Hypertension: Essential Hypertension, Insulin Resistance, Sugar, and Salt

Human Health Strategies® | Patient and family education | Evidence reviewed September 23, 2026

Blood pressure is the force of blood against artery walls. Hypertension means that pressure is persistently too high. It often causes no symptoms, yet over time it can injure the heart, brain, kidneys, eyes, and arteries. The encouraging fact is that accurate measurement, everyday habits, and medicines can substantially reduce risk. Treatment is not a moral judgment, and needing medication is not a failure.

This guide separates established evidence from plausible mechanisms and unresolved debate. It is general education, not a diagnosis or an individualized treatment plan. Do not stop or change a prescribed medicine because of a home reading, a diet, or something in this guide.

Key Takeaways

• “Essential” hypertension does not mean blood pressure is necessary or harmless. Historically it meant that no single disease cause was identified; today primary hypertension is understood as multifactorial.

• A diagnosis should usually rest on an average of careful readings on more than one occasion, often supported by home or ambulatory monitoring—not one rushed reading.

• For nonpregnant adults, current office categories are normal below 120/80, elevated at 120–129 and below 80, stage 1 at 130–139 or 80–89, and stage 2 at 140 or 90 and above.

• Kidney disease, primary aldosteronism, obstructive sleep apnea, thyroid disease, medicines, substances, and rarer disorders can cause or worsen hypertension. Early, severe, abrupt, or resistant hypertension deserves particular attention.

• Insulin can acutely reduce sodium excretion in human experiments. Its contribution to chronic hypertension appears variable and remains under study.

• Randomized trials show that reducing dietary sodium lowers blood pressure on average. This evidence does not support abandoning salt-reduction advice.

• Sugar-sweetened drinks are associated with later hypertension, but association is not proof of causation. Energy-matched sugar trials differ from trials that add excess calories, and food source matters.

• Lifestyle and medication are complementary. Many people need both.

• In a nonpregnant adult, new concerning symptoms with a reading higher than 180 systolic or higher than 120 diastolic require an immediate 911 call; do not delay to repeat the reading. When there are no concerning symptoms, wait at least one minute and repeat. Pregnancy has a lower urgent threshold: 160 systolic or 110 diastolic.

1. What the numbers mean

A blood-pressure reading has two numbers in millimeters of mercury, or mm Hg. Systolic pressure, the upper number, is pressure while the heart contracts. Diastolic pressure, the lower number, is pressure while the heart relaxes. Either number can place a person in a higher category.

The current 2025 AHA/ACC multisociety guideline retains these adult office categories: [1]

Category	Systolic	Diastolic
Normal	Below 120	and below 80
Elevated	120–129	and below 80
Stage 1 hypertension	130–139	or 80–89
Stage 2 hypertension	140 or higher	or 90 or higher

The words and and or matter. A reading of 118/84 is stage 1 because the diastolic number is 80–89. A reading of 134/76 is also stage 1 because the systolic number is 130–139. These categories apply to nonpregnant adults in ordinary outpatient assessment; pregnancy, childhood, and acute stroke have separate pathways.

Classification is generally based on the average of at least two careful readings on at least two occasions. Home or 24-hour ambulatory measurements can help identify a “white-coat” pattern, in which office pressure is higher, or masked hypertension, in which readings outside the clinic are higher. One elevated value during pain, anxiety, illness, caffeine use, or poor technique is information to recheck, not enough by itself to define a person's usual pressure. [1]

The guideline's general treatment goal is below 130/80 for adults, with individualized considerations for pregnancy, limited life expectancy, institutional care, medication effects, falls, and other clinical circumstances. A category is not the whole treatment decision. Cardiovascular disease, stroke, diabetes, chronic kidney disease, and estimated cardiovascular risk influence when medication is recommended. Adults averaging at least 140/90 generally need lifestyle measures plus medication; selected higher-risk adults start medication at 130/80, while lower-risk adults may first have three to six months of lifestyle treatment and reassessment. [1]

2. How common is hypertension?

In the latest dedicated national examination report, covering NHANES August 2021–August 2023, 47.7% of U.S. civilian, noninstitutionalized adults age 18 or older had hypertension. The definition was measured systolic pressure at least 130, measured diastolic pressure at least 80, or current use of blood-pressure medicine. A person controlled below 130/80 by medication was therefore still counted as having hypertension. The overall 47.7% is a crude, survey-weighted estimate; the age-adjusted estimate was 44.5%. [2]

Age	Total	Men	Women
All adults 18+ (crude)	47.7%	50.8%	44.6%
18–39	23.4%	30.0%	16.4%
40–59	52.5%	55.9%	49.0%
60+	71.6%	72.7%	70.6%

These are weighted population percentages, not the proportion of the raw 6,084-person examination sample. They describe broad groups, not an individual's destiny. NCHS reports sex as men and women and does not provide a gender-identity breakdown in this table. The survey excluded pregnant women and institutionalized populations. [2]

CDC also cites 119.9 million adults, or 48.1%, as a weighted estimate. That count comes from the older NHANES 2017–March 2020 period. It should not be presented as the head count corresponding to the newer 47.7% survey result. [3]

3. “Essential” once meant unknown; “primary” now means multifactorial

For much of medical history, hypertension without a demonstrable disease cause was called essential or idiopathic hypertension. “Essential” was a classification of what could not be attributed to a specific secondary disorder; it never meant that high pressure was essential for health. Primary hypertension is now the clearer term. [4]

Current evidence describes primary hypertension as a final common pattern produced by interacting influences: inherited susceptibility; kidney handling of sodium and water; blood-vessel structure and function; sympathetic nervous and hormonal systems; aging; adiposity and metabolic health; sodium and potassium intake; alcohol; activity; sleep; medicines; and social or environmental exposures. Different mixtures may dominate in different people, and no clinical test can usually assign exact percentages to each contributor. [1] [5]

Primary and secondary contributors can coexist. A person with a family history and longstanding primary hypertension may also develop sleep apnea, kidney disease, or a medication effect. Likewise, finding insulin resistance does not end the search when clinical clues point to another cause.

4. Secondary causes and why early onset matters

Secondary hypertension is elevated pressure attributable to another condition, medication, substance, or exposure. Finding one may change treatment, but testing should be guided by history, examination, basic laboratory results, and probability rather than indiscriminate scans and hormone panels. Clues include onset at a young age, abrupt onset or sudden worsening, very high readings, pressure uncontrolled despite an appropriate multi-drug regimen, low potassium, impaired kidney function, or organ injury out of proportion to the apparent duration. Measurement error, inconsistent medicine use, and white-coat effect should also be considered before labeling hypertension resistant. [6] [5]

Important groups include:

• Kidney disease. Kidney disease can cause hypertension, and hypertension can damage kidneys. Evaluation commonly includes creatinine/eGFR, electrolytes, and urine testing. Renal-artery disease is considered when clues such as abrupt severe hypertension, asymmetric kidneys, recurrent sudden pulmonary edema, or a marked creatinine rise after an ACE inhibitor or ARB are present.

• Primary aldosteronism. One or both adrenal glands produce aldosterone autonomously, causing renal sodium retention, volume expansion, suppressed renin, and increased cardiovascular risk. Potassium may be low, but normal potassium does not rule it out. The 2025 AHA/ACC guideline strongly recommends screening adults with resistant hypertension regardless of potassium. A separate 2025 Endocrine Society guideline conditionally suggests screening all people with hypertension when resources and feasibility allow. These recommendations differ in breadth and strength. Aldosterone, renin, potassium, sodium intake, kidney function, and medicines affect interpretation; this is clinician-directed testing, not a stand-alone mail-order result. [1] [7]

• Obstructive sleep apnea. Loud snoring, witnessed breathing pauses, daytime sleepiness, obesity, and resistant hypertension are reasons to discuss assessment, often including a sleep study. OSA can contribute, but it is not the explanation for every case, and its treatment does not automatically replace blood-pressure treatment.

• Thyroid and other endocrine disorders. Thyroid disease is an important consideration. Pheochromocytoma/paraganglioma, Cushing syndrome, and other endocrine conditions are much less common and usually warrant symptom- or sign-directed testing.

• Structural or vascular conditions. These include renal-artery fibromuscular dysplasia, especially in some younger adults, and coarctation of the aorta.

• Medicines and substances. Examples include NSAID pain relievers, decongestants and other stimulants, amphetamines and cocaine, systemic corticosteroids, estrogen-containing contraceptives, androgens, calcineurin inhibitors, some antidepressants and psychiatric medicines, certain cancer therapies, nicotine, heavy alcohol exposure, ephedra/ma huang, and licorice. An example on this list is not proof it caused one person's pressure. Review prescriptions, over-the-counter products, supplements, recreational substances, nicotine, and alcohol with a clinician or pharmacist. Do not stop a prescribed medicine on your own. [6] [1]

“Young-onset” has no single universal cutoff. Reviews often use under age 40, while some U.S. recommendations particularly flag onset before 30. Primary hypertension remains common in young adults, but selected young-onset populations have a higher frequency of secondary causes. A thoughtful assessment confirms persistent elevation, reviews family and pregnancy history, sleep, diet, alcohol and drugs, and obtains baseline kidney, electrolyte, urine, glucose/metabolic, lipid, and organ-effect information. Additional testing follows the clues. [8] [5]

5. Insulin: normal work, compensation, and a plausible kidney pathway

Insulin is a normal, necessary hormone. After eating, it helps move glucose into cells and coordinates energy storage and use. Insulin resistance means that tissues respond less effectively to a given insulin signal. The pancreas may compensate by producing more insulin, maintaining glucose for a time. This compensatory hyperinsulinemia is common in—but not identical to—obesity, prediabetes, and type 2 diabetes.

The kidney also responds to insulin. In short, tightly controlled human experiments, raising insulin while holding glucose normal reduced urinary sodium excretion. Reviews report that this acute antinatriuretic response may remain present even when insulin's glucose-lowering action is resistant, sometimes called selective insulin resistance. Proposed renal transport pathways are biologically plausible, although much transporter detail comes from cells and animals. [9] [10]

An acute fall in urinary sodium is not the same finding as chronic hypertension. Long-term pressure depends on continued sodium and water balance, vascular effects, nervous and hormonal responses, kidney function, and compensations. Chronic animal experiments have been mixed: several euglycemic insulin infusions in dogs did not produce sustained sodium retention or hypertension, and some rat results depended on accompanying hyperglycemia. Historical insulinoma observations—people with marked chronic insulin excess who did not consistently have hypertension—are counterevidence to a simple “more insulin always raises pressure” model, though insulinoma is rare and physiologically unusual. [10]

Human population evidence suggests a relationship without proving one universal pathway. A meta-analysis of prospective cohorts found a higher incidence of hypertension in the highest versus lowest fasting-insulin groups, but studies differed, residual confounding remained, and obesity, diet, activity, sleep, kidney function, and shared genetics can affect both insulin and pressure. A 2024 Mendelian-randomization study supported a modest possible causal contribution from a composite insulin-resistance phenotype; it did not isolate insulin alone or renal sodium retention. [11] [12]

Intervention evidence also counsels humility. In one randomized trial of nondiabetic hypertensive men with central adiposity, metformin lowered fasting insulin more than placebo without significantly lowering blood pressure. That narrow trial cannot exclude every role for insulin, but it shows why “lower insulin and pressure must fall” is not a reliable rule. [13]

Research on insulin, sodium handling, and blood pressure spans decades. Human antinatriuresis experiments established the short-term kidney effect, and observational youth work was published in the 1990s. In the Bogalusa cohort, persistently higher insulin tracked with higher later pressure, while the same group also had dramatically more obesity. The finding therefore reflects clustered exposures and cannot isolate insulin's contribution. Available evidence does not define insulin-mediated sodium retention as a universal mechanism of hypertension in healthy young people. [14]

The practical conclusion is balanced: insulin resistance and compensatory high insulin may contribute to blood-pressure elevation in some people, alongside adiposity-related kidney compression, sympathetic activation, renin-angiotensin-aldosterone signaling, sleep apnea, diet, and other factors. Fasting insulin or HOMA-IR is not a validated test that identifies the cause of an individual's hypertension.

6. Sugar and sugary drinks: what trials and cohorts can say

“Sugar causes hypertension” combines different exposures and different questions. Added sugar in soda, naturally occurring sugar in whole fruit, 100% juice, table sugar, and isolated fructose do not arrive in the same food matrix or necessarily have the same effects. Study design also changes the conclusion.

In controlled feeding research, isocaloric substitution means fructose-containing sugars replace other carbohydrate while calories are kept matched. A 2023 systematic review found no significant increase in systolic or diastolic pressure for total fructose-containing sugars under substitution conditions. In addition trials, sugars add calories above the comparison diet. The pooled addition result concealed opposite food-source patterns: fruit and 100% juice trials pulled estimates downward, while high-dose mixed sources that included sugar-sweetened beverages raised pressure. Many trials were short, interventions varied, and author competing interests were extensive. The accurate lesson is that energy surplus and food source both matter; fructose is not an inevitable pressure-raising molecule in every context. [15]

Prospective cohorts answer a different question: what happens later among people who report different intakes? A large meta-analysis found no clear overall association between highest and lowest total fructose intake, while another found that higher sugar-sweetened beverage intake was associated with incident hypertension. In the beverage analysis, highest intake—generally at least one daily serving—had a relative risk of 1.12 compared with none, with substantial between-study heterogeneity. Diet was not randomized; sodium, overall dietary pattern, weight, access, activity, and other factors can confound the association. A relative risk is not the percentage of cases caused by beverages and does not give one person's absolute risk. [16] [17]13717-8/pdf)

In the PREMIER lifestyle study, reducing sugary drinks was associated with lower pressure, and the estimate became smaller after adjustment for weight change. Participants were not randomized specifically to beverage reduction, so this does not prove a direct beverage effect or identify insulin as mediator. [18]

Evidence from a 2026 youth-to-adult cohort

The GUTS cohort followed 25,749 participants from a mean age near 12 to a mean age near 36 and recorded 1,625 reported hypertension diagnoses. Cumulative intake of at least two sugar-sweetened beverage servings per day, compared with fewer than three per week, was associated with a hazard ratio of 1.52. High juice intake was also associated; whole fruit and the highest-versus-lowest total-fructose comparison were not clearly associated. This strengthens temporal evidence but remains an observational cohort based on repeated self-report. The accessible publisher material is an abstract, not full-text access, and reports no insulin measurements, renal sodium balance, or insulin-mediation analysis. Accordingly, the study estimates beverage associations but does not establish the mechanism that produced them. [19]

Reducing frequently consumed sugary drinks is a reasonable practical step, especially when it also reduces excess energy. Water, unsweetened drinks, or other suitable replacements can help. Current federal guidance also limits added sugar for broader health reasons, but that numerical guidance was not derived solely from blood-pressure trials. Avoid turning a population recommendation into blame for a child or adult's diagnosis. [20]

7. Salt and sodium: strong blood-pressure evidence, variable response

Sodium is not the same as salt. Table salt is sodium chloride and is about 40% sodium by weight. Therefore:

• 2,300 mg sodium is about 5.8 g salt.

• 1,500 mg sodium is about 3.8 g salt.

• A label listing 600 mg sodium does not mean 600 mg of salt.

The 2025 AHA/ACC guideline advises staying below 2,300 mg sodium per day and aiming for an ideal limit below 1,500 mg for most adults. The National Academies advises reducing intake when it is above 2,300 mg/day for people age 14 and older. Individual clinical circumstances still matter. [1] [21]

Randomized trials directly support lower sodium for blood pressure. In DASH-Sodium, 412 adults received high, intermediate, and low sodium periods under controlled feeding. Pressure fell as sodium fell, and the DASH eating pattern provided an additional benefit. Compared with high-sodium control, low-sodium DASH produced a substantially lower average systolic pressure, with larger average differences among participants who had hypertension. The trial lasted 30 days per sodium period and did not test long-term cardiovascular events or real-world adherence. [22]

In a 2023 crossover trial of 213 adults ages 50–75, one week on a low-sodium diet versus a high-sodium diet produced an 8 mm Hg average between-group systolic difference. Pressure declined in about three quarters, but individual response varied. The unusually separated diets, short duration, and partially uncontrolled food intake mean 8 mm Hg is not a promise for every person. [23]

Salt sensitivity is a continuum, not a dependable home yes/no diagnosis. Pressure naturally varies, research cutoffs are arbitrary, and age, kidney function, environment, and cardiometabolic conditions influence response. One home experiment cannot prove someone is “salt resistant.” [24]

Most sodium reduction should not depend on throwing away the salt shaker. Packaged, prepared, and restaurant foods can deliver large amounts. Compare sodium per serving, check servings actually eaten, choose lower-sodium versions, rinse canned foods when appropriate, cook more often from basic ingredients, and build flavor with herbs, spices, citrus, vinegar, garlic, or chiles. Make changes gradually so taste can adapt.

What about potassium and salt substitutes?

Potassium-rich foods can support blood-pressure control when safe, and DASH naturally emphasizes fruits, vegetables, beans, and other potassium-containing foods. The current guideline describes 3,500–5,000 mg/day from food when clinically appropriate. Kidney disease and some medicines can impair potassium excretion, so a high-potassium diet or supplement is not automatically safe. [1]

In SSaSS, 20,995 high-risk people in rural China were assigned by village to regular salt or a substitute containing 75% sodium chloride and 25% potassium chloride. Over 4.74 years, the substitute group had fewer strokes, major cardiovascular events, and deaths. The intervention changed both sodium and potassium; participants at important hyperkalemia risk were excluded, and the setting had high discretionary salt use. It does not establish safety for every U.S. patient. [25]

WHO conditionally suggests a potassium-containing lower-sodium salt substitute for eligible adults who choose to use table salt. The recommendation excludes children, pregnant people, people with kidney impairment, and people with conditions or circumstances that compromise potassium excretion, including potassium-sparing diuretics or potassium supplements. Ask a clinician or pharmacist before use if kidney function is reduced or any medicine can raise potassium. [26]

Interpreting the low-sodium “J curve”

Some observational studies report higher cardiovascular risk at both high and very low estimated sodium intake. This is a legitimate controversy, and sodium is difficult to measure: one urine collection poorly represents usual intake, spot-urine formulas can change the apparent curve, illness can both lower intake and raise mortality, and unmeasured differences may remain. Possible adverse physiology at extremely low intake has been proposed, and every question is not settled. Randomized evidence nevertheless shows that lowering sodium reduces pressure, while SSaSS provides outcome evidence in its studied population. Together, these findings support recommended—not extreme—targets and individualized advice rather than deliberate sodium increases based on observational curves. [27] [22]

8. A practical treatment plan

Treatment usually combines several modest, sustainable changes:

• Eat a DASH-style pattern. Emphasize vegetables, fruits, whole grains, beans, nuts, and low-fat dairy as appropriate; limit saturated fat and choose lower-sodium foods. DASH and lower sodium can work independently and together.

• Move regularly. Aerobic activity, resistance exercise, and reducing sedentary time can help. Build up safely if inactive or if heart, lung, joint, neurologic, or pregnancy concerns affect exercise.

• Address weight without shame. For people with excess adiposity, sustained weight reduction often lowers pressure and improves insulin sensitivity. Weight is not the only cause, and health-supporting habits matter even when weight changes little.

• Protect sleep. A consistent sleep schedule and evaluation of snoring, witnessed apneas, or marked daytime sleepiness may identify a treatable contributor.

• Limit or avoid alcohol. The guideline's best-health goal is abstinence. If alcohol is used, reducing intake—no more than two drinks daily for men or one for women—is recommended; less may be better. Do not start drinking for heart health.

• Avoid tobacco and nicotine exposure. Smoking and nicotine acutely stress the cardiovascular system and compound overall risk.

• Manage stress realistically. Breathing practice, counseling, social support, time outdoors, and adequate sleep can complement treatment. Stress reduction is not a substitute for medication when medication is indicated.

Medicines lower pressure through different pathways. Common first-line groups include thiazide-type diuretics, ACE inhibitors, angiotensin receptor blockers, and calcium-channel blockers. Choice depends on kidney function, potassium, pregnancy potential, other diseases, side effects, cost, and interactions. Stage 2 hypertension often needs two complementary medicines. Requiring two or more drugs does not mean a person failed at lifestyle; biology frequently requires combination treatment. [1]

If you are planning pregnancy or recognize that you are pregnant, contact the prescriber promptly for a medication-safety review. Many usual blood-pressure medicines require different planning in pregnancy; ACE inhibitors, ARBs, and direct renin inhibitors can harm the fetus, particularly in the second and third trimesters. Do not stop, switch, or change the dose on your own. Labetalol and extended-release nifedipine are preferred first-line options when treatment is indicated for people planning pregnancy or already pregnant, but any change must be selected and supervised by the treating clinician. Medication safety after delivery and during breastfeeding also needs a separate review. [1]

Take medicine consistently and discuss cost, side effects, missed doses, and daily routines honestly. Do not double a dose after a missed tablet unless the prescription instructions say to. Do not stop suddenly because pressure improved—the medicine may be why it improved. If diet, activity, weight, alcohol intake, or another medicine changes substantially, pressure and medication needs may change; arrange monitoring and prescriber review.

9. Measuring at home correctly

Use a validated automatic upper-arm monitor with the correct cuff size. Wrist and finger monitors are less reliable, and cuffless smartwatch readings should not currently be used alone for diagnosis or treatment decisions. Bring the device to a visit to compare it and your technique with the office equipment. [28] [1]

For a useful reading:

1. For 30 minutes beforehand, avoid smoking or nicotine, caffeine, and exercise.

2. Empty your bladder.

3. Sit quietly for at least five minutes. Do not talk, text, or watch a stressful screen.

4. Put the cuff on bare skin. Support your back; keep feet flat and uncrossed.

5. Support the arm so the cuff is at heart level.

6. At the times recommended for you, take two readings one minute apart and record both, along with date and time.

Look for an average and pattern rather than reacting to the single highest or lowest value. Home monitoring complements clinical care; it does not authorize unsupervised medication changes. Ask how many days to measure, when to report results, and what range should trigger a call. [28]

10. Complications and why control matters

Untreated or inadequately controlled hypertension can thicken and weaken the heart, accelerate coronary artery disease, contribute to heart attack and heart failure, injure small vessels and promote stroke, damage kidney filters, affect retinal vessels and vision, and worsen disease in leg arteries. Risk generally rises over time and alongside smoking, diabetes, high cholesterol, kidney disease, and older age. Complications are serious possibilities, not inevitable outcomes.

Lowering pressure reduces risk even when no single “root cause” can be named. A plan may also include cholesterol treatment, diabetes care, kidney monitoring, smoking cessation, or sleep-apnea treatment. Blood-pressure medicine and lifestyle are not competing philosophies; they address risk through overlapping and complementary routes.

11. Urgent readings and pregnancy safety

What should a nonpregnant adult do with a reading above 180/120?

If systolic is higher than 180 OR diastolic is higher than 120 and there is chest pain, shortness of breath, back pain, numbness, weakness, a vision change, difficulty speaking, or another new concerning symptom, call 911 immediately. Do not delay the call to repeat the reading, and do not wait for the pressure to fall on its own. When there are no new concerning symptoms, wait at least one minute and repeat the measurement using correct technique. [29]

If the repeat remains above 180/120 but there are no new concerning symptoms, contact a health professional as soon as possible. Severe hypertension without evidence of acute organ injury often needs timely oral-medication initiation, restarting, or adjustment rather than rapid unsupervised lowering. Never take extra tablets unless a clinician has given a specific plan. Absence of the listed symptoms is not a guarantee of safety. [29] [1]

What if the person is pregnant or recently gave birth?

Pregnancy uses a lower severe threshold. Systolic 160 or higher OR diastolic 110 or higher is severe and requires urgent obstetric assessment; current guidance calls for confirmation within 15 minutes and prompt treatment. Do not wait for 180/120. Severe headache, vision change, upper abdominal pain, shortness of breath, chest pain, confusion, seizure, marked swelling with illness, or feeling seriously unwell warrants emergency obstetric guidance even when a home threshold card seems ambiguous. The general >180/120 pathway is for nonpregnant adults. [1]

Frequently Asked Questions

What do “essential” and “primary” hypertension mean?

The older word essential meant that clinicians could not identify one secondary disease cause; it did not mean that elevated pressure was necessary. Primary hypertension is the clearer current term. It is harmful when persistent and treatable even when its exact mix of contributors cannot be separated. [4]

How does insulin resistance fit into an individual evaluation?

A home test or one fasting-insulin value cannot identify the cause of an individual's hypertension. Insulin resistance may contribute in some people, but fasting insulin varies with timing, laboratory method, body composition, and other factors. It does not measure kidney sodium handling. A complete evaluation considers measurement quality, kidney health, sleep, medicines, aldosterone, and other clinical clues.

How does insulin-related sodium retention affect dietary advice?

An internal sodium-retention mechanism does not make dietary sodium harmless. Controlled trials show that eating less sodium lowers blood pressure on average, including among many people taking medication. The response varies, and current evidence supports recommended sodium reduction rather than deliberate salt increases. [22] [23]

What evidence links sugar-sweetened drinks and hypertension in young people?

Sugary-drink intake is associated with later hypertension, including in a cohort followed from childhood. Because that study was observational and did not establish insulin mediation, it cannot determine the independent causal contribution of the drinks or a specific pathway. Genetics, kidney and vascular regulation, adiposity, sleep, sodium, activity, medicines, alcohol, and social conditions can all matter. [19]

Does a normal potassium level rule out primary aldosteronism?

No. Many affected people do not have low potassium. Resistant hypertension is a strong reason to discuss screening; broader screening is conditionally suggested by the Endocrine Society when feasible. Testing and interpretation must account for medicines, potassium status, kidney function, and the assay. [7]

Can I stop medicine if my home pressure becomes normal?

No—not without the prescriber's plan. Normal readings may show that the medicine and lifestyle plan are working. Stopping can cause pressure to rise, and some medicines should not be stopped abruptly. Record your readings and ask whether supervised adjustment is appropriate. [28]

Is a potassium salt substitute safer than ordinary salt?

It can reduce sodium and helped a defined high-risk population, but it is not safer for everyone. Potassium accumulation can be dangerous in kidney impairment or with medicines and supplements that reduce potassium excretion. Pregnancy and childhood are outside WHO's recommendation. Ask before switching. [26] [25]

Evidence limitations

Evidence was reviewed through September 23, 2026. This guide is general education, not individualized medical advice or a substitute for a clinician's evaluation. The sources include the live corrected 2025 AHA/ACC guideline, federal surveillance, major randomized sodium trials, systematic reviews, and clearly labeled observational or mechanistic work.

Important limitations remain. Many diet and mechanism trials are short. Long-term trials cannot perfectly isolate sodium, calories, weight, insulin, sleep, and every co-exposure. Cohort studies are vulnerable to measurement error and confounding. Mendelian randomization depends on genetic-instrument assumptions. The 2026 youth-to-adult cohort was available in publisher abstract scope, not full manuscript scope. Secondary-cause evaluation and medication choice require patient-specific information; population evidence cannot predict one person's response or replace urgent care.

Sources

1. 2025 AHA/ACC Multisociety Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults

American Heart Association and American College of Cardiology | 2025-08-14; corrected through 2026-05-26 | current multisociety clinical practice guideline

https://www.ahajournals.org/doi/full/10.1161/CIR.0000000000001356

2. Hypertension Prevalence, Awareness, Treatment, and Control Among Adults Age 18 and Older: United States, August 2021–August 2023

CDC National Center for Health Statistics | 2024-10 | primary federal surveillance report

https://www.cdc.gov/nchs/products/databriefs/db511.htm

3. Estimated Hypertension Prevalence, Treatment, and Control Among U.S. Adults

CDC Million Hearts | reviewed 2023-05-12 | official population estimate

https://millionhearts.hhs.gov/data-reports/hypertension-prevalence.html

4. Primary Hypertension (Formerly Known as Essential Hypertension)

Cleveland Clinic | 2022-05-05 | academic medical center patient education

https://my.clevelandclinic.org/health/diseases/22024-primary-hypertension-formerly-known-as-essential-hypertension

5. Investigation and Treatment of High Blood Pressure in Young People

American Heart Association | 2020 | peer-reviewed scientific review

https://www.ahajournals.org/doi/full/10.1161/HYPERTENSIONAHA.119.13820

6. Secondary Hypertension: Discovering the Underlying Cause

American Academy of Family Physicians | 2017-10-01 | peer-reviewed clinical review

https://www.aafp.org/pubs/afp/issues/2017/1001/p453.html

7. Primary Aldosteronism: An Endocrine Society Clinical Practice Guideline

Endocrine Society | 2025-07-14 | clinical practice guideline

https://www.endocrine.org/clinical-practice-guidelines/primary-aldosteronism-2

8. Investigating hypertension in younger patients

BMJ | 2022-03-24 | peer-reviewed clinical review

https://www.bmj.com/content/376/bmj-2021-067924

9. Renal effects of insulin in man

Journal of Nephrology | 1997 | peer-reviewed human physiology review

https://europepmc.org/article/MED/9377725

10. Sodium-retaining effect of insulin in diabetes

American Journal of Physiology—Regulatory, Integrative and Comparative Physiology | 2012 | peer-reviewed mechanistic review

https://europepmc.org/articles/PMC3533616

11. Fasting insulin concentrations and incidence of hypertension, stroke, and coronary heart disease: a meta-analysis of prospective cohort studies

American Journal of Clinical Nutrition | 2013 | systematic review and meta-analysis of prospective cohorts

https://europepmc.org/articles/PMC3831539

12. Mendelian randomization study on insulin resistance and risk of hypertension and cardiovascular disease

Scientific Reports | 2024 | two-sample Mendelian randomization study

https://www.nature.com/articles/s41598-023-46983-3

13. Treatment with metformin of non-diabetic men with hypertension, hypertriglyceridaemia and central fat distribution: the BIGPRO 1.2 trial

BIGPRO Study Group | 2000 | randomized placebo-controlled trial abstract

https://europepmc.org/article/MED/10707032

14. Persistent elevation of plasma insulin levels is associated with increased cardiovascular risk in children and young adults

Bogalusa Heart Study / Circulation | 1996 | longitudinal observational cohort

https://www.ahajournals.org/doi/10.1161/01.cir.93.1.54

15. Fructose-containing food sources and blood pressure: a systematic review and meta-analysis of controlled feeding trials

PLOS ONE | 2023-08-15 | systematic review and meta-analysis of controlled feeding trials

https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0264802

16. Total fructose intake and risk of hypertension: a systematic review and meta-analysis of prospective cohorts

Journal of the American College of Nutrition | 2014 | systematic review and meta-analysis of prospective cohorts

https://europepmc.org/articles/PMC4261182

17. Sugar-sweetened beverage consumption and incident hypertension: a systematic review and meta-analysis of prospective cohorts

American Journal of Clinical Nutrition | 2015-08-12 | systematic review and meta-analysis of prospective cohorts

https://ajcn.nutrition.org/article/S0002-9165(23)13717-8/pdf

18. Reducing consumption of sugar-sweetened beverages is associated with reduced blood pressure

Circulation | 2010 | prospective secondary analysis

https://www.ahajournals.org/doi/full/10.1161/CIRCULATIONAHA.109.911164

19. Consumption of Fructose-Containing Food and Beverage Sources in Childhood Through to Adulthood and Risk of Hypertension: A Prospective Cohort Study

Circulation | online 2026-06-22 | prospective observational cohort; publisher abstract

https://www.ahajournals.org/doi/10.1161/CIRCULATIONAHA.125.077666

20. Get the Facts: Added Sugars

Centers for Disease Control and Prevention | reviewed 2026-04-29 | current federal nutrition guidance summary

https://www.cdc.gov/nutrition/php/data-research/added-sugars.html

21. Sodium and Potassium Dietary Reference Intake Values Updated in New Report

National Academies of Sciences, Engineering, and Medicine | 2019-03-05 | official evidence-summary release

https://www.nationalacademies.org/news/sodium-and-potassium-dietary-reference-intake-values-updated-in-new-report

22. Effects on Blood Pressure of Reduced Dietary Sodium and the DASH Diet

DASH-Sodium Collaborative Research Group / New England Journal of Medicine | 2001-01-04 | randomized controlled feeding trial

https://www.nejm.org/doi/full/10.1056/NEJM200101043440101

23. Effect of Dietary Sodium on Blood Pressure: A Crossover Trial

JAMA | 2023-12-19 | prospectively allocated crossover trial

https://europepmc.org/articles/PMC10640704

24. Salt Sensitivity of Blood Pressure: A Scientific Statement From the American Heart Association

American Heart Association | 2016-07-21 | scientific statement

https://www.ahajournals.org/doi/10.1161/HYP.0000000000000047

25. Effect of Salt Substitution on Cardiovascular Events and Death

Salt Substitute and Stroke Study / New England Journal of Medicine | 2021-09-16 | cluster-randomized clinical outcome trial
https://www.nejm.org/doi/full/10.1056/NEJMoa2105675

26. Use of lower-sodium salt substitutes: WHO guideline

World Health Organization | 2025-01 | intergovernmental guideline
https://www.who.int/publications/i/item/9789240105591

27. Sodium and health—concordance and controversy

BMJ | 2020-06-26 | peer-reviewed narrative and methods review
https://www.bmj.com/content/369/bmj.m2440

28. Home Blood Pressure Monitoring

American Heart Association | reviewed 2025-08-14 | professional patient guidance
https://www.heart.org/en/health-topics/high-blood-pressure/understanding-blood-pressure-readings/monitoring-your-blood-pressure-at-home

29. When To Call 911 About High Blood Pressure

American Heart Association | reviewed 2025-08-14 | professional emergency patient guidance
https://www.heart.org/en/health-topics/high-blood-pressure/understanding-blood-pressure-readings/when-to-call-911-for-high-blood-pressure