

1 **Review article**

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3 **Hot-water immersion: a (not so) new therapy for the primary and secondary prevention of**
4 **hypertension?**

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33 **Abstract**

34 Hypertension affects over 30% of adults worldwide, significantly increasing the risk of
35 cardiovascular, cerebrovascular, and renal diseases. While regular physical activity is a well-
36 established strategy for lowering blood pressure, additional therapeutic approaches may
37 help individuals who struggle to achieve target blood pressure levels. Hot-water immersion
38 is garnering attention due to its potential cardiovascular benefits. Historically practiced for
39 therapeutic and cultural purposes, hot-water immersion induces physiological responses
40 that share key similarities with physical activity. Accumulating evidence suggests that hot-
41 water immersion may contribute to blood pressure reduction. However, while small-scale
42 studies report promising acute and chronic blood pressure lowering effects, critical gaps
43 remain in the literature. This review summarises current evidence on the anti-hypertensive
44 effects of hot-water immersion, outlining key areas for future research. Hot-water
45 immersion may emerge as an accessible and culturally relevant adjunct therapy for
46 hypertension management.

47

48 **Introduction**

49 Hypertension affects more than 30% of adults, increasing the risk of heart, brain, and kidney
50 diseases (1). Globally, the number of adults living with hypertension has doubled since 1990,
51 now exceeding 1.2 billion, and projections suggest this figure will continue to climb (2).
52 Defined by the American Heart Association as a consistent elevation in blood pressure with
53 systolic values ≥ 130 mm Hg and/or diastolic values ≥ 80 mm Hg, hypertension becomes
54 increasingly prevalent with advancing age, affecting roughly half of adults aged 65 years and
55 older (3). A 10 mm Hg reduction in systolic blood pressure is associated with a 20% relative
56 risk reduction in major cardiovascular events and almost half the risk of stroke (4, 5).
57 International guidelines recommend regular physical activity for the primary and secondary
58 prevention of hypertension. Unfortunately, due to a number of factors (e.g., neighbourhood
59 built environment, lack of time, lack of enjoyment) (6, 7), these guidelines are not being met
60 by at least a quarter of the global adult population (8, 9). With hypertension prevalence
61 predicted to increase further, declining physical activity rates must be reversed, and *adjunct*
62 therapies are necessary to mitigate future burden on healthcare systems.

64 **Hot-water immersion**

65 One such strategy is hot-water immersion; a form of passive heat therapy that has been
66 rejuvenated in recent research due to its health benefits. Hot-water immersion or bathing
67 has been used for centuries as a form of healing (10-12), with deeply rooted cultural and
68 social significance in many parts of the world. For example, communal bathing traditions
69 such as the Japanese onsen and sento, Turkish and Roman baths, and geothermal bathing
70 among the New Zealand Māori and Nordic populations have long been associated with
71 physical restoration, mental wellbeing, and social connection. Epidemiological data from
72 Japan have shown that frequent bathing is associated with a reduced risk of developing
73 hypertension and ~28% lower cardiovascular disease risk (13, 14). Data on the accessibility
74 of bathing facilities, particularly bathtubs, are limited. However, bathing practices are
75 common worldwide and occur in various forms in the home or communal bathing facilities,
76 including in low/middle-income regions (15). Thus, while the feasibility of hot-water
77 immersion may vary by infrastructure and socioeconomic context, the general practice of
78 bathing is culturally widespread and versatile.

79

Hot-water immersion is characterised as bathing in water temperatures of ~39-40 °C, with thermoneutral or sham exposures considered 33-37 °C. The benefits of the hot water (compared to thermoneutral) appear to come from the consequential rise in body core temperature and the physiological cascade that ensues (16). In fact, there are many similarities in this response to that of an acute exercise bout. In 1909, Hill and Flack (17) first reported that 15-30 min of hot-water immersion (40.5-43 °C) elicited concurrent increases in rectal temperature (up to 40 °C), heart rate (up to 160 bpm), and ventilation (up to 50 L·min⁻¹), along with a decrease in systolic blood pressure to as low as 60 mm Hg. As with exercise, core temperature rises during acute hot-water immersion, prompting cardiovascular and thermoregulatory responses designed to restore thermostasis. Cardiac output can double to support increased skin blood flow, elevating shear stress on the vascular endothelium (16, 18). This is driven partly by a sympathetically mediated increase in heart rate. Shifts in blood volume to the periphery (i.e., skin for thermoregulation) and ultrafiltration to the interstitial fluid, along with sweating-induced fluid losses from all compartments, including plasma, would normally reduce venous return and thus stroke volume. However, unique to water immersion is the hydrostatic force exerted by water, aiding venous return and preserving stroke volume (18). The rise in core and muscle temperatures *may* mediate other pleiotropic effects such as skeletal muscle adaptations (e.g., increased mitochondrial density, capillarisation) and haematological changes (e.g., plasma and blood volume expansion); these likely contribute to previously reported increases in sub-maximal and maximal cardiorespiratory fitness (19, 20), but may also be involved in any hypotensive benefits of hot-water immersion (detailed below).

Aim of this review

This narrative review is not proposing that heat therapy replace pharmaceuticals or be used as an alternative to regular physical activity. Based on current data and understanding of likely mechanisms, there are both similarities and differences in how hot-water immersion and physical activity mediate any anti-hypertensive effects. Importantly, hot-water immersion may complement and potentially enhance the therapeutic effect of anti-hypertensive medication (21). While direct comparative data on adherence to hot-water immersion versus physical activity are limited, emerging evidence suggests that it is

perceived as enjoyable and relaxing (22, 23). Where intervention attendance data has been reported, adherence is high (>90%; (20, 24, 25)), albeit within supervised research settings.

Despite these promising indications, previous reviews have thus far examined passive heat therapies collectively (i.e., Finnish sauna, Waon therapy, water-perfused suit, and hot-water immersion), and their broader cardiovascular effects, but with limited focus on blood pressure specifically. Although cohort studies indicate cardiovascular and mortality benefits of Finnish sauna bathing (26), the evidence base remains dominated by epidemiological data from a single dataset (Kuopio Ischemic Heart Disease Risk Factor Study). Randomized trials are scarce, and the only repeated-exposure trial of Finnish sauna showed no effect on resting blood pressure over 8 weeks (27). In contrast, multiple studies report hypotensive effects following repeated hot-water immersion (i.e., > 5 sessions; Table 1). Therefore, this review examines evidence that hot-water immersion elicits hypotensive benefits beyond those attributable to heating alone, likely resulting from the additive influence of hydrostatic pressure, and explores current literature on its adaptive effects and the physiological mechanisms underpinning these responses, while highlighting opportunities for future research.

Relevant literature was identified through a comprehensive search of PubMed, Scopus, and Web of Science databases up to October 2025, using combinations of terms including *hot-water immersion*, *passive heat therapy*, *blood pressure*, *hypertension*, and *vascular function*. Reference lists of key articles and reviews were also screened for additional studies. Emphasis was placed on original research involving adult human participants that examined blood pressure outcomes, mechanisms of action, or comparisons with exercise or pharmacological interventions. For intervention studies in Table 1, inclusion was limited to those involving repeated exposures (≥ 5 sessions) using hot-water immersion at temperatures $\geq 39^\circ\text{C}$.

Hypotensive effects of hot-water immersion – an overview

A recent meta-analysis reported a modest reduction in systolic blood pressure of approximately 2 mm Hg following bathing interventions, based on only four included studies (28). However, this analysis did not capture the full extent of the hot-water immersion

literature, as more than ten intervention studies have explored the adaptive effects of hot-water immersion on blood pressure (Table 1). During and immediately following immersion, increases in vascular shear stress, peripheral vasodilation, increased skin blood flow, and reductions in systemic vascular resistance contribute to transient decreases in arterial pressure. Beyond these vascular responses, acute immersion also evokes marked autonomic, renal, and hormonal adjustments that may underpin longer-term hypotensive adaptations. Repeated exposures may further enhance nitric oxide bioavailability, antioxidant defences, and vagal tone while reducing oxidative stress, contributing to sustained improvements in vascular function and resting blood pressure. Many of these acute responses have been described in detail by Crandall and Wilson (16) and Romero et al. (29). We highlight in Figure 1, and discuss throughout this review, the acute responses and compensatory adaptations that we believe may collectively contribute to the sustained reductions in resting blood pressure associated with repeated hot-water immersion.

To our knowledge, the first intervention study to investigate the adaptive effects of hot-water immersion was an 8-week home-based intervention in young and healthy individuals (30). The authors reported that despite five, 10-min baths per week and achieving mean heart rates during immersion of ~130 bpm, resting blood pressure was unchanged. In the first study in a clinical population, a two-week intervention (five, 10-min sessions/wk) in patients with heart failure reduced resting mean arterial pressure by 4 mm Hg (85 to 81 mm Hg), but not in the control group (shower only; 95 to 93 mm Hg) (31). Since then, hypotensive effects have been reported across interventions ranging from 1-12 weeks and in sedentary or overweight individuals (32, 33), patients with hypertension (34), peripheral arterial disease (24), type 2 diabetes mellitus (35), polycystic ovarian syndrome (35), heart failure (36, 37) and osteoarthritis (21). Akerman et al., (24) reported a greater reduction in systolic and diastolic blood pressure, than supervised exercise in patients with peripheral artery disease. More recent work reported systolic and diastolic blood pressure decreased by 9 mm Hg and 4 mm Hg across twelve weeks of regular hot-water immersion in patients with lower-limb osteoarthritis (21). To put the magnitude of this reduction in context, Cochrane reviews report similar reductions in systolic (8-9 mm Hg) and diastolic blood pressure (4-6 mm Hg) with maximum dosages of common anti-hypertensive pharmaceuticals (e.g., ace inhibitors (38) and angiotensin receptor blockers (39)). Although

the reported reduction by Roxburgh et al. (21) was larger than the home-based exercise comparator group, it was not different to that of high-intensity interval training. It should be noted that the hot-water immersion interventions utilised by Akerman et al. (24) and Roxburgh et al. (21) concluded each session with light callisthenic exercise, which may have also had complementary hypotensive effects.

Although these adaptive hypotensive responses appear promising, blood pressure was not the primary outcome in these studies, and none utilised the gold-standard assessment of blood pressure (i.e., 24-h ambulatory monitoring). The only study in which blood pressure was the primary outcome *and* assessed using ambulatory monitoring reported that 8–10 weeks of hot-water immersion was not effective for reducing ambulatory blood pressure in adults with untreated hypertension (25); curiously, the comparator, aerobic exercise training did not reduce ambulatory blood pressure either. Further adequately powered studies with these methodological considerations are necessary to determine if previous studies that have shown benefit were biased by less robust blood pressure assessment, or if there is a potential synergistic relationship in people who are also pharmaceutically treated for hypertension (21).

Mechanisms

Vascular function

Contributory mechanisms appear complex, particularly when trying to delineate apparent modulators (e.g., water temperature, immersion depth, immersion duration). Upon immersion in hot water, blood flow is redistributed in part due to the hydrostatic effect displacing fluid from the extremities, as well as to support convective and conductive cooling. Specifically, for every 30 cm of immersion depth, the pressure exerted on the body increases by ~22 mm Hg (40); the net effect is an approximate 700 mL increase in central blood volume with head out whole-body hot-water or thermoneutral immersion (41). This acute blood flow effect increases shear stress on the endothelium, a potent stimulus for endothelial nitric oxide synthase. The ensuing increase in nitric oxide bioavailability promotes vasodilation and supports vascular function and health (42). Immediately following acute hot-water immersion exposure, antegrade shear increases to a similar degree as acute exercise; however, recovery from hot-water immersion produces a greater

reduction in retrograde shear, than exercise does (43, 44). Since retrograde shear is considered proatherogenic (45), this reduction may promote favourable long-term adaptations in endothelial function and a more anti-atherogenic vascular profile. Such differences in shear stress profiles may contribute to different haemodynamic outcomes of hot-water interventions (compared to exercise), but this remains speculative.

To our knowledge, no study has investigated the effects of repeated hot-water immersion on vascular smooth muscle function. A single 40 °C immersion, compared with thermoneutral immersion at 36 °C, augments both endothelium-dependent (acetylcholine-mediated) and endothelium-independent (sodium nitroprusside-mediated) vasodilation (46), implying that sufficient thermal stress can acutely enhance vascular smooth muscle responsiveness. Ayme et al. (47) also reported that a single 60-min thermoneutral (34 °C) water exposure did not differ from ambient air in its effects on vascular reactivity (assessed via nitroglycerin-mediated dilation), suggesting temperature may be a key driver.

Intervention studies assessing changes in endothelial function have reported favourable, though variable results. Bailey et al. (20) and Carter et al. (48) reported modest improvements in flow-mediated dilation (1.7 and 2.5% respectively) in healthy female and healthy adult cohorts, across eight weeks. Epidemiological data suggest that each 1% increase in FMD is associated with roughly a 10–13% reduction in future cardiovascular events (49). By contrast, Brunt et al., reported a 5.3% increase in flow-mediated dilation in sedentary participants across eight weeks (32), a change greater than that typically reported following exercise interventions (50); the larger effect may relate to the higher total “dose” of heat exposure in their study (54 h vs. 12 h per participant).

In clinical populations, repeated hot-water immersion may elicit central hemodynamic adaptations without improving endothelial function. James et al. (51) conducted a relatively short-term (8–10 sessions over 14 days) intervention in individuals with type 2 diabetes and observed reductions in systolic blood pressure and resting heart rate, but no improvement in macro- or microvascular endothelial function. Similarly, Akerman et al. (24), reported reductions in both systolic and diastolic blood pressure following twelve weeks of repeated hot-water immersion, despite no reliable change in flow-mediated dilation. Given the

participants' underlying peripheral arterial disease, it is likely their vessels are "unadaptable", potentially masking endothelial benefits. Collectively, these findings suggest that while repeated hot-water immersion can induce clinically meaningful reductions in blood pressure and beneficial central cardiovascular adaptations, the evidence for adaptive enhancement of endothelial function in clinical populations remains limited, and may not be a requisite mechanism underlying the hypotensive effects.

Angiogenesis

Hot-water immersion also stimulates the release of angiogenic factors, likely driven by increased tissue temperature and vascular shear stress. Notably, vascular endothelial growth factor (VEGF) has been shown to increase by approximately 60% across twelve weeks of hot-water immersion (24). VEGF promotes endothelial nitric oxide synthase activation and exerts potent hypotensive effects in animal models (52-54). In humans, inhibition of VEGF is associated with marked increases in systolic (~12 mm Hg) and diastolic blood pressure (~10 mm Hg) (55), likely due to decreased nitric oxide bioavailability and increased peripheral resistance. Interestingly, Brunt and colleagues observed that serum from young healthy adults following 8 wk of hot-water immersion stimulated endothelial cell angiogenesis in vitro, despite no concurrent increase in VEGF (56). The authors hypothesised that other pro-angiogenic factors such as basic fibroblast growth factor and transforming growth factor may stimulate endothelial nitric oxide synthase-dependent angiogenesis, independently of VEGF. Nevertheless, these findings suggest that circulating, heat-induced factors mediate nitric oxide-dependent angiogenic signalling in endothelial cells.

Hormonal and fluid-regulatory effects

Central fluid shifts during immersion stimulate a cascade of neurohormonal responses that modulate blood pressure, vascular resistance, and renal sodium handling. One proposed mechanism involves the stimulation of atrial natriuretic peptide (ANP) release, triggered by increased venous return and right atrial stretch during immersion (47, 57). ANP has both vasodilatory and natriuretic effects and also suppresses key fluid-regulating hormones such as renin, aldosterone and vasopressin, while increasing prostaglandin E (58). This response

has been extensively described with thermoneutral-water immersion (59), but its role in hot-water immersion remains less clear.

In a study using 38 °C water, aldosterone decreased slightly during prolonged immersion (~4 h), but increased markedly after immersion (60). More recently, Kissling et al. (61), reported that ANP levels tended to be higher following thermoneutral compared to hot-water immersion. Additionally, aldosterone increased following acute hot-water immersion exposure, but decreased slightly with thermoneutral-water immersion. After five days of repeated exposure, aldosterone levels remained higher following hot-water immersion, compared to thermoneutral-water immersion. This pattern suggests enhanced aldosterone responsiveness with repeated heat exposures and aligns with post-exercise hypotension (62, 63), where plasma volume supercompensates following repeated exercise exposures. Consistent with this adaptation, albumin concentration remained stable during repeated hot-water immersion, while plasma volume–adjusted albumin increased over five days, indicating a rise in total albumin content proportional to plasma volume expansion (61). This suggests enhanced intravascular protein retention or synthesis with hot-water immersion, supporting the osmotic drive necessary to maintain the expanded plasma volume characteristic of heat acclimation.

Autonomic mechanisms

At rest, expanded plasma volume increases stroke volume via the Frank-Starling mechanism, leading to a compensatory decrease in resting heart rate and improved autonomic balance (64, 65). Repeated hot-water immersion has also been associated with reductions in muscle sympathetic nerve activity (MSNA), aligning with improvements in endothelial function and resting blood pressure (35). Although speculative, one potential mechanism is an altered baroreflex function. Notably, studies have shown divergent outcomes regarding baroreflex adaptations, with both improved baroreflex sensitivity (35), and unchanged sensitivity described, the latter with a resetting of the baroreflex operating point to lower MSNA and heart rate (66). These discrepancies may arise from differences in subject populations, heating protocols, or baroreflex assessment methods.

Oxidative and inflammatory mechanisms

Passive heating via hot-water immersion reduces oxidative stress and enhances antioxidant defences, both acutely and chronically (67, 68). A single 60-min session that raises core temperature above 38.5°C increases serum antioxidant capacity, upregulates haemeoxygenase-1 (HO-1), and attenuates superoxide generation in endothelial cells, partly through suppression of nuclear factor-kappa-B (NF-κB) activation (67). Over longer interventions (4-8 weeks), repeated hot-water immersion induces adaptive antioxidant responses, including sustained elevations of HO-1 and heat shock proteins (HSPs) (67).

Recent human work in a muscle-injury model also supports these molecular pathways. Dablainville et al. (69) demonstrated that daily lower-limb hot-water immersion elevated HSP27 and HSP70 expression, reduced NF-κB activity, and increased the anti-inflammatory cytokine interleukin-10, whereas cold- or thermoneutral-water immersion did not. While this study targeted skeletal muscle recovery, the observed rapid shift toward an anti-inflammatory profile and preservation of cellular function align with proposed vascular mechanisms and may partly explain blood pressure reductions seen with repeated heat exposure. Specifically, these reductions in oxidative stress and pro-inflammatory markers enhance endothelial nitric oxide bioavailability and vasodilation, contributing to lower peripheral resistance and blood pressure.

Given the multifactorial nature of these responses, some extending beyond the scope of this review, rigorous mechanistic studies are needed to clarify the pathways through which hot-water immersion confers any hypotensive effects.

Dose response

For hot-water immersion to become a credible non-pharmaceutical therapy for the primary and secondary prevention of hypertension, more dose-response data are required. Specifically, very little is known about how manipulating the “training” principles of hot-water immersion (i.e., frequency, intensity, time, type and immersion depth) influence adaptive responses. Acutely, increased duration appears to increase the hypotensive response *during* exposure; 20-min immersion in chest depth 40°C water decreased mean arterial pressure by 13 mm Hg, and an additional 10-min immersion decreased it further by

another 6 mm Hg in older adults with osteoarthritis (21). Importantly, repeated exposures across 12 weeks do not appear to diminish this acute response. Despite this apparent dose-response relationship during immersion, a recent study reported that both 20- and 40-min hot-water immersions produced a similar reduction in 24-h ambulatory systolic blood pressure (6-7 mm Hg) in people with hypertension, compared to no immersion (70). Interestingly, a similar 24-h reduction was observed following both 40-min thermoneutral (36.5°C) and hot-water immersion (40°C), suggesting that duration and temperature may not be the only determinants of benefit.

Adaptive responses are less consistent. For example, more intensive interventions (i.e., greater volume) have not necessarily produced larger or more reliable reductions in blood pressure. Hartinah et al. (34) reported as few as five, 15-min sessions were effective for reducing resting systolic (14 mm Hg) and diastolic blood pressure (9 mm Hg) in people with hypertension. At the opposite end of the dosage spectrum, Brunt et al. (32), examined the effect of thirty-six, 60-90 min sessions of hot-versus temperate-water immersion in young sedentary adults. Despite the intensive exposure, they observed only small non-statistically significant reductions in systolic blood pressure (4 mm Hg vs. 3 mm Hg in the sham condition). Ely et al. (35) employed a similar protocol in women with obesity and polycystic ovary syndrome and showed a 5 and 2 mm Hg reduction in systolic and diastolic blood pressure after 4-5 wk, then a further 5 and 4 mm Hg reduction after 8-10 wk. It is difficult to fully understand why similar “doses” elicited such divergent responses in blood pressure in the two latter studies. These inconsistencies likely reflect interindividual differences in physiology and health status, as well as variations in baseline autonomic or vascular function. Thus, acute and adaptive dose-responses likely differ across a variety of patient populations, exposing another area of necessary research to provide greater insight.

Is the dose response consistent across different groups?

The previous section raises key questions: are blood pressure responses to hot-water immersion in young, fit or healthy individuals similar to those in older, less fit or clinical populations? From exercise intervention literature, patients with hypertension have greater reductions in resting blood pressure with aerobic exercise training, compared to people who

are normotensive (71), so the same may apply with immersion. However, the data are limited.

Acute hypotensive effects have been documented in patients with hypertension (70, 72), peripheral arterial disease (73), but no acute effect is seen in patients with type 2 diabetes (74, 75). After repeated exposure, Kaiser et al. (25) reported no effect on resting blood pressure in people with untreated hypertension (8-10 wk). In contrast, a sub-analysis of a trial involving older adults with osteoarthritis, those with hypertension (taking at least one anti-hypertensive medication) experienced substantial reductions in resting systolic and diastolic blood pressure (14 mm Hg and 5 mm Hg, respectively) following 12 weeks of hot-water immersion (21). The individuals without hypertension showed no change (~0 mm Hg). A third intervention study in people pharmaceutically treated for hypertension showed a similar reduction in systolic blood pressure with as little as five sessions of ankle deep immersion (34). From a mechanistic perspective, Coruzzi et al. (76) reported that during thermoneutral immersion, people with hypertension experience greater natriuresis and larger acute reductions in systolic blood pressure, compared to normotensive individuals. Collectively, these variable findings highlight the need to reconcile differences across study designs, participant characteristics, and treatment status, to better define when and for whom hot-water immersion may exert meaningful blood pressure benefits.

A major challenge moving forward is delineating the effects of aging and declining cardiorespiratory fitness on these responses. Both factors reduce cardiac baroreflex sensitivity (77, 78), and play a critical role in regulating cardiac output and systemic vascular resistance during perturbations in blood pressure. It is possible that younger or fitter individuals retain a more responsive baroreflex, enabling them to better buffer the fluid shifts and decreased peripheral resistance that occur during immersion. Supporting this, healthy older adults (~70 y) have larger reductions in systolic blood pressure than younger adults (20-24 y) following 8-min (39 °C water; 26 vs. 8 mm Hg) and 15-min (41 °C water; 30 vs. 10 mm Hg) chest-deep immersions (79, 80). Nagasawa and colleagues reported divergent heart rate variability profiles in older (75 y) and younger (27 y) men during 10 min of shoulder deep hot-water immersion (81). Specifically, younger adults showed a decrease in parasympathetic activity (indicated by a decrease in the high frequency (HF) component

of heart rate variability), while older adults did not. In addition, the low-frequency/high-frequency (LF/HF) ratio decreased in older adults. Collectively these *acute* responses may explain some of the limited hypotensive benefit with repeated exposures in young adults (30, 66, 82), but the more pronounced and sustained reductions in healthy older adults (83).

With respect to cardiorespiratory fitness level, data are limited and mostly confined to acute thermoneutral exposures. Two studies reported that untrained individuals exhibit greater diuresis and larger decreases in vasopressin and aldosterone following thermoneutral immersion (35 °C), than endurance trained individuals (84, 85). These differences may reflect altered fluid regulatory control in trained individuals, namely lower volume sensitivity and/or a reduced osmotic threshold for vasopressin secretion. Thus, trained individuals may experience diminishing hormonal responses to immersion, having already undergone similar adaptations through regular exercise.

Both aging and cardiorespiratory fitness also influence myocardial compliance, and this may influence ANP release during immersion. Acute immersion increases venous return and right atrial pressure, stimulating ANP secretion via atrial wall stretch (47, 57). Fitter individuals typically have larger blood volumes and may experience greater absolute venous return during immersion. However, their larger and more compliant atria can accommodate this volume shift with a relatively smaller rise in pressure, potentially resulting in a more moderate ANP response. Conversely, in older or less fit individuals with smaller atrial volumes, the same volume shift may result in relatively greater chamber stretch and stronger ANP release. These anatomical and mechanical differences may contribute to the interindividual variability in natriuretic and blood pressure responses to acute and repeated immersion.

Although these acute response data are helpful, more longitudinal work is needed to clarify how age, fitness level, and comorbidities influence adaptive blood pressure adaptations to hot-water immersion. Additionally, identifying the mechanisms that underpin individual variability in blood pressure regulation, such as baroreflex function, vascular compliance, and hormonal reactivity, remains an important avenue for future research (Figure 2).

Can research on acute responses predict long-term adaptive responses?

It is evident from the previous paragraph that further research is required to better understand the dose-response relationship to hot-water immersion, ensuring it can be prescribed as personalised therapy. As proposed previously (29), in the absence of these longer-term follow-up data, responses to a single acute exposure may provide insight on how certain populations adapt across an intervention or repeated exposures. Importantly, identifying associations between acute and chronic hypotensive responses may help determine individual responsiveness, aiding in the stratification of who may benefit most.

Several cross-sectional studies have reported strong correlations between the magnitude of the post-exercise hypotensive response and the adaptive change in blood pressure across an intervention. Liu et al. (86) first reported that the change in blood pressure following a single bout of aerobic exercise was strongly correlated with the change in systolic ($r = 0.89$) and diastolic blood pressure ($r = 0.75$) after eight weeks of continuous moderate-intensity exercise training in adults with pre-hypertension. Subsequent studies have corroborated this association across interventions ranging from 4-wk to 6-month interventions (87, 88). A similar acute-vs-chronic relationship has been reported for hot-water immersion (21), although, the correlations were more modest (systolic blood pressure: $r = 0.67$; diastolic blood pressure: $r = 0.54$). Additionally, Kissling et al. (61) showed that the change in plasma volume following a single session of hot-water immersion, was strongly correlated with adaptation across five repeated exposures ($r = 0.80$). As alluded to above, this increase in plasma volume and concomitant shift toward increased parasympathetic (and/or decreased sympathetic) activity, may be one mechanism contributing to longer-term reductions in blood pressure (65).

In an era of precision medicine, this approach allows for the dose (i.e., duration, intensity or frequency) to be manipulated and individualised to optimise the acute post-exposure hypotensive response, on the assumption this maximises the longer-term adaptive benefit. Conversely, if no or minimal acute post-exposure hypotension is observed, other interventions may represent a more efficient and effective use of time and resource.

Combining hot-water immersion and exercise

Regular physical activity such as exercise is a fundamental lifestyle strategy for hypertension prevention and management (89). There are many similarities in the systemic physiological response to an acute exposure of exercise or hot-water immersion as noted above (e.g., rise in core temperature, heart rate, peripheral vasodilation). However, there are also several differences in how these are achieved. For example, during exercise, core temperature rises as a result of metabolic heat production from contracting skeletal muscle (i.e., active heating). In contrast, hot-water immersion induces an intense convective heat transfer from the water (i.e., passive heating), while convection ceases to function as a cooling mechanism, and evaporative heat loss is largely negated. Additionally, water immersion introduces hydrostatic pressure, and this exerts its own unique cardiovascular effects not present in other forms of passive heating.

Heat acclimation research has explored the combined effects of exercise followed immediately by hot-water immersion. This strategy appears to offer synergistic benefits, outperforming either therapy alone for improving cardiorespiratory fitness (90, 91). Scoon et al. (90) was the first to demonstrate that exercise followed by sauna resulted in superior effects on endurance performance than exercise alone; specifically, run time to exhaustion was improved by ~30%. Dalleck et al. (91) demonstrated greater increases in maximal oxygen consumption with 15 sessions of exercise (combination of continuous moderate-intensity exercise training and high-intensity interval training) followed by hot-water immersion ($+3.4 \text{ mL min}^{-1} \text{ kg}^{-1}$), compared to exercise alone ($+1.1 \text{ mL min}^{-1} \text{ kg}^{-1}$). Given the strong epidemiological link between increasing fitness and reductions in resting blood pressure (92), it is plausible that combining these therapies elicits greater hypotensive effects, than either therapy performed independently. Cross-sectional findings from Ely and colleagues (93) report that an acute bout of exercise followed by 30-min hot-water immersion reduces mean arterial blood pressure by 6 mm Hg below pre-exercise levels; there were no differences in the exercise only comparator group at this time point. Steward et al., (94) reported that 8 weeks of exercise followed by hot-water or thermoneutral immersion in previously physically inactivate middle-aged adults increased fitness similarly in both groups (2.7 vs. $2.5 \text{ mL min}^{-1} \text{ kg}^{-1}$), but reductions in systolic and diastolic blood

pressure were greatest with exercise and hot-water immersion (8 and 5 mm Hg vs 3 and 1 mm Hg respectively).

It is imperative to recognise that water immersion cannot replicate many of the health- and fitness-related benefits that physical activity such as exercise offers. For example, the cardiac strain (measured by rate pressure product) is more than two-fold lower during hot-water immersion, compared to exercise (21). While this is beneficial for patients with angina or heart failure – allowing for cardiovascular improvements with a reduced risk of exceeding angina thresholds or overloading a failing heart - it likely also explains the absence of structural cardiac adaptations with repeated hot-water immersion. Furthermore, the superior increases in muscle blood flow and intramuscular muscle temperature achieved through active heating seem to promote greater peripheral adaptations than passive heating (95, 96). Accordingly, combining exercise with hot-water immersion may provide complementary benefits, with both stimuli eliciting haemodynamic and thermal stress through distinct mechanisms that together may potentiate cardiovascular and peripheral adaptations.

Beyond the physiological

Pharmaceutical approaches for hypertension address only a small aspect of physical health. However, hypertension arises from a complex interplay of physiological processes, with psychosocial factors also playing a role (97). For example, poor mental health and poor sleep quality are recognised risk factors for hypertension. Notably, improvements in psychological well-being have favourable effects on blood pressure (98). Unlike pharmaceuticals, hot-water immersion is a holistic approach to health, with benefits for both physical and non-physical aspects of health. Interventions of hot-water immersion have reported improved sleep quality (99, 100), acute reductions in stress markers (101) and longer-term psychological health (99, 102). Favourable alterations in parasympathetic or sympathetic activity arising from the quiet time and ambience, or the social interaction, remain possible factors.

Ethnicity is another significant non-physiological risk factor for hypertension. Although it is a non-modifiable risk factor, hot-water immersion presents a culturally appropriate therapeutic option that may help address disparities in hypertension management and outcomes, particularly among Indigenous populations (103). For example, Japanese have long embraced hot-water immersion and bathing (e.g., Onsen), and epidemiological evidence reporting that older Japanese adults who practice regular Onsen bathing have lower rates of hypertension (14). Similarly, before colonisation, indigenous New Zealanders (Māori) commonly used natural hot springs for holistic medicinal benefits. Unlike traditional Western approaches to health, Māori – along with many other indigenous cultures - view physical, mental, social and spiritual health as interconnected. For Māori, hot-water bathing extends beyond its physical benefits; it promotes psychological restoration, fosters social connections (can be performed with family and friends) and holds spiritual significance by reconnecting them with their ancestors, who are believed to be present within the water (104). However, due to the lasting effects of colonisation and other complex factors, Māori now rarely practice hot-water bathing and experience disproportionately higher rates of hypertension compared to non-Māori (105, 106). Revitalisation of this traditional practice provides a culturally relevant and natural approach to hypertension management for Indigenous groups with historical ties to hot-water immersion as a form of healing.

Safety considerations

Although hot-water immersion is generally safe for healthy individuals when performed under controlled conditions, caution is warranted at higher water temperatures, longer immersion durations, or in populations with impaired cardiovascular, autonomic, or thermoregulatory function. In Japan, drowning while bathing is the most common cause of accidental death at home, with an estimated 19,000 fatalities annually, predominantly among older adults during winter months (107). This elevated risk is associated with the traditional Japanese-style bathing practice, characterized by immersion in very hot water (42-43 °C), often in cold indoor environments (10 °C). Steward et al. (108) suggest practical strategies such as gradual exit from the bath, facial fan cooling, partial limb exposure (e.g., keeping arms out of the water), ensuring adequate hydration, and allowing a seated recovery period before standing can reduce the incidence of dizziness. These precautions, combined with moderate water temperatures (≤ 39 – 40 °C) and session durations of ≤ 30 min,

provide a pragmatic framework for safer implementation of hot-water immersion across populations.

Conclusion

Hot-water immersion has the potential to serve as an adjunct therapy for both the primary and secondary management of hypertension; however, its clinical efficacy remains uncertain. Although preliminary findings indicate promising blood pressure reductions and favourable benefits for health, some recent findings underscore the need for cautious interpretation. Rigorously designed clinical studies are required to determine the true therapeutic value of hot-water immersion, to identify which patient populations are most likely to benefit, and clarify underlying mechanisms and dose-response relationships. Despite the difficulties in persuading the general population to be physically active, this should remain the primary strategy for improving health. Nonetheless, hot-water immersion may provide additive benefits when used alongside physical activity. Future research should explore its combined effects with physical activity as part of a healthy lifestyle.

Figure 1: Potential physiological mechanisms contributing to the hypotensive response following hot water immersion. ADH = antidiuretic hormone; ANP = atrial natriuretic peptide; eNOS = endothelial nitric oxide synthase; NO = nitric oxide; SNS = sympathetic nervous system. Figure created using Biorender.

Figure 2: Future research directions for evaluating hot-water immersion as a strategy to reduce blood pressure

591 **Table 1: Study characteristics and resting blood pressure outcomes**

Study	Patient cohort	Age (y)	Number of participants in HWI group (n)	Study duration (wk)	Frequency / number of sessions	Session duration (min)	Volume (h per participant)	Water temperature (°C)	Immersion depth	Change in BP (mm Hg)
Akerman et al. (24)	Peripheral arterial disease	76	11	12	3 x wk	30	18	40	Nipple	SBP: -7 DBP: -4
Bailey et al. (20)	Healthy females	25	9	8	3 x wk	30	12	42	Top sternum	MAP: -3
Brunt et al. (32)	Sedentary	22	10	8	36 sessions	up to 90	54	40.5	Shoulder and then waist*	SBP: -4 DBP: -2
Brunt et al. (83)	Healthy older adults	67	6	8-10	30 sessions	60	30	40.5	N/A	SBP: -10 DBP: N/A
Campbell et al. (109)	Healthy younger adults	23	13	1	5 sessions	60	5	40	Shoulder and then waist*	SBP: -3 DBP: -3
Cheng et al. (82)	Healthy younger adults	23	15	8	3 x wk	45	18	43	Ankle	SBP: +1 DBP: -1
Cui et al.	Healthy	59	9	4	20 sessions	30	10	39-42	Mid chest	SBP: -2

(66)	older adults									DBP: -1
Ely et al. (35)	PCOS	27	9	8-10	30 sessions	60	30	40.5	Shoulder and then waist*	SBP: -10 DBP: -9
Hartinah et al. (34)	Hypertensi on	N/A	10	1	5 sessions	15	1.25	40.5 - 43	Ankle	SBP: -14 DBP -9
Hoekstra et al. (33)	Overweight	33	10	2	5 x wk	5 @ 45 5 @ 60	8.75	39	Neck	SBP: -10 DBP: -4
James et al. (51)	Type 2 diabetes mellitus	65	14	2	10 sessions	60	10	40	Shoulder and then waist*	SBP: - 9 DBP: -1
Kaiser et al. (25)	Untreated Hypertensi on	48	21	8-10	30 sessions	45	22.5	40	Mid- sternum	SBP: -1 DBP: 0
Kudo et al. (36)	Heart failure	75	13	2	4 x wk	10	1.33	40	Sternum (or below)	MAP: -4
Luria et al. (30)	Healthy adults	32	11	8	5 x wk	10	6.67	42-44	Umbilicus	No change
Oyama et al. (37)	Heart failure	69	16	2	5 x wk	10	1.33	40	Xiphoid process	SBP: -1 DBP: -3
Roxburgh	Osteoarthri	66	27	12	3 x wk	30	18	40	Nipple	SBP: -9

et al. (21)	tis								depth	DBP: -4
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592 PCOS = polycystic ovarian syndrome; DBP = diastolic blood pressure; SBP = systolic blood pressure; * = Depth was manipulated to maintain a
593 rectal temperature of 38.5–39 °C. Pre–post mean differences were manually calculated when no statistical analysis was reported in the original
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Acute

Adaptive



Vascular

↑ Shear stress = ↑ eNOS = ↑ NO = Vasodilation
↑ Cutaneous blood flow / redistribution of blood from core to periphery
↑ Arterial compliance

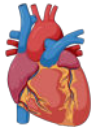
↑ Shear-stress-mediated vascular remodeling
Improved endothelial function
↑ Angiogenesis
↑ Capillary density
↑ Arterial compliance



Neurohormonal / Autonomic

↓ ADH & aldosterone = ↑ diuresis, ↑ natriuresis
↑ ANP release from atrial stretch = vasodilation, natriuresis

↓ SNS activity and plasma norepinephrine
↑ Vagal tone and baroreflex sensitivity
↑ Baseline aldosterone



Cardiac / Haematological

↑ Venous return (hydrostatic pressure) = ↑ preload = ↑ ANP

Plasma volume expansion ("supercompensation") = ↑ venous return and stroke volume
↓ Resting heart rate



Cellular

↑ Heat shock proteins
↑ Antioxidant capacity
↑ Inflammatory and immune signaling

↓ Pro-inflammatory cytokine production
↓ Oxidative stress
↑ Antioxidant capacity

STUDY DESIGN

- Conduct adequately powered randomized controlled trials, with resting blood pressure as the primary outcome
- Utilize “gold standard” 24-h ambulatory monitoring for robust blood pressure assessment
- Include follow-up assessments after cessation of hot-water immersion to determine whether any hypotensive benefits are sustained, or require ongoing exposure
- Explore synergetic effects with pharmaceuticals or exercise

MECHANISMS

- Investigate potential mechanisms:
 - Structural (cardiac, vascular, musculoskeletal remodeling)
 - Neurohormonal (renin-angiotensin-aldosterone system, natriuretic peptides)
 - Autonomic regulation

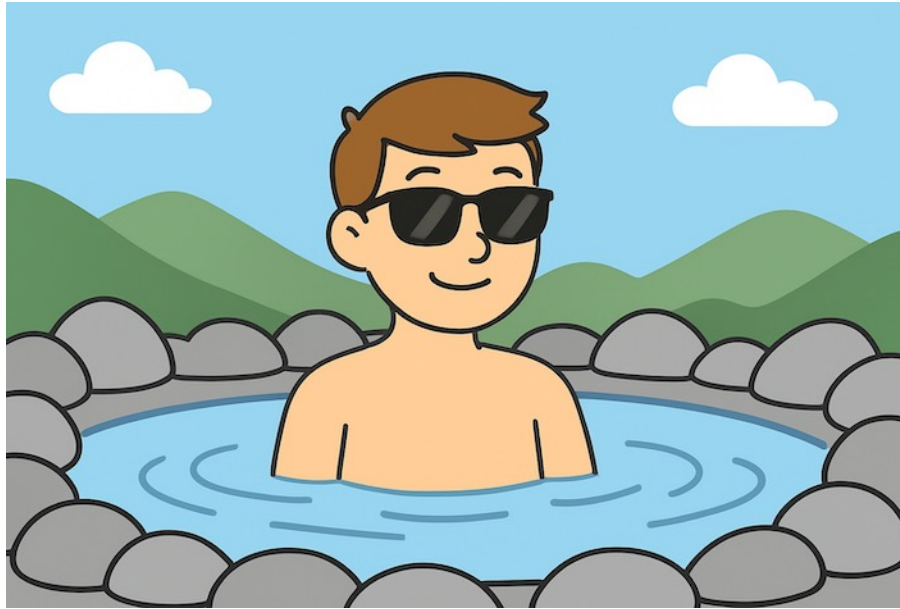
DOSE RESPONSE

- Identify factors influencing individual responses (age, sex, fitness, baseline resting blood pressure, comorbidities)
- Assess dose-response relationships (session frequency, temperature, immersion duration/volume)

SOCIETAL / ENVIRONMENTAL CONCERNS

- Evaluate more accessible and sustainable approaches (natural hot springs, lower-limb immersion) to reduce energy and water use
- Translate findings to culturally and more ecologically relevant settings (i.e., traditional bathing practices, lagoon bathing) to capture “non-physiological” contributors to blood pressure

Hot-water immersion as an anti-hypertensive therapy



Mechanisms: Hot-water immersion transiently lowers blood pressure via:

- increased shear stress
- vasodilation
- autonomic adjustments
- neurohormonal changes

Repeated exposures may sustain reductions in resting blood pressure.

Dose-response and individual variability: Blood pressure responses and mechanisms may depend on:

- immersion duration and frequency
- water temperature
- individual characteristics (age, fitness, comorbidities)

CONCLUSION

Hot-water immersion shows promise as an adjunct therapy for hypertension, however, its clinical efficacy remains uncertain, and further rigorous studies are needed to define optimal protocols, target

populations, and underlying mechanisms.