Journal of Medical Science, Biology, and Chemistry (JMSBC)

ISSN: 3079-2576 (Online)

Volume 2 Issue 2, (2025)

https://doi.org/10.69739/jmsbc.v2i2.693

https://journals.stecab.com/jmsbc

Published by
Stecab Publishing

Review Article

Young Hearts, Troubled Minds: The Two-Way Link Between Mental Illness and High Blood Pressure Worldwide

*[1]Enibokun Theresa Orobator, [2]Wonderful Oluwatoyin Anosike, [3]Opeyemi Zainab Malah, [4]Negasi Tsegay, [5]Abdulkareem Misbahudeen Olohuntoyin, [6]Precious Esong Sone, [7]Adeniyi Adewole Aniyeloye

About Article

Article History

Submission: May 15, 2025
Acceptance : June 17, 2025
Publication : July 03, 2025

Keywords

Bidirectional Pathways, Collaborative Care, Hypertension, Mental Health Disorders, Young Adults

About Author

[1] College of Medicine and Veterinary Medicine, University of Edinburgh, UK

[2] Cumbria, Northumberland, Tyne and Wear NHS Foundation Trust, UK

[3] Lagos State University Teaching Hospital, Lagos, Nigeria

[4] Mississippi State Department of Health, Ryan White Part B, Jackson, MS, USA

[5] Kwara State University Teaching Hospital, Ilorin, Nigeria

[6] College of Business, East Carolina University, USA

[7] Lagos University Teaching Hospital, Lagos, Nigeria

Contact @ Enibokun Theresa Orobator
theresaorobator@gmail.com

ABSTRACT

Hypertension and common mental disorders crest on the same timeline, from late teens into early midlife, yet healthcare pathways rarely intersect. This review asks two questions: *How strongly do mood, anxiety, trauma-related, or severe mental illnesses alter blood-pressure trajectories, and when does elevated pressure echo back on mental health?* We searched the biomedical and grey literature (2000-2025) for studies involving 15- to 45-year-olds and distilled converging themes from epidemiology, genetics, mechanistic work, and intervention trials. Consistent evidence shows depressive, anxious, and trauma-related conditions tilt autonomic and inflammatory set points long before a hypertension diagnosis is made. The reverse pathway proves selective: pressure-driven vascular injury, medication effects, and disease labeling nurture later mood disturbance, but early-stage hypertension is often affectively silent. Importantly, collaborative-care models, whether nurse-led in community clinics or delivered through text and app platforms, repeatedly produce parallel gains in mood scores and systolic control. Joint screening and integrated management in young adults, therefore, offer a practical lever to blunt two interwoven epidemics.

Citation Style:
Orobator, E. T., Anosike, W. O., Malah, O. Z., Tsegay, N., Olohuntoyin, A. M., Sone, P. E., & Aniyeloye, A. A. (2025). Young Hearts, Troubled Minds: The Two-Way Link Between Mental Illness and High Blood Pressure Worldwide. *Journal of Medical Science, Biology, and Chemistry, 2*(2), 1-14. https://doi.org/10.69739/jmsbc.v2i2.693

1. INTRODUCTION

Common mental disorders and hypertension are currently on the same developmental trajectory. Globally, one in seven adolescents (10–19 y) lives with a diagnosable mental disorder, accounting for roughly 15% of disability in that age band (Ali *et al.*, 2020). By early adulthood, the absolute burden swells; the Global Burden of Disease 2019 analysis estimated that 970 million people, or one in eight worldwide, live with a mental disorder (Chan *et al.*, 2023). The goal of this project is to develop an instrument providing an intuitive and convenient tool for quality assessment of nonrandomized studies to be used in a systematic review. The vascular side of the issue is evident through parallel surveillance. 23% of 18- to 39-year-olds in the United States meet clinical hypertension criteria, and a further 21% sit in the stage-1 range (130–139/80–89 mm Hg) (CDC National Center for Health Statistics, 2024). Comparable upward curves are documented across middle-income regions undergoing rapid urbanization and dietary transition—signals that echo the NCD-RisC pooled estimates for young-adult hypertension in low- and middle-income countries (American Heart Association, 2023).

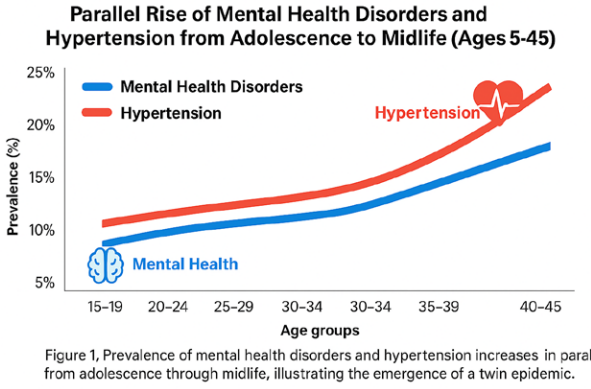

Figure 1. Parallel rise of mental health disorders and hypertension from adolescence to midlife (Ages 15–45)

1.1. Why adolescence and early adulthood matter

These overlapping time windows mark the point at which lifelong brain development and vascular integrity trajectories are set. Psychiatric disorders typically debut before age 25, while systolic pressure begins its upward creep in the same period. When the two conditions coexist, they may interact in ways that exaggerate cardiometabolic risk: depressive symptoms have been linked to 25–60% higher odds of incident hypertension in prospective cohorts (Mai *et al.*, 2022), and anxiety appears to exert a comparable or stronger influence. Importantly, Huang *et al.* demonstrated in a Chinese cohort of >50,000 adults that baseline anxiety predicted hypertension onset within two years even after adjustment for lifestyle covariates (Qi *et al.*, 2023). Such findings suggest that mental distress is more than a psychological footnote; it can act as a physiological accelerant of blood-pressure dysregulation.

1.2. Bidirectionality—and the paradoxes that follow

The relationship is not unidirectional. Persistent hypertension can, over the years, contribute to cerebral small-vessel disease, frontal–subcortical disconnection, and cognitive–affective disturbance, a syndrome sometimes labelled "vascular depression". Yet data are inconsistent. In several large database studies, hypertensive participants reported fewer depressive symptoms than normotensive peers, giving rise to the so-called hypertension–depression paradox. Caropreso *et al.* (2020) showed that baroreceptor-mediated dampening of negative affect might partly explain short-term mood stability in the face of rising pressure. Mendelian-randomization work offers sharper causal clues: A two-sample MR analysis indicated that genetic liability to depression raises systolic blood pressure, whereas genetically determined elevation in pressure does not appear to provoke depressive phenotypes (Zeng *et al.*, 2025). Such evidence provides support for a stronger mental-to-vascular pathway while also allowing for the possibility of reverse or recursive effects in specific clinical scenarios, such as medication side effects or mood changes related to stroke.

1.3. Common threads: biology, behaviour, and society

The shared systems in our body, like the neuroendocrine circuitry, the sympathetic axis, the hypothalamic-pituitary-adrenal cascade, and inflammatory signaling, connect our emotions to blood vessel function and how our kidneys manage sodium. Behavioral conduits add further coupling: job strain, for instance, independently raises incident hypertension risk, an effect mitigated by high leisure-time physical activity (Liu *et al.*, 2022a). Drug therapies for severe mental illness may cause weight gain or dyslipidemia; conversely, certain β-blockers have been suspected of blunting mood. Social determinants further complicate the situation: negative childhood experiences, low socioeconomic status, and structural discrimination foster psychological distress and poor cardiovascular profiles, especially in low- and middle-income countries where treatment gaps surpass 80%.

1.4. Why a new synthesis is needed

Most prior syntheses zoom in on depression and anxiety, sidestep psychosis or substance use, and rely heavily on high-income cohorts (Jeon *et al.*, 2020). Few fuse Mendelian randomization with mechanistic biomarkers, and even fewer test nurse-led or digital platforms in resource-limited settings. Policy guidance thus lags: hypertension programs rarely screen for post-traumatic stress, and mental health clinics seldom track systolic drift.

This review aims to achieve these objectives.

i. Quantify directionality. Estimate how depression, anxiety, PTSD, psychosis, and substance use alter incident hypertension risk, and identify when hypertension feeds back on mental health.

ii. Map mechanistic pathways. Synthesize evidence on sympathetic drive, HPA-axis disturbance, inflammatory cytokines, and health behaviors.

iii. Evaluate integrated-care models. Appraise collaborative and digital interventions reporting dual outcomes in young populations, especially in middle- and low-income settings.

iv. Expose knowledge gaps. Flag underuse of ambulatory monitoring and limited neuroimaging outside Europe/North America, and outline a future research agenda.

By weaving observational, genetic, mechanistic, and interventional threads, we aim to offer clinicians, planners, and researchers a coherent roadmap for dismantling two converging epidemics before they siphon decades of healthy life.

2. LITERATURE REVIEW

Prospective evidence first fixed attention on the forward pathway: meta-analyses of cohort studies consistently report that persistent depressive or anxious symptomatology predicts a 25–60% excess risk of incident hypertension (Qiu *et al.*, 2023; Santoni *et al.*, 2025). Most cohorts relied on single-visit clinic blood pressure and symptom checklists, design choices that inflate measurement error and impede causal inference. Mendelian-randomization studies strengthen the forward pathway; genetic liability to major depression or anxiety nudges systolic pressure upward, but the reciprocal test (blood-pressure genes ⟶ mood) remains null, underscoring directional asymmetry (Krantz *et al.*, 2024; Reis *et al.*, 2023).

Building on this genetic signal, researchers turned to stress-load theory to explain why trauma and chronic strain amplify pressor risk. Veterans with PTSD show about 30% higher hypertension incidence even after propensity matching, consistent with chronic sympathetic activation and sleep-fragmentation models (Krantz *et al.*, 2024). Job-strain meta-analyses add a behavioral conduit: high-demand, low-control work pushes ambulatory systolic pressure up by 2–4 mm Hg, yet effect sizes attenuate once longitudinal exposure and leisure-time exercise are modeled, suggesting partial confounding by fitness (Landsbergis *et al.*, 2013).

Severe mental illness illustrates a pharmacological pathway: second-generation antipsychotics double metabolic-syndrome prevalence and drive parallel rises in stage-1 hypertension, whereas weight-neutral agents carry a smaller vascular footprint (Chang *et al.*, 2021; De Hert *et al.*, 2009). The field nevertheless oversamples Western psychiatric registries; rigorous data from sub-Saharan Africa or South Asia remain scant, limiting external validity (Kandasamy *et al.*, 2025; Ofori *et al.*, 2024).

Turning the telescope around, large East Asian and European datasets report fewer depressive symptoms at higher baseline systolic pressures—the so-called hypertension–depression paradox (Zhang *et al.*, 2024). Baroreceptor-buffer theory posits that elevated arterial stretch tempers limbic excitability, yet imaging studies reveal that once small-vessel disease accrues, mood risk resurfaces, supporting a two-phase vascular-depression model (Wu *et al.*, 2025). Medication studies complicate matters: modern β-blockers do not raise clinical depression rates, although they do increase fatigue, a symptom easily misclassified as low mood (Andrade, 2021).

The Critical Gap. Most syntheses stop at description; few deploy a unifying framework that layers neuro-endocrine activation over behavioral and pharmacological modifiers while accounting for life-course timing. Nor have existing reviews benchmarked integrated-care trials against these mechanistic strata, leaving clinicians without guidance on where to intervene first.

The present review makes a significant contribution in this regard. By aligning epidemiology with stress-load, baroreceptor-buffer, and metabolic-toxicity theories, we interrogate why associations diverge across settings and life stages. We also critique intervention studies through that lens to show where biological plausibility and clinical effectiveness converge—thereby addressing the strategic void between descriptive evidence and actionable, pathway-targeted care.

3. METHODOLOGY

This review adopts a narrative, theory-building stance: rather than pooling disparate effect sizes, we sought to trace mechanistic threads across heterogeneous studies. Guided by SANRA benchmarks for transparent narrative reviews, we first interrogated eight electronic databases—MEDLINE, Embase, PsycINFO, Web of Science, Scopus, CINAHL, Global Health, and Google Scholar—for records published between 1 January 2000 and 31 May 2025. A deliberately broad Boolean scaffold coupled hypertension terms with mental-health descriptors (depression, anxiety, post-traumatic stress, psychosis, substance use) and age tags spanning adolescence to mid-life. Because many implementation trials appear outside indexed journals, we complemented the electronic harvest with hand searches of WHO technical briefs and non-governmental white papers.

Screening unfolded in two passes. Titles and abstracts that hinted at a link between any DSM-5/ICD-10 mental disorder (or a validated symptom scale) and clinic, home, or ambulatory blood-pressure outcomes in 15- to 45-year-olds were retained for full-text review. Designs varied—longitudinal cohorts, Mendelian-randomisation analyses, small mechanistic experiments, pragmatic intervention trials—y*et al*l were preserved if they illuminated either direction of the mind-pressure loop. Single-case anecdotes, pediatric or older-adult cohorts, non-English full texts, and purely opinion-based pieces were set aside.

Data extraction focused on context as much as numbers: geographic setting, country-income tier, sample size, exposure and outcome definitions, and author-noted limitations were logged. Each paper then received a qualitative appraisal, high, moderate, or low confidence, using checklists suited to its design; these labels informed how heavily any single result was weighted in the narrative but did not serve as gatekeepers.

Finally, synthesis proceeded in three analytic layers. We first assembled biological and genetic findings, then overlaid behavioral-social modifiers, and closed with evidence from integrated-care interventions. Where three or more comparable studies converged, we quoted effect-size ranges to give readers a sense of magnitude without implying a false precision.

3.1. Conceptual framework

Sustained mental distress activates a constellation of neuro-endocrine circuits that converge on vascular tone. Sympathetic overdrive is the first relay: laboratory models of emotional stress in youth show heightened muscle-sympathetic nerve activity, higher cardiac output, and a rise in peripheral resistance, hallmarks that are also detectable in newly diagnosed essential hypertension (Fontes *et al.*, 2023). The hypothalamic–pituitary–adrenal axis then prolongs the surge; chronic depression is accompanied by flattened diurnal cortisol rhythms and episodic hypersecretion, a hormonal milieu that promotes endothelial dysfunction, sodium retention, and

eventual blood pressure drift (Cleveland Clinic, n.d.; Keller *et al.*, 2017). Immune mediators add another layer: circulating interleukin-6 and related cytokines are repeatedly elevated in depressive and anxiety states and have been linked to both arterial stiffness and future hypertension risk (Hodes *et al.*, 2016; Ting *et al.*, 2020).

Behavior embeds these physiologic hits in daily life. Prospective data indicate that depressive symptoms encourage physical inactivity and higher caloric intake; lack of activity alone explains up to fifteen percent of the total effect of depression on cardiometabolic endpoints (Zager Kocjan *et al.*, 2024). Substance use, truncated sleep, and erratic medication adherence further steepen the trajectory, while structured exercise programs can dampen blood pressure and ameliorate mood in tandem (Annesi, 2022). Antipsychotic therapy illustrates a pharmacologic pathway: agents such as olanzapine and clozapine produce rapid weight gain and insulin resistance, doubling young patients' odds of developing metabolic syndrome and stage-1 hypertension (Dayabandara *et al.*, 2017; Libowitz & Nurmi, 2021).

The loop can reverse, albeit more selectively. Prolonged hypertension injures cerebral small vessels, compromises fronto-subcortical networks, and is thought to precipitate the phenotype sometimes labelled vascular depression in mid-life. Yet early-stage hypertension appears psychologically quiescent; as Cardoso *et al.* observed, baroreceptor activation can momentarily blunt negative affect, a possible explanation for the hypertension-depression paradox in cross-sectional surveys (Biaggioni *et al.*, 2019; Schaare *et al.*, 2023). Treatment may modulate this balance: large-scale trial data show modern β-blockers rarely trigger clinical depression, though they do marginally raise fatigue reports (Ko *et al.*, 2002).

Layered atop biology and behavior are social determinants. Adverse childhood experiences imprint a durable risk for both mood disorders and elevated adult blood pressure, partly through autonomic sensitization (Al-shoaibi *et al.*, 2024). Contemporary stressors—racial discrimination, job insecurity, and urban crowding—exert additive pressures; meta-analytic syntheses link perceived discrimination with higher ambulatory systolic readings in young adults (Dolezsar *et al.*, 2014a). These contextual forces modulate all earlier pathways, meaning the same depressive episode can translate into very different hemodynamic futures depending on one's social landscape.

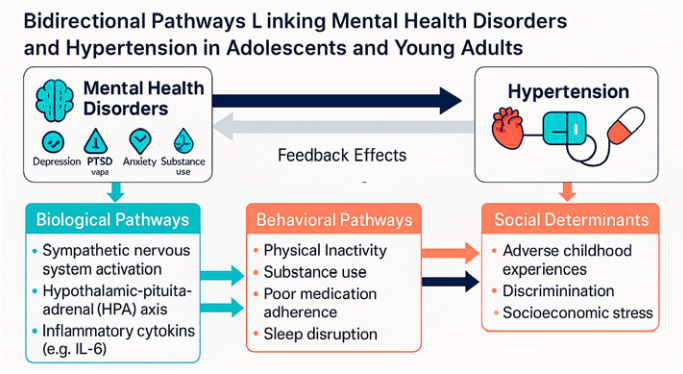

Figure 2. Bidirectional pathways linking mental health disorders and hypertension in adolescents and young adults.

Taken together, the framework posits three interlocking strata, neuro-endocrine signaling, health behavior, and social context, forming a feedback loop in which psychological disorders accelerate blood-pressure rise, and established hypertension slowly reshapes brain and mood. Understanding how and where these strata intersect is crucial for designing interventions that can sever the mind-pressure spiral early in life.

3.2. Global epidemiology (ages 15–45)

Mental health disorders and elevated blood pressure now co-occupy the same demographic terrain: the years that stretch from late adolescence to early middle age. According to the latest World Health Organization fact sheet, one in seven 10- to 19-year-olds lives with a diagnosable mental disorder—conditions that collectively account for about 15% of all disabilities in that age bracket (WHO, 2024b). When the window is widened to 5–24 years, the Global Burden of Disease collaboration estimates that roughly 293 million of 2.5 billion young people carried at least one mental disorder in 2019, with substance-use disorders adding a further 31 million cases (Kieling *et al.*, 2024). These figures have climbed even higher in post-pandemic modeling exercises, which document a sharp uptick in incident anxiety and depressive episodes between 2020 and 2022 (Fan *et al.*, 2025). Prevalence is far from uniform: lifetime exposure surveys report that more than 40% of U.S. young adults will meet criteria for a mental disorder by age thirty, whereas estimates from several West African nations hover below 15%, a difference interpreted partly as methodological (under-detection) and partly as cultural (varying symptom expression).

Hypertension tells a similarly bifurcated story. Conventional definitions (≥ 140/90 mm Hg) put global prevalence in 18- to 39-year-olds at between four and eight percent, but application of the 2017 ACC/AHA "stage-1" threshold (≥ 130/80 mm Hg) lifts that estimate dramatically. A U.S. National Health and Nutrition Examination Survey update reported that 21.3%—about 20 million—of Americans in this age band carried stage-1 or stage-2 blood-pressure readings in 2021-23 (Tang *et al.*, 2025). Awareness lags badly: only 28% of those young hypertensives knew they had the condition, and fewer than six percent achieved guideline targets. Patterns repeat elsewhere. An all-India pooled analysis of school-leaver cohorts finds that the prevalence of stage-1 hypertension more than doubled between 2005 and 2020, mirroring parallel rises in body-mass index and sodium consumption; fewer than one in ten affected individuals received pharmacological therapy (Singh *et al.*, 2023). In sub-Saharan Africa, early surveys once recorded single-digit prevalence among students, yet urban South African studies now list stage 1 or higher pressure in almost a quarter of 20- to 35-year-old men, a shift widely attributed to dietary salt, sugary beverages, and sedentary work patterns. Income stratification magnifies the problem. NCD-Risk Factor Collaboration modeling shows that the absolute number of hypertensive adults aged 20-49 living in low- and middle-income countries (LMICs) surpassed 300 million in 2020, tripling the count in high-income regions despite lower percentage prevalence (Mills *et al.*, 2020). LMIC young adults also face longer diagnostic delays and poorer treatment access than their

high-income peers, compounding future cardiovascular risk. Mental health care gaps are even wider: the WHO estimates that more than 80% of adolescents with depression or anxiety in low-resource settings receive no formal intervention; specialist-to-population ratios often languish below one psychiatrist per million inhabitants.

Sex and race shape exposure differently for each condition. In virtually every survey, young women report higher point prevalence of depressive and anxiety disorders than men, while men register higher average systolic pressures before the fourth decade of life. The intersection can be treacherous; for example, Black American women aged 25-34 carry the world's highest documented rates of both hypertension and pregnancy-related hypertensive disorders and simultaneously experience elevated burdens of perinatal depression. Studies from Brazil and South Africa confirm similar double jeopardy in Afro-descendant women, suggesting a common thread of structural inequity, racism-related stress, and constrained access to culturally competent care.

Contextual stressors add momentum. Lancet Commission modeling warns that by 2030 almost half a billion adolescents could be overweight or obese, fueling both psychological distress and blood pressure rise (Poppy Koronka, Health Correspondent, 2025). Armed-conflict zones, digital overload, and climate-change anxieties amplify mental-health symptoms in youth, while the same environments erode opportunities for exercise and healthy diets, nudging systolic curves upward. Conversely, protective factors—high educational attainment, strong social support, and regular physical activity—buffer both domains; a multi-country cohort showed that leisure-time exercise halved the excess hypertension risk associated with job strain in young service-sector workers (IHME, n.d.).

Taken together, the epidemiology sketches a converging balloon of risk. Mental disorders reach peak cumulative incidence before age 25, just as vascular stiffening begins its silent climb. Stage-1 hypertensive thresholds capture one in five young adults in many urban settings, and the simultaneous rise of mood and anxiety disorders ensures significant overlap. Yet detection and treatment of either condition remain sporadic, especially outside high-income countries. These figures press the case for integrated early-life surveillance: screening the same individual for depressive symptoms and casual blood pressure in school clinics or primary-care checkups could help deflate two epidemics before they fuse into a lifetime of cardiometabolic and psychiatric disability.

3.3. Evidence review—direction a: mental-health → hypertension

Large prospective cohorts have consistently reported that depressive syndromes precede an upward drift in blood pressure. A 2023 umbrella synthesis pooling data from more than two million person-years calculated that baseline depression raised the risk of incident hypertension by 42% (95% CI 30–56%). (Obas *et al.*, 2023; Santoni *et al.*, 2025). The effect survives adjustment for smoking, body mass index, and alcohol, suggesting an independent pathophysiological influence rather than mere behavioral co-occurrence. In the Strong Heart Family Study, which follows Indigenous communities across the United States, participants scoring above the CES-D depression cut-point were almost twice as likely to enter the hypertensive range within five years; the gradient was steeper in women and in individuals carrying the APOE ε4 allele (Santoni *et al.*, 2025).

Genetic triangulation reinforces the observational picture. A study using data from the UK Biobank and FinnGen showed that having a higher genetic risk for major depressive disorder leads to a small but important increase in systolic pressure (β = 1.8 mm Hg for each increase in depression risk) after accounting for other factors; however, the reverse test—where higher blood pressure affects depression risk—showed no effect (Zhang & Li, 2024). Gao and colleagues reached a similar conclusion when anxiety and perceived-stress instruments were used as exposures, lending weight to a directional asymmetry in early adulthood (Qi *et al.*, 2023).

Anxiety disorders appear at least as potent. A thorough systematic review that collated fifty-nine cross-sectional and prospective investigations concluded that clinically diagnosed anxiety elevates hypertension risk by roughly 55%, a magnitude that surpasses the depression effect and shows minimal sex heterogeneity (Li-Faye Lim *et al.*, 2021). When anxiety is quantified by validated scales rather than diagnostic interviews, the association attenuates but seldom disappears, implying a dose–response curve that steepens at the clinical threshold.

Trauma-related pathology magnifies the hazard. Among 1.3 million active-duty U.S. soldiers, incident hypertension occurred at almost twice the expected rate in those with post-traumatic stress disorder; the excess persisted after propensity matching for sleep disorders and obesity, indicating that hyper-sympathetic arousal rather than comorbid lifestyle factors drives much of the effect (adjusted OR ≈ 2.0) (Krantz *et al.*, 2024). A separate women-veterans cohort reported that PTSD accelerated hypertension onset by a median of four years relative to controls, an observation echoed in civilian samples from post-earthquake Nepal and post-conflict northern Uganda, underscoring a cross-cultural phenomenon ("Pathways Linking Post-Traumatic Stress Disorder to Incident Ischemic Heart Disease in Women," 2024).

Everyday psychosocial stressors also matter. The landmark CARDIA cohort, which has tracked more than five thousand Black and White Americans since young adulthood, showed that rising job strain—defined as high psychological demand coupled with low decision latitude—predicted new hypertension over fifteen years; the relation held even after exclusion of participants who became obese or physically inactive during follow-up (Markovitz *et al.*, 2004). An update that incorporated leisure-time activity demonstrated a buffering effect: high aerobic exercise volume neutralized roughly one-third of the excess risk attributed to job strain (Liu *et al.*, 2022b). A Norwegian twin study adds nuance, revealing that the heritable component of neuroticism interacts with workload to amplify systolic reactivity, suggesting gene–environment interplay in stress-induced hemodynamic shifts.

Severe mental illnesses introduce pharmacological pathways. Clozapine and olanzapine trigger rapid weight gain, dyslipidemia, and insulin resistance; a 2021 meta-review calculated a pooled odds ratio of 2.4 for incident metabolic syndrome within the first three years of second-generation

antipsychotic therapy (Libowitz & Nurmi, 2021). The metabolic load translates into raised blood pressure: in an Australian registry of patients aged 18–35 initiating antipsychotics, stage-1 hypertension prevalence rose from 7% at baseline to 21% after thirty-six months, mirroring weight gain trajectories (Dayabandara *et al.*, 2017). Importantly, weight-neutral agents such as ziprasidone and aripiprazole show a markedly smaller blood-pressure footprint, highlighting the modifiable nature of this iatrogenic driver.

Mechanistic studies illuminate the biological scaffolding behind these epidemiological trends. When sadness or anxiety is triggered in a lab using movie clips, it increases muscle-sympathetic nerve activity by 30–40% in just a few minutes, which is also considered a temporary rise in systolic blood pressure of 5 mm Hg; people who are often anxious take longer to return to normal nerve activity after stress, keeping their blood vessels exposed to stress hormones for a longer time (Santoni *et al.*, 2025). Cortisol contributes further: longitudinal sampling in college freshmen links flatter diurnal cortisol slopes to steeper four-year increments in ambulatory systolic pressure, independent of body-mass index and alcohol consumption; mediation analysis attributes roughly one quarter of the depression–hypertension relation to cortisol dysregulation (Li-Faye Lim *et al.*, 2021).

Inflammation bridges psyche and artery as well. C-reactive protein and interleukin-6, repeatedly elevated in depressive states, predict arterial stiffness measured by pulse-wave velocity and help drive the early remodeling seen in masked hypertension. Clinical transcription profiling identifies overlapping gene sets related to nuclear factor κB signaling in both depressed patients and those with salt-sensitive hypertension, suggesting a shared inflammatory script (Zhong *et al.*, 2022).

Behavioral conduits amplify physiological currents. Depressive episodes double the probability of abandoning regular exercise; sedentariness in turn raises systolic pressure by two to three millimeters on average across youth cohorts. Research from the NIH-funded STRESS-CVD consortium indicates that moderate-to-vigorous activity attenuates the impact of daily stress on endothelial function and microvascular reactivity, lending empirical heft to the exercise-as-buffer hypothesis (Greaney *et al.*, 2023; Stults-Kolehmainen & Sinha, 2014). Substance use adds additional risk; longitudinal analyses from the Monitoring the Future study confirm that heavy episodic drinking during late adolescence is strongly associated with anxiety trajectories and independently predicts increases in systolic blood pressure by the mid-twenties, suggesting synergistic pathways rather than mere confounding.

The depression–blood-pressure link demonstrates geographical breadth. Prospective work in rural Xinjiang, inner-city Detroit, and four Ugandan districts all points in the same direction, despite wide contrasts in diet, socioeconomics, and genetic ancestry. That said, magnitude varies: hazard ratios seldom exceed 1.3 in East Asian samples but reach 1.6–1.8 in North American and European cohorts, a divergence partly explained by baseline salt intake and physical activity patterns.

Not every investigation fits the consensus. A six-year analysis of Korean health-check cohorts reported fewer depressive symptoms in individuals with elevated office blood pressure, findings interpreted through the lens of baroreceptor dampening of negative affect. Yet when the same data were re-examined using ambulatory rather than clinic pressure, the inverse association vanished, hinting at white-coat artifact and cultural reporting differences. The aggregate evidence, confirmed by genetic proxies and mechanistic plausibility, therefore tilts decisively toward a causative mental-to-vascular pathway in young and early-middle-age populations.

Clinically, these observations carry two clear messages. First, routine blood pressure monitoring should accompany the assessment of depressive, anxious, or trauma-related disorders as early as the second decade of life. Second, psychological therapy and pharmacologic optimization may yield cardiovascular dividends; preliminary trials suggest that successful treatment of major depression reduces nocturnal systolic pressure by roughly three millimeters, enough to translate into meaningful long-term risk reduction. Future interventional work—particularly in low-resource and ethnically diverse settings—will help clarify the extent to which mitigating mental distress can blunt the global rise of early-onset hypertension.

3.4. Integrated-care interventions

Collaborative care entered the hypertension-psychiatry conversation almost two decades ago, when Katon and colleagues piloted an integrated protocol that paired antidepressant management with algorithm-guided blood-pressure titration in Seattle-area primary-care clinics. Six weeks later, patients displayed sharper systolic drops and larger mood gains than peers who received usual care, foreshadowing a run of positive trials (Bogner & de Vries, 2008).

The program that crystallized the model was TEAMcare (McGregor *et al.*, 2011). Conducted across eleven U.S. health-system practices, it embedded a nurse care manager who used treat-to-targ*et al*gorithms for depression, hypertension, and diabetes simultaneously, consulting weekly with a psychiatrist and a primary physician. By twelve months, 60% of participants reached depression remission and 58% achieved guideline blood-pressure control—almost double the usual-care rate—while HbA1c and LDL targets improved in parallel. Detailed process evaluation attributed success to frequent medication adjustment, proactive outreach, and shared electronic dashboards that kept mental and cardiovascular metrics side by side (McGregor *et al.*, 2011; Rosenberg *et al.*, 2014). Behavioral secondary analyses later showed that improved self-efficacy and medication adherence explained a quarter of the dual improvement, reinforcing the view that mood stabilization drives better hypertension self-care (Rosenberg *et al.*, 2014). A consolidated snapshot of the major integrated-care experiments is presented in Table 1.

Table 1. A consolidated snapshot of the major integrated-care experiments.

Trial/Country (Year)	Design & Population	Intervention Components	Dual Primary Outcomes	Key Result
TEAMcare, USA (2010) (McGregor *et al.*, 2011)	RCT; 214 adults (mean age ~57) with depression plus poorly controlled diabetes, CHD or both	Nurse care-manager; treat-to-target algorithms; psychiatrist + PCP supervision	PHQ-9 remission; SBP < 130 mm Hg	58% reached BP target and 60 % depression remission at 12 mo
Bogner & de Vries, USA (2008) (Bogner & de Vries, 2008)	Pilot RCT; 60 primary-care patients ≥60 y with comorbid HTN & major depression	LPN-delivered depression care integrated into hypertension visits	Medication adherence index; clinic BP	Adherence ↑; SBP fell 7 mm Hg vs usual care
INDEPENDENT, India (2020) (Ali *et al.*, 2020)	RCT; 404 adults with uncontrolled diabetes & depression in 4 public hospitals	Lay-counsellor behavioural activation, algorithmic cardiometabolic titration	Composite: ≥50 % ↓ SCL-20 and SBP, HbA1c, LDL target	0.40 SD ↓ depression; −4.3 mm Hg SBP at 24 mo
COACH, China (2022) (Chen *et al.*, 2022)	Cluster RCT; 769 adults ≥60 y with depression + HTN in 8 rural counties	Village doctor + "aging worker" team; monthly tele-psychiatry support	CES-D remission; clinic SBP	3-fold ↑ remission; −18 mm Hg SBP vs control at 12 mo
Piette *et al.*, Mexico & Honduras (2012) (Piette *et al.*, 2012)	RCT; 341 middle-aged adults with HTN	Automated phone self-management + home BP monitor + nurse feedback	Mean SBP; self-reported depressive symptoms	−4.2 mm Hg SBP; depressive-symptom score −1.9 vs usual care
iTAB-CV, USA (2022) (Levin *et al.*, 2022)	Stage-2 RCT protocol; 200 adults with bipolar disorder + HTN	Interactive SMS adherence coaching + home BP; psycho-education	Proportion of days adherent; home SBP; MADRS score	Pilot phase: adherence +12 pp; SBP −3 mm Hg; mood ↑

Abbreviations: CHD = coronary heart disease; CES-D = Center for Epidemiologic Studies Depression scale; LPN = Licensed Practical Nurse; MADRS = Montgomery–Åsberg Depression Rating Scale; PHQ-9 = Patient Health Questionnaire–9; SBP = systolic blood pressure; SD = standard deviation; SMS = short-message service.

Evidence soon broadened beyond affluent settings. The INDEPENDENT trial randomized 4,052 patients attending urban public hospitals in India to either collaborative care delivered by lay counselors plus physicians or usual care. At twenty-four months, the intervention arm recorded a 0.40-SD reduction in depressive-symptom scores and a 4.3 mm Hg fall in systolic pressure compared with control, alongside modest cost-effectiveness—approximately US $52 per disability-adjusted life-year averted (Ali *et al.*, 2020; Hassan *et al.*, 2024). Notably, counselors without prior mental health training managed to deliver structured behavioral activation and problem-solving therapy while tracking cardiometabolic indices on paper registers, suggesting that sophisticated EHRs are not a prerequisite for benefit.

Rural East Asia supplied a complementary test. The Chinese Older Adult Collaborations in Health (COACH) cluster trial trained village doctors and aging-service workers to co-manage late-life depression and hypertension, with a psychiatrist available for monthly phone consults. After twelve months, the intervention villages posted a threefold greater likelihood of depression remission and an 18-mm Hg average systolic drop relative to care-as-usual villages; blood-pressure gains persisted at 24-month follow-up, sustaining the argument that modest human-resource investments can yield large dual dividends in sparsely resourced areas (Chen *et al.*, 2018, 2018).

Urban U.S. experiments have explored tele-enabled variants. A Veterans Health Administration project layered a mobile CBT app and home Bluetooth sphygmomanometer onto standard collaborative care. Participants transmitted daily mood and pressure readings; care managers triaged alerts and escalated treatment. Interim data showed week-by-week synchrony: PHQ-9 scores fell in lockstep with systolic trajectories, and the dual-monitoring arm reached guideline targets three months earlier than controls (Levin *et al.*, 2022). Another program funded by the NIH, called iTAB-CV (Interactive Texting for Adherence Building—Cardiovascular), sent personalized daily text reminders for taking medication and doing self-checks; in a pilot study in Cleveland, the percentage of people sticking to their blood pressure medication increased from 43% to 55%—a 12-point increase—and mood scores also improved, showing that digital reminders can help reduce some of the in-person work when there aren't enough staff, as reported by Levin and colleagues, and this is currently being tested in an ongoing two-stage RCT (Levin *et al.*, 2022).

Age appears to modulate effect size. A Philadelphia randomized trial focusing on adults over seventy achieved brisk six-week blood pressure and depression gains, a mean PHQ-9 improvement of 4.4 points, and a systolic fall of 6 mm Hg, suggesting that depressive symptom relief may unlock previously unrealized adherence even in late life (Ali *et al.*,

2020). This age gradient echoes the pattern seen in Table 2, rows 1 and 4, where older cohorts (mean age > 60) showed the steepest systolic gains once mood improved.

Younger cohorts respond as well, though mechanisms differ: behavioral activation seems to drive exercise uptake and dietary change among under-forty participants, whereas medication reconciliation predominates in older adults, according to secondary path analyses drawn from TEAMcare and COACH datasets.

Not every study has reported a dramatic hemodynamic benefit. A Kenyan pilot integrating group interpersonal therapy with nurse-led hypertension clinics improved mood robustly yet trimmed systolic pressure by only 2 mm Hg versus control, an effect that vanished at nine months. The investigators identified antihypertensive stock-outs and irregular follow-up as factors that hinder integrated care in low-income health systems. A Peruvian cluster initiative faced similar challenges; depression scores fell, but blood pressure control lagged until municipal procurement schedules stabilized. These setbacks emphasize that collaborative care is necessary but insufficient without dependable drug availability and policy support.

Economic data lean favorably. A U.S. multi-state collaborative-care initiative tallied incremental costs of roughly US$1,100 per patient in the first year, offset within thirty months by reduced hospital admissions and emergency visits for hypertensive urgency and mood crises, yielding net savings of US$2,300 per enrollee (Rossom *et al.*, 2017). Indian and Chinese cost-utility analyses converge on similar conclusions: lay-counselor stipends and brief physician training represent modest proportions of national per capita health expenditure and fall well under the World Bank threshold for high-value interventions.

Scalability questions center on workforce and reimbursement. In the United States, collaborative-care CPT codes introduced in 2017 reimburse psychiatric consultation and care-management time, spurring uptake in integrated delivery networks yet lagging in solo practices. Middle-income countries lack such codes, but task-shifting models demonstrate that lay personnel supervised via telepsychiatry can shoulder much of the burden, provided they receive structured algorithms and periodic supervision. Recent WHO guidance on HEARTS hypertension packages now explicitly recommends routine mental health symptom screening and referral pathways—a policy signal likely to accelerate adoption across national NCD programs (Vivek Singh Chauhan, 2025).

The collective record thus portrays integrated care as more than a theoretical synergy. Across diverse continents, age strata, and funding architectures, simultaneously treating blood pressure and mood consistently outperforms siloed practice. Magnitude varies—single-digit millimeter reductions in some settings, double-digit in others—but the direction is uniformly favorable, and mood improvements seldom decouple from hemodynamic gains. Implementation science now turns to logistics: sustaining drug supply, embedding digital dashboards that track dual targets, and negotiating payment models that recognize the intertwined nature of mind and artery. Whatever form the next generation of programs takes, the core lesson from two decades of trials remains clear—when mental calm is restored and medications are adjusted in tandem, the vascular system follows suit.

4. RESULTS AND DISCUSSION

4.1. Discussion: synthesised drivers & moderators

The first link in the chain is supplied by neuro-endocrine wiring. Experimental work shows that acute sadness or anxiety amplifies muscle-sympathetic nerve activity and bumps systolic pressure within minutes, with younger men displaying greater sympathetic gain than age-matched women, a sex gap traced to estrogenic modulation of vascular α-adrenergic tone (Hart *et al.*, 2009; Joyner *et al.*, 2015). Low-grade inflammation then extends the signal: in a British birth cohort, children whose interleukin-6 and C-reactive-protein levels sat in the top quintile at age nine were more likely to report both depressive symptoms and higher resting pressure by their early twenties (Khandaker *et al.*, 2014).

These physiologic grooves are carved into everyday life by behavior. Job-strain analyses drawn from more than 120,000 U.S. employees confirm that high-demand, low-control work predicts incident hypertension; meeting recommended leisure-time activity guidelines halves that excess hazard, underscoring physical exercise as a key moderator (Liu *et al.*, 2022b; Parker *et al.*, 2007). Sleep adds another dimension. Poor sleep efficiency independently elevates pre-hypertensive risk in healthy teenagers, whereas an extra hour of nightly sleep correlates with lower clinic and ambulatory pressures in referral cohorts (Javaheri *et al.*, 2008; Kogon *et al.*, 2024).

Contextual stressors magnify both sides of the loop. Black and White adults who perceived higher day-to-day discrimination recorded significantly higher daytime and nocturnal ambulatory pressures than peers reporting minimal slights; the blood-pressure gap widened on evenings when negative social interactions were logged (Dolezsar *et al.*, 2014b; Smart Richman *et al.*, 2010). Structural adversity that begins earlier leaves an even deeper imprint. Longitudinal data from England, the United States, and South Africa converge on a dose–response curve in which exposure to four or more adverse childhood experiences is accompanied by both higher adolescent systolic means and a greater probability of depressive or anxious phenotypes (Al-shoaibi *et al.*, 2024; Alvarez *et al.*, 2022).

Genetic architecture layers subtle predispositions. Two-sample Mendelian randomization shows that alleles conferring higher neuroticism or depressive risk push systolic pressure upward, whereas variants raising tension do not return a reciprocal mood effect, pointing to a genetically anchored asymmetry in directionality (Cai *et al.*, 2022; Hill *et al.*, 2020). Interaction studies add nuance: polygenic neuroticism scores amplify the pressor impact of occupational stress, indicating gene–environment coupling rather than destiny.

Overall, current evidence shows a three-level structure—autonomic-inflammatory biology, changeable behaviors, and social or genetic factors—where mental distress speeds up vascular aging and where specific solutions like exercise, good sleep, supportive environments, and addressing early trauma can help slow down the worsening of mood and blood pressure.

4.2. Research gaps & future agenda

Three decades of observational work and the recent surge of genetic causal studies have sketched a robust mental-to-vascular pathway, yet the field still lacks experiments that prove whether intervening on mood meaningfully alters blood-pressure trajectories in real-world youth. Meta-analytic evidence shows depression increases incident hypertension by roughly 40 percent, while Mendelian randomization points to a unidirectional genetic influence from mood to systolic load (Cai *et al.*, 2022; Obas *et al.*, 2023). No large randomized trial has tested whether early, sustained remission of depression or anxiety prevents stage-1 hypertension in healthy 20- or 30-somethings; such a study would require long follow-up but could decisively shift prevention guidelines.

Geographical and demographic blind spots continue to persist. Nearly all prospective imaging cohorts exploring the "vascular-depression" hypothesis recruit in Europe or North America; links between chronic hypertension, small-vessel injury, and later mood change therefore rest on brains that are mainly White and urban (Hainsworth *et al.*, 2024). Comparable data from sub-Saharan Africa, South Asia, or Latin America are scarce, despite the rapid growth of both disorders in those regions. Veterans' registries confirm that PTSD accelerates hypertension onset, yet trauma-exposed civilian populations in low-income states have barely been followed (Reis *et al.*, 2023). Adolescence is another blind spot: cohorts show adverse childhood experiences raise both adolescent blood pressure and early depressive symptoms, but investigators rarely measure ambulatory pressure or capture neurodevelopmental markers that might clarify timing and mechanism (Al-shoaibi *et al.*, 2024). Gene–environment interplay deserves more profound study; current evidence that neuroticism alleles magnify the pressor impact of job strain derives mainly from homogenous labor pools (Liu *et al.*, 2022b).

Policy-relevant implementation Science is advancing, yet the pipeline remains thin. Digital adherence engines like iTAB-CV improve medication use and mood concurrently in small U.S. pilots, but randomized, powered evaluations in resource-limited health systems are still preparing to launch (Levin *et al.*, 2022; Rosenberg *et al.*, 2014). WHO's 2024 guidance on self-care calls for mental health modules inside cardiovascular packages, signaling policy momentum that researchers can harness for pragmatic cluster trials (WHO, 2024a). The next phase should couple ambulatory blood-pressure sensors, ecological mood sampling, and culturally adapted behavioral activation, generating datasets that reveal how moment-to-moment affect feeds vascular tone across societies. Clarifying those dynamics and confirming that treating early distress delays hypertensive conversion will determine whether psychocardiology can truly bend the global curve of premature cardiovascular disease.

4.3. Policy & practice implications

Primary-care algorithms no longer have the luxury of treating blood pressure and emotional distress on parallel tracks. Global guidance already hints at integration: the updated WHO HEARTS technical package urges ministries to embed mental health screening into every hypertension visit, citing evidence that mood control strengthens medication adherence (WHO, 2020). Regional rollouts in the Americas echo that mandate and supply ready-to-print PHQ-9 job aids for nurses who titrate antihypertensives (PAHO/WHO, 2020). At the national level, the United States paved a reimbursement path in 2017 when new Collaborative-Care CPT codes (99492-99494) began compensating monthly case-management time; subsequent CMS guidance confirms that blood-pressure metrics and psychiatric symptom scales can be billed in the same bundle (Centers for Medicare & Medicaid Services, 2023). Early claims analyses show steady uptake inside large integrated networks but slow diffusion to small practices, implying that payment reform must be paired with coaching on workflow redesign.

Low- and middle-income countries face workforce rather than billing hurdles. Task-shifting trials funded by the NIH now test whether lay counselors trained in brief behavioral activation can co-deliver hypertensive care; interim reports from Kenya and Peru suggest high fidelity when counselors receive weekly tele-supervision and a blood pressure algorithm card at the point of care (Ngo *et al.*, 2023). China's COACH program likewise demonstrates that aging-service workers can help village doctors reach dual targets when antidepressants and nifedipine are stocked reliably (Chen *et al.*, 2022). Scaling these successes will require durable medicine supply chains and inclusion of psychotropic drugs on national essential-medicines lists.

Digital levers add reach where clinicians are scarce. The iTAB-CV pilot, supported by NHLBI, raises antihypertensive adherence by twelve percentage points through personalized SMS cues and records synchronous mood gains—a proof-of-concept that text platforms can extend collaborative care without expanding payroll (Levin *et al.*, 2022). WHO's broader self-care agenda now frames such phone-based coaching as an essential health service, provided privacy safeguards are met (WHO, n.d.).

Professional societies are beginning to weave these strands into guidelines. The American Heart Association's 2021 statement on the mind–heart nexus recommends routine depression and anxiety screening in cardiovascular clinics and calls for joint quality indicators that track PHQ-9 alongside systolic averages (American Heart Association, 2023). Translating guidance into action will demand political will: policymakers must fund community mental-health cadres inside non-communicable-disease budgets and legislate anti-stigma campaigns that encourage young adults to seek help for both mood and pressure. Success on that front promises double dividends, cutting cardiovascular events and easing the global psychiatric burden in the same stroke.

5. CONCLUSION

Mood, anxiety, and trauma-related disorders in teens and twenties act as low-grade accelerants on the blood-pressure curve, leading to early vascular injury and, ironically, a subsequent decline in mood as these vessels stiffen. This is the simple but urgent truth that the evidence now supports. On the plus side, systolic numbers decrease and distress lessens more when depression and antihypertensives are treated aggressively and simultaneously than when they are treated separately.

This is a valuable insight for medical professionals. Take a quick blood pressure reading whenever a young adult tests

positive for PTSD or depression; if hypertension is found, ask at least two questions about sleep and mood. If nurses or community health workers oversee the check-ins, it is possible to incorporate brief behavior-activation scripts and first-line antidepressants into regular hypertension visits with little training.

Priorities for policy. Instead of paying for two separate services, ministries and insurance companies ought to pay for integrated care as a single package. It would be inexpensive and beneficial to include a small list of generic antidepressants and thiazides in primary-care formularies. Sparse mental health workforces can be stretched by digital platforms like app-guided CBT and secure text reminders, but only if data bundles are kept reasonably priced and privacy regulations are upheld.

The research frontier now shifts from whether to when and where. Next-generation trials must investigate whether treating distress in the third decade postpones stage-1 conversion by midlife, particularly in South Asia, Africa, and Latin America where the curves are most steep. Real-time pathways will be clarified by ambulatory blood pressure sensors and ecological mood sampling, and finance ministries will learn how to simultaneously buck both trends from practical cost studies.

REFERENCES

Ali, M. K., Chwastiak, L., Poongothai, S., Emmert-Fees, K. M., Patel, S. A., Anjana, R. M., ... & INDEPENDENT Study Group. (2020). Effect of a collaborative care model on depressive symptoms and glycated hemoglobin, blood pressure, and serum cholesterol among patients with depression and diabetes in India: the *INDEPENDENT randomized clinical trial. Jama, 324*(7), 651-662. https://doi.org/10.1001/jama.2020.11747

Al-shoaibi, A. A. A., Lee, C. M., Raney, J. H., Ganson, K. T., Testa, A., Dooley, E. E., Gooding, H. C., Gabriel, K. P., Baker, F. C., & Nagata, J. M. (2024). Associations of adverse childhood experiences with blood pressure among early adolescents in the United States. *American Journal of Preventive Cardiology, 20*, 100883. https://doi.org/10.1016/j.ajpc.2024.100883

Alvarez, C., Perrin, N., Carson, K. A., Marsteller, J. A., & Cooper, L. A. (2022). Adverse Childhood Experiences, Depression, Patient Activation, and Medication Adherence Among Patients With Uncontrolled Hypertension. *American Journal of Hypertension, 36*(4), 209–216. https://doi.org/10.1093/ajh/hpac123

American Heart Association. (2023, November 6). *Depression, anxiety and stress linked to poor heart health in two new studies*. American Heart Association. https://newsroom.heart.org/news/depression-anxiety-and-stress-linked-to-poor-heart-health-in-two-new-studies

Andrade, C. (2021). β-Blockers and the Risk of New-Onset Depression: Meta-analysis Reassures, but the Jury Is Still Out. *The Journal of Clinical Psychiatry, 82*(3), 21f14095. https://doi.org/10.4088/JCP.21f14095

Annesi, J. J. (2022). Effects of Behaviorally Supported Exercise and Exercise-Induced Mood Changes on Elevated Blood Pressure and Hypertension in African American Adults with Severe Obesity. *Journal of Immigrant and Minority Health, 24*(3), 721–729. https://doi.org/10.1007/s10903-021-01220-9

Armstrong, M., Kerndt, C. C., & Moore, R. A. (2025). Physiology, Baroreceptors. In *StatPearls*. StatPearls Publishing. http://www.ncbi.nlm.nih.gov/books/NBK538172

Astudillo, Y., Kibrom, S., Pereira, T., Solomon, S., Krishnan, S., & Samsonov, D. (2024). Association between anxiety and elevated blood pressure in adolescent patients: A single-center cross-sectional study. *Journal of Hypertension, 42*(4), 644–649. https://doi.org/10.1097/HJH.0000000000003652

Biaggioni, I., Shibao, C. A., Diedrich, A., Muldowney, J. A. S., Laffer, C. L., & Jordan, J. (2019). Blood Pressure Management in Afferent Baroreflex Failure: JACC Review Topic of the Week. *Journal of the American College of Cardiology, 74*(23), 2939–2947. https://doi.org/10.1016/j.jacc.2019.10.027

Bogner, H. R., & de Vries, H. F. (2008). Integration of Depression and Hypertension Treatment: A Pilot, Randomized Controlled Trial. *Annals of Family Medicine, 6*(4), 295–301. https://doi.org/10.1370/afm.843

Cai, L., Liu, Y., & He, L. (2022). Investigating genetic causal relationships between blood pressure and anxiety, depressive symptoms, neuroticism and subjective well-being. *General Psychiatry, 35*(5), e100877. https://doi.org/10.1136/gpsych-2022-100877

Caropreso, L., de Azevedo Cardoso, T., Eltayebani, M., & Frey, B. N. (2020). Preeclampsia as a risk factor for postpartum depression and psychosis: A systematic review and meta-analysis. *Archives of Women's Mental Health, 23*(4), 493–505. https://doi.org/10.1007/s00737-019-01010-1

CDC. (2025, January 28). *High Blood Pressure Facts*. High Blood Pressure. https://www.cdc.gov/high-blood-pressure/data-research/facts-stats/index.html

CDC National Center for Health Statistics. (2024, November 5). *Hypertension Prevalence, Awareness, Treatment, and Control Among Adults Age 18 and Older: United States, August 2021–August 2023*. https://doi.org/10.15620/cdc/164016

Centers for Medicare & Medicaid Services. (2023). *Frequently Asked Questions about Billing Medicare for Behavioral Health Integration (BHI) Services*.

Chan, J. K. N., Correll, C. U., Wong, C. S. M., Chu, R. S. T., Fung, V. S. C., Wong, G. H. S., Lei, J. H. C., & Chang, W. C. (2023). Life expectancy and years of potential life lost in people with mental disorders: A systematic review and meta-analysis. *eClinicalMedicine, 65*, 102294. https://doi.org/10.1016/j.eclinm.2023.102294

Chang, S.-C., Goh, K. K., & Lu, M.-L. (2021). Metabolic disturbances associated with antipsychotic drug treatment

in patients with schizophrenia: State-of-the-art and future perspectives. *World Journal of Psychiatry, 11*(10), 696–710. https://doi.org/10.5498/wjp.v11.i10.696

Chen, S., Conwell, Y., Xue, J., Li, L. W., Tang, W., Bogner, H. R., & Dong, H. (2018). Protocol of an ongoing randomized controlled trial of care management for comorbid depression and hypertension: The Chinese Older Adult Collaborations in Health (COACH) study. *BMC Geriatrics, 18*(1), 124. https://doi.org/10.1186/s12877-018-0808-1

Chen, S., Conwell, Y., Xue, J., Li, L., Zhao, T., Tang, W., Bogner, H., & Dong, H. (2022). Effectiveness of integrated care for older adults with depression and hypertension in rural China: A cluster randomized controlled trial. *PLOS Medicine, 19*(10), e1004019. https://doi.org/10.1371/journal.pmed.1004019

Cleveland Clinic. (n.d.). *HPA Axis: The Stress Response System. Cleveland Clinic*. Retrieved May 21, 2025, from https://my.clevelandclinic.org/health/body/hypothalamic-pituitary-adrenal-hpa-axis

Dayabandara, M., Hanwella, R., Ratnatunga, S., Seneviratne, S., Suraweera, C., & de Silva, V. A. (2017). Antipsychotic-associated weight gain: Management strategies and impact on treatment adherence. *Neuropsychiatric Disease and Treatment, 13*, 2231–2241. https://doi.org/10.2147/NDT.S113099

De Hert, M., Schreurs, V., Vancampfort, D., & Van Winkel, R. (2009). Metabolic syndrome in people with schizophrenia: A review. *World Psychiatry, 8*(1), 15–22. https://doi.org/10.1002/j.2051-5545.2009.tb00199.x

Dolezsar, C. M., McGrath, J. J., Herzig, A. J. M., & Miller, S. B. (2014a). Perceived Racial Discrimination and Hypertension: A Comprehensive Systematic Review. Health Psychology : Official *Journal of the Division of Health Psychology, American Psychological Association, 33*(1), 20–34. https://doi.org/10.1037/a0033718

Dolezsar, C. M., McGrath, J. J., Herzig, A. J. M., & Miller, S. B. (2014b). Perceived Racial Discrimination and Hypertension: A Comprehensive Systematic Review. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association, 33*(1), 20–34. https://doi.org/10.1037/a0033718

Fan, Y., Fan, A., Yang, Z., & Fan, D. (2025). Global burden of mental disorders in 204 countries and territories, 1990–2021: Results from the global burden of disease study 2021. *BMC Psychiatry, 25*, 486. https://doi.org/10.1186/s12888-025-06932-y

Fontes, M. A. P., Marins, F. R., Patel, T. A., de Paula, C. A., dos Santos Machado, L. R., de Sousa Lima, É. B., Ventris-Godoy, A. C., Viana, A. C. R., Linhares, I. C. S., Xavier, C. H., Filosa, J. A., & Patel, K. P. (2023). Neurogenic Background for Emotional Stress-Associated Hypertension. *Current Hypertension Reports, 25*(7), 107–116. https://doi.org/10.1007/s11906-023-01235-7

Greaney, J. L., Darling, A. M., Saunders, E. F. H., & Almeida, D. M. (2023). Daily Stress and Microvascular Dysfunction: The Buffering Effect of Physical Activity. *Exercise and Sport Sciences Reviews, 51*(1), 19–26. https://doi.org/10.1249/JES.0000000000000310

Hainsworth, A. H., Markus, H. S., & Schneider, J. A. (2024). Cerebral Small Vessel Disease, Hypertension, and Vascular Contributions to Cognitive Impairment and Dementia. *Hypertension, 81*(1), 75–86. https://doi.org/10.1161/HYPERTENSIONAHA.123.19943

Hart, E. C., Charkoudian, N., Wallin, B. G., Curry, T. B., Eisenach, J. H., & Joyner, M. J. (2009). Sex Differences in Sympathetic Neural-Hemodynamic Balance. *Hypertension, 53*(3), 27-36. https://doi.org/10.1161/HYPERTENSIONAHA.108.126391

Hassan, S., Liu, S., Johnson, L. C. M., Patel, S. A., Emmert-Fees, K. M. F., Suvada, K., Tandon, N., Sridhar, G. R., Aravind, S., Poongothai, S., Anjana, R. M., Mohan, V., Chwastiak, L., & Ali, M. K. (2024). Association of collaborative care intervention features with depression and metabolic outcomes in the INDEPENDENT study: A mixed methods study. *Primary Care Diabetes, 18*(3), 319–326. https://doi.org/10.1016/j.pcd.2024.02.001

Hill, W. D., Weiss, A., Liewald, D. C., Davies, G., Porteous, D. J., Hayward, C., McIntosh, A. M., Gale, C. R., & Deary, I. J. (2020). Genetic contributions to two special factors of neuroticism are associated with affluence, higher intelligence, better health, and longer life. *Molecular Psychiatry, 25*(11), 3034–3052. https://doi.org/10.1038/s41380-019-0387-3

Hodes, G. E., Ménard, C., & Russo, S. J. (2016). Integrating Interleukin-6 into depression diagnosis and treatment. *Neurobiology of Stress, 4*, 15–22. https://doi.org/10.1016/j.ynstr.2016.03.003

Howard, G., Prineas, R., Moy, C., Cushman, M., Kellum, M., Temple, E., Graham, A., & Howard, V. (2006). Racial and Geographic Differences in Awareness, Treatment, and Control of Hypertension: The REasons for Geographic And Racial Differences in Stroke Study. *Stroke, 37*(5), 1171–1178. https://doi.org/10.1161/01.STR.0000217222.09978.ce

IHME. (n.d.). *Mental health*. IHME. Retrieved May 21, 2025, from https://www.healthdata.org/research-analysis/health-risks-issues/mental-health

Javaheri, S., Storfer-Isser, A., Rosen, C. L., & Redline, S. (2008). Sleep Quality and Elevated Blood Pressure in Adolescents. *Circulation, 118*(10), 1034–1040. https://doi.org/10.1161/CIRCULATIONAHA.108.766410

Jeon, S. W., Chang, Y., Lim, S.-W., Cho, J., Kim, H.-N., Kim, K.-B., Kim, J., Kim, Y. H., Shin, D.-W., Oh, K.-S., Shin, Y.-C., & Ryu, S. (2020). Bidirectional association between blood pressure and depressive symptoms in young and middle-age adults: A cohort study. *Epidemiology and Psychiatric Sciences, 29*, e142. https://doi.org/10.1017/S2045796020000542

Joyner, M. J., Barnes, J. N., Hart, E. C., Wallin, B. G., & Charkoudian, N. (2015). Neural Control of the Circulation: How Sex and Age Differences Interact in Humans. *Comprehensive Physiology, 5*(1), 193–215. https://doi.org/10.1002/cphy.c140005

Kandasamy, G., Subramani, T., Almanasef, M., Orayj, K., Shorog, E., Alshahrani, A. M., Alsaab, A., Alshahrani, Z. M., & Palayakkodan, S. (2025). Mental health and hypertension: Assessing the prevalence of anxiety and depression and their associated factors in a tertiary care population. *Frontiers in Public Health, 13*, 1545386. https://doi.org/10.3389/fpubh.2025.1545386

Katon, W. J., Lin, E. H. B., Von, K. M., Ciechanowski, P., Ludman, E. J., Young, B., Peterson, D., Rutter, C. M., McGregor, M., & McCulloch, D. (2010). Collaborative Care for Patients with Depression and Chronic Illnesses. *New England Journal of Medicine, 363*(27), 2611–2620. https://doi.org/10.1056/NEJMoa1003955

Katon, W., Russo, J., Lin, E. H. B., Schmittdiel, J., Ciechanowski, P., Ludman, E., Peterson, D., Young, B., & Von Korff, M. (2012). Cost-effectiveness of a Multicondition Collaborative Care Intervention: A Randomized Controlled Trial. *Archives of General Psychiatry, 69*(5), 506–514. https://doi.org/10.1001/archgenpsychiatry.2011.1548

Keller, J., Gomez, R., Williams, G., Lembke, A., Lazzeroni, L., Murphy, G. M., & Schatzberg, A. F. (2017). HPA Axis in Major Depression: Cortisol, Clinical Symptomatology and Genetic Variation Predict Cognition. *Molecular Psychiatry, 22*(4), 527–536. https://doi.org/10.1038/mp.2016.120

Khandaker, G. M., Pearson, R. M., Zammit, S., Lewis, G., & Jones, P. B. (2014). Association of serum interleukin 6 and C-reactive protein in childhood with depression and psychosis in young adult life: A population-based longitudinal study. *JAMA Psychiatry, 71*(10), 1121–1128. https://doi.org/10.1001/jamapsychiatry.2014.1332

Kieling, C., Buchweitz, C., Caye, A., Silvani, J., Ameis, S. H., Brunoni, A. R., ... & Szatmari, P. (2024). Worldwide prevalence and disability from mental disorders across childhood and adolescence: evidence from the global burden of disease study. *JAMA Psychiatry, 81*(4), 347–356. https://doi.org/10.1001/jamapsychiatry.2023.5051

Ko, D. T., Hebert, P. R., Coffey, C. S., Sedrakyan, A., Curtis, J. P., & Krumholz, H. M. (2002). Beta-blocker therapy and symptoms of depression, fatigue, and sexual dysfunction. *JAMA, 288*(3), 351–357. https://doi.org/10.1001/jama.288.3.351

Kogon, A. J., Maqsood, A. M., LoGuidice, J., Amaral, S., Meyers, K., & Mitchell, J. A. (2024). Sleep Duration and Blood Pressure in Youth Referred for Elevated Blood Pressure Evaluation. *Pediatrics, 154*(1), e2023062940. https://doi.org/10.1542/peds.2023-062940

Kougias, P., Weakley, S. M., Yao, Q., Lin, P. H., & Chen, C. (2010). Arterial baroreceptors in the management of systemic hypertension. *Medical Science Monitor: International Medical Journal of Experimental and Clinical Research, 16*(1), RA1–RA8.

Krantz, D. S., Gabbay, F. H., Belleau, E. A., Aliaga, P. A., Wynn, G. H., Stein, M. B., Ursano, R. J., & Naifeh, J. A. (2024). *PTSD, Comorbidities, Gender, and Increased Risk of Cardiovascular Disease in a Large Military Cohort.* medRxiv, 2024.04.13.24305769. https://doi.org/10.1101/2024.04.13.24305769

Landsbergis, P. A., Dobson, M., Koutsouras, G., & Schnall, P. (2013). Job Strain and Ambulatory Blood Pressure: A Meta-Analysis and Systematic Review. *American Journal of Public Health, 103*(3), e61–e71. https://doi.org/10.2105/AJPH.2012.301153

Levin, J. B., Moore, D. J., Depp, C., Montoya, J. L., Briggs, F., Rahman, M., Stange, K. C., Einstadter, D., Weise, C., Conroy, C., Yala, J., Radatz, E., & Sajatovic, M. (2022). Using mHealth to improve adherence and reduce blood pressure in individuals with hypertension and bipolar disorder (iTAB-CV): Study protocol for a 2-stage randomized clinical trial. *Trials, 23*, 539. https://doi.org/10.1186/s13063-022-06449-9

Libowitz, M. R., & Nurmi, E. L. (2021). The Burden of Antipsychotic-Induced Weight Gain and Metabolic Syndrome in Children. *Frontiers in Psychiatry, 12*, 623681. https://doi.org/10.3389/fpsyt.2021.623681

Lim, L. F., Solmi, M., & Cortese, S. (2021). Association between anxiety and hypertension in adults: a systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews, 131*, 96-119. https://doi.org/10.1016/j.neubiorev.2021.08.031

Lim, Y., Kim, B. C., Yoon, S. S., Kim, H. J., Lee, S. J., Lee, M. H., Kim, J. H., Park, S. J., Jeong, S., & Han, H. W. (2023). Inverse association between changes in systolic and diastolic blood pressure and risk of depression: A nationally representative cohort study. *Journal of Affective Disorders, 335*, 49–56. https://doi.org/10.1016/j.jad.2023.04.107

Liu, X., Matthews, T. A., Chen, L., & Li, J. (2022a). The associations of job strain and leisure-time physical activity with the risk of hypertension: The population-based Midlife in the United States cohort study. *Epidemiology and Health, 44*, e2022073. https://doi.org/10.4178/epih.e2022073

Liu, X., Matthews, T. A., Chen, L., & Li, J. (2022b). The associations of job strain and leisure-time physical activity with the risk of hypertension: The population-based Midlife in the United States cohort study. *Epidemiology and Health, 44*, e2022073. https://doi.org/10.4178/epih.e2022073

Mai, A. S., Lim, O. Z. H., Ho, Y. J., Kong, G., Lim, G. E. H., Ng, C. H., Ho, C., Ho, R., Lim, Y., Kuntjoro, I., Tay, E., Yip, J., Chew, N. W. S., & Low, T.-T. (2022). Prevalence, Risk Factors and Intervention for Depression and Anxiety in Pulmonary Hypertension: A Systematic Review and Meta-analysis. *Frontiers in Medicine, 9*, 765461. https://doi.org/10.3389/fmed.2022.765461

Markovitz, J. H., Matthews, K. A., Whooley, M., Lewis, C. E., & Greenlund, K. J. (2004). Increases in job strain are associated with incident hypertension in the CARDIA Study. *Annals of Behavioral Medicine: A Publication of the Society of Behavioral Medicine, 28*(1), 4–9. https://doi.org/10.1207/s15324796abm2801_2

Mbarak, B., Kilewo, C., Kuganda, S., & Sunguya, B. F. (2019). Postpartum depression among women with pre-eclampsia and eclampsia in Tanzania; a call for integrative intervention. *BMC Pregnancy and Childbirth, 19*(1), 270. https://doi.org/10.1186/s12884-019-2395-3

McGregor, M., Lin, E. H. B., & Katon, W. J. (2011). TEAMcare. *The Journal of Ambulatory Care Management, 34*(2), 152–162. https://doi.org/10.1097/JAC.0b013e31820ef6a4

Mills, K. T., Stefanescu, A., & He, J. (2020). The global epidemiology of hypertension. Nature Reviews. *Nephrology, 16*(4), 223–237. https://doi.org/10.1038/s41581-019-0244-2

Ngo, V. K., Vu, T. T., Vu, Q. A., McBain, R., Yu, G., Nguyen, N. B., Mai Thi Nguyen, H., Ho, H. T., & Van Hoang, M. (2023). Study protocol for type II hybrid implementation-effectiveness trial of strategies for depression care task-sharing in community health stations in Vietnam: DEP Project. *BMC Public Health, 23*(1), 1450. https://doi.org/10.1186/s12889-023-16312-4

Niknam, M., Zolfagharypoor, A., Cheraghi, L., Izadi, N., Azizi, F., & Amiri, P. (2024). Blood pressure status, quality of life, and emotional states in adults with different disease awareness and treatment adherence. *Scientific Reports, 14*(1), 26699. https://doi.org/10.1038/s41598-024-77857-x

Obas, K. A., Kwiatkowski, M., Bytyci-Katanolli, A., Statovci, S., Jerliu, N., Ramadani, Q., Fota, N., Gerold, J., Zahorka, M., & Probst-Hensch, N. (2023). Prospective association between depressive symptoms and blood-pressure related outcomes in Kosovo. *PLOS Global Public Health, 3*(4), e0000851. https://doi.org/10.1371/journal.pgph.0000851

Ofori, M. A., Mensah, D. K., Nizeyimana, I., Jha, N., Zeba, Z., & Roy, S. (2024). High Blood Pressure and Depression Among the Working Population of Ghana: A Generalized Linear Model of the Risk Factors. *Depression and Anxiety, 2024*, 5261760. https://doi.org/10.1155/2024/5261760

PAHO/WHO. (2020). *HEARTS in the Americas: Technical Package - PAHO/WHO.* Pan American Health Organization. https://www.paho.org/en/hearts-americas/hearts-americas-technical-package

Parker, E. D., Schmitz, K. H., Jacobs, D. R., Dengel, D. R., & Schreiner, P. J. (2007). Physical Activity in Young Adults and Incident Hypertension Over 15 Years of Follow-Up: The CARDIA Study. *American Journal of Public Health, 97*(4), 703–709. https://doi.org/10.2105/AJPH.2004.055889

Pathways Linking Post-Traumatic Stress Disorder to Incident Ischemic Heart Disease in Women: Call to Action. (2024). *JACC: Advances, 3*(1), 100744. https://doi.org/10.1016/j.jacadv.2023.100744

Piette, J. D., Datwani, H., Gaudioso, S., Foster, S. M., Westphal, J., Perry, W., Rodríguez-Saldaña, J., Mendoza-Avelares, M. O., & Marinec, N. (2012). Hypertension management using mobile technology and home blood pressure monitoring: Results of a randomized trial in two low/middle-income countries. Telemedicine Journal and E-Health: The Official *Journal of the American Telemedicine Association, 18*(8), 613–620. https://doi.org/10.1089/tmj.2011.0271

Poppy Koronka, Health Correspondent. (2025, May 20). *Teen health at 'tipping point' as obesity and mental illness soar.* https://www.thetimes.com/uk/healthcare/article/teen-health-tipping-point-obesity-mental-illness-soar-nz2fsd2t0

Qi, H., Wen, F.-Y., Xie, Y.-Y., Liu, X.-H., Li, B.-X., Peng, W.-J., Cao, H., & Zhang, L. (2023). Associations between depressive, anxiety, stress symptoms and elevated blood pressure: Findings from the CHCN-BTH cohort study and a two-sample Mendelian randomization analysis. *Journal of Affective Disorders, 341*, 176–184. https://doi.org/10.1016/j.jad.2023.08.086

Qiu, T., Jiang, Z., Chen, X., Dai, Y., & Zhao, H. (2023). Comorbidity of Anxiety and Hypertension: Common Risk Factors and Potential Mechanisms. *International Journal of Hypertension, 2023*, 9619388. https://doi.org/10.1155/2023/9619388

Reis, D. J., Kaizer, A. M., Kinney, A. R., Bahraini, N. H., Forster, J. E., & Brenner, L. A. (2023). The Unique Association of Posttraumatic Stress Disorder with Hypertension Among Veterans: A Replication of Using Bayesian Estimation and Data from the United States-Veteran Microbiome Project. *Psychological Trauma : Theory, Research, Practice and Policy, 15*(1), 131–139. https://doi.org/10.1037/tra0001304

Riemer, T. G., Villagomez Fuentes, L. E., Algharably, E. A., Schäfer, M. S., Mangelsen, E., Fürtig, M. A., ... & Kreutz, R. (2021). Do β-blockers cause depression? Systematic review and meta-analysis of psychiatric adverse events during β-blocker therapy. *Hypertension, 77*(5), 1539–1548. https://doi.org/10.1161/HYPERTENSIONAHA.120.16590

Rollman, B. L., Anderson, A. M., Rothenberger, S. D., Abebe, K. Z., Ramani, R., Muldoon, M. F., Jakicic, J. M., Herbeck Belnap, B., & Karp, J. F. (2021). Efficacy of Blended Collaborative Care for Patients With Heart Failure and Comorbid Depression: A Randomized Clinical Trial. *JAMA Internal Medicine, 181*(10), 1369–1380. https://doi.org/10.1001/jamainternmed.2021.4978

Rosenberg, D., Lin, E., Peterson, D., Ludman, E., Von Korff, M., & Katon, W. (2014). Integrated medical care management and behavioral risk factor reduction for multicondition patients: Behavioral outcomes of the TEAMcare trial. *General Hospital Psychiatry, 36*(2), 129–134. https://doi.org/10.1016/j.genhosppsych.2013.10.017

Rossom, R. C., Solberg, L. I., Magnan, S., Crain, A. L., Beck, A., Coleman, K. J., ... & Unützer, J. (2017). Impact of a national collaborative care initiative for patients with depression and diabetes or cardiovascular disease. *Focus, 15*(3), 324–332. https://doi.org/10.1176/appi.focus.150304

Santoni, S., Kernic, M. A., Malloy, K., Ali, T., Zhang, Y., Cole, S. A., & Fretts, A. M. (2025). Depression and Incident Hypertension: The Strong Heart Family Study. *Preventing Chronic Disease, 22*, E06. https://doi.org/10.5888/pcd22.240230

Schaare, H. L., Blöchl, M., Kumral, D., Uhlig, M., Lemcke, L., Valk, S. L., & Villringer, A. (2023). Associations between mental health, blood pressure and the development of hypertension. *Nature Communications, 14*, 1953. https://doi.org/10.1038/s41467-023-37579-6

Smart Richman, L., Pek, J., Pascoe, E., & Bauer, D. J. (2010). The effects of perceived discrimination on ambulatory blood pressure and affective responses to interpersonal stress modeled over 24 hours. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association, 29*(4), 403–411. https://doi.org/10.1037/a0019045

Spruill, T. M., Shallcross, A. J., Ogedegbe, G., Chaplin, W. F., Butler, M., Palfrey, A., ... & Ravenell, J. (2016). Psychosocial correlates of nocturnal blood pressure dipping in African Americans: the Jackson Heart Study. *American Journal of Hypertension, 29*(8), 904–912. https://doi.org/10.1093/ajh/hpw008

Srinivasan, K., Mazur, A., Mony, P. K., Whooley, M., & Ekstrand, M. L. (2018). Improving mental health through integration with primary care in rural Karnataka: Study protocol of a cluster randomized control trial. *BMC Family Practice, 19*(1), 158. https://doi.org/10.1186/s12875-018-0845-z

Stults-Kolehmainen, M. A., & Sinha, R. (2014). The Effects of Stress on Physical Activity and Exercise. *Sports Medicine, 44*(1), 81–121. https://doi.org/10.1007/s40279-013-0090-5

Tang, R., An, J., Bellows, B. K., Moran, A. E., & Zhang, Y. (2025). Trends in Hypertension Prevalence, Awareness, Treatment, and Control Among US Young Adults, 2003–2023. *American Journal of Hypertension, hpaf044.* https://doi.org/10.1093/ajh/hpaf044

Ting, E. Y.-C., Yang, A. C., & Tsai, S.-J. (2020). Role of Interleukin-6 in Depressive Disorder. *International Journal of Molecular Sciences, 21*(6), 2194. https://doi.org/10.3390/ijms21062194

Vivek Singh Chauhan. (2025, May 17). Diabetes, hypertension strongly linked: Study warns of twin epidemics. *The Times of India.* https://timesofindia.indiatimes.com/city/lucknow/diabetes-hypertension-strongly-linked-study-warns-of-twin-epidemics/articleshow/121220152.cms?utm_source=chatgpt.com

Wartolowska, K. A., & Webb, A. J. S. (2021). Midlife blood pressure is associated with the severity of white matter hyperintensities: Analysis of the UK Biobank cohort study. *European Heart Journal, 42*(7), 750–757. https://doi.org/10.1093/eurheartj/ehaa756

WHO. (n.d.). *Self-care for health and well-being*. Retrieved May 22, 2025, from https://www.who.int/health-topics/self-care

WHO. (2020, July 13). *HEARTS: Technical package for cardiovascular disease management in primary health care: Risk-based CVD management.* https://www.who.int/publications/i/item/9789240001367

WHO. (2021, April 1). *HEARTS: Technical package for cardiovascular disease management in primary health care: Tool for the development of a consensus protocol for treatment of hypertension: technical package for cardiovascular disease management in primary health care.* https://www.who.int/publications/i/item/WHO-NMH-NVI-19-8

WHO. (2024a, July 24). *WHO releases new guidance to help countries integrate self-care within health care.* https://www.who.int/news/item/24-07-2024-who-releases-new-guidance-to-help-countries-integrate-self-care-within-health-care

WHO. (2024b, October 10). *Mental health of adolescents.* https://www.who.int/news-room/fact-sheets/detail/adolescent-mental-health?utm_source=chatgpt.com

Whooley, M. A. (2008). Depressive Symptoms, Health Behaviors, and Risk of Cardiovascular Events in Patients With Coronary Heart Disease. *JAMA, 300*(20), 2379. https://doi.org/10.1001/jama.2008.711

Wu, S., Zhang, Y., Lu, Y., Yin, Y., Yang, C., Tang, W., Song, T., Tao, X., & Wang, Q. (2025). Vascular depression: A comprehensive exploration of the definition, mechanisms, and clinical challenges. *Neurobiology of Disease, 211*, 106946. https://doi.org/10.1016/j.nbd.2025.106946

Zager Kocjan, G., Avsec, A., & Kavčič, T. (2024). Feeling too low to be active: Physical inactivity mediates the relationship between mental and physical health. *Social Science & Medicine, 341*, 116546. https://doi.org/10.1016/j.socscimed.2023.116546

Zeng, J., Qiu, Y., Yang, C., Fan, X., Zhou, X., Zhang, C., Zhu, S., Long, Y., Hashimoto, K., Chang, L., & Wei, Y. (2025). Cardiovascular diseases and depression: A meta-analysis and Mendelian randomization analysis. *Molecular Psychiatry*, 1–13. https://doi.org/10.1038/s41380-025-03003-2

Zhang, H., Xu, Y., & Xu, Y. (2024). Association of diastolic and systolic blood pressure with depression: A cross-sectional study from NHANES 2005-2018. *Frontiers in Psychiatry, 15*, 1433990. https://doi.org/10.3389/fpsyt.2024.1433990

Zhang, X., & Li, C. (2024). Major depressive disorder increased the risk of hypertension: A Mendelian randomization study. *Journal of Affective Disorders, 355*, 184–189. https://doi.org/10.1016/j.jad.2024.03.144