ARTICLE

Can We Protect Our Hearts by Sweating Out Excess Sodium?

Jonathan W. Hoch[1,2] *and Joseph C. Watso*[1,2,3]

[1]Cardiovascular and Applied Physiology Laboratory, Department of Health, Nutrition, and Food Sciences, Florida State University, Tallahassee, FL; [2]Institute of Sports Sciences and Medicine, Florida State University, Tallahassee, FL; and [3]Institute for Successful Longevity, Florida State University, Tallahassee, FL

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KEY POINTS

- Excess dietary sodium increases blood pressure responses during exercise.
- Sodium is primarily removed from the body through the urine, but can also be excreted from the body via sweating.
- Higher sodium intake is associated with higher sodium concentrations in sweat.
- Interventions that increase whole-body sweating (*e.g.*, sauna, hot tub, exercise in the heat, etc.) could offset the negative effects of excess dietary sodium on exercise blood pressure, but this has not yet been investigated.

INTRODUCTION

Blood pressure (BP) during exercise provides strong prognostic value, with abnormal responses being associated with a higher risk of adverse health outcomes. Therefore, it is important to understand how modifiable factors affect exercise BP. In 1989, individuals with exaggerated exercise BP were encouraged to perform aerobic exercise training, lose weight, restrict alcohol, and restrict sodium intake before participating in sports (1). However, this concern is not limited to people participating in sports; exaggerated BP responses are highly prevalent in clinical populations (*e.g.*, chronic kidney disease, hypertension), where sodium regulation is already impaired. Current evidence supports that sodium restriction can provide protective effects by reducing BP (2) while also attenuating the exercise pressor reflex (EPR) (3). However, nine in 10 adults overconsume dietary sodium, necessitating alternative approaches to mitigate this underappreciated adverse effect of high dietary sodium. Physical activity reduces salt sensitivity through established cardiovascular adaptations (4). However, the potential contribution of exercise-induced sodium losses via increased sweating to BP regulation remains unexplored. Therefore, we critically examined evidence on whether sweating-inducing interventions could attenuate salt sensitivity of exercise BP. We hypothesize that sweating-inducing activities (*e.g.*, sauna) may attenuate salt sensitivity of exercise BP in part by providing an alternative pathway for sodium excretion, potentially impacting future cardiovascular disease (CVD) risk by reducing chronic cardiovascular strain experienced during physical activity (Fig. 1, Supplemental Digital Content 1, https://links.lww.com/ESSR/A77) (5–8).

AUTONOMIC REGULATION OF EXERCISE BLOOD PRESSURE

During exercise, perfusion pressure is elevated through a sophisticated convergence of neural and humoral mechanisms to meet the oxygen demands of active skeletal muscle.

Address for correspondence: Joseph C. Watso, Ph.D., 120 Convocation Way, Tallahassee, FL 32306 (E-mail: jwatso@fsu.edu).
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This regulation is driven by central command from the motor cortex (9), feedback from arterial, cardiopulmonary baroreflexes (9), chemoreflexes (9), and the EPR driven by skeletal muscle afferents (9). Collectively, these inputs modulate autonomic regulation of blood flow and pressure (9) as necessary to meet metabolic demands, via alterations in heart rate, cardiac contractility, and vascular resistance. Other reviews provide extended discussions of EPR mechanisms (10,11).

There is strong rationale for submaximal exercise BP as a clinically relevant risk factor (Supplemental Digital Content 1, https://links.lww.com/ESSR/A77). For example, a meta-analysis of 46,314 adults found that exaggerated submaximal exercise BP responses were associated with a higher risk for adverse cardiovascular events and mortality, independent of resting BP and other cardiovascular risk factors (10). Exaggerated systolic BP (SBP) during maximal or peak dynamic exercise predicts dose-dependent increased future hypertension risk in most (12) studies. Thus, it is important to identify the modifiable lifestyle factors (*e.g.*, dietary composition) that contribute to these exaggerated responses. Exaggerated BP does not increase CVD risk at higher workloads (13), but a higher SBP/metabolic equivalent of task (MET) slope is associated with an increased mortality risk and demonstrates superior predictive value compared with SBP alone (14). Evidence suggests that sodium accumulation augments sympathetic outflow and sensitizes skeletal muscle afferents to both mechanical and metabolic stimuli (6,7,15). This autonomic sensitization is particularly pronounced during submaximal exercise (Fig. 2) (6), where BP reactivity appears more sensitive to dietary sodium shifts than during maximal exertion.

SODIUM INTAKE AND CARDIOVASCULAR HEALTH

The 2026 U.S. Department of Agriculture recommends consuming no more than 2300 mg of sodium per day (16), and the American Heart Association advises no more than 1500 mg of sodium daily for optimal cardiovascular health (17). However, nine in 10 American adults exceed these guidelines, with an average intake of 3550 mg/d (2). Excess dietary sodium impairs endothelial function, augments autonomic cardiovascular reactivity, and impairs renal function (2). These effects contribute to an increased cardiovascular

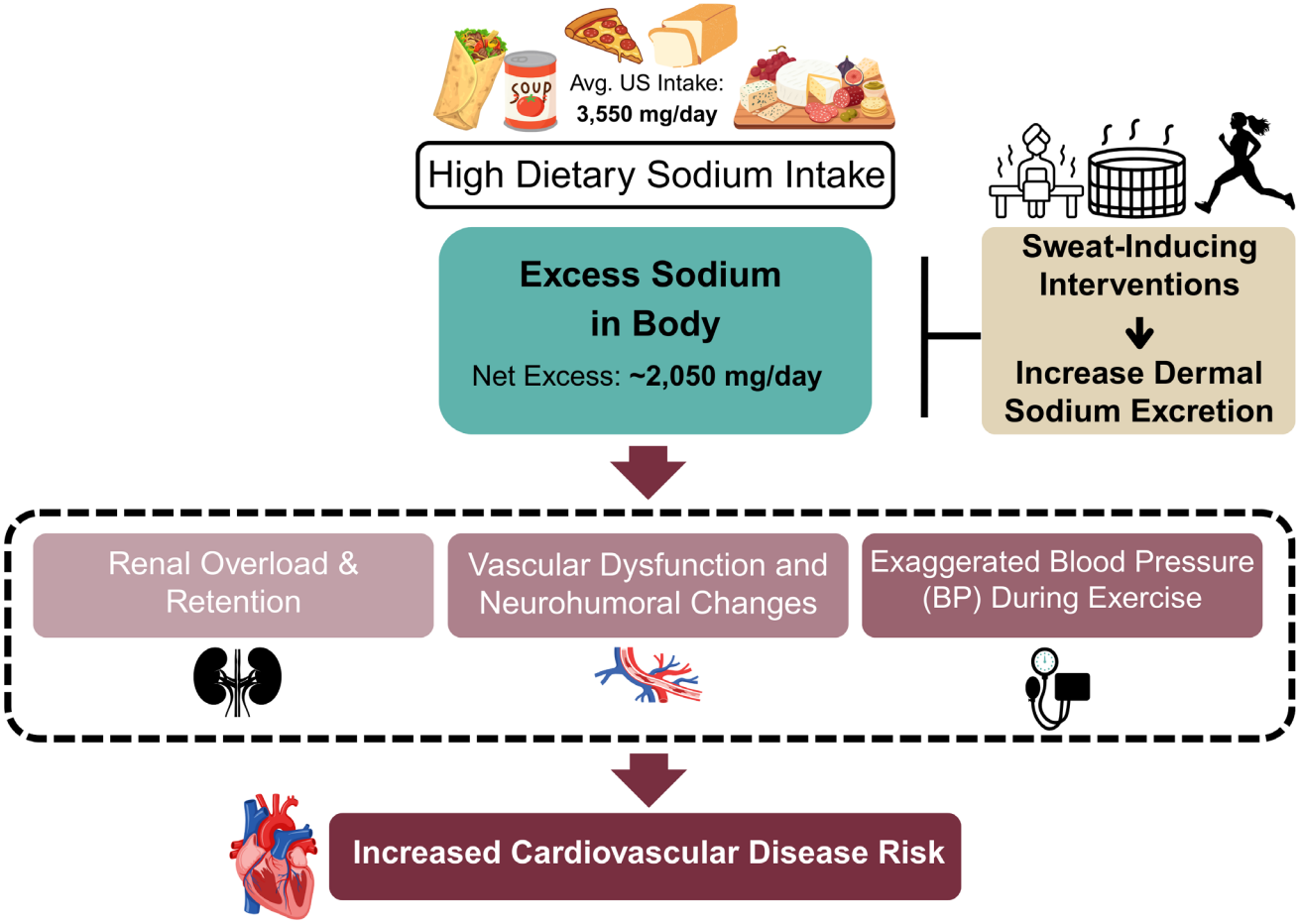

Figure 1. Conceptual figure. Chronic high sodium intake increases whole-body sodium content, with the skin serving as a primary site of storage. Excess sodium elicits vascular dysfunction (even independent of resting blood pressure changes) and alters neurohumoral regulation of blood volume and pressure (*e.g.*, renal stress) such that the sympathetic nervous system is more reactive to various stimuli. These and other effects culminate in exaggerated BP responses during exercise, which is notable as higher blood pressure responses during exercise are linked with a greater future risk for morbidity and mortality. Our novel hypothesis is simply that interventions increasing sweating can remove meaningful amounts of sodium from the body and serve as a countermeasure to mitigate at least some of the deleterious effects of high dietary sodium.

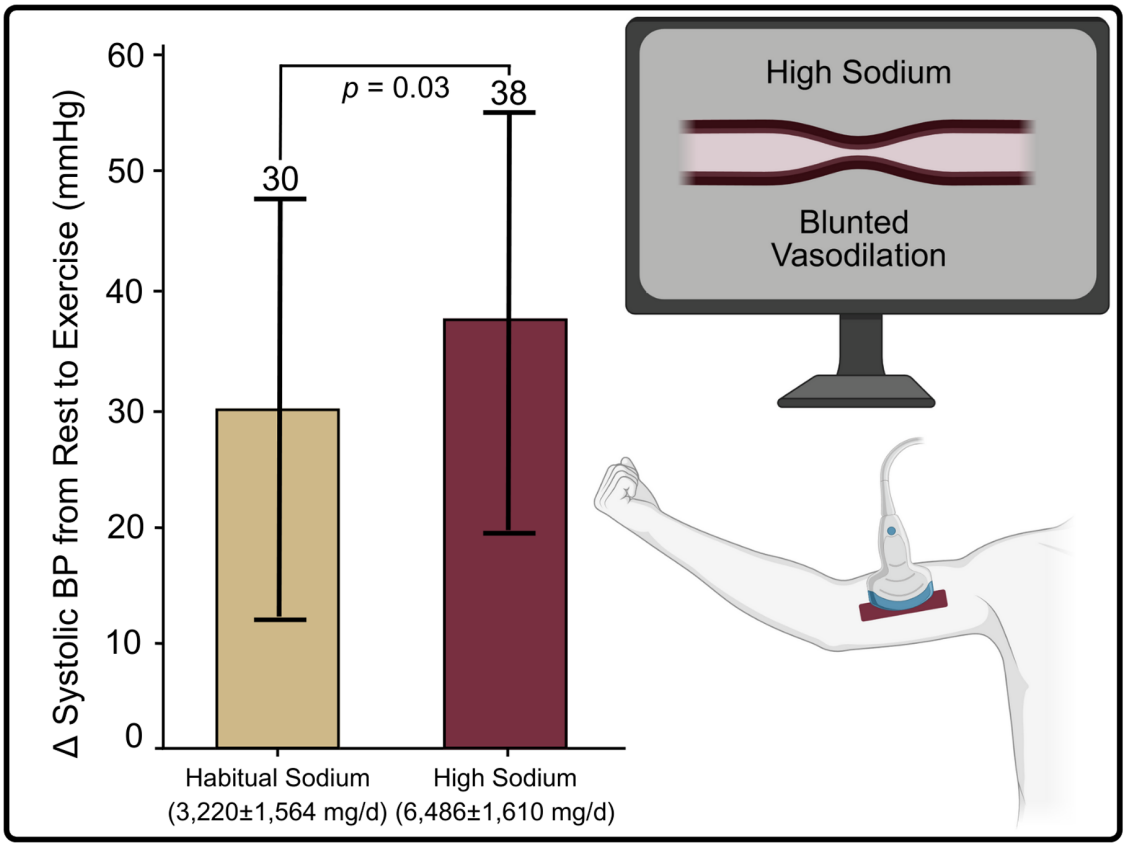

Figure 2. Dietary sodium and exercise blood pressure (BP) reactivity. Data are derived from Babcock *et al*. (6) comparing the change in systolic blood pressure (ΔSBP) from rest to steady-state submaximal cycling (60% $\dot{V}O_{2peak}$), after 10 d of low versus high dietary salt (urinary sodium excretion: 3220 ± 1564 vs 6486 ± 1610 mg·d^{-1}, $P < 0.01$). High salt intake exaggerated the pressor response compared with the low salt condition (ΔSBP: 38 ± 16 vs 30 ± 16 mm Hg, $P = 0.03$). **Inset:** High sodium intake was associated with endothelial dysfunction (assessed as blunted flow-mediated dilation [FMD]), as the augmentation in exercising SBP significantly correlated ($r = –0.71$, $P = 0.002$) with reductions in FMD.

strain at rest (2), but a less recognized consequence is an exaggerated BP response (the impaired ability of the vasculature to dilate during high-flow states) during exercise and heat stress. Such exaggerated responses have been observed during both isometric (7) and dynamic (6) exercise, contributing to increased CVD risk. This salt-sensitive exercise hypertension (SSEH) represents an additional layer of individual variability in exercise BP research.

We have shown that 10 d of a high-sodium diet (6486 ± 1610 mg/d) augmented systolic BP by 8 mm Hg during submaximal aerobic exercise when compared with a habitual-sodium (3220 ± 1564 mg/d) condition (Fig. 2) (6). Furthermore, acute elevations in serum sodium and plasma osmolality increase sympathetic outflow and mean arterial pressure during exercise. This salt-induced increase in cardiovascular reactivity represents a modifiable driver of cardiovascular risk that persists even in healthy individuals (7).

In occupational populations, dietary factors—including sodium intake — explain 24% of adverse ambulatory BP responses to maximal physical exertion in firefighters (18), highlighting the real-world significance of dietary sodium in exercise BP responses. Chronic fluid overload (linked to sodium accumulation) is common, affecting nearly half of new dialysis patients, and is associated with left ventricular hypertrophy, arrhythmias, pulmonary edema, and hospitalizations (19). Current evidence suggests inflammatory pathways may also contribute to SSEH. Short-term high-salt consumption increases circulating monocyte chemoattractant protein-1, an inflammatory biomarker, and correlates with salt-induced increases in mean BP (20), providing a potential mechanistic link between salt intake, inflammation, and exercise BP regulation.

While chronic salt intake is common, acute sodium ingestion has immediate cardiovascular consequences. A single high-salt meal rapidly impairs vascular function by reducing nitric oxide bioavailability, primarily through increased oxidative stress and the uncoupling of endothelial nitric oxide synthase (2). This dysfunction is functionally evident as a significant reduction in flow-mediated dilation), an effect that can manifest within 60 min of ingestion (21). Notably, this impairment occurs even in the absence of hypertension; flow-mediated dilation is significantly reduced in both salt-sensitive and salt-resistant normotensive adults, suggesting that excess salt exerts a direct deleterious effect on the vasculature that is independent of an individual's BP salt sensitivity (22).

This impairment extends to the smallest blood vessels, reducing microvascular vasodilation independent of changes in resting BP (23). While the vascular dysfunction is immediate, its impact on the EPR appears to be cumulative; the augmentation of exercising BP becomes more pronounced

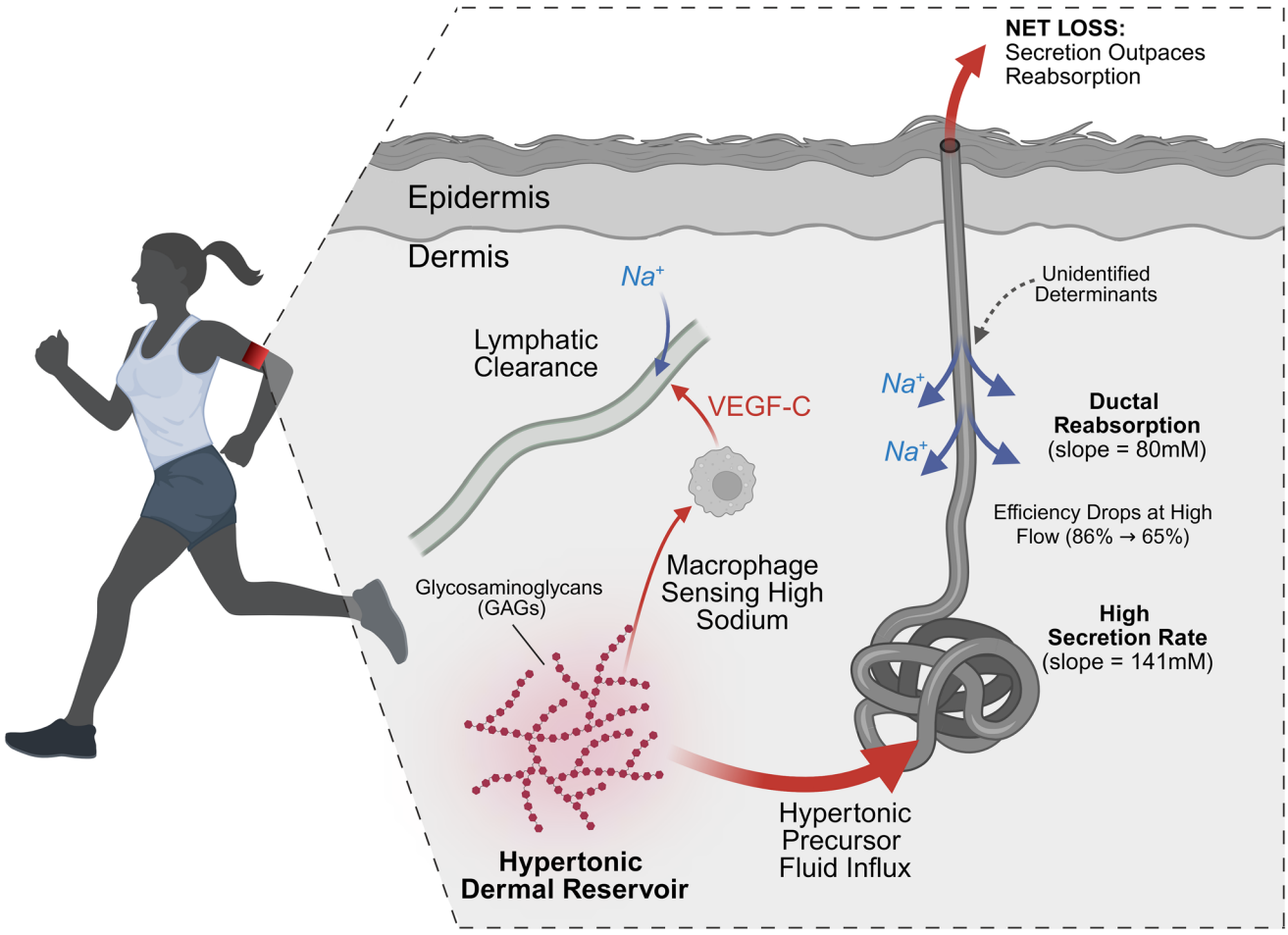

Figure 3. Mechanistic overview of the "third compartment" and sweat sodium kinetics. The eccrine sweat gland secretory coil utilizes this interstitial fluid as its precursor; thus, elevated dermal sodium directly increases the tonicity of the precursor fluid influx. Final sweat composition is dictated by the balance of secretion and reabsorption rates. Both rates increase with sweat flow, but the Na^+ secretion rate (slope = 141 mM) increases faster than the ductal reabsorption capacity (slope = 80 mM). Consequently, reabsorption efficiency declines at high-flow rates (from 86% to 65%), leading to a progressive increase in net sodium loss. The dashed gray arrow indicates that known factors (energy expenditure, sex, season) account for only ~17–23% of interindividual variation, suggesting significant determinants of sweat sodium concentration remain unidentified.

after several days of high salt intake (6,24). This repeated, acute insult to the endothelium contributes to the underlying inflammatory pathways and vascular dysfunction that is thought to drive the progressive development of hypertension over time.

Sodium Excretion

The kidneys are the primary regulators of sodium homeostasis, but modern high-salt diets place them under relentless pressure to excrete excess salt — a burden that contributes significantly to SSEH. While the kidneys regulate circulating sodium, considerable amounts can be retained in the body's largest organ, the skin, which serves as a major hypertonic salt reservoir (2). In this case, sodium is stored nonosmotically in the dermis, bound to glycosaminoglycans (Fig. 3). Because precursor sweat is derived from this local environment, dermal excretion through sweating is a powerful complementary pathway to secrete sodium, potentially offloading the kidneys. This process allows for substantial sodium removal, independent of renal function, by secreting a primary sweat that is isotonic with plasma (approximately 135–145 mM sodium, 3105–3335 $mg \cdot l^{-1}$), which then undergoes aldosterone-controlled sodium reabsorption to produce a final hypotonic sweat (25). The amount of sodium ultimately lost is substantial, with athletes losing up to ~3800 mg in a single session, and it is directly related to sweat rate, as higher rates overwhelm the duct's reabsorption capacity (25,26).

Even short-term shifts in sodium intake (*e.g.*, 10 d of salt loading) increase the urinary excretion of neutrophil gelatinase-associated lipocalin, an established biomarker of kidney tubular injury (27). This renal stress is further evidenced by a marked increase in creatinine clearance (*e.g.*, from 110 to 145 $ml \cdot min^{-1}$), indicating a state of glomerular hyperfiltration that occurs independently of changes in resting BP (27).

Chronic heat stress, whether from exercise or passive exposure, triggers profound cellular and molecular adaptations within the eccrine sweat gland. Structurally, the glands can undergo hypertrophy (an increase in size), which

is correlated with enhanced cholinergic sensitivity and a greater maximal sweat rate (25,28). This increased sensitivity leads to an earlier onset and greater responsiveness of sweating for a given core temperature (29). Simultaneously, the sweat duct becomes more efficient at sodium conservation. This is driven by an increased sensitivity to the hormone aldosterone, which upregulates the activity of Na^+/K^+–ATPase pumps on the ductal cell membrane (29,30). These pumps actively reabsorb sodium from sweat back into the extracellular fluid, resulting in a lower sodium concentration in sweat for any given sweat rate. Furthermore, heat stress induces the production of cytoprotective heat shock proteins, which likely allow for more rapid re-acclimation after a period of inactivity (31,32). Collectively, these adaptations create a sweating system that is both more powerful and more efficient, allowing for higher sustainable sweat rates during thermal stress, ultimately enabling a greater total volume of sodium to be removed from the body. However, chronic high sodium intake prevents the body from triggering the molecular adaptations of the eccrine gland (30). By maintaining high plasma sodium and suppressing aldosterone (2), high salt intake effectively signals to the eccrine glands that conservation mechanisms (*e.g.*, increased transporter density) are unnecessary, thereby blunting the adaptive decline in sweat sodium concentration typically observed with heat acclimation (28).

One retrospective analysis of 48 individuals identified energy expenditure as the primary statistical driver of sweat sodium concentration. The physiological capacity to offload sodium remains clinically robust because high sweat rates overwhelm ductal reabsorption — a kinetic limit (secretion slope = 141 vs reabsorption slope = 80) that is further exploited when chronic high salt intake, blunting normal aldosterone-mediated conservation mechanisms (33).

One study in 38 adults found that high salt intake increased sweat sodium concentration by suppressing aldosterone, which reduces the efficiency of epithelial sodium channels in the sweat duct (34). But a later study in nine male adults found no difference in sweat sodium after 9 d of low versus high salt (35). This discrepancy suggests that high sweat flow rates may overwhelm the duct's reabsorptive capacity regardless of hormonal signals, and points toward potential individual variation in how sweat sodium responds to dietary changes. Ultimately, regardless of background diet, sweating remains a robust physiological mechanism to eliminate excess salt.

Sweat-Inducing Interventions

A single session's power to increase sodium excretion is an underappreciated therapeutic tool. These interventions can be categorized as active (exercise-based) or passive (environmental heat exposure). Both methods are effective at stimulating sweat production while also inducing important chronic adaptations that enhance the body's capacity for dermal sodium excretion. The intensity of exercise-based exposures can be measured in metabolic equivalents of task (METs), where 1 MET is the energy expended at rest.

Active heat stress

Exercise training, particularly in the heat (*e.g.*, METs ≥3 and temperatures ≥30°C), is a potent stimulus for enhancing sweat efficiency and sodium conservation. Sweat rates typically range from 0.5 to 2.0 $l \cdot h^{-1}$ during voluntary exercise and moderate thermal stress (25,36), with final sweat sodium concentrations ranging from 20 to 80 mM (460–1840 $mg \cdot l^{-1}$), depending on individual factors and heat acclimation status (25,36). For a moderately trained individual with a sweat rate of 1.2 $l \cdot h^{-1}$ (typical value) and a whole-body sweat sodium concentration of 36 mM (typical value; 828 $mg \cdot l^{-1}$), sodium losses would approximate 22 mM (506 mg) in 30 min or 43 mM (1012 mg) in 60 min.

In an occupational context, average sodium losses over a single work shift in the heat can range from 4800 to 6000 mg (37). In a recreational setting, a single 90-min hot yoga session can result in an average sweat loss of 1.54 l, corresponding to a remarkable 2700 mg of sodium (38). This is because the high temperature (~40°C) and humidity (~40%) of the environment reduce the water vapor pressure gradient between the skin and the air, severely impairing evaporative cooling. To compensate, the body must produce a higher sweat rate to achieve thermoregulation, which in turn overwhelms the capacity for ductal sodium reabsorption, leading to greater net sodium loss (25,26,38)

With repeated heat exposure (*e.g.*, 10 d of exercise-heat acclimation), physiological adaptations enhance sweat efficiency. Sweat sodium concentration decreases by approximately 34%, significant reductions in calcium (~29%), and magnesium (~43%) (39). While this adaptation allows for electrolyte conservation on a per-liter basis, it is accompanied by a dramatic increase in the overall sweat rate. Crucially, while the sodium concentration per liter decreases, the total volume of sweat produced increases dramatically (often doubling). Therefore, the net effect is a substantial total sodium loss, as the increase in sweat volume far outweighs the decrease in its concentration. Notably, this adaptation for sodium conservation is more pronounced after active heat acclimation compared with passive heating, likely due to a more robust aldosterone response during exercise (32).

Passive heat stress

Passive heating interventions using sauna (dry: 70°C–90°C <10% relative humidity or wet (*i.e.*, steam room): 40°C–50°C, ~100% relative humidity), or water (*e.g.*, hot water immersion [HWI], 40°C), can induce thermoregulatory adaptations comparable to those achieved through exercise (28). While the functional outcomes are similar, the underlying stimuli may differ, with passive methods relying more on peripheral adaptations to high skin temperature, in contrast to the core temperature-driven adaptations of exercise (32).

Sauna bathing is a reliable method for inducing significant fluid loss; a dry sauna session, for example, can produce double the body mass loss of a wet sauna (0.72 vs 0.36 kg) (40). In a similar protocol, sedentary men with overweight lost 0.65 kg of body mass during a 60-min Finnish (dry) sauna session (41). While effective, the composition of sweat from

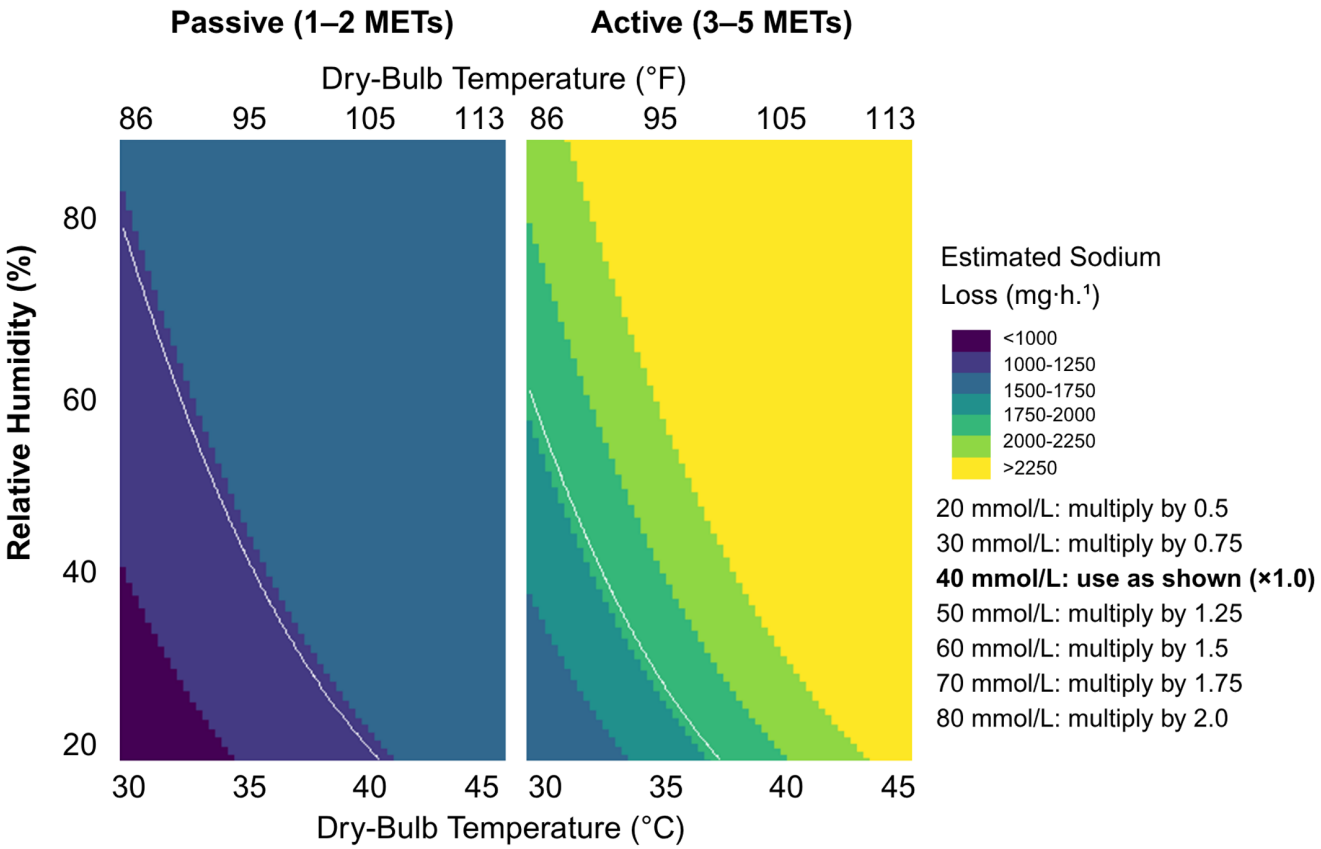

Figure 4. Conceptual heatmaps comparing sweat sodium loss from passive heating versus moderate-intensity exercise. The passive panel is modeled on a sweat rate of 1.0–1.5 l·h^{-1} (44), reflecting empirical data from traditional sauna and hot water immersion studies. The active panel, modeled on a sweat rate of 1.5–2.5 l·h^{-1} (44,45), illustrates the even greater potential for sweat loss when metabolic heat from exercise is added. This comparison highlights that intense passive heating is a potent stimulus for sodium excretion, which is further amplified by moderate-intensity exercise. The contour line on each panel denotes the relevant Wet Bulb Globe Temperature (WBGT) risk threshold for that activity level (45,46). The passive panel indicates the 28°C (82.4°F) threshold for light activity, while the active panel displays the 26°C (78.8°F) threshold, marking the point at which heat stress becomes a greater concern for moderate exercise. All calculations assume a mid-range sweat sodium concentration of 40 mM (~920 mg·l^{-1}).

passive heating may differ slightly from exercise-induced sweat, with sauna bathing producing higher concentrations of sweat magnesium and calcium (42). For instance, combining a typical dry sauna sweat rate of 0.72 kg·h^{-1} with a sweat sodium concentration of ~61 mM (1403 mg·1^{-1}) would result in a sodium loss of approximately 1000 mg·h^{-1}.

Notably, passive sweating shows direct therapeutic promise. In a clinical trial with patients experiencing congestion from heart failure, a median hourly weight loss of 0.22 kg·h^{-1} was safely induced by gently heating the skin (43). This demonstrates that enhancing sweat rate through passive means is a feasible strategy to facilitate a clinically meaningful removal of both fluid (via osmolality changes) and sodium (*e.g.*, ≥1000 mg per session), underscoring its potential as an adjunct therapy. This demonstrates the primary advantage of passive interventions: offering a lower-demand therapeutic option for clinical or non-ambulatory populations that still elicits powerful cardiovascular and thermoregulatory adaptations (31,44).

These interventions provide an additive cardiovascular benefit beyond dermal sodium excretion; passive heat therapies (sauna, HWI) independently demonstrate lowering resting BP and improving endothelial function, while habitual physical activity directly protects against BP's sensitivity to dietary salt (4,31,44). To visualize the quantifiable impact of these interventions, Figure 4 illustrates the estimated hourly sodium loss for both active and passive heat stress across a range of environmental temperatures and humidity levels.

While some modalities like HWI may be perceived as more uncomfortable for inexperienced users, passive heat therapies are generally less mechanically and metabolically demanding than exercise. By minimizing central (*i.e.*, motor) command and skeletal muscle afferent feedback, these modalities offer a valuable alternative for clinical or non-ambulatory populations (31,44) who may lack the functional capacity for physical exertion yet can tolerate thermal cardiovascular conditioning. The cultural integration and widespread accessibility of thermal therapies support their feasibility for long-term lifestyle integration. In the United States, the accessibility of these modalities varies by setting: hot tubs are widely prevalent in residential communities (*e.g.*, apartment complexes), hospitality, and home settings, whereas saunas and steam rooms are commonly accessible through commercial fitness centers and health clubs. Conversely, warm-water therapy pools are typically restricted to clinical or rehabilitation facilities. Despite these distinct access points, the

collective availability of these venues supports the feasibility of long-term lifestyle integration. This is underscored by strong epidemiological data associating frequent sauna use (>3 times ·wk−1) with significant reductions in cardiovascular mortality, suggesting that adherence to such protocols is both achievable and highly beneficial (31).

Practical Application and Proposed Uses

The evidence above supports the strategic use of sweat-inducing interventions as a practical tool to help manage total body sodium. The optimal method for amplifying the sweat stimulus depends on the modality. For active interventions like exercise, where significant metabolic heat is already being generated, increasing the ambient humidity is a powerful way to increase the required sweat rate. This effect is highlighted in hot yoga studies, where the humid environment impairs evaporative cooling, resulting in a profuse sweat rate that leads to an exceptionally high sweat sodium concentration of ~82 mM (1886 $mg \cdot L^{-1}$), compared with the 40–50 mM (920–1150 $mg \cdot L^{-1}$) often observed in drier conditions (38). Conversely, for passive interventions, increasing the absolute dry temperature appears to be the most effective driver. A hot and dry sauna session produced double the sweat loss of a less-hot, wet sauna (0.72 vs 0.36 kg) (40).

The goal of inducing sweat is not to create a deficit that requires aggressive replacement with high-salt electrolyte supplements, but rather to help normalize total body sodium toward a healthier baseline. For context, the "normal ancestral level" of sodium intake is defined as less than 1000 mg/d, an amount consistent with diets in many hunter-gatherer societies where hypertension remains rare (47). This is clinically meaningful, as the average U.S. adult consumes 3550 mg of sodium daily, resulting in a surplus of over 1200 mg relative to the Food and Drug Administration's upper limit and over 2000 mg relative to the American Heart Association's ideal limit of 1500 mg. Therefore, a single intervention that removes 1000 mg of sodium can offset a significant portion of this daily excess, helping to align an individual's net sodium balance more closely with a healthier ancestral target. For rehydration, the evidence suggests that for most individuals, simply salting food to taste is sufficient to restore the necessary electrolytes without negating the excretory benefit of the intervention (48).

The sensitivity of thirst responses provides an important safety mechanism during sweat-based interventions. Thirst is highly sensitive to increases in plasma sodium concentration and osmolality, requiring only a 2–3% increase to induce feelings of thirst (24). This physiological safeguard helps prevent excessive dehydration during controlled thermal interventions. Systematic reviews of thirst responses to plasma osmolality changes identify significant individual variation, with studies including participants ranging from healthy controls to those with diabetes insipidus, diabetes mellitus, and chronic kidney disease (49). This heterogeneity suggests that personalized approaches to thermal interventions may be necessary for optimal effectiveness and safety.

Potential Sources of Interindividual Variation

The efficacy and safety of sweat-based interventions are influenced by individual factors, including sex and physical training status.

While men often exhibit higher whole-body sweat rates, these differences are largely attributed to their greater body mass and higher metabolic heat production, and tend to disappear when individuals are matched for body size and fitness (25,28). A more significant factor is hormonal variation in females; during the luteal phase of the menstrual cycle, core temperature is regulated at a higher set point, which increases the threshold for initiating both sweating and vasodilation (24,32). This suggests that while total sodium excretion potential may be similar, the timing and intensity of an intervention may need to be personalized.

Additionally, trained individuals display a more robust sweating response, characterized by an earlier onset and a higher sweat rate (25,28). While the effect of training on sweat sodium concentration is inconclusive, any observed increase is likely due to the higher sweat *rate* overwhelming the capacity for sodium reabsorption, not a decrease in efficiency (30). Consequently, trained individuals may possess a greater capacity for total sodium excretion in a given session. Therefore, while sex and training status influence the dynamics of sweating, they do not negate the therapeutic potential; rather, they influence the intensity and duration required to achieve the target sodium loss similar to dietary sodium intake.

Safety Considerations

The convergence of physiological efficacy with behavioral feasibility positions sweat-inducing interventions as a promising adjunct therapy for SSEH. The evidence suggests minimal risk of compensatory salt-seeking behaviors or caloric overconsumption, supporting further investigation of structured thermal therapy protocols. However, implementation should incorporate systematic monitoring of individual responses and electrolyte balance to optimize safety and effectiveness across diverse populations.

Safe implementation necessitates careful participant screening and systematic monitoring. Protocols should exclude individuals with unstable cardiovascular, metabolic, or renal disease, and those on specific medications like blood thinners (32,38). A critical first step is confirming baseline euhydration, typically via a urine specific gravity measurement of ≤1.025 (32,44). During interventions, key variables for monitoring physiological strain include continuous core temperature, heart rate, and BP, supplemented by pre- and post-session nude body mass to quantify total sweat loss (28,44).

For healthy, acclimated individuals, sweat-inducing interventions are generally safe when appropriate hydration and cooling strategies are adopted (28). However, they induce significant cardiovascular strain and are not without risk for individuals with pre-existing conditions. In patients with heart failure, the thermal load of a sauna or HWI session is equivalent to a moderate physical load of 60–100 W cycling,

which can precipitate high-output heart failure in severe cases (28,41). Furthermore, individuals with unstable coronary artery disease may be at risk for myocardial ischemia (31). These risks are compounded by common medications; diuretics can exacerbate dehydration, while beta-blockers can impair the body's primary cooling mechanism of cutaneous vasodilation (28,50). However, for stable clinical populations limited by orthopedic or other constraints (*e.g.*, obesity), passive heat therapy may serve as a beneficial "modified stimulus." In these groups, it allows for clinically meaningful sodium excretion without the mechanical stress of dynamic exercise, provided that physiological strain is monitored.

A primary challenge is overcoming "voluntary dehydration," as individuals drinking to thirst often replace only a fraction of fluid losses (38). This issue is compounded by the fact that many individuals begin exercise already in a state of mild dehydration, potentially experiencing symptoms like headaches (26,51). Restoring the extracellular fluid lost through sweat does require sodium (24). This has led to the widespread use of (largely unnecessary) high-salt electrolyte supplements (*e.g.*, LMNT, WIRED, Liquid IV, etc.). This can create a misleading feedback loop: an individual who starts dehydrated may consume a high-sodium product, experience relief as the excess sodium drives fluid retention, and incorrectly attribute the benefit to a high-sodium need, rather than simply the correction of their dehydration (28,48). The evidence suggests this is often unnecessary; for most individuals, simply salting food to taste ("season to taste") provides sufficient sodium to facilitate effective rehydration and restore fluid balance after an intervention (48).

KNOWLEDGE GAPS AND FUTURE RESEARCH DIRECTIONS

Age-Related Variations in Sweat Response and Cardiovascular Strain

Aging significantly impairs dermal sodium excretion capacity through multiple mechanisms that collectively increase cardiovascular strain during thermal stress. The age-related decline in sweating rate is attributed primarily to sweat glands' decreased sensitivity to cholinergic stimulation—a factor strongly linked to reduced aerobic fitness—rather than aging per se, with older individuals (>60 yr) demonstrating attenuated cutaneous vasodilatory capacity, less effective sweat responses, and decreased thermoreceptor sensitivity compared with younger adults (25,28). These age-related impairments in thermoregulatory function, combined with reduced total body water, impaired plasma vasopressin regulation, and higher thirst sensation thresholds, create a compounding effect that limits the effectiveness of dermal sodium excretion as a regulatory mechanism and increases the risk of developing hyperthermia and cardiovascular complications during exercise in older populations (28). This presents a critical clinical paradox: While the risks of thermal stress are elevated, the potential benefit of using dermal excretion to offload the kidneys, whose function also declines with age, is substantial. Future research is therefore needed to establish safe protocols for this population. Consequently, while older adults stand to benefit significantly from renal offloading, their reduced thermoregulatory capacity requires that sweating interventions be less aggressive and more carefully monitored than in younger cohorts.

Individual Salt Sensitivity

A more fundamental knowledge gap is the direct relation between an individual's vascular salt sensitivity and their capacity for dermal sodium excretion. While aldosterone is known to influence both vascular function and sweat gland sodium reabsorption, no studies explicitly link an individual's BP response to dietary salt with their dermal sodium handling (30). Viewing this through the classic Guytonian framework, salt sensitivity is driven by a shifted pressure–natriuresis curve, where the kidneys fail to excrete daily sodium loads at normal BPs. This state is often characterized by volume expansion and a compensatory suppression of the renin–angiotensin–aldosterone system. Because the eccrine sweat duct shares functional homology with the renal distal tubule — specifically relying on aldosterone to drive sodium reabsorption — this systemic suppression of the renin–angiotensin–aldosterone system could theoretically reduce ductal reabsorption efficiency. Future research is needed to determine if salt-sensitive individuals possess a distinct dermal sodium handling profile and to clarify the metabolic consequences of the skin's role as a sodium reservoir (19).

Management of Exercise Hypertension

Furthermore, while thermal therapies have been shown to lower resting BP, their long-term efficacy for specifically managing exercise hypertension remains to be established. Prospective studies are required to confirm whether regular interventions can chronically attenuate the exaggerated BP responses to exercise. Such research is critically needed in diverse and high-risk populations — including individuals with obesity, diabetes, or chronic kidney disease — who are underrepresented in heat therapy studies and often take medications like diuretics or angiotensin-converting enzyme inhibitors that alter thermoregulation and fluid balance (44,50).

Optimal Protocols

Finally, optimal protocols for combining thermal therapy with dietary sodium restriction are unknown (44). More work is needed to understand the mixed results regarding dietary salt intake on sweat sodium concentration and to determine if strategies like permissive dehydration could enhance the adaptive response (28,30). Bridging the gap between short-term physiological studies and long-term mechanistic research is crucial for defining the most effective and sustainable protocols for clinical use.

CONCLUSIONS

This review has synthesized the evidence for a complementary, non-nephrocentric approach: the use of sweat-inducing interventions to facilitate dermal sodium excretion.

This pathway is powerful, quantifiable, and highly modifiable, offering a unique advantage as it operates independently of renal function or diuretic efficacy (43). By stimulating the body's largest organ, these interventions allow for the direct manipulation of the "third compartment" — sodium stored nonosmotically in the skin — targeting a major part of the sodium and water imbalance (19). This strategy represents a potential shift in the treatment of fluid overload and salt sensitivity. By leveraging the skin as a major excretory organ, thermal therapies hold significant promise as a future therapeutic approach to mitigate the cardiovascular consequences of excess dietary sodium.

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