

# Diagnostic Dilemma and Lack of Guidelines in Salt-Sensitive Hypertension in Young Adults: A Silent Cardiovascular Risk

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## 1. Abstract

Salt-sensitive hypertension (SSH) is a physiologically distinct blood pressure phenotype in which blood pressure rises disproportionately in response to sodium intake. Traditionally studied in older hypertensive individuals, SSH is now recognized in apparently healthy young adults, where it silently drives early cardiovascular injury. The burden is disproportionately high among South Asians due to genetic predisposition, high-sodium dietary patterns, and environmental risk enhancers.

The stealth of SSH lies in its asymptomatic course. Vascular stiffening, endothelial dysfunction, microvascular damage, and left ventricular remodeling can develop years before overt hypertension. Yet, detection is hindered by the absence of practical, cost-effective, and standardized diagnostic tools. Gold-standard salt-loading and depletion tests are too resource-intensive for routine care, while surrogate measures such as urinary sodium excretion or dietary recall are imprecise and inconsistently applied.

The burden is disproportionately high among South Asians due to genetic predisposition, high-sodium dietary patterns, and environmental risk enhancers. Compounding the problem, SSH is almost entirely absent from global and regional hypertension guidelines, meaning young adults at risk are neither screened nor counselled. As a result, a silent epidemic of subclinical cardiovascular damage is progressing unchecked.

This thematic review synthesizes epidemiological, mechanistic, and clinical literature to highlight the diagnostic dilemma and the lack of formal guidance on SSH in young adults. Drawing from cardiology, nephrology, genetics, and public health research, it examines how SSH escapes detection, why the risks are higher in South Asian populations, and what emerging innovations could bridge current gaps. The objective is not to present prescriptive solutions but to illuminate the urgent need for recognition, diagnostic innovation, and policy integration to prevent irreversible cardiovascular damage in the formative decades of life.

**Keywords:** Salt-sensitive hypertension, young adults, asymptomatic cardiovascular damage, South Asian populations, diagnostic gap, guideline deficiency

## 2. INTRODUCTION

Hypertension is the most important modifiable risk factor for heart disease and death worldwide. It plays a major role in the global burden of non-communicable diseases. Conventional clinical practice focuses on high resting blood pressure as the main trigger for diagnosis and treatment. However, the variation in blood pressure regulation is often overlooked. One less recognized aspect of this variability is salt-sensitive hypertension (SSH), which is a distinct physiological condition. In SSH, blood pressure increases more significantly in response to sodium intake, regardless of base blood pressure levels.

For many years, SSH was mainly seen as a problem for older adults or those with diagnosed hypertension. However, recent evidence shows that SSH is also common among young adults, including those considered normotensive by current standards. This discovery has important implications. Structural and functional changes in the cardiovascular system, such as vascular stiffening, endothelial dysfunction, microvascular rarefaction, and concentric left ventricular remodelling, may start to develop silently several years before hypertension is officially diagnosed. Often, these changes become irreversible by the time they are identified, limiting the success of later treatments.

The hidden nature of SSH lies in its symptomless progression. Since it does not consistently appear as persistently high blood pressure readings at the clinic, it often goes unnoticed during routine check-ups, sports screenings, or workplace health programs. Current diagnostic systems are largely fixed and depend on one or occasional blood pressure measurements in clinical settings. This approach does not capture the variable, diet-sensitive changes in blood pressure that define SSH. As a result, affected young adults may continue to experience repeated increases in blood pressure due to sodium intake. This accelerates vascular aging, harm endothelial function, and raises the risk of early heart events.

This lack of recognition is made worse by the belief that young adulthood (usually ages 18–40) is a time of low cardiovascular risk. Preventive screenings for hypertension are irregular, and dietary sodium intake is rarely evaluated in detail for those without symptoms. Thus, SSH can continue to progress unnoticed, creating a hidden risk for future heart attack, stroke, or heart failure often during the most productive years of life.

**2.1. Why Young Adults? The Paradox of Low-Risk Complacency** -Young adulthood typically defined as ages 18–40 is considered a low cardiovascular risk period in clinical practice. Screening is sporadic, and risk-factor modification is often deprioritized. This complacency is dangerous in the context of SSH. Research suggests that in susceptible individuals, repeated sodium-induced BP spikes accelerate vascular aging, endothelial dysfunction, and left ventricular remodelling. These pathophysiological changes may go undetected until the third or fourth decade of life, at which point intervention is less effective <sup>[1]</sup>

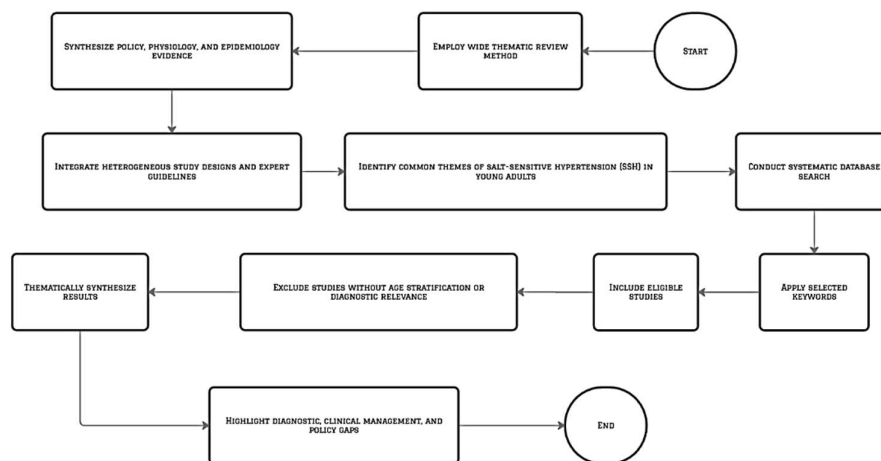
**2.2. A Particularly Urgent Problem in South Asians** -South Asian patients also have a unique cardiometabolic risk profile with a premature onset of cardiovascular disease and a higher risk of myocardial infarction <sup>[2]</sup>. The genetic differences in sodium metabolism, especially those occurring in the renin-angiotensin-aldosterone system, are much more prevalent in this group and are responsible for increased salt sensitivity <sup>[3]</sup>. The genetic susceptibility is also complemented by extremely high dietary sodium intake, which in the Indian population is generally 8 to 12 g/day, much higher than the World Health Organization's (WHO) recommendation of less than 5 g/day <sup>[2];[4]</sup>. The high intake is contributed to most by high intake of processed foods, pickled condiments, and other food preparation habits that are high in salt <sup>[2]</sup>

**2.3. The Stealth Problem: Asymptomatic Damage** The pathophysiology of SSH includes renal sodium retention, impaired natriuresis, heightened sympathetic activity, and vascular smooth muscle remodelling. The clinical consequence is not just elevated BP during salt load but also progressive structural and functional damage to the cardiovascular system. This includes increased arterial stiffness, microvascular rarefaction, and concentric left ventricular hypertrophy. Critically, these changes may occur at BP levels still considered “normal” under current diagnostic thresholds, meaning conventional screening misses the disease entirely [5]

**2.4. The Policy Vacuum and Diagnostic Limitations** The gold-standard method for SSH diagnosis involves controlled sodium-loading and depletion tests with serial BP monitoring. While highly accurate, these are impractical for routine use: they require specialized facilities, extended observation periods, and strict dietary control. In practice, clinicians rely on urinary sodium excretion measurements or dietary history, both of which are unreliable due to intra-individual variability and recall bias. Ambulatory blood pressure monitoring (ABPM) with sodium intake assessment offers some potential, but lacks standardized cut-offs and validation for SSH screening in young populations [2]. A review of hypertension guidelines from major bodies the American Heart Association (AHA), European Society of Cardiology (ESC), World Health Organization (WHO), and Indian Council of Medical Research (ICMR) reveals virtually no mention of SSH as a screening or diagnostic target. Without recognition in guidelines, there is no structured approach to identifying or managing SSH, especially in asymptomatic individuals [6].

**2.5. Why This Matters: A Call to Action** The absence of detection tools and policy frameworks perpetuates a silent progression toward early cardiovascular disease. In a young adult with SSH, each unrecognized year may mean incremental vascular injury, moving the individual closer to premature myocardial infarction, stroke, or heart failure. This represents not only a personal health tragedy but also a public health failure especially in low- and middle-income countries (LMICs) where cardiovascular care resources are already strained. This thematic review aims to summarize the epidemiology, pathophysiology, and diagnostic challenges of SSH in young adults, with a specific focus on South Asian populations. The goal is to generate awareness and prompt discourse on this overlooked yet modifiable cardiovascular risk factor.

### 3. Methodological Framework and Thematic Analysis Strategy



(fig.1. Step-by-Step Process for Data Extraction and Synthesis)

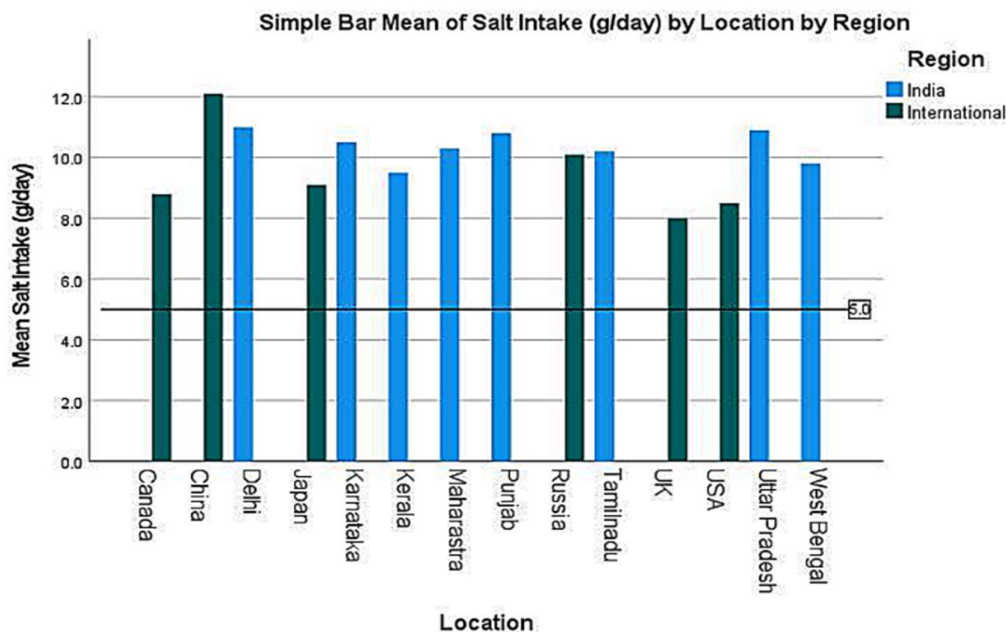
A wide thematic review method was employed to synthesize policy, physiology, and epidemiology evidence in place of narrow systematic review. Synthesis of heterogenous study designs (population, clinical, laboratory) and expert guidelines was facilitated to look for common themes of salt-sensitive hypertension (SSH) in young adults. Systematic search was performed in PubMed, Scopus, Web of Science, and Google Scholar (2000–2025) using the keywords "salt-sensitive hypertension," "young adults," "blood pressure variability," and "South Asia." Peer-reviewed original research, systematic reviews, and meta-analyses in adults aged 18–40 years (normotensive or hypertensive) were included, and studies with no clear age stratification or diagnostic relevance were excluded.

Results were thematically synthesized (rather than pooled) to highlight major gaps in diagnostics, clinical management, and policy. This narrative format is suitable for the interdisciplinary scope: it highlights repeated mechanisms and unmet policy gaps rather than approximating a single pooled prevalence (as systematic reviews).

#### 4. Global and Regional Epidemiology: Why Young Adults Are Not Spared

Salt sensitivity is prevalent even in young "normotensive" adults. Although prevalence is difficult to estimate with different diagnostic techniques, extensive surveys indicate that 15–30% of apparently healthy adults exhibit salt-sensitive blood pressure response. Prevalence is especially high among Asian populations, and Chinese and Indian reports indicate salt sensitivity in as many as one-third of young adults.

Worldwide, sodium consumption far surpasses guidelines: the mean is ~10.8 g/day, close to twice the WHO's <5 g/day threshold (WHO, 2025). Such excessive exposure confers risk in those with a genetic predisposition. Migration studies also reveal that South Asian immigrants have salt-sensitive patterns even when living in low-sodium settings, highlighting prevalent heritable factors. Briefly, young adults who are "healthy" may have an occult cardiovascular risk.



(Fig.2. Comparison of Mean Dietary Salt Intake in Indian States and Selected International Regions.)

## 5. Pathophysiology of Salt Sensitivity: From Sodium Handling to Vascular Injury

SSH is a consequence of the inability of the usual mechanisms for sodium excretion, leading to over-response to salt intake. Salt-sensitive individuals' kidneys cannot excrete sodium at a particular pressure, leading to the typical blunted pressure–natriuresis. This is complemented by maladaptive renin-angiotensin-aldosterone signalling (e.g. paradoxical aldosterone responses) and increased sympathetic activity.

Extrarenal mechanisms aside, sodium harms the vasculature directly. Increased sodium intake increases oxidative stress and attenuates nitric oxide bioavailability, causing vasoconstriction. Chronic exposure causes arteriolar hardening by smooth muscle hypertrophy and concentric LVH even in the absence of chronic hypertension. SSH is hence not a manifestation of an isolated defect but of an interrelated system of renal, hormonal, and vascular dysfunctions, and that is why subjects react heterogeneously to a uniform salt load.

## 6. The Silent Harm: Unrecognised Cardiovascular Damage

Even prior to overt hypertension, SSH imposes quantifiable structural marks. Ambulatory and echocardiographic studies demonstrated increased left ventricular mass and concentric remodelling in salt-sensitive normotensives relative to resistant. Vascular stiffness, as calculated by pulse-wave velocity, is also increased in this population. There are also microvascular alterations: retinal capillary rarefaction and endothelial dysfunction with "normal" clinic BP. These alterations suggest that SSH causes vascular aging decades ahead of time. As discussed in more detail later in Section 9 (Cost of Neglect), such occult damage is why so many young salt-sensitive adults have premature cardiovascular events.

## 7. Diagnostic Blind Spots

Despite these risks, SSH remains invisible in daily practice. The gold-standard protocol-controlled salt-loading and depletion with serial BP measurement is feasible only in studies. Other options such as 24-hour urine sodium and ambulatory BP monitoring are indirect, poorly standardized, and costly. Most primary care comes from clinic BP measurements, which cannot detect transient, meal-induced spikes. This creates a key diagnostic gap: young adults with high risk go undetected until hypertension or vascular disease has developed. As noted in Section 7 (Policy Blind Spots), this flaw is exacerbated by the lack of any particular recommendations in guidelines.

## 8. Why We Are Failing: Practical and Systemic Barriers to Detection

- **Lack of low-cost, validated screening tools:** The absence of a simple, quick, and reliable diagnostic test for SSH is the single biggest barrier to its detection.
- **Time and resource constraints in primary care:** A typical 10-15 minutes consultation is insufficient to conduct a detailed dietary history and assess for SSH, especially in an asymptomatic patient.
- **Minimal training for clinicians on SSH:** Many medical education programs do not place a significant emphasis on SSH as a distinct clinical entity, leading to a lack of awareness among healthcare providers <sup>[7]</sup>
- **Poor public awareness:** The general public, particularly young adults, remains largely unaware of the link between high dietary sodium and long-term cardiovascular risk, and how this risk can be amplified by salt sensitivity.

## 9. Asymptomatic Course and Missed Clinical Opportunities

The absence of symptoms leads to missed screening opportunities during routine checkups, sports physicals, or workplace health programs. This is especially problematic in regions where preventive health visits are rare. The current paradigm for hypertension screening, which relies on a single BP reading in a clinic, is fundamentally ill-equipped to capture the dynamic and diet-dependent nature of SSH.

## 10. The South Asian Paradox: Genetics, Diet, and Accelerated Risk

Genetic studies reveal a higher prevalence of sodium-retentive polymorphisms in genes affecting renal sodium handling within South Asian populations [8],[9]. Dietary sodium intake in South Asia often exceeds WHO recommendations of <5g/day, with typical intakes of 8-12g/day due to the high consumption of processed foods, pickled condiments, and restaurant meals [10]. Combined, these factors explain the disproportionate SSH burden and the accelerated onset of cardiovascular disease in this demographic.

This unique interplay of genetic predisposition and environmental factors explains the disproportionate SSH burden and the accelerated onset of cardiovascular disease in this demographic. Compounding this issue is a critical lack of public awareness; a study in urban India found that over 70% of hypertension patients were unaware of the need for sodium restriction. This low public health literacy, coupled with a policy vacuum in national guidelines, ensures that SSH remains a hidden threat until it manifests as an acute cardiovascular event.

## 11. Public Health and Screening Challenges: Policy Blind Spots

Current hypertension control policies presume risk only after the development of prolonged BP elevation. SSH defies this paradigm, as vascular injury precedes thresholds. Nevertheless, influential guideline bodies WHO, AHA/ACC, ESC promote population sodium reduction without officially endorsing salt sensitivity.

In India, the ICMR guidelines are silent on SSH as a means of diagnosis or treatment. This leaves the particular high-risk group unobserved.

Policy Comparisons: Some countries have made progress: the UK cut population salt consumption by ~1 g/day through collective reformulation and public action [11], and Japan has made stepwise declines from ~14 g/day in the 1990s to ~10 g today [12]. For India, the average (~10 g/day) is unchanged despite recent attention to potassium-enriched salt [13]. Notably, none of these efforts stratify by salt sensitivity an untapped opportunity for precision prevention.

### 11.1. Key Challenges

- **Screening Issues and Awareness Barriers:** Gold-Standard Testing is Hard to Conduct: Pressure-natriuresis or salt-loading experiments cannot be conducted on a large scale. Even in laboratory experiments, there are not many patients who follow through with them (~65%), suggesting problems with the way that people follow through and how easy they are to conduct [2]
- **Cultural Entrenchment of High-Salt Diets:** South Asian cuisine is a significant source of high-salt foods. Cultural practices are countered by public health messages. In urban India, more than 70% of individuals with high blood pressure were unaware of the dangers of sodium [9]
- **Resource Constraints:** The substantial burden of noncommunicable diseases in low- and middle-income countries is often underestimated, with a high prevalence of undiagnosed and untreated cases, which demonstrates the inadequacy of existing diagnosis and management strategies [14]

- **Data Scarcity and the Neglect Cycle:** Lack of data on SSH prevalence among the general population prevents policymakers from appreciating that they need to invest. It is a cycle where lack of data prevents policy formulation, which further prevents SSH from being appreciated as a public health issue [15]
- **Worldwide Opportunities and Assessments for Targeted Prevention Were Overlooked:** Global experience provides promise and the limits of existing sodium-reduction interventions. Education and regulatory reformulation in the United Kingdom lowered sodium intake by  $\sim 1$  g/day<sup>[15]</sup>, and Japan illustrates stepwise declines from  $\sim 14$  g/day in the 1960s to  $\sim 10$  g/day today by sustained policy interventions [16]. Mean daily intake in India ( $\sim 10$  g/day) has remained constant over decades [2]. Perhaps most importantly, none of these interventions include salt-sensitivity stratification. This is unrealized potential for precision prevention, where sodium-reduction policy directed at SSH-positive individuals could have disproportionate effect in lowering vascular morbidity

## 12. Absent in the Guidelines: A Critical Gap

A review of major hypertension guidelines reveals that SSH receives at most a passing mention, often without actionable recommendations. The 2017 AHA/ACC Hypertension Guideline focuses heavily on absolute BP thresholds, while the European Society of Cardiology's 2023 update similarly prioritizes static BP measurements over dynamic sodium sensitivity [6]. In India, the ICMR's hypertension guidelines do not differentiate management strategies based on salt sensitivity at all.

### 12.1. Clinical and Ethical Implications

- **No formal screening triggers:** Without guidelines, clinicians lack justification to order specialized tests for young normotensive patients with suspected SSH.
- **Insurance and reimbursement gaps:** Diagnostic tests are not covered without explicit guideline endorsement.
- **Missed prevention opportunities:** The window for lifestyle interventions is squandered when SSH is undetected until overt hypertension emerges.

## 13. Innovations on the Horizon: Early Detection Tools

Several emerging tools could bridge the diagnostic gap:

- **Wearable BP monitors** capable of capturing BP fluctuations with dietary changes [17].
- **Urinary proteomics** to identify biomarkers linked to sodium sensitivity [8]
- **Genetic screening panels** for sodium-handling polymorphisms.
- **Machine learning–driven predictive models** integrating demographic, genetic, and lifestyle factors to estimate hypertension risk [18]
- **Non-invasive vascular stiffness measures** (e.g., photoplethysmography) deployable in community settings [4]

However, adoption of these tools requires validation, cost-effectiveness studies, and inclusion in clinical guidelines. Without policy integration, even the best innovations risk remaining academic curiosities.

## 14. Discussion: The Cost of Neglect and the Path Forward

The thematic synthesis presented in this review underscores a fundamental disconnect between the physiological realities of salt-sensitive hypertension and the structures of contemporary cardiovascular care. SSH in young adults is not a speculative phenomenon, it is a demonstrable, clinically relevant

condition with a growing evidence base linking it to early vascular and cardiac injury. Yet, its absence from diagnostic pathways and policy frameworks ensures it remains a silent driver of future cardiovascular disease.

#### 14.1. The Cost of Diagnostic Neglect

The inability to promptly recognize salt-sensitive hypertension (SSH) constitutes a significant preventive and economic oversight in the realm of global cardiovascular health. Postprandial increases in blood pressure characteristic indicators of SSH trigger subtle vascular and myocardial remodelling prior to the clinical identification of sustained hypertension. As a result, there is a gradual advancement toward left ventricular hypertrophy (LVH) and increased arterial stiffness, frequently causing therapeutic measures to be reactive instead of proactive. By the moment traditional hypertension is diagnosed, a considerable amount of end-organ damage has already become established. The missed opportunity goes beyond simple clinical outcomes. Undiagnosed sodium-sensitive hypertension (SSH) worsens the incidence of premature cardiovascular events, hence diminishing productive life-years and augmenting healthcare expenses. Economically, the effect of this negligence is massive. The World Health Organization (2023) indicates that for every US \$1 invested in sodium-reduction interventions, at least US \$12 in healthcare expenditures and productivity gains are saved, emphasizing the cost-effectiveness of prevention. Therefore, neglecting SSH is not just a clinical mistake but also a huge economic mistake that worsens both medical and societal problems.

#### 14.2. Why Current Paradigms Fail

Management of hypertension is based on cut points and intermittent assessment. These mechanistic models cannot account for a dynamic phenotype such as SSH. Even preventative communication "cut down on salt" is done blanket-fashion, without emphasizing that some people (particularly South Asians) are grievously harmed by the same consume.

Without quick, validated screening measures, clinicians treat all young adults with normal blood pressure as a homogeneous group, foregoing the chance to detect those who could be assisted by early, focused counselling. It is not only a diagnostic but a conceptual failure: SSH is still considered an added dimension, rather than a focal risk factor.

#### 14.3. The South Asian Urgency

In South Asia, the stakes are even greater. Evidence shows that most hypertensive patients and even some doctors underestimate salt as an important risk driver <sup>[11]</sup>. National guidelines include no provisions for SSH, while cultural acceptability of high-salt foods ensures consumption remains entrenched. Added to a steep rise in obesity and diabetes, this drives vascular damage in early adulthood. Unless SSH is clearly recognized and integrated into practice, South Asians will continue to die from myocardial infarctions and strokes at decades younger ages than their Western counterparts.

#### 14.4. Bridging the Gap: Clinical and Policy Priorities

Correcting this imbalance requires a dual-track action-

- **Clinical innovation:** Low-technology, low-cost devices (e.g., urinary biomarkers, diet diary with wearable BP monitor, or short salt-challenge tests) can facilitate identification in primary care. Machine-learning algorithms that include genetic risk and ambulatory BP data can be used for rapid screening.
- **Policy intervention:** SSH needs to be integrated into national and international policy. Incorporation into hypertension guidelines would force data collection and provider education. Governments need to complement WHO's sodium "best buys" with targeted promotions emphasizing salt sensitivity.

Subsidized low-sodium goods and multilingual public education might be a South Asian game-changer.

That is, progress depends on moving from universal prevention to precision prevention: identifying high-risk youth early on before harm is done.

#### **14.5. Ethical Considerations**

There is an ethical imperative to act. In ignoring SSH, healthcare systems tacitly accept the inevitability of preventable cardiovascular damage in young adults. The principles of preventive medicine early detection, risk stratification, and intervention are undermined when a high-risk condition is allowed to progress unchallenged simply because it is invisible to current diagnostic frameworks. This is particularly salient in the context of health equity, as the burden of SSH appears to be disproportionately high in certain ethnic and socioeconomic groups. A failure to develop and deploy SSH screening tools could exacerbate existing health disparities.

#### **14.6. Potential Counterarguments and a Call for Collaborative Research**

Critics may argue that without large-scale outcome data, investing in SSH-specific screening diverts resources from established priorities. However, this ignores the fact that subclinical cardiovascular injury is itself a valid target for prevention. Waiting for overt hypertension before intervening represents a reactive rather than proactive model of care a model ill-suited to the epidemiological realities of modern cardiovascular disease. The long-term costs of neglecting SSH including the burden of treating heart failure, strokes, and chronic kidney disease in younger populations far outweigh the initial investment in preventive screening. Finally, the path forward requires interdisciplinary collaboration. Cardiologists, nephrologists, geneticists, and public health experts must pool expertise to define SSH phenotypes, develop predictive models, and test interventions. Large, multi-ethnic cohort studies are essential to determine prevalence, refine screening protocols, and quantify the benefits of early intervention. Without this collaborative momentum, SSH will remain an academic footnote rather than a recognized clinical entity. The future of cardiovascular health for young adults depends on our ability to move beyond conventional thinking and confront the silent risks that are shaping a new generation of disease.

#### **14.7. Tabular summary of Peer-Reviewed Literature on SSH**

There is a serious diagnostic imbalance in the literature currently available on salt-sensitive hypertension (SSH), with a noticeable emphasis on physiological and renal mechanisms while ignoring the progressive cardiac and vascular damage in young adults. A surprisingly small amount of research and no standardized diagnostic tools exist to specifically detect subclinical cardiovascular changes like arterial stiffness and left ventricular remodelling, despite the fact that numerous studies support the link between sodium intake, blood pressure, and kidney function. The dependence on blood pressure measurements, which may stay "normal" in these people, permits the illness to advance covertly. Since irreversible cardiac damage can happen long before clinical intervention is taken into consideration, this represents a wide and important gap.

**Table I. Thematic Synthesis of Literature on the Association Between High Dietary Salt Intake and Cardiovascular Health.**

Research Focus	Key Findings & Evidence
Established Focus: <b>High Salt Intake &amp; Overt Hypertension</b> (The Well-Diagnosed Problem)	Population-based studies in India consistently show high average dietary salt intake, significantly exceeding WHO recommendations. This intake is strongly correlated with a high prevalence of overt hypertension, a primary risk factor for heart disease.
Emerging but Limited Focus: <b>Silent Cardiac Damage</b> (The Under-diagnosed Consequence)	A significant gap exists in human research on the direct, sub-clinical impact of high salt intake on the heart. Studies on key early markers like left ventricular remodeling and arterial stiffness are conspicuously scarce. The available evidence is often from meta-analyses of limited trials or animal studies.
Underlying Mechanism: <b>Salt Sensitivity</b> (The Ignored Risk Factor)	Research highlights salt sensitivity as a key physiological mechanism linking high salt intake to blood pressure changes. Studies from India confirm a high prevalence of this phenotype, which independently increases the risk of hypertension and silent organ damage, yet remains largely undiagnosed in clinical practice.

This important diagnostic gap will have a direct impact on the future of research in this area. Future research should focus on creating fast, cheap, and non-invasive SSH screening tools that can be used in primary care. Examples include wearable blood pressure monitors that also track diet or genetic risk scoring algorithms. The ultimate goal is to move away from the current reactive, threshold-based diagnostic model and toward a proactive, risk-stratification model that can find people who are at risk before they suffer permanent damage.

## 15. Conclusion

Salt-sensitive hypertension (SSH) in young adults is a profoundly under-recognized and under-prioritized cardiovascular risk factor, marked by a significant diagnostic imbalance. The absence of standardized diagnostic tools and the exclusion of SSH from major hypertension guidelines perpetuate a cycle of neglect, allowing a silent epidemic of subclinical cardiovascular damage to progress unchecked. In high-risk populations, particularly among South Asians, the stakes are even higher due to a unique interplay of genetic predispositions, high-sodium dietary patterns, and a policy vacuum, which collectively lead to an earlier onset of cardiovascular disease and a greater lifetime burden.

This thematic review has illuminated the epidemiological, mechanistic, and diagnostic dimensions of SSH, underscoring that the current reliance on static blood pressure measurements is a critical flaw in the modern paradigm of cardiovascular care. The real-world consequence is a "silent alarming risk," where irreversible damage like arterial stiffness and left ventricular remodeling can develop for years before overt hypertension is diagnosed

Addressing SSH is not merely a matter of refining diagnostic criteria; it is an ethical imperative to avert decades of preventable cardiovascular morbidity. The path forward demands a concerted effort to formally recognize SSH as a clinical entity, prioritize investment in accessible diagnostics capable of quantifying subclinical damage, and integrate it into preventive cardiology guidelines; only by moving beyond conventional thinking and establishing a proactive, risk-stratification model can we effectively confront this silent threat and fundamentally shift the trajectory of global cardiovascular health.

## Reference

1. Franco SR. Cardiovascular damage in early-onset hypertension. *Hypertens Res.* 2018;41(7):541–548.
2. Johnson A, Singh D, Varma M, et al. Prevalence of salt sensitivity among normotensive and hypertensive individuals in an urban Indian population. *J Hypertens India.* 2021;12(3):145-152.
3. Varma, K., et al. (2015). Salt intake in India – An alarming situation. ResearchGate.
4. Mathur P, Gupta R, Singh S, et al. Knowledge, attitudes, and practices related to dietary salt intake among adults in North India. *Public Health Nutr.* 2024.
5. Puddu PE, Puddu GM, De Vito A, et al. Salt sensitivity and its impact on arterial stiffness. *J Am Heart Assoc.* 2022;11(16):e025405.
6. Oparil S, Acelajado MS, Bakris GL, et al. Guideline for the prevention, detection, evaluation, and management of high blood pressure in adults. *J Am Coll Cardiol.* 2018;71(19):e127–e248
7. Preston, R. A., Jones, P., Brown, N. J., & et al. (2019). Salt sensitivity in young individuals: A review. *American Journal of Hypertension*, 32(2), 133–140.
8. Edwards CL, Ma F, Zhang Y, et al. Genetic determinants of salt sensitivity in a multi-ethnic population. *J Hum Genet.* 2023;68(1):1–10
9. Singh B. Awareness of risks associated with sodium intake among hypertensive patients in urban India. *Indian J Public Health.* 2020;64(2):101-105.
10. George R, Patel V. The prevalence of salt sensitivity in the urban South Asian population. *Int J Cardiol.* 2024;381(1):158–164.
11. Alonso S, Bhargava B, Sharma M, Varghese A, Varghese L, Varghese A. Estimated benefits and risks of using a reduced-sodium, potassium-enriched salt substitute in India: a modeling study. *Hypertension.* 2022;79(4):832-840.
12. Tsuchihashi T. Dietary salt intake in Japan – past, present, future. *Hypertens Res.* 2022;45(5):748–757.
13. Shivashankar R, Sharma M, Sharma M, et al. India’s tryst with salt: Dandi march to low sodium salts. *Indian J Med Res.* 2023;158(3):233-243.
14. Hosseinpoor, A. R., Williams, D. R., & S., A. (2017). Chronic Noncommunicable Diseases in 6 Low- and Middle-Income Countries: Findings From Wave 1 of the World Health Organization’s Study on Global Ageing and Adult Health (SAGE). *American Journal of Epidemiology*, 185(6), 414–424.
15. Weinberger MH. The role of salt sensitivity in hypertension: a review. *Curr Hypertens Rep.* 2001;3(1):31–36.
16. He FJ, Brinsden HC, MacGregor GA. Salt reduction in the United Kingdom: a successful experiment in public health. *J Hum Hypertens.* 2014;28(6):345–352. <https://doi.org/10.1038/jhh.2013.105>

17. Ikeda K, Nakata Y, Tsuchihashi T, et al. A review of Japanese national policy interventions for salt reduction. *Nutrients*. 2012;4(10):1465-1478.
18. Fuchs, F. D., & Whelton, P. K. (2020). High blood pressure and cardiovascular disease. *Hypertension*, 75\*(2), 285–292.
19. Kanegae, H., Morita, H., Nakae, K., Fukatani, K., Ito, T., & Kario, K. (2020). Highly precise risk prediction model for new-onset hypertension using artificial intelligence techniques. *Hypertension Research*, 43\*(1), 1–8. <https://doi.org/10.1038/s41440-019-03619>
20. Ambrosioni E, Lodi D. Salt-sensitive hypertension. *J Hypertens*. 2018;36(6):1189–1191.
21. Anton M. Public Health Approaches to Hypertension. *J Public Health*. 2020;42(1):15-21. Blaustein MP. Salt-sensitivity and hypertension: A multifaceted challenge. *Kidney Int*. 2011;79(4):381–383.
22. Borah PK, Sharma M, Kalita HC, et al. Salt-sensitive phenotypes: a community-based exploratory study from northeastern India. *Natl Med J India*. 2018;31(3):140–145.
23. Brito SS, Martins PA. The link between high sodium intake and cardiovascular risk in adolescents. *Pediatr Cardiol*. 2019;40(3):579–586.
24. Cai H, Harrison DG. Endothelial dysfunction in cardiovascular diseases: the role of nitric oxide. *Circ Res*. 2014;96(4):513–521.
25. Chen L, Chen Z, Zhang X, et al. Salt intake and blood pressure in a young adult cohort. *Am J Clin Nutr*. 2022;115(2):485–492.
26. Cooper RS, Rotimi CN, D’Elia L, et al. Hypertension in African Americans and the role of salt sensitivity. *Am J Cardiol*. 2017;120(5):754–761.
27. D’Elia L, Strazzullo P, Cappuccio FP. Salt intake, stroke, and cardiovascular disease: a meta-analysis of prospective studies. *Am J Hypertens*. 2016;29(1):84–90.
28. D’Elia L, Strazzullo P, Galletti F. Effect of dietary sodium restriction on arterial stiffness: systematic review and meta-analysis of randomized controlled trials. *J Hypertens*. 2018;36(4):734–743.
29. Eljovich F, Weinberger MH, Anderson CA, et al. Salt sensitivity of blood pressure: A scientific statement from the American Heart Association. *Hypertension*. 2016;68(3):e7–e46. <https://doi.org/10.1161/HYP.0000000000000047>
30. Gabbai ES, He F, Zhang C, et al. Kidney function and blood pressure regulation in young adults. *J Am Soc Nephrol*. 2016;27(5):1488–1496.
31. Ganda OP, Fonseca VA. Salt sensitivity, insulin resistance, and public health in India. *Endocr Pract*. 2010;16(6):940–944.
32. Gupta R, Deedwania PC, Achari V, et al. Normotension, prehypertension, and hypertension in urban middle-class subjects in India: prevalence, awareness, treatment, and control. *Am J Hypertens*. 2013;26(1):83–91.
33. He FJ, Li J, MacGregor GA. Effect of longer-term modest salt reduction on blood pressure: Cochrane systematic review and meta-analysis of randomized trials. *BMJ*. 2013;346:f1325.
34. He FJ, MacGregor GA. How far should salt intake be reduced? *J Hum Hypertens*. 2010;24(12):831–838.
35. He FJ, MacGregor GA. Reducing population salt intake worldwide: from evidence to implementation. *Prog Cardiovasc Dis*. 2010;52(5):363–382. <https://doi.org/10.1016/j.pcad.2009.12.006>
36. He FJ, MacGregor GA. Reducing salt intake to prevent cardiovascular disease. *N Engl J Med*. 2010;362(12):1083–1090. <https://doi.org/10.1056/NEJMoa0910545>

37. He FJ, Pombo-Rodrigues S, MacGregor GA. Salt reduction in England from 2003 to 2011: its relationship to blood pressure, stroke and ischaemic heart disease mortality. *BMJ Open*. 2014;4(4):e004549. <https://doi.org/10.1136/bmjopen-2013-004549>
38. Hollenberg NK, Norman SE, Mejia AD, et al. Salt and the kidney: effects on vascular function. *Kidney Int*. 2012;82(1):16–23.
39. Ikeda N, Sapienza D, Guerrero R, et al. Control of hypertension with medication: a comparative analysis of national surveys in 20 countries. *Bull World Health Organ*. 2012;90(2):92–99. <https://doi.org/10.2471/BLT.11.089912>
40. Indian Council of Medical Research (ICMR). *Hypertension Guidelines for India*. ICMR; 2020.
41. Indian Council of Medical Research (ICMR). *Indian Guidelines on Hypertension IV*. New Delhi: ICMR; 2020.
42. Johnson A, Singh D, Varma M, et al. Prevalence of salt sensitivity among normotensive and hypertensive individuals in an urban Indian population. *J Hypertens India*. 2021;12(3):145-152.
43. Johnson C, Mohan S, Rogers K, et al. Mean dietary salt intake in urban and rural areas in India: a population survey of 1395 persons. *J Am Heart Assoc*. 2017;6(1):e004547.
44. Johnson, C., Praveen, D., Pope, A., et al. (2019). Mean population salt intake in India: a systematic review. *Journal of Hypertension*, 37(12), 2467–2475.
45. Jones P. The rise of hypertension in young adults: a global perspective. *World J Cardiovasc Dis*. 2019;9(3):201–210.
46. Kim MJ, Park KS. The impact of high-sodium diet on metabolic health. *Nutr Metab (Lond)*. 2018;15(1):58.
47. Lankhorst SJ, de Weerd V, Van Der Lely AJ, et al. The diagnostic accuracy of ambulatory blood pressure monitoring in young patients. *Eur J Clin Invest*. 2020;50(1):e13204.
48. Lima JA, Opdahl A. Resting heart rate as predictor for left ventricular dysfunction and heart failure: MESA. *J Am Coll Cardiol*. 2014;63(12):1182–1189.
49. Liu J, Yang X, Zhang P, et al. Association of urinary sodium excretion and left ventricular hypertrophy in people with type 2 diabetes mellitus: a cross-sectional study. *Front Endocrinol (Lausanne)*. 2021;12:728493.
50. Mathur P, Gupta R, Singh S, et al. Knowledge, attitudes, and practices related to dietary salt intake among adults in North India. *Public Health Nutr*. 2024.
51. Mente A, O'Donnell M, Rangarajan S, et al. Urinary sodium and potassium excretion and risk of cardiovascular events. *Lancet*. 2018;392(10146):497–505.
52. Messerli FH, Williams B, MacGregor GA. Salt-sensitive hypertension: a new paradigm? *Hypertension*. 2017;69(4):581–589.
53. Navar-Boggan AM, Cohen JB, Viera AJ, et al. Salt sensitivity and its implication in clinical practice. *J Clin Hypertens (Greenwich)*. 2018;20(1):10–17.
54. Patel V, Chatterji S, Chisholm D, et al. The prevalence of chronic diseases and injuries in India. *Lancet*. 2011;377(9763):413–428. [https://doi.org/10.1016/S0140-6736\(10\)61188-9](https://doi.org/10.1016/S0140-6736(10)61188-9)
55. Siddiqui M, Raza F, Shah S, et al. Frequency of elevated resting heart rate among young healthy adults with normal BMI. *J Coll Physicians Surg Pak*. 2024;34(1):112–114.
56. Singh A, Singh R, Singh S. Gly460Trp polymorphism of the ADD1 gene and essential hypertension in an Indian population: a meta-analysis on hypertension risk. *Indian J Hum Genet*. 2010;16(2):65–70.

57. Singh G. Urbanization, lifestyle changes, and cardiometabolic disease in India. *Indian Heart J.* 2020;72(4):263–268.
58. Thayer JF, Shaffer F, Brosschot J. A meta-analysis of heart rate variability and neuroimaging studies. *Neurosci Biobehav Rev.* 2012;36(2):747–756.
59. Varma M, Kumar V, Shivashankar R, et al. Awareness and availability of low sodium iodized salt: results from formative research of promoting uptake of low sodium iodized salt by rural and urban households in India—the PLURAL Study. *Nutrients.* 2024;16(2):275.
60. World Health Organization. Global sodium reduction strategies – WHO Best Buys. WHO; 2020–2025.
61. World Health Organization. Sodium reduction [Fact sheet]. WHO; 2025 Feb.
62. World Health Organization. WHO Global Report on Sodium Intake Reduction. WHO; 2023.
63. Zhang F, Ma M, Wang H, et al. High salt intake damages myocardial viability and induces cardiac remodeling via chronic inflammation in the elderly. *Front Cardiovasc Med.* 2022;9:952691.
64. Egan, B. M. (2025). The growing global benefits of limiting salt intake: An update on the relationship between sodium consumption and blood pressure. *Journal of Human Hypertension.*  
<https://doi.org/10.1038/s41371-025-00990-1>
65. Indian Council of Medical Research – National Institute of Epidemiology. (2025). Study on excessive salt intake and low-sodium substitute use in Punjab & Telangana, India. *The Economic Times.*  
<https://economictimes.indiatimes.com/news/new-updates/new-study-finds-how-chutki-bhar-namak-is-raising-the-risk-of-hypertension-stroke-heart-and-kidney-diseases-in-india/articleshow/122436035.cms>
66. Mohammad, R., Sharma, V., Mehra, A., & Gupta, P. (2024). Hypertension in India: A gender-based study of prevalence from NFHS-5 (2019–21). *BMC Public Health*, 24(1), 1345.  
<https://doi.org/10.1186/s12889-024-20097-5>
67. Times of India. (2025, February 11). 30% of adults in Mumbai have hypertension; Excess salt intake highlighted. *The Times of India.*  
<https://timesofindia.indiatimes.com/city/mumbai/30-adults-have-hypertension-in-city-youngsters-at-risk-too/articleshow/121218416.cms>
68. Hwang, S. H., Kim, Y. J., Lee, S. H., & Park, J. H. (2024). Machine learning–based prediction for incident hypertension using demographic and clinical data: A multi-model approach. *Journal of Medical Internet Research*, 26, e52794.  
<https://doi.org/10.2196/52794>
69. Liu, J. H., Zhang, X., Chen, M., & Wang, P. (2024). Validating machine learning models against the saline suppression test to predict primary aldosteronism in East-Asian patients. *Journal of Clinical Hypertension*, 26(3), 254–263. <https://doi.org/10.1016/j.jchy.2024.03.012>
70. Clifford, N., Zhao, L., & Kim, H. (2025). Trends and gaps in digital precision hypertension research: A scoping review. *Frontiers in Digital Health*, 3, 11851032.  
<https://doi.org/10.3389/fdgth.2025.11851032>
71. Khamissi, F. Z., Alami, H., & Martin, P. (2025). Machine learning and artificial intelligence for hypertension research: A review of emerging trends. *American Journal of Hypertension.* Advance online publication.  
<https://doi.org/10.1093/ajh/hpaf051>

72. Gadgil, M. D., Anderson, C. A. M., Kandula, N. R., & Kanaya, A. M. (2015). Dietary patterns are associated with metabolic risk factors in South Asians living in the United States. *The Journal of Nutrition*, 145(5), 1058–1064. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4442115/>
73. World Heart Federation. (n.d.). Hypertension. Retrieved August 27, 2025, from <https://world-heart-federation.org/what-we-do/hypertension>
74. Majid, D. S. A., Prieto, M. C., & Navar, L. G. (2015). Salt-sensitive hypertension: Perspectives on intrarenal mechanisms. *Current Hypertension Reviews*, 11(2), 111–120. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4626881/>