118–120], circadian rhythm [121,122], and menstrual cycle [122–125] have been shown to modify the onset and/or sensitivity of the sudomotor response (see Table 1). However, modifications in the onset and/or sensitivity of regional sweating in relation to body core temperature are not necessarily associated with significant differences in overall whole-body sweat losses during exercise. Two examples of this were noted above, with respect to the impact of sex and chronological age on sweating. Another example is the menstrual cycle: during the luteal phase regional sweating rate is lower at a given body core temperature (increased threshold and decreased slope) [122–125], but there are no differences in whole-body sweating rate across the menstrual cycle phases [123,126–129]. Additionally, for trained females their menstrual phase is of little physiological or performance consequence during exercise in the heat [103,130].

Some of the variability in sweating rate can be explained by differences in the structure of sweat glands. For example, with habitual activation, sweat glands show some plasticity in their size and neural/hormonal sensitivity [18,19]. Sato and colleagues have shown that glandular size (volume) can vary by as much as fivefold between individuals [9,131], and there is a significant positive correlation between the size of isolated sweat glands and their methacholine sensitivity and maximal secretory rate [131] (see Figure 4). Sweat gland hypertrophy and increased cholinergic sensitivity have been reported to occur with aerobic training [131] and heat acclimation [38] (see Table 1 for more information).

Eccrine sweat composition

Sweat is a very complex aqueous mixture of chemi- cals. Although sweat is mostly water and NaCl, it also contains a multitude of other solutes in varying concentrations [6,136–139]. Tables 3 and 4 list some of the micronutrients and non-micronutrients, respectively, present in sweat. This is obviously not an exhaustive list but includes some of the more commonly researched constituents. Tables 3 and 4 include the range in sweat constituent concentra- tions, mechanisms of secretion and reabsorption, and functional role in health, where known or applicable. Micronutrients include the electrolytes Na and Cl, which are the constituents found in the highest concentrations in sweat, as well as K, vita- mins, and trace minerals. Non-micronutrient ingre- dients listed in Table 4 include products of metabolism, proteins, amino acids, and toxicants. It is important to note that concentrations listed in these tables are approximate ranges and are not intended to reflect normal reference ranges. There are insufficient data, perhaps with the exception of Na, Cl, and K, to inform normative ranges for sweat constituents at this time. Instead the ranges listed are meant to provide some context in terms of relative order of magnitude of concentrations across all of the constituents, in order of higher (e.g. NaCl) to lower (e.g. trace minerals and heavy metals) concen- trations (in mmol/L). For some constituents, higher values outside the range listed have been reported, but are relatively rare, involve individuals with med- ical conditions, or may be inflated because of meth- odological issues; all of these points are discussed in more detail in later sections of this paper.

Because interstitial fluid is the precursor fluid for primary sweat, it follows that many components of final sweat originate from this fluid space. However, the exact mechanisms of secretion are largely unknown for most consti- tuents other than Na and Cl. Potential mechan- isms and supporting references are listed in Tables 3 and 4 and may include active or pas- sive (diffusion across membranes or paracellu- lar transport) mechanisms of transport. Some sweat constituents do not originate from the interstitial fluid, but instead, appear in sweat as a result of sweat gland metabolism (e.g. lac- tate) [140]. Yet others (e.g. antimicrobial pep- tides, proteolytic enzymes) are thought to be produced by the sweat gland and play a func- tional role in skin health (Table 4). It should be noted that many other chemicals (not in Tables 3 and 4), such as cortisol [141,142] neuropep- tides, bradykinin, cyclic AMP, angiotensins, and histamines [9,15] are also present in sweat. Some researchers have hypothesized that one or more of these ingredients may be biologi- cally functional, and involved in the regulation of sweat gland and/or ductal function; however, support for this notion is currently limited [9]. For a more comprehensive list of sweat consti- tuents, the reader is referred to other published reviews [6,134] and studies [139,143], including metabolomic analysis of sweat [136–138].

Abbreviations

ASGD activated sweat gland density
AVP arginine vasopressin
ATP adenosine triphosphate
BPA bisphenol-A
Ca calcium
CFTR cystic fibrosis transmembrane conductance regulator
Cl chloride
Cu copper
E_{req} evaporative requirement for heat balance
Fe iron
HCO_3 bicarbonate
K potassium
Mg magnesium
Mn mangenese
Na sodium
NH_3 ammonia
RSR regional sweating rate
SGO sweat gland output
WBSR whole body sweating rate
Zn zinc

Disclosure statement

The author is employed by the Gatorade Sports Science Institute, a division of PepsiCo, Inc. The views expressed in this article are those of the authors and do not necessarily reflect the position or policy of PepsiCo, Inc.

Funding

This work was supported by the Gatorade Sports Science Institute, a division of PepsiCo, Inc.

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