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Titel	Does Sauna Improve Cardiovascular Health?
Subtitel	Acute cardiovascular effects, possible adaptations and what long-term observational studies can—and cannot—prove
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Short answer: Regular sauna bathing is associated with favourable cardiovascular outcomes, but the strength of the evidence depends on the question. A single sauna session clearly produces acute cardiovascular effects: skin vessels dilate, vascular resistance falls, heart rate and cardiac output rise, and blood pressure and arterial stiffness can decrease during recovery. Repeated passive heat exposure may produce vascular and thermoregulatory adaptations, but randomized intervention trials remain relatively small and heterogeneous. The strongest long-term findings come from prospective Finnish cohort studies, in which more frequent sauna bathing was associated with lower rates of fatal cardiovascular events and cardiovascular mortality. These are important associations, not proof that sauna itself caused the risk reduction. Sauna should therefore be considered a potentially useful component of a healthy lifestyle—not a substitute for exercise, blood-pressure treatment or established cardiovascular prevention.

Few sauna questions illustrate the difference between physiological plausibility and clinical proof as clearly as cardiovascular health. The acute effects of heat on the circulation are well established. Long-term observational studies are striking. Yet neither of those facts automatically proves that sauna prevents cardiovascular disease.

A scientifically useful answer therefore has to separate three levels of evidence: what happens during and immediately after one sauna session; what may adapt after repeated heat exposure; and what long-term epidemiological studies observe in habitual sauna users.

Acute effects, repeated adaptations and long-term disease outcomes are three different scientific questions.

What happens during a single sauna session?

Whole-body heat exposure redistributes blood toward the skin. Cutaneous vessels dilate, systemic vascular resistance falls, heart rate rises and cardiac output increases to support thermoregulation. Sweating simultaneously begins to reduce body water and plasma volume.

These acute responses are not speculative. They are fundamental features of human heat physiology and have been measured repeatedly in sauna and other passive-heating experiments [1–3].

The cardiovascular system is therefore strongly engaged during sauna bathing, even though the skeletal muscles are not performing the mechanical work of exercise.

What happens to blood pressure?

Blood pressure does not have one universal response during sauna because it reflects the balance between cardiac output and vascular resistance. During heating, vasodilation tends to lower resistance while increasing heart rate and cardiac output can help maintain arterial pressure.

In a study of 102 adults with at least one cardiovascular risk factor, a 30-minute sauna at approximately 73 °C and 10–20% relative humidity reduced mean systolic blood pressure from 137 to 130 mmHg and diastolic pressure from 82 to 75 mmHg immediately after sauna. Systolic pressure remained below baseline after 30 minutes of recovery [4].

This is an acute effect. It does not by itself demonstrate treatment of chronic hypertension.

What happens to arterial stiffness?

The same experimental programme found an acute improvement in measures of arterial compliance after a single sauna session [5]. Arterial stiffness is clinically relevant because stiff arteries increase pulsatile load on the cardiovascular system.

An acute reduction is biologically interesting, but a temporary change after one exposure is not equivalent to a permanently healthier arterial system. To establish durable adaptation, repeated-exposure trials are required.

Why might repeated heat exposure affect vascular function?

Repeated increases in skin blood flow expose the vascular endothelium to altered shear stress. Heat also repeatedly challenges vascular tone, cardiac output, plasma volume regulation and thermoregulatory control.

These mechanisms provide plausible pathways through which repeated passive heating could influence endothelial function, arterial stiffness and blood-pressure regulation. Experimental hot-water immersion studies have reported improvements in vascular function after repeated exposure [6].

Plausibility, however, is not proof of a clinical cardiovascular benefit. A mechanism can exist without producing a meaningful reduction in heart attacks, stroke or mortality.

Does repeated sauna lower resting blood pressure?

Some intervention studies suggest that repeated whole-body heat exposure can reduce blood pressure in selected populations. But the evidence is not uniform.

A 2025 systematic review and meta-analysis of 20 randomized controlled trials of passive heating lasting 2–15 weeks found no statistically significant pooled effect for most cardiometabolic and vascular outcomes. The overall pooled effect on systolic blood pressure was also not statistically significant, although subgroup analyses suggested reductions with systemic whole-body heating and among people with coronary risk or cardiovascular disease [7].

A separate 2025 meta-analysis of heat thermotherapy reported small reductions in diastolic and mean arterial pressure, but substantial heterogeneity remained across protocols and populations [8].

> **The intervention evidence is promising in places, but it is not a simple story of 'sauna lowers blood pressure'.**

What about endothelial function?

Endothelial function describes the ability of the vessel lining to participate in vascular regulation. Repeated passive heating can increase blood flow and shear stress, mechanisms known to influence endothelial signalling.

Individual experimental studies have reported improvements, but the 2025 randomized-trial meta-analysis did not find a significant pooled improvement in flow-mediated dilation across the available passive-heating studies [7].

This discrepancy between plausible mechanism, positive individual studies and an inconclusive pooled result is exactly why evidence hierarchy matters.

What do long-term Finnish studies show?

The most widely discussed sauna-health findings come from the Kuopio Ischaemic Heart Disease Risk Factor Study, a prospective population cohort in eastern Finland. Participants reported their habitual sauna use and were then followed for cardiovascular outcomes over many years.

These studies found that men reporting more frequent sauna bathing had lower rates of several fatal cardiovascular outcomes than men reporting one sauna session per week. Later analyses also found that sauna frequency and objectively measured cardiorespiratory fitness were independently associated with lower cardiovascular and all-cause mortality [9,10].

These are prospective data, which are stronger than simple cross-sectional comparisons. They still remain observational.

What does 'associated with' mean?

An association means that two variables occur together more often than expected after statistical adjustment. It does not establish that one variable caused the other.

Frequent sauna users may differ from infrequent users in physical activity, fitness, socioeconomic factors, alcohol use, diet, social behaviour, underlying health or other characteristics. Statistical models can adjust for measured differences, but they cannot guarantee elimination of every confounder.

> **A lower cardiovascular risk among frequent sauna users does not automatically mean that sauna caused the lower risk.**

Why are the Finnish findings still important?

Observational evidence should not be dismissed simply because it cannot prove causality. Long follow-up, prospective design, clinically meaningful endpoints and dose-related associations can provide valuable signals.

The Finnish studies are especially interesting because the epidemiological associations point in the same general direction as several plausible physiological mechanisms: lower vascular resistance, altered arterial stiffness, repeated endothelial shear stress, autonomic effects and possible blood-pressure changes [3,9].

The convergence strengthens the hypothesis. It does not convert the hypothesis into a randomized clinical trial.

How large were the observed associations?

In one analysis of 2,277 men followed for a median of 26.1 years, sauna bathing 3–7 times per week, compared with no more than twice weekly, was associated with a lower risk of cardiovascular mortality after multivariable adjustment. Higher cardiorespiratory fitness was also independently associated with lower risk [10].

Another analysis of 2,291 men found lower sudden-cardiac-death risk among those reporting more frequent sauna bathing, while the combination of higher fitness and frequent sauna use was associated with particularly low observed risk [11].

The appropriate wording is important: these studies measured risk associations in a specific Finnish male cohort. They did not randomly assign people to decades of sauna use.

Can these results be generalized to everyone?

Not automatically. Much of the best-known long-term evidence comes from middle-aged Finnish men, predominantly using traditional Finnish sauna. Findings from that population cannot simply be assumed to apply with the same magnitude to women, younger adults, different ethnic groups, people with different disease profiles or very different bathing practices.

More recent research has broadened the evidence base, but the epidemiological literature remains much less diverse than would be desirable for universal recommendations.

Is frequency itself the active ingredient?

The cohort studies often use sauna frequency as the exposure variable because it is practical to measure. Frequency, however, is not the same as physiological dose.

A sauna session can differ in temperature, humidity, duration, number of heat phases, Aufguss intensity, cooling, hydration and individual tolerance. Two people reporting four sessions per week may therefore receive very different thermal exposures.

Frequency is useful epidemiologically, but sauna science should not confuse it with a precisely quantified biological dose.

Does longer sauna exposure mean greater cardiovascular benefit?

Observational studies have reported associations involving both frequency and duration, but they cannot establish that deliberately extending each heat exposure will improve health.

Longer exposure also increases heat storage, fluid loss and circulatory strain. The existence of a statistical association in habitual sauna users is not a licence to maximise session length.

Health-oriented sauna bathing aims for a repeatable and recoverable thermal stimulus, not maximal endurance.

Does a higher temperature produce greater benefit?

There is no convincing clinical evidence that progressively higher sauna temperatures produce progressively greater cardiovascular benefit. Most Finnish evidence comes from customary high-temperature sauna use, but the body responds to total thermal dose rather than the thermometer alone.

Passive-heating studies using hot-water immersion and other lower-temperature modalities can also produce vascular responses. This supports the view that heat transfer and accumulated physiological strain matter more than a particular prestige temperature.

How does sauna compare with exercise?

Sauna and exercise share some acute cardiovascular features: heart rate and cardiac output rise, skin blood flow increases and vascular shear stress changes. This overlap has encouraged the description of passive heating as an 'exercise mimetic'.

That term should be used cautiously. Exercise also produces skeletal-muscle contraction, major metabolic demand, mechanical loading and adaptations in cardiorespiratory fitness that passive heating does not reproduce.

In the Finnish cohort, cardiorespiratory fitness and sauna use were independently associated with outcomes [10]. That is a strong reason not to frame sauna as a replacement for exercise.

> **Sauna may complement physical activity. It does not reproduce the full cardiovascular biology of exercise.**

Could sauna help people who cannot exercise?

This is an important research question. Passive heating can produce cardiovascular stimuli without requiring locomotor exercise and is therefore being studied in older adults and people with cardiovascular disease or limited exercise capacity.

A 2025 review of passive heat therapy in cardiovascular disease identified 18 eligible intervention studies using several modalities, including Finnish sauna and Waon therapy. The authors concluded that the field is promising but that efficacy remains uncertain because studies are heterogeneous and often small [12].

This is a potential clinical application, not an established substitute for evidence-based cardiac rehabilitation.

What about heart failure and Waon therapy?

Repeated low-temperature sauna-based protocols, often termed Waon therapy, have been studied particularly in Japan in people with chronic heart failure. Some studies report improvements in symptoms, vascular function and haemodynamic measures.

These protocols differ substantially from ordinary recreational Finnish sauna bathing: temperature, duration, post-heating rest, patient selection and medical supervision are defined. Their results therefore should not be transferred uncritically to unsupervised public sauna use.

A therapeutic heat protocol is not automatically equivalent to a normal sauna visit.

Can sauna prevent heart attacks or stroke?

The observational literature is compatible with lower cardiovascular risk among frequent sauna users, but prevention is a causal claim. At present, long-term randomized trials showing that sauna itself prevents myocardial infarction, stroke or cardiovascular death are lacking.

It is therefore scientifically more accurate to say that habitual sauna bathing is associated with lower cardiovascular event and mortality rates in several prospective Finnish analyses, while causal prevention remains unproven.

What does the randomized evidence add?

Randomized trials are particularly valuable because allocation reduces confounding. Current passive-heating trials demonstrate that repeated heat exposure can modify some cardiovascular variables, but

the 2025 meta-analyses also show how uncertain the magnitude and consistency of these changes remain [7,8].

Most trials measure surrogate endpoints such as blood pressure, arterial stiffness or endothelial function rather than heart attacks, stroke or cardiovascular mortality. Improvement in a surrogate marker is not identical to proof of fewer clinical events.

Why surrogate endpoints matter—and why they are not enough

Blood pressure and arterial stiffness are genuine cardiovascular risk factors. If an intervention reliably improves them, that is biologically meaningful.

But cardiovascular disease develops over years and is influenced by lipids, smoking, diabetes, physical activity, body composition, genetics, inflammation and many other factors. A short-term change in one marker cannot by itself quantify long-term disease prevention.

What about autonomic regulation?

Heat exposure changes sympathetic and parasympathetic cardiovascular control. During heating, heart rate rises and sympathetic cardiovascular demands change; during recovery, autonomic balance shifts again.

Repeated exposure may influence autonomic regulation, and this has been proposed as one pathway linking habitual sauna bathing with cardiovascular outcomes [3]. The clinical significance and magnitude of this mechanism remain incompletely established.

Is sauna safe for the cardiovascular system?

For most healthy people, conventional sauna bathing is generally well tolerated when used sensibly. Reviews also report that many people with stable cardiovascular disease can tolerate sauna under appropriate conditions [3].

That statement does not apply automatically to unstable disease. Recent acute myocardial infarction, unstable angina, severe symptomatic hypotension, decompensated heart failure, significant uncontrolled arrhythmia or other unstable cardiovascular conditions require individual medical assessment.

Alcohol, dehydration and extreme thermal exposure can add risk and should not be confused with ordinary health-oriented sauna bathing.

What role does cooling play?

Cooling changes the cardiovascular state again. Cold exposure causes cutaneous vasoconstriction, increases peripheral vascular resistance and can raise blood pressure. An ice-cold plunge is therefore not merely the absence of heat; it is a second cardiovascular stimulus.

The cardiovascular dose of a sauna visit includes both heating and cooling. Very abrupt cold exposure may be inappropriate for some individuals even when the preceding heat phase was well tolerated.

How should the evidence be summarized?

The acute physiology is strong: sauna causes major but normally regulated cardiovascular adjustments. Evidence for repeated vascular adaptation is plausible and supported by some intervention studies, but pooled randomized evidence remains heterogeneous and does not show uniform improvement across all cardiovascular markers.

The epidemiology is striking: frequent habitual sauna bathing is associated with lower cardiovascular mortality and several adverse cardiovascular outcomes in long-term Finnish cohorts. But those studies establish association, not causality.

The strongest scientific position is therefore neither 'sauna is proven to prevent cardiovascular disease' nor 'the evidence is only anecdotal'. The evidence is substantial but uneven across levels of inference.

So, does sauna improve cardiovascular health?

It can acutely improve several haemodynamic and vascular measurements, and repeated heat exposure may promote cardiovascular adaptations. Habitual sauna use is also associated with favourable long-term cardiovascular outcomes in prospective cohorts.

What remains unproven is the strongest version of the claim: that sauna itself causally reduces heart attacks, stroke or cardiovascular mortality in the general population.

> **The cardiovascular evidence for sauna is promising and biologically coherent. Association is not causation, and physiological plausibility is not clinical proof.**

Conclusion

Sauna bathing is a meaningful cardiovascular stimulus. During a heat phase, vasodilation lowers peripheral resistance while heart rate and cardiac output rise. Acute studies demonstrate changes in blood pressure and arterial stiffness, and repeated passive heating may influence vascular function and thermoregulatory adaptation.

Long-term Finnish cohort studies consistently report lower cardiovascular and mortality risks among more frequent sauna users. These findings are important, but they remain observational and arise largely from specific populations and bathing traditions.

Randomized passive-heating trials provide a more cautious picture: some cardiovascular markers improve, while others show no consistent pooled effect. Sauna can therefore reasonably be viewed as a potentially beneficial adjunct to an otherwise healthy lifestyle, while exercise, smoking cessation, blood-pressure control, lipid management and indicated medical treatment remain established foundations of cardiovascular prevention.

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Editorial note: This article distinguishes acute physiological effects, intervention evidence and observational epidemiology. It is intended for scientific and educational information and does not replace individual cardiovascular diagnosis or treatment.