Stroke

FOCUSED UPDATES: BLOOD PRESSURE

Blood Pressure and Stroke: A Review of Sex- and Ethnic/Racial-Specific Attributes to the Epidemiology, Pathophysiology, and Management of Raised Blood Pressure

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ABSTRACT: Raised blood pressure (BP) is the leading cause of death and disability worldwide, and its particular strong association with stroke is well established. Although systolic BP increases with age in both sexes, raised BP is more prevalent in males in early adulthood, overtaken by females at middle age, consistently across all ethnicities/races. However, there are clear regional differences on when females overtake males. Higher BP among males is observed until the seventh decade of life in high-income countries, compared with almost 3 decades earlier in low- and middle-income countries. Females and males tend to have different cardiovascular disease risk profiles, and many lifestyles also influence BP and cardiovascular disease in a sex-specific manner. Although no hypertension guidelines distinguish between sexes in BP thresholds to define or treat hypertension, observational evidence suggests that in terms of stroke risk, females would benefit from lower BP thresholds to the magnitude of 10 to 20 mm Hg. More randomized evidence is needed to determine if females have greater cardiovascular benefits from lowering BP and whether optimal BP is lower in females. Since 1990, the number of people with hypertension worldwide has doubled, with most of the increase occurring in low- and-middle-income countries where the greatest population growth was also seen. Sub-Saharan Africa, Oceania, and South Asia have the lowest detection, treatment, and control rates. High BP has a more significant effect on the burden of stroke among Black and Asian individuals than Whites, possibly attributable to differences in lifestyle, socioeconomic status, and health system resources. Although pharmacological therapy is recommended differently in local guidelines, recommendations on lifestyle modification are often very similar (salt restriction, increased potassium intake, reducing weight and alcohol, smoking cessation). This overall enhanced understanding of the sex- and ethnic/racial-specific attributes to BP motivates further scientific discovery to develop more effective prevention and treatment strategies to prevent stroke in high-risk populations.

Key Words: blood pressure ■ ethnicity hypertension ■ race ■ sex ■ systematic review

Raised blood pressure (BP) is a complex disorder involving multiple organ systems and is the primary modifiable risk factor for stroke, which remains the second leading cause of death and disability worldwide.[1] A 20 mm Hg increase in systolic BP (SBP) is associated with a 35% greater risk for ischemic stroke (95% CI, 1.28–1.42), 44% (95% CI, 1.32–1.58) for intracerebral hemorrhage, and 43% (95% CI, 1.25–1.63) for subarachnoid hemorrhage.[2] The mortality from stroke doubles with each 20 mm Hg increase of SBP or 10 mm Hg increase of diastolic BP.[3]

See related articles, p 1052, 1054, 1065, 1074, 1085, 1104

Since 1990, the number of people with hypertension worldwide has doubled, with most of the increase occurring in low- and middle-income countries.[4] In high-income countries, prevalence has declined while health systems have achieved treatment rates of up to 80% and control rates of up to 60%.[4] However, in low- and middle-income countries, only 1 in 3 are aware of their hypertension status, and ≈8% have their BP controlled.[5]

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Supplemental Material is available at https://www.ahajournals.org/doi/suppl/10.1161/STROKEAHA.121.035852.
For Sources of Funding and Disclosures, see page 1124.

Stroke is available at www.ahajournals.org/journal/str

Nonstandard Abbreviations and Acronyms

ACC	American College of Cardiology
ACE	angiotensin-converting enzyme
ALLHAT	Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial
AT1R	angiotensin II type 1 receptor
BP	blood pressure
CCB	calcium channel blocker
CVD	cardiovascular disease
ESC/ESH	the European Society of Cardiology and the European Society of Hypertension
HDP	hypertensive disorders of pregnancy
HELIUS	Healthy Life in an Urban Setting
HIC	high-income country
HR	hazard ratio
INTERSTROKE	Importance of Conventional and Emerging Risk Factors of Stroke in Different Regions and Ethnic Groups of the World
MMM	May Measurement Month
NHANES	National Health and Nutrition Examination Survey
RAS	renin-angiotensin system
REGARDS	Geographic and Racial Differences in Stroke
SBP	systolic blood pressure

Mounting evidence has also highlighted differences between females and males in the manifestation of common cardiovascular diseases (CVDs). Although premenopausal females have a lower incidence and severity of hypertension—and therefore a lower incidence of CVD—than males, the risk increases sharply after menopause.[6] Nevertheless, females remain under-represented in preclinical animal studies and clinical trials in humans.[7,8] There is evidence that females are undertreated both in primary and secondary prevention of CVDs compared with males.[9]

In this review, we discuss the available evidence regarding sex and ethnic/racial differences in the epidemiology, pathophysiology, and management of raised BP, especially in relation to stroke. We are using sex assuming the biological context has been reported, ethnicity in the restricted geographic sense, and race is a culturally structured systematic way of looking at, perceiving, and interpreting reality.[10–12] However, we acknowledge that modern thinking on sex/sex and race/ethnicity postdates most of the studies we review such that distinguishing one from the other is challenging. We performed a systematic review for each topic, with the methods presented in the Supplemental Data. These included a PRISMA flow diagram for each review (Flow diagram S1 through S7).

SEX DIFFERENCES

Hypertension Prevalence by Sex

The Non-Communicable Disease Risk Factor Collaboration used data from population-representative studies on people aged 30 to 79 years in 184 countries, covering 99% of the global population.[4] The estimated global age-standardized prevalence of hypertension (defined as SBP/diastolic BP≥140/90 mm Hg, or current use of antihypertensive medication)[4] was 32% (95% CI, 30%–34%) in females and 34% (95% CI, 32%–37%) in males in 2019. This finding is consistent with a meta-analysis including 135 population-based studies from 90 countries,[13] where the age-standardized prevalence of hypertension was 30% in females (95% CI, 29%–32%) and 32% in males (95% CI, 30%–34%) aged ≥20 years. Despite lower prevalence rates in females, diagnosis, treatment and control rates are higher than in males. Globally, more females (59% [95% CI, 55–62]) than males (49% [95% CI, 46–52]) were diagnosed with hypertension. The treatment rate was 47% (95% CI, 43–51) in females and 38% (95% CI, 35–41) in males. Less than half of those treated had achieved hypertension control, leading to global control rates of only 23% (95% CI, 20–27) for females and 18% (95% CI, 16–21) for males with hypertension.[4]

Hypertension Prevalence by Sex and Age

Of 31 meta-analyses reviewed (Table S1), 26 meta-analyses did not find sex differences in hypertension prevalence in the general population. Four studies analyzed data from population-based studies in south Asia[14–16] and the Niger Delta,[17] reporting that males were more likely to have hypertension than females. However, most of the included studies enrolled relatively young patients of <65 or 70 years with little consideration of confounders that might explain the greater prevalence of hypertension among males than females in the analyses. Six studies[13,18–22] reported hypertension in a sex- and age-dependent manner, showing that hypertension prevalence increases with age in both sexes, but it is more prevalent in males at early adulthood and in females beginning at middle age, which is consistent across all regions. However, the quality and publication bias of the included studies were not assessed, which might impact the validity of the conclusions. Mills et al[13] estimated that among 20- to 29-year-old adults globally, 9% (95% CI, 7%–12%) of females but 15% (95% CI, 11%–18%) of males had hypertension, but a steeper rise in hypertension rates is seen in the female after menopause. This is reflected in the hypertension prevalence rates in the

elderly of over 70 years, with the estimated rate of 76% (95% CI, 73%–80%) and 69% (95% CI, 65%–72%) for females and males, respectively. A similar trend was also observed in the Non-Communicable Disease Risk Factor Collaboration study[22] (Figure 1). Those were further confirmed by the MMM (May Measurement Month) 2018 study, which was an opportunistic cross-sectional BP screening study during the month of May in over 1.5 million volunteers aged ≥18 years in over 89 countries.[23] MMM 2018 found a positive linear association between age and SBP in untreated hypertensive males and females, with the mean BP in females overtaking that in males after 75 years of age.

The Association Between BP and Incident Stroke by Sex

The association between usual SBP and the risk of stroke have long been reported to be similar for males and females, such that every 10 mm Hg increment in SBP is associated with an increased risk of stroke of ≈25%[24] (Table S2). In the REGARDS (Geographic and Racial Differences in Stroke) Study[25] of 26 641 adults aged ≥45 years, the magnitude of the association between SBP>140 mm Hg and incident stroke were comparable for females (hazard ratio [HR], 1.25 [95% CI, 1.16–1.34) and males (1.14, 1.05–1.23) (*P*=0.09 for interaction). This is further confirmed by an observational analysis of the ALLHAT (Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial)[26] involving participants with 4 to 7 SBP measurements during 22 months (n=24,309). Those with sustained BP control, defined as SBP <140 mm Hg, at <50% of study visits, compared with those with SBP control at 100% visits, were more likely to have a stroke for both females and males (*P*=0.08 for interaction).

However, an analysis of 471 971 UK Biobank participants demonstrated that females with hypertension had a greater excess risk of the first stroke than their male counterparts with increasing severity of hypertension during 9 years of follow-up.[27] The multiple-adjusted female-to-male ratios of HRs associated with stage 2 hypertension were 1.36 (95% CI, 1.26–1.47) for stroke. This is further supported by studies investigating ambulatory BP monitoring, which found a steeper relationship between higher ambulatory BP and CVD risk in females than in males.[28,29] Boggia et al[29] performed a cohort study of 9357 participants from 11 populations, with a median of 11.2-years follow-up and found that a 15 mm Hg increase in 24-hour SBP increased the risk of a cardiovascular event by 56% in females, compared with 32% in males. The proportion of stroke events preventable by BP control has been found much higher in females (38.3%) than males (25.9%).[29] Those results were not able to be pooled to generate estimates using meta-analysis because of the inconsistent presentation of data.

Management

Other than special recommendations for the management of hypertension during pregnancy,[30–33] there is

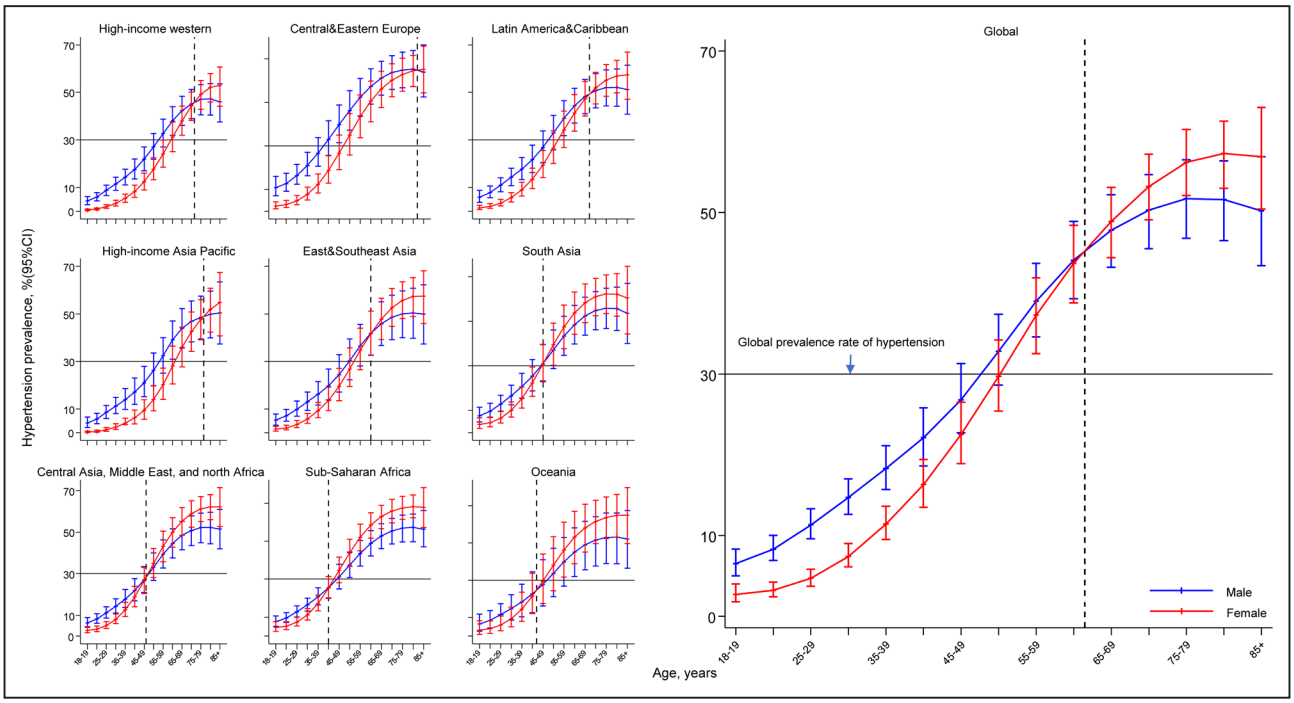

Figure 1. Global hypertension prevalence by region and sex.
Worldwide trends in blood pressure from 1975 to 2015: A pooled analysis of 1479 population-based measurement studies with 19.1 million participants. Reproduced from Zhou et al[22] with permission. Copyright ©2017.

no evidence that the BP threshold for initiating drug treatment, the treatment target, the choice of initial antihypertensive medication, or the combination of medications for lowering BP differs for females versus males.[34,35] Randomized evidence has shown that BP-lowering treatment provided broadly similar protection against major cardiovascular events in females and males.[35] Differences in cardiovascular risks between sexes do not reflect differences in response to BP-lowering treatment (Table S3).[35] However, females were underrepresented in most of the trials and were underpowered to examine sex differences in BP-lowering treatment effects.

Different CVD risk profiles between the sexes may influence the choice of antihypertensive treatment. For example, thiazide diuretics are useful in the management of osteoporosis[36] and reported to significantly increase the total body bone mineral density in postmenopausal females,[37] but not in males.[38] ACE (angiotensin-converting enzyme) inhibitors and angiotensin receptor blockers (ARBs) are contraindicated for females who are or intend to become pregnant because of the risk of fetal developmental abnormalities.[39] Antihypertensive drugs may also have sex-specific side effects.[40] Females experience more frequent electrolyte disturbances (eg, hyponatramia[41,42] and hypokalamia[43]).[44] Gout[45] is more common in females taking a diuretic than in males. ACE inhibitor-induced dry cough is 2 to 3 times more frequent in females than in males,[46,47] and calcium channel blockers (CCBs)-related edema[48] is much more common in females than males.

Recent large-scale observational studies suggest that females would have greater cardiovascular benefits from reducing BP to lower targets[29] and imply that optimal BP may be lower in females than in males.[28,49] A prospective study[28] of 3344 participants (1626 females) concluded that the optimal outcome–based ambulatory BP threshold for males was 135/85 mm Hg during the day and 120/70 mm Hg during the night, which align with current hypertension guidelines.[32,50] However, these thresholds are substantially higher than the optimal thresholds for females, which was found to be 125/80 mm Hg during the day and 110/65 mm Hg during the night.[28] This sex discrepancy in optimal BP levels is further confirmed by a community-based cohort study[49] of over 27 000 participants (54% female) in which the magnitude of stroke risk (HR, 1.53 [95% CI, 1.07–2.21]) in females with SBP 120 to 129 mm Hg was comparable with risk in males (1.50: 0.85–1.64) with SBP 140 to 149 mm Hg. This study found that stroke risk increased from an SBP of 120 mm Hg in males, but the equivalent threshold in females was 110 mm Hg or lower (Figure 2). Since the 2021 European Society of Hypertension practice guidelines[50] recommend ambulatory BP monitoring thresholds of daytime and nighttime hypertension to align with those identified for males, namely 135/85 mm Hg and 120/70 mm Hg, respectively, it would be important to review this recommendation and to determine based on all available evidence whether more appropriate targets should be recommended for females.

Females tend to have more traditional CVD risk factors than males, including central obesity, elevated total cholesterol, and low high-density lipoprotein cholesterol.[51–54] The association between hypertension and obesity is sex and age-related, and the prevalence of hypertension and obesity is lower in premenopausal females than in males.[55,56] In addition, many lifestyles also influence BP and CVD in a sex-specific manner. Females are prone to physical inactivity,[57] while males are prone to a higher rate of smoking and alcohol consumption.[6] The second Nurses' Health Study[58] identified 6 low-risk factors for incident hypertension, including a body mass index of <25 kg/m^2, 30 minutes daily vigorous exercise, a high score on the dietary approaches to stop hypertension diet, modest alcohol intake up to 10 g/day, use of nonnarcotic analgesics less than once per week, and intake of 400 µg/day or more of supplemental folic acid. The authors suggested that 78% of new-onset hypertension in this population could have been prevented if all females had these 6 low-risk factors. We have systematically reviewed papers based on sex-specific differences when adopting healthy lifestyles recommended in the hypertension guidelines (Table 1). We found that these are equally effective in lowering BP for males and females. Collectively, these findings suggest that management strategies for hypertension that are tailored according to sex and their risk profiles could lead to improved health outcomes.

ETHNIC/RACIAL DIFFERENCES

Hypertension Prevalence by Ethnicity/Race

Figure 1 demonstrates that fewer females than males have hypertension at young ages, but the prevalence increases markedly with age.[22] It is clear that there are major regional differences on when females overtake males. Higher prevalence rates among males than females are observed until ≈70 to 80 years of age in high-income Western, European, and Asia-Pacific countries, but in low- and middle-income settings, this change occurs much earlier at 40 to 50 years. The MMM study also shows substantial regional differences in hypertension with the highest prevalence in sub-Saharan Africa,[98] while the lowest rate of hypertensive awareness,[23] hypertensives on medications,[23] and controlled BP.[23,98,99] Non-Communicable Disease Risk Factor Collaboration further confirms the substantial regional differences in hypertension with Sub-Saharan

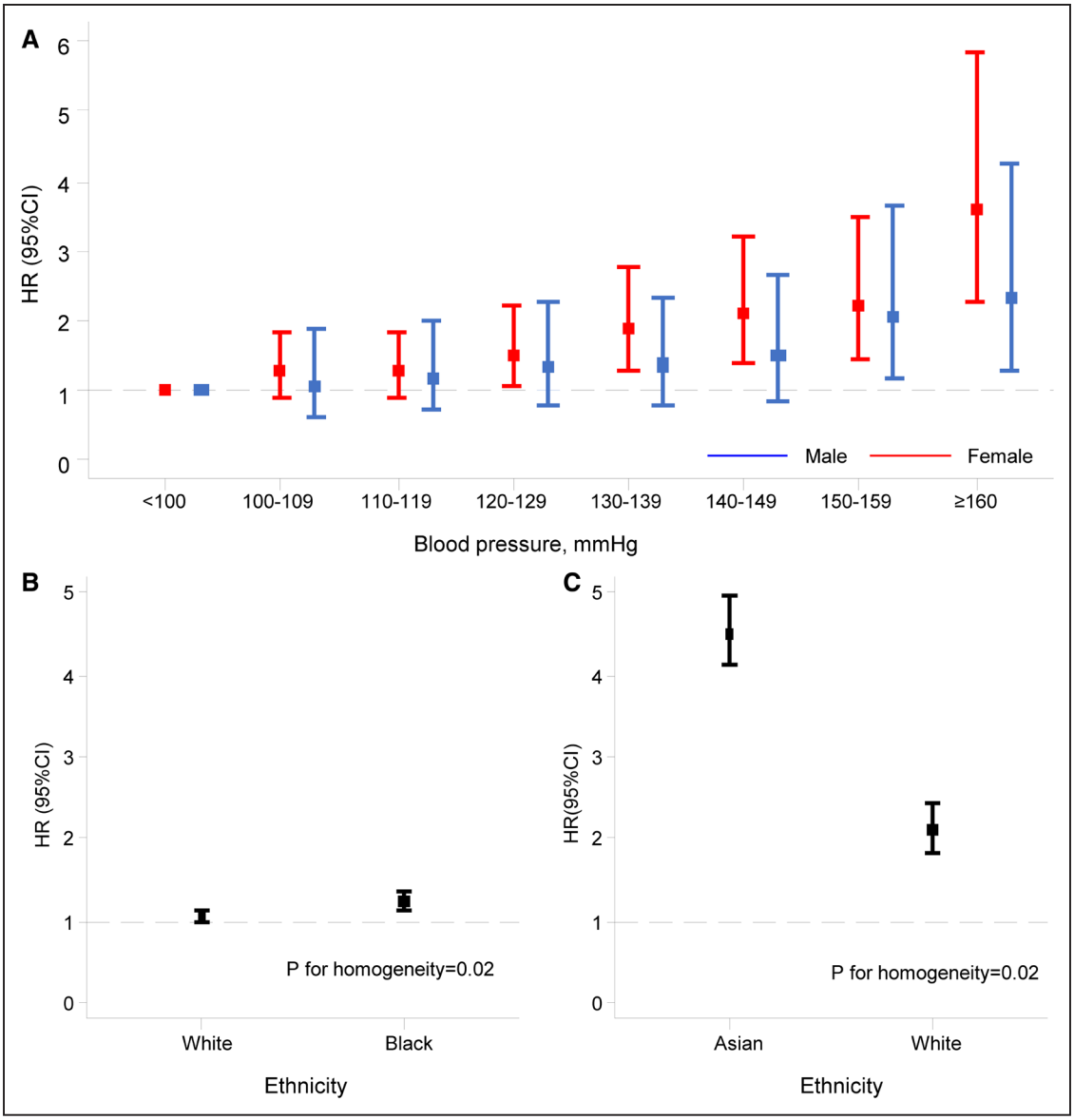

Figure 2. Sex differences and ethnic differences for the association between blood pressure and the risk of stroke. Hazard ratio (HR) is for the risk of stroke in **A**. HR is for per 10 mm Hg increase in systolic blood pressure for the risk the stroke in **B**. HR is for ≥140/90 vs <120/80 mm Hg (reference) for the risk of stroke in **C**. The center of each solid box is plotted against the point estimate, and the horizontal lines are drawn to the 95% confidence limits. Areas of the boxes are proportional to the reciprocal of the variance of the estimates. **A**, Reproduced from Ji et al with permission, Copyright ©2021, Wolters Kluwer Health, Inc. **B**, Reproduced from Howard et al with permission. Copyright©2013, American Medical Association. All rights reserved. **B**, Reproduced from Arima et al with permission. Copyright©2013, Wolters Kluwer Health, Inc.

Africa, Oceania, and South Asia having the lowest detection, treatment, and control rates.[4]

Regional differences in hypertension prevalence rates are likely driven by socioeconomics and ethnicity/race, as reflected by 32 studies listed in Table S3. In the United States, Black adults have the highest, and White adults have the lowest prevalence of hypertension.[100,101] South Asian[102–104] and Hispanic adults[102,103,105] have a higher prevalence of hypertension compared with White adults, while hypertension is less prevalent among Mexican adults.[100–103] Most studies in the United Kingdom also report a higher prevalence in Black and South Asian adults than White adults.[106–108] Studies from other countries, including both developed[109–111] and developing countries,[112–116] have generally found that ethnic/racial minorities have higher levels of prevalent hypertension than the majority population.

Hypertension Prevalence by Ethnicity/Race and Age

With consistent ethnic/racial differences in BP at all ages in many countries, disparities in raised BP represents a lifetime consideration.[117,118] Raised BP is associated with changes in vascular function and structure that are more pronounced than the changes that would be expected as

 Stroke. 2022;53:1114–1133. DOI: 10.1161/STROKEAHA.121.035852

Table 1. Comparison Between Female and Male, and Ethnicities/Races According to the Lifestyle in the Hypertension Guidelines[30–32,59–68]

Guideline recommendations	Evidence level (class, level)	Sex differences		Ethnic/racial differences	
		Epidemiological evidence	Interventional evidence	Epidemiological evidence	Interventional evidence
Salt restriction <5 g per d	I, A	Salt consumption: male>female[69]	Salt restriction is effective on BP control in both males and females, with no significant heterogeneity[70,71]	Salt consumption: Asians>Westerners[72] Blacks>White[73] Salt sensitivity: Blacks>Whites[74] Asians>Westerners[75]	Salt restriction is effective on BP control, and the magnitude of the effect size: Blacks>Asians>Whites[70]
Moderation of alcohol consumption to: <14 units per week for males. Less than 8 units per wk for females	I, A	Alcohol consumption: male>female[76]	Alcohol reduction is effective on BP control in male,[77,78] but only limited data are available in female[78]	Alcohol consumption: Asians>Westerners[76] Blacks>Whites[80]	Only limited information is available on the effect of alcohol reduction on BP in Blacks[79]
Increased consumption of vegetables, fresh fruits, fish, nuts, and unsaturated fatty acids (olive oil); low consumption of red meat; and consumption of low-fat dairy products The DASH diet: a diet rich in fruits, vegetables, whole grains, and low-fat dairy products, with reduced content of saturated and total fat.	I, A	DASH diet consumption: female>male[81]	The DASH diet was effective in reducing BP in both males and females, and no significant heterogeneity[82]	DASH diet consumption: Westerners>Asians[83] Whites>Blacks[85]	The DASH diet was more effective in Blacks than Whites.[82] And it is also effective in Asian populations.[84]
Potassium supplementation (3500–5000 mg/d), preferably in dietary modification	I, A	Potassium consumption: female>male[81,86]	Potassium interventions are effective in lowering BP, and the magnitude of the effect size: female>male[87]	Dietary potassium consumption: Whites>Blacks[85,88] Westerns>Asians[76]	Potassium interventions are effective in lowering BP with the magnitude of the effect size: Blacks>Whites.[89] It is also effective in Asians.[87,90]
Body-weight control to avoid obesity (BMI >30 kg/m^2 or waist circumference >102 cm in males and >88 cm in females) Regular physical activity, for example, at least 30 min of moderate dynamic exercise on 5 to 7 d per wk	I, A	Overweight: female>[9,91]	Weight reduction is effective in lowering BP in both males and females without significant heterogeneity[92]	Overweight: Blacks> Whites>Asians[75,93]	Weight reduction is effective in lowering BP and the magnitude of the effect size: Asians>Blacks>Whites[92]
Smoking cessation	I, B	Smoking rate: male>female	Incident hypertension was high among smokers compared with nonsmokers in both male[94] and female[95]	Smoking rate: Westerners<East Asians[75] Blacks>Whites[6]	Incident hypertension was high among smokers compared with nonsmokers in Western countries,[94,95] Asia,[96] and Africa[97]

BMI indicates body mass index; BP, blood pressure; and DASH, Dietary Approaches to Stop Hypertension.

part of a normal aging process. This process is referred to as early vascular aging, which reflects increased arterial stiffness at younger chronological ages.[119,120] Most studies performing ethnic/racial-specific comparisons in arterial stiffness report that populations of African descent (and often also Hispanic populations) have higher arterial stiffness than White populations from as young as 6[121] to 70 years of age.[122] Raised BP often accompanies arterial stiffness in Black populations.[123] Data from National Health and Nutrition Examination Survey (NHANES) demonstrated that Black adults had significantly higher rates of hypertension compared with Whites, Asians, and Mexican Americans, at all ages and for both sexes.[124–126] Black individuals tend to have an earlier age of onset, a longer duration, and greater severity in terms of BP levels and organ damage than in Whites, resulting in a higher incidence of CVD and mortality.[127] An assessment of US children aged 8 to 17 years found SBPs to be 2.9 and 1.6 mm Hg higher in Black boys and girls, compared with age-matched White boys and girls.[128] An observational study in South Africa demonstrated that Black boys from 6 to 8 years of age have higher arterial stiffness throughout the arterial tree along with higher diastolic BP when compared with White boys of the same age.[121]

The Association Between Raised BP and Incident Stroke by Ethnicity/Race

High BP is a major cardiovascular risk factor and one of the top contributors to ethnic/racial disparities in stroke.[129,130] The REGARDS study, including 27 748 participants ≥45 years, found the impact of higher BP levels on stroke was 3 times greater for Black (24% increase per 10 mm Hg increment in SBP) than for White (8%) participants over 4.5 years of follow-up.[131] (Figure 2) The disparities of higher prevalence and greater risks from high BP are most evident with the population attributable risks, which are nearly twice as big for Black than White adults.[132] The estimated benefits from modest population-wide decrements in SBP for stroke were also twice as large for Blacks (12 events reduced per 1 mm Hg decrease) than for Whites (5 events).[133] In addition, the Northern Manhattan Study (n=3298) found

the population attributable risk for stroke resulting from hypertension to be greater among Hispanics (50.6%) than Whites (2.6%).

The Global Burden of Disease Study 2019[134] indicated that high SBP had a large effect on the burden of stroke in Asian countries (eg, 69% in Mongolia). This is much higher than the burden reported in the developed countries, eg, United Kingdom (47%) and the United States (45%) (Table S4). The Asia Pacific Cohort Studies Collaboration[135–137] found hypertension (≥140/90 mm Hg), compared with normal BP (<120/80 mm Hg), was associated with a 1.5-fold increased risk of ischemic stroke among non-Asian participants from Australia and New Zealand, but a much greater increased risk of 3.4 fold among Asian participants (Table S5, Figure 2). The disparity was even more remarkable for intracerebral hemorrhage, with a risk of 2.5 and 9.7 times for non-Asians and Asians, respectively. This was further confirmed by the INTERSTROKE (Importance of Conventional and Emerging Risk Factors of Stroke in Different Regions and Ethnic Groups of the World) Study.[138]

Management

There are many areas where ethnic/racial/regional disparities in hypertension have been shown, as this article illustrates. Therefore, we searched national (eg, high-risk countries in Asia and Africa in terms of ethnic/racial population) and international guidelines to see if there were any ethnic/racial-specific management strategies to close the disparities gap (Table 2; Table S6). Of the 16 regional guidelines identified, 3 were from East Asia (China, South Korea, and Japan), 2 from Southeast Asia (Malaysia and Thailand), 1 from South Asia (India), 9 from Africa (South Africa, Sierra Leone, Rwanda, Tanzania, Somalia, Ethiopia, Kenya, Zambia, and Zimbabwe), and 1 for Latin American countries. Almost all the guidelines provided an explicit numerical diagnostic threshold for hypertension, defined as a clinic-based BP ≥140/90 mm Hg and included distinct stages for classifying hypertension. In terms of BP-lowering goals, the majority (14 out 16) of national guidelines recommend a clinic-based BP target of <140/90 mm Hg for general hypertensive patients and <130/80 mm Hg, if tolerated or in a high-risk population for CVD. This target is aligned with the European Society of Cardiology and the European Society of Hypertension 2018 guidelines,[31] the 2020 International Society of Hypertension global hypertension practice guidelines[32] and World Health Organization (WHO) 2021 guidelines.[33] Japan[63] and India[64] recommend a lower target of <130/80 mm Hg in younger patients, which is in line with the American College of Cardiologys and American Heart Association 2017 guidelines.[30] Guidelines in Asian countries and Latin America recommend the first-line agents of 5 major drug classes: ACEIs, ARBs, beta-blockers, CCBs, and diuretics, except Japan, Thailand, and India, who do not recommend beta-blockers. In African guidelines, a diuretic and/or a CCB is recommended initially for Black people because of a better response rate compared with an ACE inhibitor,[68] except Somalia, where the cheapest option with the fewest side effects is recommended.[139] Black patients may combine a diuretic with a CCB, or a Renin-angiotensin system (RAS) blockade, making CCB/*RAS* more effective.[140] Angioedema seems more common with ACEIs in Black patients, which may favor the preferred use of ARBs in this population.[31] Single-pill combination drugs are preferred to improve patient adherence, home BP monitoring, and a combined CV risks and BP levels-based antihypertensive treatment algorithm is recommended in almost all the guidelines.

As shown in Table 2, the recommended pharmacological intervention options for the management of hypertension are largely similar despite some variations among different guidelines. It has been reported that Black hypertensive patients exhibit a very similar, impressive, benefit from a reduction of cardiovascular and renal events in response to BP-lowering treatment as White patients, with somewhat different treatment modalities.[141] Table S7 summarizes 19 trials comparing the effect of different classes of antihypertensive agents on incident stroke in Asian populations, consistent with the previous findings,[142] suggesting that reduction in stroke risk could be achieved irrespective of the class of antihypertensive agent used (no evidence of publication bias; Figure S1). The pooled HR was 1.14 (95% CI, 0.84–1.56) for CCB versus angiotensin receptor blocker, 1.08 (95% CI, 0.82–1.42) for non- angiotensin receptor blocker therapy versus angiotensin receptor blocker. Two trials[143,144] compared CCB with diuretics and all reported neutral results. In addition, some Japanese studies reported that diuretics are effective for salt-sensitive hypertension[145] and preventing stroke.[144] Brewster et al[146] performed a systematic review of antihypertensive drug therapy in patients of South Asian ethnicity/race and found no evidence of different efficacy of antihypertensive drugs. However, there were no trials with morbidity and mortality outcomes. Optimal treatment combinations (required by most patients) are not identified for black, South Asian or patients from East Asia.[147] While the CREOLE trial[140] indicates that the combination of amlodipine with either hydrochlorothiazide or perindopril is superior to perindopril plus hydrochlorothiazide in reducing 24-hour BP among black patients from sub-Saharan Africa with hypertension, trials with hard outcomes such as cardiovascular morbidity and mortality are required for a clear recommendation for each major ethnic/racial group. Regarding optimal BP thresholds and targets for different ethnic/racial groups, current randomized evidence suggests that the treatment effects of different BP targets on cardiovascular events were similar for different ethnicities/races (Table S3). However, those studies

 Stroke. 2022;53:1114–1133. DOI: 10.1161/STROKEAHA.121.035852

Table 2. BP Management in Hypertension Guidelines by Region

	Region					
	East Asia[59–63]	South Asia[64]	Southeast Asia[65,66]	Latin America[67]	Africa[68]	Western[30,31] and International guidelines[32,33]
Hypertension definition	≥140/90 mm Hg	≥140/90 mm Hg	≥140/90 mm Hg	≥140/90 mm Hg	≥140/90 mm Hg	≥140/90 mm Hg ACC/AHA: ≥130/80 mm Hg
BP target	Japan: <75 y, <130/80 mm Hg South Korea: <65 y: 140/90 mm Hg China: 65–79 y <150/90 and <140/80mmHg if tolerable	<60 y: <130/80 mm Hg	<50 y: BP<130/80 mm Hg	130–140/90 mm Hg	<140/80 mmHg	ACC/AHA: <130/80 mm Hg ESC/ESH: <140/90 mm Hg and further <130/80 mm Hg, if tolerated
	Japan: ≥75 y: <140/90 mm Hg South Korea: ≥65 y: 140/90 mm Hg China: ≥80 y <150/90 mm Hg	≥60 y: the target should be individualized	60–80 y: <140–150/90 mm Hg. ≥80 y: <150/90 mm Hg		≥80y: 140–150 mm Hg	ACC/AHA: <130/80 mm Hg ESC/ESH: <65y 120–129mmHg ≥65y 130–139 mm Hg
First-line BP-lowering drugs therapy	Japan: ACEI, ARBs, CCBs, or diuretics, ≥20/10 mm Hg above target BP: drugs combination recommended South Korea and China: ACEIs, ARBs, CCBs, diuretics, and beta-blockers BP≥160/100 mm Hg, or ≥20/10 mm Hg above target BP, or at high risk: drugs combination recommended	<60 y: ACEIs and ARBs. ≥60 y CCB and diuretics BP≥160/100 mm Hg: two drugs combination in a singer pill recommended	ACEIs, ARBs, CCBs, diuretics, or beta-blocker BP≥160/100 mm Hg, or at high risk: drugs combination recommended	ACEIs, ARBs, CCBs, diuretics or Beta-blocker BP≥160/100 mm Hg: drugs combination recommended	Diuretics, CCB, ACEI or ARB. BP≥160/100 mm Hg: drugs combination recommended A diuretic and/or a CCB is recommended for Black patients	ACC/AHA: diuretics, CCBs, and ACEI or ARBs; 2 drugs combination recommended if BP≥140/90 mm Hg, or ≥20/10 mm Hg above target BP. ESC/ESH: ACEIs, ARBs, Beta-blockers, CCBs, and diuretics Combination treatment is recommended for most hypertensive patients as initial therapy comprising a RAS blocker (either an ACEI or an ARB) with a CCB or diuretic. Beta-blockers are combined with any of the other major drug classes for specific clinical situations, eg, angina, MI, HF

ACC/AHA indicates American College of Cardiology/American Heart Association; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BP: blood pressure; CCB, calcium channel blocker; ESC/ESH, the European Society of Cardiology and the European Society of Hypertension; HF, heart failure; MI, myocardial infarction; and RAS, renin-angiotensin-system.

were underpowered to examine ethnic/racial differences in BP-lowering treatment effects.

Nonpharmacological, nonpersonal interventions are also important for hypertension prevention and control. Recommended nonpharmacological strategies of healthy lifestyles are summarized in Table 1. For example, higher salt sensitivity, even with mild obesity and higher salt intake, is an Asian characteristic of hypertension.[148] Similarly, a recent systematic review examining salt intakes in sub-Saharan Africa, including the data from 13 countries, suggested that over 80% of adult populations consume more than the WHO recommended 5 g salt or 2 g sodium each day.[149] To achieve effective BP control, salt restriction is particularly important in high-risk populations.[141,150] Other strategies to improve prevention and control of hypertension at the individual level include efforts towards increased awareness and self-care skills, availability of, and adherence to, quality antihypertensive therapies, availability of university health coverage and access to health care.[147] Health care–related reasons for poor hypertension control include accepted standards and goals in hypertension treatment and control, physician education and familiarity with therapeutic options, physician-to-patient ratio or nurse-to-patient ratio, antihypertensive regime complexity, provider-patient interaction, and adequate patient follow-up.[151] Broader efforts to investigate the challenges in effective implementation of evidence-based care are likely to address social and ethnic/racial differences in hypertension treatment and control.[152]

THE INTERACTION BETWEEN SEX AND ETHNICITY/RACE

National surveys, such as NHANES, have highlighted the heterogeneity in hypertension based on sex and ethnicity/race.[102,126,153,154] This is similar to findings in European ethnic/racial minority groups(Table S8).[109] In the HELIUS (Healthy Life in an Urban Setting) Study in Amsterdam,[109] compared with people of Dutch origin, the prevalence ratio of hypertension was higher in all the ethnic/racial minority groups, except for Moroccan females, ranging from 1.29 to 3.57 in males and from 1.28 to 1.90 in females. However, data on the impact of the interaction between sex and ethnicity/race on hypertension prevalence, awareness, treatment, and control are lacking, although they may have significant implications for patient care. Data from NHANES III and NHANES 1999 to 2004[126] showed there was some improvement in awareness of hypertension, which was most clear in White males but not improved among Mexican American or White females. Treatment and control rates among Mexican American persons remain substantially lower than for other ethnic/racial groups. NHANES 2011 to 2014 showed the percentage of males with controlled hypertension was lower than that for females among

Black and Hispanic adults.[154] There were also ethnic and sex disparities identified in response to antihypertensive therapies[155] and the association of hypertension with stroke.[156] Social and physical environments have been implicated as major determinants of cardiovascular health. Certain social and physical environments tend to promote a cause-and-effect chain of events that contribute to developing CVD.[157] Community characteristics, including racial segregation, employment opportunities, neighbourhood safety, lack of access to timely and quality health care, low education and income levels and poor social support, influence different ethnicities/races in a different way.[158] Some ethnic/racial disparities in CVD risk factors were explained by differences in individual and community characteristics, but other disparities persisted even after controlling for these factors.[157] Greater understanding of how sex and ethnicity/race influence the prevalence, diagnosis and management of hypertension is needed given the health impact and economic burden of hypertension are only expected to increase as the population ages.

HYPERTENSIVE DISORDERS IN PREGNANCY

Prevalence

An important sex-specific aspect related to BP and stroke is hypertensive disorders of pregnancy (HDP), which is a common complication in females during pregnancy with an overall prevalence of 10% to 20%.[159–162] This includes chronic hypertension, gestational hypertension, preeclampsia, eclampsia, and chronic hypertension with superimposed preeclampsia.[159] HDP is the second most common direct cause of maternal mortality worldwide (14% [95% CI, 11%–17%]).[163] Chronic hypertension complicates 0.2% to 3% of deliveries,[159,164–168] whereas gestational hypertension develops in 2% to 10% of pregnancies.[18,159,160,169,170] Preeclampsia affects 2% to 10% of pregnancies[159,160,167–169,171–173] and eclampsia develops in ≈1% of pregnant females (Table S9).[172]

Ethnic/Racial Differences

Studies have shown apparent ethnic/racial disparities in developing HDP (Table S10).[174–176] HDP is estimated to cause 10% to 15% of maternal deaths in Asia, 16% to 17% in Africa and 22% in Latin America and the Caribbean.[163] Ghosh et al[177] examined 56617 nulliparous females with singleton deliveries and found that Black females experienced more chronic hypertension and mild, severe, and superimposed preeclampsia, White Hispanic females and Asian/Pacific Islanders had an overall decreased risk of HDP compared with White females. Among females with chronic hypertension or preeclampsia, all minority females in the United States (Blacks, Hispanics, and Asian/Pacific Islanders) had a higher risk of stroke than White females.[178,179] Some of these ethnic/racial differences may be attributable to differences in socioeconomic position[180–182] or different prevalences of other CVD risk factors, such as obesity, smoking, or physical inactivity.[183,184] In addition, minority females were also at risk for delay in seeking prenatal care.[185,186] Studies from Australia,[187] New Zealand,[175] Norway,[188] and the Netherlands[176] found immigration was generally associated with reduced risk of HDP compared with nonimmigrant females, which was further confirmed by a systematic review.[189] A plausible explanation for the lower prevalence of HDP among immigrant females could be related to a disproportionately higher under-diagnosis of HDP among immigrants because of a lack of access or underutilization of health care services.[190]

Long-Term Risk for Stroke

We updated the previous systematic reviews on the risk of HDP for future stroke (Table S11)[191–198] and found that females who previously experienced HDP were at increased odds of a stroke (Figure 3A, HR, 1.6 [95% CI, 1.28–1.86]). Preeclampsia (Figure 3B, HR, 1.7 [95% CI, 1.43–1.94]) is now recognized by the American Heart Association/American Stroke Association as a sex-specific risk factor for future stroke, recommending that all females be evaluated for a history of preeclampsia as part of routine CVD risk assessment.[199] However, the pooled data showed significant heterogeneity (Figure 3) and the analysis of HDP and stroke showed significant publication bias, indicating the quality of the evidence is low (Supplementary Figure I). The risk of stroke in females with previous gestational hypertension was higher than for other females (Figure S2, HR, 1.5 [95% CI, 1.26–1.70]). In a meta-analysis, the excess risk of stroke was greater with early (HR, 5.08 [95% CI, 2.09–12.35]), compared with late (after 37 weeks), preeclampsia (HR, 0.98 [95% CI, 0.50–1.92]).[191]

PATHOPHYSIOLOGY OF RAISED BP

Sex

Fewer females have hypertension at young ages than males, but over the age of 75 years, ≈81% of females and ≈73% of males have hypertension.[6] This marked reversal of sex difference in the prevalence of hypertension in older people may, in part, be attributable to males with hypertension-related CVD frequently dying before the age of 75.[200] However, loss of cardiorenal protective mechanisms with age may also contribute to the sharp increase in hypertension in postmenopausal females.[200] Defects in these protective pathways may contribute to the development of vascular diseases that are unique to younger premenopausal females (such as

 Stroke. 2022;53:1114–1133. DOI: 10.1161/STROKEAHA.121.035852

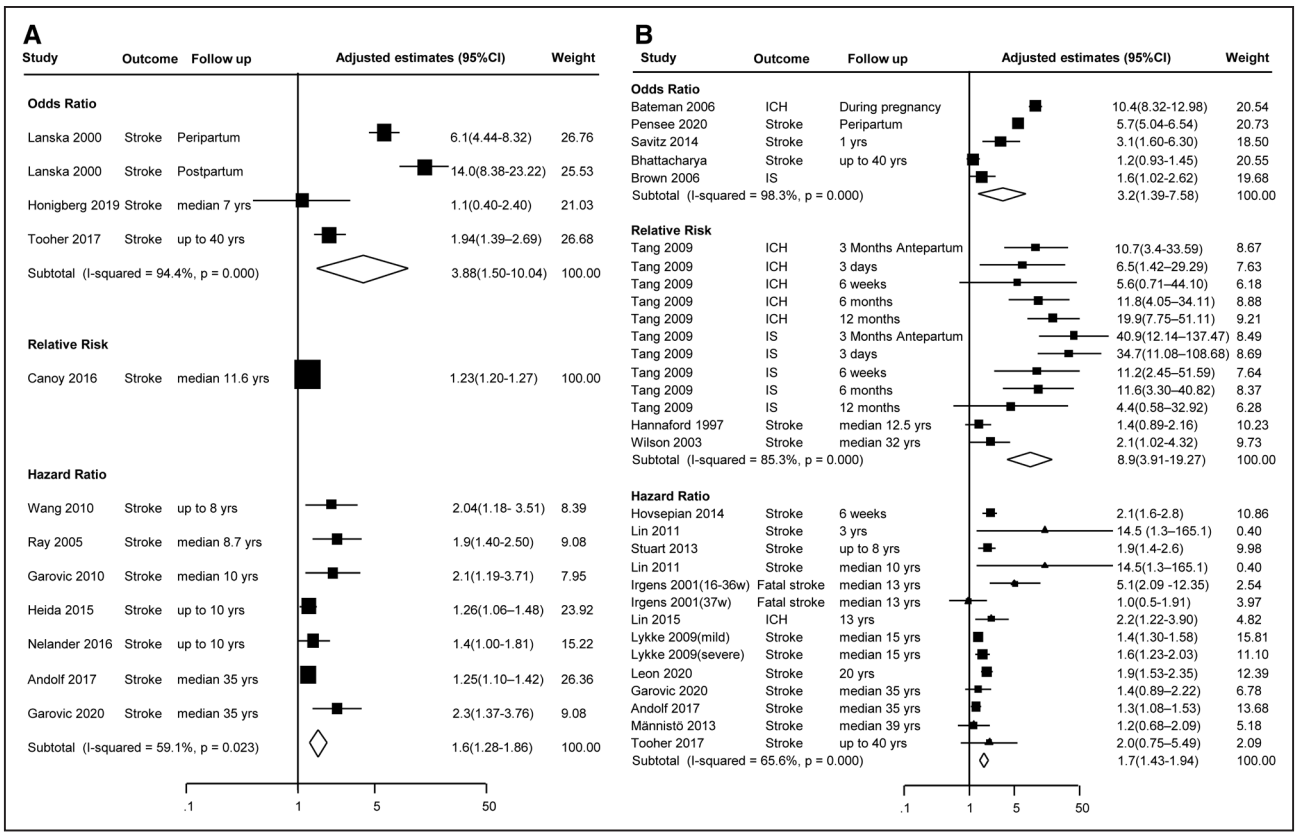

Figure 3. Meta-analysis for the association between hypertensive disorders of pregnancy (A)/preeclampsia (B) and the risk of stroke.
ICH indicates intracerebral hemorrhage; and IS, ischemic stroke

HDP and gestational diabetes).[200] Moreover, this may be related, in part, to sex differences in vascular tone and possible vascular protective effects of the female sex hormones estrogen and progesterone.[201] In addition, numerous systems contribute to the control of BP, including the vasculature, the nervous system, and the kidney,[202] and each of these systems exhibit sex differences in hypertension.[203]

Sex differences in the RAS system are well established. Male have greater expression levels and physiological responses to activation of the classical RAS (Ang II, angiotensin II type 1 receptor [AT1R], ACE). However, females have greater expression and physiological responses to activation of the nonclassical RAS (Ang [1–7], angiotensin II type 2 receptor [AT2R], Mas receptor, ACE2).[203–205] Sex hormones, particularly testosterone and estrogen, have also been well documented to impact not only BP, renal, central, and vascular function but also numerous pathways linked to BP control.[206] Greater AT2R expression in females compared with males is dependent on both estrogen and sex chromosome complement,[207]and there is growing evidence to support a sex-specific role for the AT2R in offering cardiovascular protection to females.[208] Other molecular mechanisms driving sex differences in hypertension include oxidative and endoplasmic reticulum stress, nitric oxide, inflammation, and the endothelin system.[208]

Ethnicity/Race

Hypertension prevalence, treatment, and control rates vary significantly according to ethnicity/race. Such difference is driven by a complex set of gene/gene (not modifiable), environment/environment (modifiable), and gene/environment interactions.[209] Blacks tend to have a suppressed renin-angiotensin-aldosterone system activity, including low renin status and accordingly, low renin hypertension is common.[210,211] Other hypothesized etiology for more prevalent hypertension among Blacks include sodium abnormalities, epithelial sodium channel changes, altered genes regulating the renin-angiotensin-aldosterone system, increased peripheral vascular resistance, increasing obesity, early vascular aging (large artery stiffness), and underweight phenotype.[120,210] East Asians have a genetically higher salt sensitivity.[75,212] The Gly460Trp variant of the α-adducin gene has been associated with renal sodium retention and salt-sensitive hypertension through enhancement of the activity of the sodium pump.[75] α-adducin Gly460Trp polymorphism was only associated with salt sensitivity in Asians, but not in Whites, indicating that BP response to sodium varies among ethnic/racial groups.[213]

CONCLUSIONS

This review synthesizes the most up-to-date evidence on sex- and ethnic/racial-specific aspects of raised BP

and related stroke risk (Table 3). We highlight the need for more randomized evidence on optimal BP thresholds and targets in females and males since observational evidence suggests that, in terms of stroke risk, females would benefit from lower BP thresholds to the magnitude of 10 to 20 mm Hg in males. Also, with a substantially increased stroke risk in females with HDP, there will be benefits from the development of strategies, serving pregnant females from all backgrounds to eradicate the barriers to accessing clinical care throughout the continuum of pregnancy and beyond. Prevention and control of hypertension require multi-faceted strategies at the individual, community, and population levels. Interventions targeting high-risk ethnicities/races across the life course would be beneficial to reduce the burden of raised BP and subsequent stroke risk. Prospective studies are needed to investigate the impact of the interaction between sex and ethnicity/race in contributing to hypertension prevalence, treatment and control.

Gender/sex and ethnicity/race are often used interchangeably in the literature, and it is challenging to distinguish one from the other, while attitudes toward the dichotomy between sex and gender and between ethnicity and race have evolved over time. This compromises our ability to describe specific associations with BP.

Table 3. Executive Summary

Executive summary
Sex differences in hypertension
Higher hypertension prevalence rates among males than females are observed until the 7th decade of life in high-income Western, European, and Asia-Pacific countries (HICs), but the change occurs almost 3 decades earlier in LMICs.
Observational evidence suggests females derive greater cardiovascular benefits at lower BP thresholds than males, implying that optimal BP may be lower in females than males.
Ethnic/racial differences in hypertension
In HICs, prevalence has declined while health systems have achieved treatment rates of up to 80% and control rates of up to 60%. However, in LMICs, only 1 in 3 are aware of their hypertension status, and ~8% have their BP controlled.
The highest prevalence of hypertension occurs in LMICs, with Sub-Saharan Africa, Oceania, and South Asia have the lowest detection, treatment, and control rates.
Black individuals tend to have an earlier age of onset, a longer duration, and greater severity in terms of BP levels and organ damage than Whites.
The impact of higher BP levels on stroke was greater for Blacks and Asians than for Whites
In general, immigrant females presented with a lower prevalence of hypertensive disorders of pregnancy compared to nonimmigrant females, likely due to under-diagnosis. Among females with chronic hypertension or preeclampsia in the United States, all minority females (Blacks, Hispanics, and Asian/Pacific Islanders) had a higher risk of stroke than White females.
Actions required
More inclusion of females and reporting sex-specific data in BP-lowering trials, including females of different ethnic/racial groups and regions to identify optimal treatments across diverse ethnic/racial groups, are needed.
Developing appropriate policies across the life course to eliminate social and economic disparities, which reduce the access to the management and control of raised BP is needed.
Prospective studies of how the interaction of sex and ethnicity/race influence the prevalence, diagnosis and management of hypertension are needed.
Ethnicity/race needs to be clearly defined in these types of studies.
Strategies are required to better access clinical care throughout the continuum of pregnancy and potentially reimagine a care delivery model that serves pregnant females from all backgrounds.

BP indicates blood pressure; HIC, high-income country; and LMIC, low- and middle-income country.

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Acknowledgments

Dr Wang wrote the first draft of the article; other authors critically revised the article. All authors read and approved the final version.

Sources of Funding

Dr Wang holds an investigator grant from the National Health and Medical Research Council (NHMRC) of Australia (APP1195237); Dr Carcel is supported by the National Heart Foundation 102741 and NHMRC of Australia grants APP2009726; Dr Woodward is supported by NHMRC grants APP1149987 and APP1174120.

Disclosures

Dr Woodward is a consultant to Amgen, Freeline and Kyowa Kirin. Dr Schutte has consultant fees from Abbott and speaker honoraria from Omron, Novartis, Takeda, Servier, Sun Pharmaceuticals, and Sanofi.

Supplemental Material

Methodology for the systematic reviews
Tables S1–S12
Figures S1–S2
Flow diagrams 1–7
References138,214–327

REFERENCES

1. Krishnamurthi RV, Ikeda T, Feigin VL. Global, regional and country-specific burden of ischaemic stroke, intracerebral haemorrhage and subarachnoid haemorrhage: a systematic analysis of the global burden of disease study 2017. *Neuroepidemiology*. 2020;54:171–179. doi: 10.1159/000506396
2. Rapsomaniki E, Timmis A, George J, Pujades-Rodriguez M, Shah AD, Denaxas S, White IR, Caulfield MJ, Deanfield JE, Smeeth L, et al. Blood pressure and incidence of twelve cardiovascular diseases: lifetime risks, healthy life-years lost, and age-specific associations in 1·25 million people. *Lancet*. 2014;383:1899–1911. doi: 10.1016/S0140-6736(14)60685-1
3. National high blood pressure education program. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Bethesda (md): National heart, lung, and blood institute (us); 2004 aug. Blood pressure and cardiovascular risk. Accessed March 16, 2022. https://www.Ncbi.Nlm.Nih.Gov/books/nbk9634/.
4. Zhou B, Carrillo-Larco RM, Danaei G, Riley LM, Paciorek CJ, Stevens GA, Gregg EW, Bennett JE, Solomon B, Singleton RK, et al. Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019: A pooled analysis of 1201 population-representative studies with 104 million participants. *Lancet*. 2021;398:957–980. doi: 10.1016/S0140-6736(21)01330-1
5. Schutte AE, Srinivasapura Venkateshmurthy N, Mohan S, Prabhakaran D. Hypertension in low- and middle-income countries. *Circ Res*. 2021;128:808–826. doi: 10.1161/CIRCRESAHA.120.318729

6. Benjamin EJ, Muntner P, Alonso A, Bittencourt MS, Callaway CW, Carson AP, Chamberlain AM, Chang AR, Cheng S, Das SR, et al; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics-2019 update: a report from the American Heart Association. *Circulation*. 2019;139:e56–e528. doi: 10.1161/CIR.0000000000000659
7. Kim AM, Tingen CM, Woodruff TK. Sex bias in trials and treatment must end. *Nature*. 2010;465:688–689. doi: 10.1038/465688a
8. Beery AK, Zucker I. Sex bias in neuroscience and biomedical research. *Neurosci Biobehav Rev*. 2011;35:565–572. doi: 10.1016/j.neubiorev.2010.07.002
9. Woodward M. Cardiovascular disease and the female disadvantage. *Int J Environ Res Public Health*. 2019;16:1165. doi: 10.3390/ijerph16071165
10. Flanagin A, Frey T, Christiansen SL; AMA Manual of Style Committee. Updated guidance on the reporting of race and ethnicity in medical and science journals. *JAMA*. 2021;326:621–627. doi: 10.1001/jama.2021.13304
11. Gravlee CC. How race becomes biology: embodiment of social inequality. *Am J Phys Anthropol*. 2009;139:47–57. doi: 10.1002/ajpa.20983
12. Https://www.Genome.Gov/genetics-glossary/race. Accessed 03 Feb 2022.
13. Mills KT, Bundy JD, Kelly TN, Reed JE, Kearney PM, Reynolds K, Chen J, He J. Global disparities of hypertension prevalence and control: a systematic analysis of population-based studies from 90 Countries. *Circulation*. 2016;134:441–450. doi: 10.1161/CIRCULATIONAHA.115.018912
14. Meiqari L, Essink D, Wright P, Scheele F. Prevalence of hypertension in vietnam: a systematic review and meta-analysis. *Asia Pac J Public Health*. 2019;31:101–112. doi: 10.1177/1010539518824810
15. Neupane D, McLachlan CS, Sharma R, Gyawali B, Khanal V, Mishra SR, Christensen B, Kallestrup P. Prevalence of hypertension in member countries of South Asian Association for Regional Cooperation (SAARC): systematic review and meta-analysis. *Medicine (Baltimore)*. 2014;93:e74. doi: 10.1097/MD.0000000000000074
16. Dhungana RR, Pandey AR, Shrestha N. Trends in the prevalence, awareness, treatment, and control of hypertension in nepal between 2000 and 2025: a systematic review and meta-analysis. *Int J Hypertens*. 2021;2021:6610649. doi: 10.1155/2021/6610649
17. Ezejimofor M, Uthman O, Chen YF, Ezejimofor B, Ezeabasili A, Stranges S, Kandala NB. Magnitude and pattern of hypertension in the Niger Delta: a systematic review and meta-analysis of community-based studies. *J Glob Health*. 2018;8:010420. doi: 10.7189/jogh.08.010420
18. Berhe AK, Kassa GM, Fekadu GA, Muche AA. Prevalence of hypertensive disorders of pregnancy in Ethiopia: a systemic review and meta-analysis. *BMC Pregnancy Childbirth*. 2018;18:34. doi: 10.1186/s12884-018-1667-7
19. Adeloye D, Owolabi EO, Ojji DB, Auta A, Dewan MT, Olanrewaju TO, Ogah OS, Omoyele C, Ezeigwe N, Mpazanje RG, et al. Prevalence, awareness, treatment, and control of hypertension in Nigeria in 1995 and 2020: A systematic analysis of current evidence. *J Clin Hypertens (Greenwich)*. 2021;23:963–977. doi: 10.1111/jch.14220
20. Zurique-Sánchez MS, Zurique-Sánchez CP, Camacho-López PA, Sánchez-Sanabria M, Hernández-Hernández SC. Prevalence of arterial hypertension in colombia. Systematic review and meta-analysis. *Acta Medica Colombiana*. 2019;44:20–33. https://doi.org/10.36104/amc.2019.1293
21. Akl C, Akik C, Ghattas H, Obermeyer CM. Gender disparities in midlife hypertension: a review of the evidence on the Arab region. *Womens Midlife Health*. 2017;3:1. doi: 10.1186/s40695-017-0020-z
22. Zhou B, Bentham J, Di Cesare M, Bixby H, Danaei G, Cowan MJ, Paciorek CJ, Singh G, Hajifathalian K, Bennett JE, et al. Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19·1 million participants. *Lancet*. 2017;389:37–55. doi: 10.1016/S0140-6736(16)31919-5
23. Beaney T, Burrell LM, Castillo RR, Charchar FJ, Cro S, Damasceno A, Kruger R, Nilsson PM, Prabhakaran D, Ramirez AJ, et al; MMM Investigators. May Measurement Month 2018: a pragmatic global screening campaign to raise awareness of blood pressure by the International Society of Hypertension. *Eur Heart J*. 2019;40:2006–2017. doi: 10.1093/eurheartj/ehz300
24. Peters SA, Huxley RR, Woodward M. Comparison of the sex-specific associations between systolic blood pressure and the risk of cardiovascular disease: a systematic review and meta-analysis of 124 cohort studies, including 1.2 million individuals. *Stroke*. 2013;44:2394–2401. doi: 10.1161/STROKEAHA.113.001624
25. Madsen TE, Howard G, Kleindorfer DO, Furie KL, Oparil S, Manson JE, Liu S, Howard VJ. Sex differences in hypertension and stroke risk in the REGARDS study: a longitudinal cohort study. *Hypertension*. 2019;74:749–755. doi: 10.1161/HYPERTENSIONAHA.119.12729
26. Bowling CB, Davis BR, Luciano A, Simpson LM, Sloane R, Pieper CF, Einhorn PT, Oparil S, Muntner P. Sustained blood pressure control and coronary heart disease, stroke, heart failure, and mortality: An observational analysis of ALLHAT. *J Clin Hypertens (Greenwich)*. 2019;21:451–459. doi: 10.1111/jch.13515
27. Peters SAE, Carcel C, Millett ERC, Woodward M. Sex differences in the association between major risk factors and the risk of stroke in the UK Biobank cohort study. *Neurology*. 2020;95:e2715–e2726. doi: 10.1212/WNL.0000000000010982
28. Hermida RC, Ayala DE, Mojón A, Fontao MJ, Chayán L, Fernández JR. Differences between men and women in ambulatory blood pressure thresholds for diagnosis of hypertension based on cardiovascular outcomes. *Chronobiol Int*. 2013;30:221–232. doi: 10.3109/07420528.2012.701487
29. Boggia J, Thijs L, Hansen TW, Li Y, Kikuya M, Björklund-Bodegård K, Richart T, Ohkubo T, Jeppesen J, Torp-Pedersen C, et al; International Database on Ambulatory blood pressure in relation to Cardiovascular Outcomes Investigators. Ambulatory blood pressure monitoring in 9357 subjects from 11 populations highlights missed opportunities for cardiovascular prevention in women. *Hypertension*. 2011;57:397–405. doi: 10.1161/HYPERTENSIONAHA.110.156828
30. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, DePalma SM, Gidding S, Jamerson KA, Jones DW, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 2018;71:e13–e115. doi: 10.1161/HYP.0000000000000065
31. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, Clement DL, Coca A, de Simone G, Dominiczak A, et al; ESC Scientific Document Group. 2018 ESC/ESH Guidelines for the management of arterial hypertension. *Eur Heart J*. 2018;39:3021–3104. doi: 10.1093/eurheartj/ehy339
32. Unger T, Borghi C, Charchar F, Khan NA, Poulter NR, Prabhakaran D, Ramirez A, Schlaich M, Stergiou GS, Tomaszewski M, et al. 2020 International Society of Hypertension Global Hypertension Practice Guidelines. *Hypertension*. 2020;75:1334–1357. doi: 10.1161/HYPERTENSIONAHA.120.15026
33. Https://apps.Who.Int/iris/bitstream/handle/10665/344424/9789240033986-eng.Pdf. Accessed october 7, 2021.
34. Gueyffier F, Boutitie F, Boissel JP, Pocock S, Coope J, Cutler J, Ekbom T, Fagard R, Friedman L, Perry M, et al. Effect of antihypertensive drug treatment on cardiovascular outcomes in women and men. A meta-analysis of individual patient data from randomized, controlled trials. The INDANA Investigators. *Ann Intern Med*. 1997;126:761–767. doi: 10.7326/0003-4819-126-10-199705150-00002
35. Turnbull F, Woodward M, Neal B, Barzi F, Ninomiya T, Chalmers J, Perkovic V, Li N, MacMahon S; Blood Pressure Lowering Treatment Trialists' Collaboration. Do men and women respond differently to blood pressure-lowering treatment? Results of prospectively designed overviews of randomized trials. *Eur Heart J*. 2008;29:2669–2680. doi: 10.1093/eurheartj/ehn427
36. Hamdy RC. Thiazides and osteoporosis: an addition to the armamentarium? *South Med J (Birmingham, Ala.)*. 2008;101:342–343. doi: 10.1097/SMJ.0b013e318167d5f3
37. Bolland MJ, Ames RW, Horne AM, Orr-Walker BJ, Gamble GD, Reid IR. The effect of treatment with a thiazide diuretic for 4 years on bone density in normal postmenopausal women. *Osteoporos Int*. 2007;18:479–486. doi: 10.1007/s00198-006-0259-y
38. Legroux-Gerot I, Catanzariti L, Marchandise X, Duquesnoy B, Cortet B. Bone mineral density changes in hypercalciuretic osteoporotic men treated with thiazide diuretics. *Joint Bone Spine*. 2004;71:51–55. doi: 10.1016/j.jbspin.2003.09.009
39. Igho Pemu P, Ofili E. Hypertension in women: part I. *J Clin Hypertens (Greenwich)*. 2008;10:406–410. doi: 10.1111/j.1751-7176.2008.06552.x
40. Tamargo J, Rosano G, Walther T, Duarte J, Niessner A, Kaski JC, Ceconi C, Drexel H, Kjeldsen K, Savarese G, et al. Gender differences in the effects of cardiovascular drugs. *Eur Heart J Cardiovasc Pharmacother*. 2017;3:163–182. doi: 10.1093/ehjcvp/pvw042
41. Sharabi Y, Illan R, Kamari Y, Cohen H, Nadler M, Messerli FH, Grossman E. Diuretic induced hyponatraemia in elderly hypertensive women. *J Hum Hypertens*. 2002;16:631–635. doi: 10.1038/sj.jhh.1001458
42. Siegler EL, Tamres D, Berlin JA, Allen-Taylor L, Strom BL. Risk factors for the development of hyponatremia in psychiatric inpatients. *Arch Intern Med*. 1995;155:953–957.

43. Hendriksen LC, van der Linden PD, Lagro-Janssen ALM, van den Bemt PMLA, Siiskonen SJ, Teichert M, Kuiper JG, Herings RMC, Stricker BH, Visser LE. Sex differences associated with adverse drug reactions resulting in hospital admissions. *Biol Sex Differ*. 2021;12:34. doi: 10.1186/s13293-021-00377-0
44. Werner U, Werner D, Heinbüchner S, Graf B, Ince H, Kische S, Thürmann P, König J, Fromm MF, Zolk O. Gender is an important determinant of the disposition of the loop diuretic torasemide. *J Clin Pharmacol*. 2010;50:160–168. doi: 10.1177/0091270009337514
45. Harrold LR, Yood RA, Mikuls TR, Andrade SE, Davis J, Fuller J, Chan KA, Roblin D, Raebel MA, Von Worley A, et al. Sex differences in gout epidemiology: evaluation and treatment. *Ann Rheum Dis*. 2006;65:1368–1372. doi: 10.1136/ard.2006.051649
46. Os I, Bratland B, Dahlöf B, Gisholt K, Syvertsen JO, Tretli S. Female sex as an important determinant of lisinopril-induced cough. *Lancet*. 1992;339:372. doi: 10.1016/0140-6736(92)91694-4
47. Mackay FJ, Pearce GL, Mann RD. Cough and angiotensin II receptor antagonists: cause or confounding? *Br J Clin Pharmacol*. 1999;47:111–114. doi: 10.1046/j.1365-2125.1999.00855.x
48. Kloner RA, Sowers JR, DiBona GF, Gaffney M, Wein M. Sex- and age-related antihypertensive effects of amlodipine. The Amlodipine Cardiovascular Community Trial Study Group. *Am J Cardiol*. 1996;77:713–722. doi: 10.1016/s0002-9149(97)89205-3
49. Ji H, Niiranen TJ, Rader F, Henglin M, Kim A, Ebinger JE, Claggett B, Merz CNB, Cheng S. Sex differences in blood pressure associations with cardiovascular outcomes. *Circulation*. 2021;143:761–763. doi: 10.1161/CIRCULATIONAHA.120.049360
50. Stergiou GS, Palatini P, Parati G, O'Brien E, Januszewicz A, Lurbe E, Persu A, Mancia G, Kreutz R; European Society of Hypertension Council and the European Society of Hypertension Working Group on Blood Pressure Monitoring and Cardiovascular Variability. 2021 European Society of Hypertension practice guidelines for office and out-of-office blood pressure measurement. *J Hypertens*. 2021;39:1293–1302. doi: 10.1097/HJH.0000000000002843
51. Ong KL, Tso AW, Lam KS, Cheung BM. Gender difference in blood pressure control and cardiovascular risk factors in Americans with diagnosed hypertension. *Hypertension*. 2008;51:1142–1148. doi: 10.1161/HYPERTENSIONAHA.107.105205
52. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in obesity among adults in the United States, 2005 to 2014. *JAMA*. 2016;315:2284–2291. doi: 10.1001/jama.2016.6458
53. Peters SAE, Muntner P, Woodward M. Sex differences in the prevalence of, and trends in, cardiovascular risk factors, treatment, and control in the United States, 2001 to 2016. *Circulation*. 2019;139:1025–1035. doi: 10.1161/CIRCULATIONAHA.118.035550
54. Pinho-Gomes AC, Peters SAE, Thomson B, Woodward M. Sex differences in prevalence, treatment and control of cardiovascular risk factors in England. *Heart*. 2021;107:462–467. doi: 10.1136/heartjnl-2020-317446
55. Yanes LL, Reckelhoff JF. Postmenopausal hypertension. *Am J Hypertens*. 2011;24:740–749. doi: 10.1038/ajh.2011.71
56. Abarca-Gómez L, Abdeen ZA, Hamid ZA, Abu-Rmeileh NM, Acosta-Cazares B, Acuin C, Adams RJ, Aekplakorn W, Afsana K, Aguilar-Salinas CA, et al. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *Lancet*. 2017;390:2627–2642. doi: 10.1016/S0140-6736(17)32129-3
57. National center for health statistics. National health interview survey, 2015. Public-use data file and documentation: Nchs tabulations. Http://www.Cdc.Gov/nchs/nhis/nhis_2015_data_release. Htm. Accessed July 18, 2016.
58. Forman JP, Stampfer MJ, Curhan GC. Diet and lifestyle risk factors associated with incident hypertension in women. *JAMA*. 2009;302:401–411. doi: 10.1001/jama.2009.1060
59. Joint Committee for Guideline R. 2018 chinese guidelines for prevention and treatment of hypertension-a report of the revision committee of chinese guidelines for prevention and treatment of hypertension. *J Geriatr Cardiol*. 2019;16:182–241. doi: 10.11909/j.issn.1671-5411.2019.03.014
60. Kim HC, Ihm SH, Kim GH, Kim JH, Kim KI, Lee HY, Lee JH, Park JM, Park S, Pyun WB, et al. 2018 Korean Society of Hypertension guidelines for the management of hypertension: part I-epidemiology of hypertension. *Clin Hypertens*. 2019;25:16. doi: 10.1186/s40885-019-0121-0
61. Kim KI, Ihm SH, Kim GH, Kim HC, Kim JH, Lee HY, Lee JH, Park JM, Park S, Pyun WB, et al. 2018 Korean society of hypertension guidelines for the management of hypertension: part III-hypertension in special situations. *Clin Hypertens*. 2019;25:19. doi: 10.1186/s40885-019-0123-y
62. Lee HY, Shin J, Kim GH, Park S, Ihm SH, Kim HC, Kim KI, Kim JH, Lee JH, Park JM, et al. 2018 Korean Society of Hypertension Guidelines for the management of hypertension: part II-diagnosis and treatment of hypertension. *Clin Hypertens*. 2019;25:20. doi: 10.1186/s40885-019-0124-x
63. Kario K. Key Points of the 2019 Japanese Society of Hypertension Guidelines for the Management of Hypertension. *Korean Circ J*. 2019;49:1123–1135. doi: 10.4070/kcj.2019.0246
64. Shah SN, Munjal YP, Kamath SA, Wander GS, Mehta N, Mukherjee S, Kirpalani A, Gupta P, Shah H, Rohatgi R, et al. Indian guidelines on hypertension-IV (2019). *J Hum Hypertens*. 2020;34:745–758. doi: 10.1038/s41371-020-0349-x
65. Malaysian society of hypertension, ministry of health malaysia, academy of medicine of malaysia. Clinical practice guidelines: Management of hypertension. 2018. Accessed March 16, 2022. https://www.moh.gov.my/moh/resources/penerbitan/CPG/MSH%20Hypertension%20CPG%202018%20V3.8%20FA.pdf.
66. Buranakitjaroen P, Sitthisook S, Wataganara T, Ophascharoensuk V, Bunnag P, Roubsanthisuk W, et al. 2015 thai hypertension guideline; 2015:3. Accessed March 16, 2022. http://www.thaihypertension.org/files/2015%20Thai%20Hypertension%20Guideline.pdf.
67. Task Force of the Latin American Society of Hypertension. Guidelines on the management of arterial hypertension and related comorbidities in Latin America. *J Hypertens*. 2017;35:1529–1545. doi: 10.1097/HJH.0000000000001418
68. Seedat YK, Rayner BL, Veriava Y; Hypertension guideline working group. South African hypertension practice guideline 2014. *Cardiovasc J Afr*. 2014;25:288–294. doi: 10.5830/CVJA-2014-062
69. Department of Health. Assessment of dietary sodium levels among adults (aged 19–64) in England, 2011, June 2012. Accessed March 16, 2022. https://www.gov.uk/government/publications/assessment-of-dietary-sodium-levels-among-adults-aged-19-64-in-england-2011.
70. Huang L, Trieu K, Yoshimura S, Neal B, Woodward M, Campbell NRC, Li Q, Lackland DT, Leung AA, Anderson CAM, et al. Effect of dose and duration of reduction in dietary sodium on blood pressure levels: systematic review and meta-analysis of randomised trials. *BMJ*. 2020;368:m315. doi: 10.1136/bmj.m315
71. Neal B, Wu Y, Feng X, Zhang R, Zhang Y, Shi J, Zhang J, Tian M, Huang L, Li Z, et al. Effect of Salt Substitution on Cardiovascular Events and Death. *N Engl J Med*. 2021;385:1067–1077. doi: 10.1056/NEJMoa2105675
72. Chang HC, Cheng HM, Chen CH, Wang TD, Soenarta AA, Turana Y, Teo BW, Tay JC, Tsoi K, Wang JG, et al. Dietary intervention for the management of hypertension in Asia. *J Clin Hypertens (Greenwich)*. 2021;23:538–544. doi: 10.1111/jch.14116
73. Fulgoni VL 3rd, Agarwal S, Spence L, Samuel P. Sodium intake in US ethnic subgroups and potential impact of a new sodium reduction technology: NHANES Dietary Modeling. *Nutr J*. 2014;13:120. doi: 10.1186/1475-2891-13-120
74. Wigertz K, Palacios C, Jackman LA, Martin BR, McCabe LD, McCabe GP, Peacock M, Pratt JH, Weaver CM. Racial differences in calcium retention in response to dietary salt in adolescent girls. *Am J Clin Nutr*. 2005;81:845–850. doi: 10.1093/ajcn/81.4.845
75. Kokubo Y. Prevention of hypertension and cardiovascular diseases: a comparison of lifestyle factors in Westerners and East Asians. *Hypertension*. 2014;63:655–660. doi: 10.1161/HYPERTENSIONAHA.113.00543
76. Zhou BF, Stamler J, Dennis B, Moag-Stahlberg A, Okuda N, Robertson C, Zhao L, Chan Q, Elliott P; INTERMAP Research Group. Nutrient intakes of middle-aged men and women in China, Japan, United Kingdom, and United States in the late 1990s: the INTERMAP study. *J Hum Hypertens*. 2003;17:623–630. doi: 10.1038/sj.jhh.1001605
77. Puddey IB, Beilin LJ, Vandongen R, Rouse IL, Rogers P. Evidence for a direct effect of alcohol consumption on blood pressure in normotensive men. A randomized controlled trial. *Hypertension*. 1985;7:707–713. doi: 10.1161/01.hyp.7.5.707
78. Roerecke M, Kaczorowski J, Tobe SW, Gmel G, Hasan OSM, Rehm J. The effect of a reduction in alcohol consumption on blood pressure: a systematic review and meta-analysis. *Lancet Public Health*. 2017;2:e108–e120. doi: 10.1016/S2468-2667(17)30003-8
79. Xin X, He J, Frontini MG, Ogden LG, Motsamai OI, Whelton PK. Effects of alcohol reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*. 2001;38:1112–1117. doi: 10.1161/hy1101.093424
80. Https://pubs.Niaaa.Nih.Gov/publications/arh40/152-160.Htm. Access date:2021/08/21.

81. Grzymisławska M, Puch EA, Zawada A, Grzymisławski M. Do nutritional behaviors depend on biological sex and cultural gender? *Adv Clin Exp Med*. 2020;29:165–172. doi: 10.17219/acem/111817
82. Svetkey LP, Simons-Morton D, Vollmer WM, Appel LJ, Conlin PR, Ryan DH, Ard J, Kennedy BM. Effects of dietary patterns on blood pressure: subgroup analysis of the Dietary Approaches to Stop Hypertension (DASH) randomized clinical trial. *Arch Intern Med*. 1999;159:285–293. doi: 10.1001/archinte.159.3.285
83. Song Y, Lobene AJ, Wang Y, Hill Gallant KM. The DASH Diet and cardiometabolic health and chronic kidney disease: a narrative review of the evidence in East Asian Countries. *Nutrients*. 2021;13:984. doi: 10.3390/nu13030984
84. Kawamura A, Kajiya K, Kishi H, Inagaki J, Mitarai M, Oda H, Umemoto S, Kobayashi S. Effects of the DASH-JUMP dietary intervention in Japanese participants with high-normal blood pressure and stage 1 hypertension: an open-label single-arm trial. *Hypertens Res*. 2016;39:777–785. doi: 10.1038/hr.2016.76
85. Epstein DE, Sherwood A, Smith PJ, Craighead L, Caccia C, Lin PH, Babyak MA, Johnson JJ, Hinderliter A, Blumenthal JA. Determinants and consequences of adherence to the dietary approaches to stop hypertension diet in African-American and white adults with high blood pressure: results from the ENCORE trial. *J Acad Nutr Diet*. 2012;112:1763–1773. doi: 10.1016/j.jand.2012.07.007
86. Margetts BM, Martinez JA, Saba A, Holm L, Kearney M, Moles A. Definitions of 'healthy' eating: a pan-EU survey of consumer attitudes to food, nutrition and health. *Eur J Clin Nutr*. 1997;51 Suppl 2:S23–S29.
87. Dongfeng GU, Jiang HE, Xigui WU, Xiufang D, Whelton PK. Effect of potassium supplementation on blood pressure in chinese: a effect of potassium supplementation on blood pressure in chinese: a randomized, placebo-controlled trial. *J Hypertens*. 2001;19:1325–1331. doi: 10.1097/00004872-200107000-00019
88. LANGFORD HG. Dietary potassium and hypertension: epidemiologic data. *Ann Intern Med*. 1983;98:770–772. doi: 10.7326/0003-4819-98-5-770
89. Whelton PK, He J, Cutler JA, Brancati FL, Appel LJ, Follmann D, Klag MJ. Effects of oral potassium on blood pressure. Meta-analysis of randomized controlled clinical trials. *JAMA*. 1997;277:1624–1632. doi: 10.1001/jama.1997.03540440058033
90. Kawano Y, Minami J, Takishita S, Omae T. Effects of potassium supplementation on office, home, and 24-h blood pressure in patients with essential hypertension. *Am J Hypertens*. 1998;11:1141–1146. doi: 10.1016/s0895-7061(98)00037-5
91. Stevens GA, Singh GM, Lu Y, Danaei G, Lin JK, Finucane MM, Bahalim AN, McIntire RK, Gutierrez HR, Cowan M, et al; Global Burden of Metabolic Risk Factors of Chronic Diseases Collaborating Group (Body Mass Index). National, regional, and global trends in adult overweight and obesity prevalences. *Popul Health Metr*. 2012;10:22. doi: 10.1186/1478-7954-10-22
92. Neter JE, Stam BE, Kok FJ, Grobbee DE, Geleijnse JM. Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*. 2003;42:878–884. doi: 10.1161/01.HYP.0000094221.86888.AE
93. Pan WH, Flegal KM, Chang HY, Yeh WT, Yeh CJ, Lee WC. Body mass index and obesity-related metabolic disorders in Taiwanese and US whites and blacks: implications for definitions of overweight and obesity for Asians. *Am J Clin Nutr*. 2004;79:31–39. doi: 10.1093/ajcn/79.1.31
94. Halperin RO, Gaziano JM, Sesso HD. Smoking and the risk of incident hypertension in middle-aged and older men. *Am J Hypertens*. 2008;21:148–152. doi: 10.1038/ajh.2007.36
95. Bowman TS, Gaziano JM, Buring JE, Sesso HD. A prospective study of cigarette smoking and risk of incident hypertension in women. *J Am Coll Cardiol*. 2007;50:2085–2092. doi: 10.1016/j.jacc.2007.08.017
96. Zhang DY, Huang JF, Kang YY, Dou Y, Su YL, Zhang LJ, Cheng YB, Guo QH, Huang QF, Li Y, et al. The prevalence of masked hypertension in relation to cigarette smoking in a Chinese male population. *J Hypertens*. 2020;38:1056–1063. doi: 10.1097/HJH.0000000000002392
97. Noubiap JJ, Nansseu JR, Endomba FT, Ngouo A, Nkeck JR, Nyaga UF, Kaze AD, Bigna JJ. Active smoking among people with diabetes mellitus or hypertension in Africa: a systematic review and meta-analysis. *Sci Rep*. 2019;9:588. doi: 10.1038/s41598-018-37858-z
98. Beaney T, Schutte AE, Tomaszewski M, Ariti C, Burrell LM, Castillo RR, Charchar FJ, Damasceno A, Kruger R, Lackland DT, et al. May measurement month 2017: An analysis of blood pressure screening results worldwide. *Lancet Glob Health*. 2018;6:e736–e743. doi: 10.1016/S2214-109X(18)30259-6
99. Beaney T, Schutte AE, Stergiou GS, Borghi C, Burger D, Charchar F, Cro S, Diaz A, Damasceno A, Espeche W, et al; MMM Investigators*. May Measurement Month 2019: The Global Blood Pressure Screening Campaign of the International Society of Hypertension. *Hypertension*. 2020;76:333–341. doi: 10.1161/HYPERTENSIONAHA.120.14874
100. Glover MJ, Greenlund KJ, Ayala C, Croft JB. Racial/ethnic disparities in prevalence, treatment, and control of hypertension - united states, 1999-2002. *MMWR Morb Mortal Wkly Rep*. 2005;54:7–9.
101. Parcha V, Patel N, Kalra R, Arora G, Arora P. Prevalence, awareness, treatment, and poor control of hypertension among young american adults: race-stratified analysis of the national health and nutrition examination survey. *Mayo Clin Proc*. 2020;95:1390–1403. doi: 10.1016/j.mayocp.2020.01.041
102. Angell SY, Garg RK, Gwynn RC, Bash L, Thorpe LE, Frieden TR. Prevalence, awareness, treatment, and predictors of control of hypertension in New York City. *Circ Cardiovasc Qual Outcomes*. 2008;1:46–53. doi: 10.1161/CIRCOUTCOMES.108.791954
103. Giambrone AE, Gerber LM, Rodriguez-Lopez JS, Trinh-Shevrin C, Islam N, Thorpe LE. Hypertension prevalence in New York City Adults: unmasking undetected racial/ethnic variation, NYC HANES 2004. *Ethn Dis*. 2016;26:339–344. doi: 10.18865/ed.26.3.339
104. Quan H, Chen G, Walker RL, Wielgosz A, Dai S, Tu K, Campbell NR, Hemmelgarn BR, Hill MD, Johansen H, et al; Hypertension Outcome and Surveillance Team. Incidence, cardiovascular complications and mortality of hypertension by sex and ethnicity. *Heart*. 2013;99:715–721. doi: 10.1136/heartjnl-2012-303152
105. Carson AP, Howard G, Burke GL, Shea S, Levitan EB, Muntner P. Ethnic differences in hypertension incidence among middle-aged and older adults: the multi-ethnic study of atherosclerosis. *Hypertension*. 2011;57:1101–1107. doi: 10.1161/HYPERTENSIONAHA.110.168005
106. Chaturvedi N, McKeigue PM, Marmot MG. Resting and ambulatory blood pressure differences in Afro-Caribbeans and Europeans. *Hypertension*. 1993;22:90–96. doi: 10.1161/01.hyp.22.1.90
107. Primatesta P, Bost L, Poulter NR. Blood pressure levels and hypertension status among ethnic groups in England. *J Hum Hypertens*. 2000;14:143–148. doi: 10.1038/sj.jhh.1000960
108. Modesti PA, Reboldi G, Cappuccio FP, Agyemang C, Remuzzi G, Rapi S, Perruolo E, Parati G; ESH Working Group on CV Risk in Low Resource Settings. Panethnic differences in blood pressure in europe: a systematic review and meta-analysis. *PLoS One*. 2016;11:e0147601. doi: 10.1371/journal.pone.0147601
109. Agyemang C, Kieft S, Snijder MB, Beune EJ, van den Born BJ, Brewster LM, Ujcic-Voortman JJ, Bindraban N, van Montfrans G, Peters RJ, et al. Hypertension control in a large multi-ethnic cohort in Amsterdam, The Netherlands: the HELIUS study. *Int J Cardiol*. 2015;183:180–189. doi: 10.1016/j.ijcard.2015.01.061
110. Rabanal KS, Lindman AS, Selmer RM, Aamodt G. Ethnic differences in risk factors and total risk of cardiovascular disease based on the Norwegian CONOR study. *Eur J Prev Cardiol*. 2013;20:1013–1021. doi: 10.1177/2047487312450539
111. Liew SJ, Lee JT, Tan CS, Koh CHG, Van Dam R, Müller-Riemenschneider F. Sociodemographic factors in relation to hypertension prevalence, awareness, treatment and control in a multi-ethnic Asian population: a cross-sectional study. *BMJ Open*. 2019;9:e025869. doi: 10.1136/bmjopen-2018-025869
112. Teh JK, Tey NP, Ng ST. Ethnic and gender differentials in non-communicable diseases and self-rated health in Malaysia. *PLoS One*. 2014;9:e91328. doi: 10.1371/journal.pone.0091328
113. Dong F, Wang D, Pan L, Yu Y, Wang K, Li L, Wang L, Liu T, Zeng X, Sun L, et al. Disparities in hypertension prevalence, awareness, treatment and control between Bouyei and Han: results from a bi-ethnic health survey in developing regions from South China. *Int J Environ Res Public Health*. 2016;13:233. doi: 10.3390/ijerph13020233
114. Li G, Wang H, Wang K, Wang W, Dong F, Qian Y, Gong H, Xu G, Li Y, Pan L, et al. Prevalence, awareness, treatment, control and risk factors related to hypertension among urban adults in Inner Mongolia 2014: differences between Mongolian and Han populations. *BMC Public Health*. 2016;16:294. doi: 10.1186/s12889-016-2965-5
115. Toledo NDN, Almeida GS, Matos MMM, Balieiro AADS, Martin LC, Franco RJDS, Mainbourg EMT. Cardiovascular risk factors: differences between ethnic groups. *Rev Bras Enferm*. 2020;73:e20180918. doi: 10.1590/0034-7167-2018-0918
116. Krishnadath IS, Jaddoe VW, Nahar-van Venrooij LM, Toelsie JR. Ethnic differences in prevalence and risk factors for hypertension in the Suriname

Health Study: a cross sectional population study. *Popul Health Metr*. 2016;14:33. doi: 10.1186/s12963-016-0102-4
117. Olsen MH, Angell SY, Asma S, Boutouyrie P, Burger D, Chirinos JA, Damasceno A, Delles C, Gimenez-Roqueplo AP, Hering D, et al. A call to action and a lifecourse strategy to address the global burden of raised blood pressure on current and future generations: the Lancet Commission on hypertension. *Lancet*. 2016;388:2665–2712. doi: 10.1016/S0140-6736(16)31134-5
118. Schutte AE, Botha S, Fourie CMT, Gafane-Matemane LF, Kruger R, Lammertyn L, Malan L, Mels CMC, Schutte R, Smith W, et al. Recent advances in understanding hypertension development in sub-Saharan Africa. *J Hum Hypertens*. 2017;31:491–500. doi: 10.1038/jhh.2017.18
119. Nilsson PM, Boutouyrie P, Laurent S. Vascular aging: a tale of EVA and ADAM in cardiovascular risk assessment and prevention. *Hypertension*. 2009;54:3–10. doi: 10.1161/HYPERTENSIONAHA.109.129114
120. Schutte AE, Kruger R, Gafane-Matemane LF, Breet Y, Strauss-Kruger M, Cruickshank JK. Ethnicity and arterial stiffness. *Arterioscler Thromb Vasc Biol*. 2020;40:1044–1054. doi: 10.1161/ATVBAHA.120.313133
121. Mokwatsi GG, Schutte AE, Kruger R. Ethnic differences regarding arterial stiffness of 6-8-year-old black and white boys. *J Hypertens*. 2017;35:960–967. doi: 10.1097/HJH.0000000000001267
122. Markert MS, Della-Morte D, Cabral D, Roberts EL Jr, Gardener H, Dong C, Wright CB, Elkind MS, Sacco RL, Rundek T. Ethnic differences in carotid artery diameter and stiffness: the Northern Manhattan Study. *Atherosclerosis*. 2011;219:827–832. doi: 10.1016/j.atherosclerosis.2011.08.028
123. Snijder MB, Stronks K, Agyemang C, Busschers WB, Peters RJ, van den Born BJ. Ethnic differences in arterial stiffness the Helius study. *Int J Cardiol*. 2015;191:28–33. doi: 10.1016/j.ijcard.2015.04.234
124. Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988-2000. *JAMA*. 2003;290:199–206. doi: 10.1001/jama.290.2.199
125. Fei K, Rodriguez-Lopez JS, Ramos M, Islam N, Trinh-Shevrin C, Yi SS, Chernov C, Perlman SE, Thorpe LE. Racial and ethnic subgroup disparities in hypertension prevalence, New York City Health and Nutrition Examination Survey, 2013-2014. *Prev Chronic Dis*. 2017;14:E33. doi: 10.5888/pcd14.160478
126. Cutler JA, Sorlie PD, Wolz M, Thom T, Fields LE, Roccella EJ. Trends in hypertension prevalence, awareness, treatment, and control rates in United States adults between 1988-1994 and 1999-2004. *Hypertension*. 2008;52:818–827. doi: 10.1161/HYPERTENSIONAHA.108.113357
127. Levine DA, Duncan PW, Nguyen-Huynh MN, Ogedegbe OG. Interventions targeting racial/ethnic disparities in stroke prevention and treatment. *Stroke*. 2020;51:3425–3432. doi: 10.1161/STROKEAHA.120.030427
128. Muntner P, He J, Cutler JA, Wildman RP, Whelton PK. Trends in blood pressure among children and adolescents. *JAMA*. 2004;291:2107–2113. doi: 10.1001/jama.291.17.2107
129. Wong MD, Shapiro MF, Boscardin WJ, Ettner SL. Contribution of major diseases to disparities in mortality. *N Engl J Med*. 2002;347:1585–1592. doi: 10.1056/NEJMsa012979
130. Fiscella K, Holt K. Racial disparity in hypertension control: tallying the death toll. *Ann Fam Med*. 2008;6:497–502. doi: 10.1370/afm.873
131. Howard G, Lackland DT, Kleindorfer DO, Kissela BM, Moy CS, Judd SE, Safford MM, Cushman M, Glasser SP, Howard VJ. Racial differences in the impact of elevated systolic blood pressure on stroke risk. *JAMA Intern Med*. 2013;173:46–51. doi: 10.1001/2013.jamainternmed.857
132. Lackland DT, Keil JE, Gazes PC, Hames CG, Tyroler HA. Outcomes of black and white hypertensive individuals after 30 years of follow-up. *Clin Exp Hypertens*. 1995;17:1091–1105. doi: 10.3109/10641969509033654
133. Hardy ST, Loehr LR, Butler KR, Chakladar S, Chang PP, Folsom AR, Heiss G, MacLehose RF, Matsushita K, Avery CL. Reducing the blood pressure-related burden of cardiovascular disease: impact of achievable improvements in blood pressure prevention and control. *J Am Heart Assoc*. 2015;4:e002276. doi: 10.1161/JAHA.115.002276
134. Feigin VL, Stark BA, Johnson CO, Roth GA, Bisignano C, Abady GG, Abbasifard M, Abbasi-Kangevari M, Abd-Allah F, Abedi V, et al. Global, regional, and national burden of stroke and its risk factors, 1990-2019: a systematic analysis for the global burden of disease study 2019. *Lancet Neurol*. 2021;20:795–820. doi: 10.1016/S1474-4422(21)00252-0
135. Arima H, Murakami Y, Lam TH, Kim HC, Ueshima H, Woo J, Suh I, Fang X, Woodward M; Asia Pacific Cohort Studies Collaboration. Effects of prehypertension and hypertension subtype on cardiovascular disease in the Asia-Pacific Region. *Hypertension*. 2012;59:1118–1123. doi: 10.1161/HYPERTENSIONAHA.111.187252
136. Hyun KK, Huxley RR, Arima H, Woo J, Lam TH, Ueshima H, Fang X, Peters SA, Jee SH, Giles GG, et al. A comparative analysis of risk factors and stroke risk for Asian and non-Asian men: the Asia Pacific cohort studies collaboration. *Int J Stroke*. 2013;8:606–611. doi: 10.1111/ijs.12166
137. Woodward M, Tsukinoki-Murakami R, Murakami Y, Suh I, Fang X, Ueshima H, Lam TH; Asia–Pacific Cohort Studies Collaboration. The epidemiology of stroke amongst women in the Asia-Pacific region. *Womens Health (Lond)*. 2011;7:305–317. doi: 10.2217/whe.11.25
138. O'Donnell MJ, Chin SL, Rangarajan S, Xavier D, Liu L, Zhang H, Rao-Melacini P, Zhang X, Pais P, Agapay S, et al; INTERSTROKE investigators. Global and regional effects of potentially modifiable risk factors associated with acute stroke in 32 countries (INTERSTROKE): a case-control study. *Lancet*. 2016;388:761–775. doi: 10.1016/S0140-6736(16)30506-2
139. Somali health authorities, world health organisation. Hospital and referral health centre guidelines. 2015. Accessed March 16, 2022. https://www.humanitarianresponse.info/sites/www.humanitarianresponse.info/files/documents/files/3.hospital_rhc_stgs_-_federal_government_of_somalia.pdf.
140. Ojji DB, Mayosi B, Francis V, Badri M, Cornelius V, Smythe W, Kramer N, Barasa F, Damasceno A, Dzudie A, et al; CREOLE Study Investigators. Comparison of dual therapies for lowering blood pressure in Black Africans. *N Engl J Med*. 2019;380:2429–2439. doi: 10.1056/NEJMoa1901113
141. Erlinger TP, Vollmer WM, Svetkey LP, Appel LJ. The potential impact of nonpharmacologic population-wide blood pressure reduction on coronary heart disease events: pronounced benefits in African-Americans and hypertensives. *Prev Med*. 2003;37:327–333. doi: 10.1016/s0091-7435(03)00140-3
142. Yano Y, Briasoulis A, Bakris GL, Hoshide S, Wang JG, Shimada K, Kario K. Effects of antihypertensive treatment in Asian populations: a meta-analysis of prospective randomized controlled studies (CARdiovascular protectioN group in Asia: CARNA). *J Am Soc Hypertens*. 2014;8:103–116. doi: 10.1016/j.jash.2013.09.002
143. Ogihara T, Saruta T, Rakugi H, Saito I, Shimamoto K, Matsuoka H, Shimada K, Ito S, Horiuchi M, Imaizumi T, et al; COLM Investigators. Combinations of olmesartan and a calcium channel blocker or a diuretic in elderly hypertensive patients: a randomized, controlled trial. *J Hypertens*. 2014;32:2054–63; discussiom 2063. doi: 10.1097/HJH.0000000000000281
144. group N-Es. Randomized double-blind comparison of a calcium antagonist and a diuretic in elderly hypertensives. *Hypertension*. 1999;34:1129–1133
145. Uzu T, Kimura G. Diuretics shift circadian rhythm of blood pressure from nondipper to dipper in essential hypertension. *Circulation*. 1999;100:1635–1638. doi: 10.1161/01.cir.100.15.1635
146. Brewster LM, van Montfrans GA, Oehlers GP, Seedat YK. Systematic review: antihypertensive drug therapy in patients of African and South Asian ethnicity. *Intern Emerg Med*. 2016;11:355–374. doi: 10.1007/s11739-016-1422-x
147. Jeemon P, Séverin T, Amodeo C, Balabanova D, Campbell NRC, Gaita D, Kario K, Khan T, Melifonwu R, Moran A, et al. World Heart Federation Roadmap for Hypertension - A 2021 Update. *Glob Heart*. 2021;16:63. doi: 10.5334/gh.1066
148. Kario K, Chen CH, Park S, Park CG, Hoshide S, Cheng HM, Huang QF, Wang JG. Consensus document on improving hypertension management in Asian patients, taking into Account Asian Characteristics. *Hypertension*. 2018;71:375–382. doi: 10.1161/HYPERTENSIONAHA.117.10238
149. Oyebode O, Oti S, Chen YF, Lilford RJ. Salt intakes in sub-Saharan Africa: a systematic review and meta-regression. *Popul Health Metr*. 2016;14:1. doi: 10.1186/s12963-015-0068-7
150. Mozaffarian D, Fahimi S, Singh GM, Micha R, Khatibzadeh S, Engell RE, Lim S, Danaei G, Ezzati M, Powles J; Global Burden of Diseases Nutrition and Chronic Diseases Expert Group. Global sodium consumption and death from cardiovascular causes. *N Engl J Med*. 2014;371:624–634. doi: 10.1056/NEJMoa1304127
151. Wang TJ, Vasan RS. Epidemiology of uncontrolled hypertension in the United States. *Circulation*. 2005;112:1651–1662. doi: 10.1161/CIRCULATIONAHA.104.490599
152. Gu A, Yue Y, Desai RP, Argulian E. Racial and ethnic differences in antihypertensive medication use and blood pressure control among us adults with hypertension. *Circ Cardiovasc Qual Outcomes*. 2017;10:e003166.doi: 10.1161/CIRCOUTCOMES.116.003166
153. Deere BP, Ferdinand KC. Hypertension and race/ethnicity. *Curr Opin Cardiol*. 2020;35:342–350. doi: 10.1097/HCO.0000000000000742
154. Yoon SS, Fryar CD, Carroll MD. Hypertension prevalence and control among adults: United States, 2011–2014. *NCHS data brief, no 220*. Hyattsville, MD: National Center for Health Statistics. 2015.

155. Clemmer JS, Pruett WA, Lirette ST. Corrigendum: racial and sex differences in the response to first-line antihypertensive therapy. *Front Cardiovasc Med*. 2020;7:643289. doi: 10.3389/fcvm.2020.643289
156. Howard VJ, Madsen TE, Kleindorfer DO, Judd SE, Rhodes JD, Soliman EZ, Kissela BM, Safford MM, Moy CS, McClure LA, et al. Sex and race differences in the association of incident ischemic stroke with risk factors. *JAMA Neurol*. 2019;76:179–186. doi: 10.1001/jamaneurol.2018.3862
157. McSweeney JC, Rosenfeld AG, Abel WM, Braun LT, Burke LE, Daugherty SL, Fletcher GF, Gulati M, Mehta LS, Pettey C, et al; American Heart Association Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, Council on Epidemiology and Prevention, Council on Hypertension, Council on Lifestyle and Cardiometabolic Health, and Council on Quality of Care and Outcomes Research. Preventing and experiencing ischemic heart disease as a woman: state of the science: a scientific statement from the American Heart Association. *Circulation*. 2016;133:1302–1331. doi: 10.1161/CIR.0000000000000381
158. Finkelstein EA, Khavjou OA, Mobley LR, Haney DM, Will JC. Racial/ethnic disparities in coronary heart disease risk factors among WISEWOMAN enrollees. *J Womens Health (Larchmt)*. 2004;13:503–518. doi: 10.1089/1540999041280963
159. Wu P, Chew-Graham CA, Maas AH, Chappell LC, Potts JE, Gulati M, Jordan KP, Mamas MA. Temporal changes in hypertensive disorders of pregnancy and impact on cardiovascular and obstetric outcomes. *Am J Cardiol*. 2020;125:1508–1516. doi: 10.1016/j.amjcard.2020.02.029
160. Chen JS, Roberts CL, Simpson JM, Ford JB. Prevalence of pre-eclampsia, pregnancy hypertension and gestational diabetes in population-based data: impact of different ascertainment methods on outcomes. *Aust N Z J Obstet Gynaecol*. 2012;52:91–95. doi: 10.1111/j.1479-828X.2011.01378.x
161. Moodley J, Onyangunga OA, Maharaj NR. Hypertensive disorders in primigravid black South African women: a one-year descriptive analysis. *Hypertens Pregnancy*. 2016;35:529–535. doi: 10.1080/10641955.2016.1193190
162. Walle TA, Azagew AW. Hypertensive disorder of pregnancy prevalence and associated factors among pregnant women attending ante natal care at Gondar town health Institutions, North West Ethiopia 2017. *Pregnancy Hypertens* 2019;16:79–84. doi: 10.1016/j.preghy.2019.03.007
163. Say L, Chou D, Gemmill A, Tunçalp Ö, Moller AB, Daniels J, Gülmezoglu AM, Temmerman M, Alkema L. Global causes of maternal death: a WHO systematic analysis. *Lancet Glob Health*. 2014;2:e323–e333. doi: 10.1016/S2214-109X(14)70227-X
164. Ananth CV, Duzyj CM, Yadava S, Schwebel M, Tita ATN, Joseph KS. Changes in the prevalence of chronic hypertension in pregnancy, United States, 1970 to 2010. *Hypertension*. 2019;74:1089–1095. doi: 10.1161/HYPERTENSIONAHA.119.12968
165. Savitz DA, Danilack VA, Engel SM, Elston B, Lipkind HS. Descriptive epidemiology of chronic hypertension, gestational hypertension, and preeclampsia in New York State, 1995-2004. *Matern Child Health J*. 2014;18:829–838. doi: 10.1007/s10995-013-1307-9
166. Butwick AJ, Druzin ML, Shaw GM, Guo N. Evaluation of US state-level variation in hypertensive disorders of pregnancy. *JAMA Netw Open*. 2020;3:e2018741. doi: 10.1001/jamanetworkopen.2020.18741
167. Abalos E, Cuesta C, Carroli G, Qureshi Z, Widmer M, Vogel JP, Souza JP; WHO Multicountry Survey on Maternal and Newborn Health Research Network. Pre-eclampsia, eclampsia and adverse maternal and perinatal outcomes: a secondary analysis of the World Health Organization Multicountry Survey on Maternal and Newborn Health. *BJOG*. 2014;121 Suppl 1:14–24. doi: 10.1111/1471-0528.12629
168. Ahmad AS, Samuelsen SO. Hypertensive disorders in pregnancy and fetal death at different gestational lengths: a population study of 2 121 371 pregnancies. *BJOG*. 2012;119:1521–1528. doi: 10.1111/j.1471-0528.2012.03460.x
169. Umesawa M, Kobashi G. Epidemiology of hypertensive disorders in pregnancy: prevalence, risk factors, predictors and prognosis. *Hypertens Res*. 2017;40:213–220. doi: 10.1038/hr.2016.126
170. Laine K, Murzakanova G, Sole KB, Pay AD, Heradstveit S, Räisänen S. Prevalence and risk of pre-eclampsia and gestational hypertension in twin pregnancies: a population-based register study. *BMJ Open*. 2019;9:e029908. doi: 10.1136/bmjopen-2019-029908
171. Auger N, Luo ZC, Nuyt AM, Kaufman JS, Naimi AI, Platt RW, Fraser WD. Secular trends in preeclampsia incidence and outcomes in a large Canada database: a longitudinal study over 24 years. *Can J Cardiol*. 2016;32:987.e15–987.e23. doi: 10.1016/j.cjca.2015.12.011
172. Abalos E, Cuesta C, Grosso AL, Chou D, Say L. Global and regional estimates of preeclampsia and eclampsia: a systematic review. *Eur J Obstet Gynecol Reprod Biol*. 2013;170:1–7. doi: 10.1016/j.ejogrb.2013.05.005
173. Ananth CV, Keyes KM, Wapner RJ. Pre-eclampsia rates in the united states, 1980-2010: Age-period-cohort analysis. *BMJ*. 2013;347:f6564.
174. Tanaka M, Jaamaa G, Kaiser M, Hills E, Soim A, Zhu M, Shcherbatykh IY, Samelson R, Bell E, Zdeb M, et al. Racial disparity in hypertensive disorders of pregnancy in New York State: a 10-year longitudinal population-based study. *Am J Public Health*. 2007;97:163–170. doi: 10.2105/AJPH.2005.068577
175. Anderson NH, Sadler LC, Stewart AW, Fyfe EM, McCowan LM. Ethnicity, body mass index and risk of pre-eclampsia in a multiethnic New Zealand population. *Aust N Z J Obstet Gynaecol*. 2012;52:552–558. doi: 10.1111/j.1479-828X.2012.01475.x
176. Bouthoorn SH, Gaillard R, Steegers EA, Hofman A, Jaddoe VW, van Lenthe FJ, Raat H. Ethnic differences in blood pressure and hypertensive complications during pregnancy: the Generation R study. *Hypertension*. 2012;60:198–205. doi: 10.1161/HYPERTENSIONAHA.112.194365
177. Ghosh G, Grewal J, Männistö T, Mendola P, Chen Z, Xie Y, Laughon SK. Racial/ethnic differences in pregnancy-related hypertensive disease in nulliparous women. *Ethn Dis*. 2014;24:283–289.
178. Miller EC, Zambrano Espinoza MD, Huang Y, Friedman AM, Boehme AK, Bello NA, Cleary KL, Wright JD, D'Alton ME. Maternal race/ethnicity, hypertension, and risk for stroke during delivery admission. *J Am Heart Assoc*. 2020;9:e014775. doi: 10.1161/JAHA.119.014775
179. Minhas AS, Ogunwole SM, Vaught AJ, Wu P, Mamas MA, Gulati M, Zhao D, Hays AG, Michos ED. Racial disparities in cardiovascular complications with pregnancy-induced hypertension in the United States. *Hypertension* 2021;78:480–488. doi: 10.1161/HYPERTENSIONAHA.121.17104
180. Petersen EE, Davis NL, Goodman D, Cox S, Mayes N, Johnston E, Syverson C, Seed K, Shapiro-Mendoza CK, Callaghan WM, et al. Vital signs: pregnancy-related deaths, United States, 2011-2015, and strategies for prevention, 13 States, 2013-2017. *MMWR Morb Mortal Wkly Rep*. 2019;68:423–429. doi: 10.15585/mmwr.mm6818e1
181. Carmichael SL, Kan P, Padula AM, Rehkopf DH, Oehlert JW, Mayo JA, Weber AM, Wise PH, Shaw GM, Stevenson DK. Social disadvantage and the black-white disparity in spontaneous preterm delivery among California births. *PLoS One*. 2017;12:e0182862. doi: 10.1371/journal.pone.0182862
182. Kramer MS. Socioeconomic disparities in preterm birth. *Paediatr Perinat Epidemiol*. 2015;29:169–171. doi: 10.1111/ppe.12186
183. Knuist M, Bonsel GJ, Zondervan HA, Treffers PE. Risk factors for pre-eclampsia in nulliparous women in distinct ethnic groups: a prospective cohort study. *Obstet Gynecol*. 1998;92:174–178. doi: 10.1016/s0029-7844(98)00143-4
184. Winkleby MA, Kraemer HC, Ahn DK, Varady AN. Ethnic and socioeconomic differences in cardiovascular disease risk factors: findings for women from the Third National Health and Nutrition Examination Survey, 1988-1994. *JAMA* 1998;280:356–362. doi: 10.1001/jama.280.4.356
185. Gadson A, Akpovi E, Mehta PK. Exploring the social determinants of racial/ethnic disparities in prenatal care utilization and maternal outcome. *Semin Perinatol*. 2017;41:308–317. doi: 10.1053/j.semperi.2017.04.008
186. The world health organization. Maternal mortality ratio (per 100 000 live births) 2019. Accessed March 16, 2022. https://www.who.int/data/gho/indicator-metadata-registry/imr-details/26.
187. Al-Rubaie ZTA, Malcolm Hudson H, Jenkins G, Mahmoud I, Ray JG, Askie LM, Lord SJ. The association between ethnicity and pre-eclampsia in Australia: a multicentre retrospective cohort study. *Aust N Z J Obstet Gynaecol*. 2020;60:396–404. doi: 10.1111/ajo.13069
188. Naimy Z, Grytten J, Monkerud L, Eskild A. The prevalence of pre-eclampsia in migrant relative to native Norwegian women: a population-based study. *BJOG*. 2015;122:859–865. doi: 10.1111/1471-0528.12978
189. Mogos MF, Salinas-Miranda AA, Salemi JL, Medina IM, Salihu HM. Pregnancy-related hypertensive disorders and immigrant status: a systematic review and meta-analysis of epidemiological studies. *J Immigr Minor Health*. 2017;19:1488–1497. doi: 10.1007/s10903-016-0410-6
190. Asanin J, Wilson K. "I spent nine years looking for a doctor": exploring access to health care among immigrants in Mississauga, Ontario, Canada. *Soc Sci Med*. 2008;66:1271–1283. doi: 10.1016/j.socscimed.2007.11.043
191. Bellamy L, Casas JP, Hingorani AD, Williams DJ. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis. *BMJ*. 2007;335:974. doi: 10.1136/bmj.39335.385301.BE
192. Benschop L, Duvekot JJ, Roeters van Lennep JE. Future risk of cardiovascular disease risk factors and events in women after a hypertensive disorder of pregnancy. *Heart*. 2019;105:1273–1278. doi: 10.1136/heartjnl-2018-313453
193. Brown MC, Best KE, Pearce MS, Waugh J, Robson SC, Bell R. Cardiovascular disease risk in women with pre-eclampsia: systematic review

and meta-analysis. *Eur J Epidemiol*. 2013;28:1–19. doi: 10.1007/s10654-013-9762-6
194. Cunningham MW Jr, LaMarca B. Risk of cardiovascular disease, end-stage renal disease, and stroke in postpartum women and their fetuses after a hypertensive pregnancy. *Am J Physiol Regul Integr Comp Physiol*. 2018;315:R521–R528. doi: 10.1152/ajpregu.00218.2017
195. Lee G, Tubby J. Preeclampsia and the risk of cardiovascular disease later in life–A review of the evidence. *Midwifery*. 2015;31:1127–1134. doi: 10.1016/j.midw.2015.09.005
196. Leslie MS, Briggs LA. Preeclampsia and the risk of future vascular disease and mortality: a review. *J Midwifery Womens Health*. 2016;61:315–324. doi: 10.1111/jmwh.12469
197. Lo CCW, Lo ACQ, Leow SH, Fisher G, Corker B, Batho O, Morris B, Chowaniec M, Vladutiu CJ, Fraser A, et al. Future cardiovascular disease risk for women with gestational hypertension: a systematic review and meta-analysis. *J Am Heart Assoc*. 2020;9:e013991. doi: 10.1161/JAHA.119.013991
198. Wu P, Haththotuwa R, Kwok CS, Babu A, Kotronias RA, Rushton C, Zaman A, Fryer AA, Kadam U, Chew-Graham CA, et al. Preeclampsia and future cardiovascular health: a systematic review and meta-analysis. *Circ Cardiovasc Qual Outcomes*. 2017;10:e003497. doi: 10.1161/CIRCOUTCOMES.116.003497
199. Bushnell C, McCullough LD, Awad IA, Chireau MV, Fedder WN, Furie KL, Howard VJ, Lichtman JH, Lisabeth LD, Piña IL, et al; American Heart Association Stroke Council; Council on Cardiovascular and Stroke Nursing; Council on Clinical Cardiology; Council on Epidemiology and Prevention; Council for High Blood Pressure Research. Guidelines for the prevention of stroke in women: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2014;45:1545–1588. doi: 10.1161/01.str.0000442009.06663.48
200. Colafella KMM, Denton KM. Sex-specific differences in hypertension and associated cardiovascular disease. *Nat Rev Nephrol*. 2018;14:185–201. doi: 10.1038/nrneph.2017.189
201. Orshal JM, Khalil RA. Gender, sex hormones, and vascular tone. *Am J Physiol Regul Integr Comp Physiol*. 2004;286:R233–R249. doi: 10.1152/ajpregu.00338.2003
202. Coffman TM. Under pressure: the search for the essential mechanisms of hypertension. *Nat Med*. 2011;17:1402–1409. doi: 10.1038/nm.2541
203. Zimmerman MA, Sullivan JC. Hypertension: what's sex got to do with it? *Physiology (Bethesda)*. 2013;28:234–244. doi: 10.1152/physiol.00013.2013
204. Sullivan JC. Sex and the renin-angiotensin system: inequality between the sexes in response to RAS stimulation and inhibition. *Am J Physiol Regul Integr Comp Physiol*. 2008;294:R1220–R1226. doi: 10.1152/ajpregu.00864.2007
205. Chappell MC, Marshall AC, Alzayadneh EM, Shaltout HA, Diz DI. Update on the Angiotensin converting enzyme 2-Angiotensin (1-7)-MAS receptor axis: fetal programing, sex differences, and intracellular pathways. *Front Endocrinol (Lausanne)*. 2014;4:201. doi: 10.3389/fendo.2013.00201
206. Pérez-López FR, Larrad-Mur L, Kallen A, Chedraui P, Taylor HS. Gender differences in cardiovascular disease: hormonal and biochemical influences. *Reprod Sci*. 2010;17:511–531. doi: 10.1177/1933719110367829
207. Pessôa BS, Slump DE, Ibrahimi K, Grefhorst A, van Veghel R, Garrelds IM, Roks AJ, Kushner SA, Danser AH, van Esch JH. Angiotensin II type 2 receptor- and acetylcholine-mediated relaxation: essential contribution of female sex hormones and chromosomes. *Hypertension*. 2015;66:396–402. doi: 10.1161/HYPERTENSIONAHA.115.05303
208. Gillis EE, Sullivan JC. Sex differences in hypertension: recent advances. *Hypertension*. 2016;68:1322–1327. doi: 10.1161/HYPERTENSIONAHA.116.06602
209. Jones DW, Hall JE. Racial and ethnic differences in blood pressure: biology and sociology. *Circulation*. 2006;114:2757–2759. doi: 10.1161/CIRCULATIONAHA.106.668731
210. Opie LH, Seedat YK. Hypertension in sub-Saharan African populations. *Circulation*. 2005;112:3562–3568. doi: 10.1161/CIRCULATIONAHA.105.539569
211. Gafane-Matemane LF, Kruger R, Smith W, Mels CMC, Van Rooyen JM, Mokwatsi GG, Uys AS, Brits SJ, Schutte AE. Characterization of the renin-angiotensin-aldosterone system in young healthy black adults: the African Prospective Study on the Early Detection and Identification of Hypertension and Cardiovascular Disease (African-PREDICT Study). *Hypertension*. 2021;78:400–410. doi: 10.1161/HYPERTENSIONAHA.120.16879
212. Katsuya T, Ishikawa K, Sugimoto K, Rakugi H, Ogihara T. Salt sensitivity of Japanese from the viewpoint of gene polymorphism. *Hypertens Res*. 2003;26:521–525. doi: 10.1291/hypres.26.521
213. Wang R, Zhong B, Liu Y, Wang C. Association between alpha-adducin gene polymorphism (Gly460Trp) and genetic predisposition to salt sensitivity: a meta-analysis. *J Appl Genet*. 2010;51:87–94. doi: 10.1007/BF03195715
214. Adeloye D, Basquill C. Estimating the prevalence and awareness rates of hypertension in Africa: a systematic analysis. *PLoS One*. 2014;9:e104300. doi: 10.1371/journal.pone.0104300
215. Bosu WK, Bosu DK. Prevalence, awareness and control of hypertension in Ghana: A systematic review and meta-analysis. *PLoS One*. 2021;16:e0248137. doi: 10.1371/journal.pone.0248137
216. Bosu WK, Reilly ST, Aheto JMK, Zucchelli E. Hypertension in older adults in Africa: A systematic review and meta-analysis. *PLoS One*. 2019;14:e0214934. doi: 10.1371/journal.pone.0214934
217. Kaze AD, Schutte AE, Erqou S, Kengne AP, Echouffo-Tcheugui JB. Prevalence of hypertension in older people in Africa: a systematic review and meta-analysis. *J Hypertens*. 2017;35:1345–1352. doi: 10.1097/HJH.0000000000001345
218. Kibret KT, Mesfin YM. Prevalence of hypertension in Ethiopia: a systematic meta-analysis. *Public Health Rev*. 2015;36:14. doi: 10.1186/s40985-015-0014-z
219. Tiruneh SA, Bukayaw YA, Yigizaw ST, Angaw DA. Prevalence of hypertension and its determinants in Ethiopia: a systematic review and meta-analysis. *PLoS One*. 2020;15:e0244642. doi: 10.1371/journal.pone.0244642
220. Kuate Defo B, Mbanya JC, Kingue S, Tardif JC, Choukem SP, Perreault S, Fournier P, Ekundayo O, Potvin L, D'Antono B, et al. Blood pressure and burden of hypertension in Cameroon, a microcosm of Africa: a systematic review and meta-analysis of population-based studies. *J Hypertens*. 2019;37:2190–2199. doi: 10.1097/HJH.0000000000002165
221. Bao M, Wang L. The longitudinal trend of hypertension prevalence in Chinese adults from 1959 to 2018: a systematic review and meta-analysis. *Ann Palliat Med*. 2020;9:2485–2497. doi: 10.21037/apm-19-377
222. Chen X, Li L, Zhou T, Li Z. Prevalence of hypertension in rural areas of china: a meta-analysis of published studies. *PLoS One*. 2014;9:e115462. doi: 10.1371/journal.pone.0115462
223. Chen X, Wei W, Zou S, Wu X, Zhou B, Fu L, Wang H, Shi J. Trends in the prevalence of hypertension in island and coastal areas of china: a systematic review with meta-analysis. *Am J Hypertens*. 2014;27:1503–1510. doi: 10.1093/ajh/hpu026
224. Wang X, Bots ML, Yang F, Hoes AW, Vaartjes I. Prevalence of hypertension in China: a systematic review and meta-regression analysis of trends and regional differences. *J Hypertens*. 2014;32:1919–27; discussion 1927. doi: 10.1097/HJH.0000000000000252
225. Wang Y, Peng X, Nie X, Chen L, Weldon R, Zhang W, Xiao D, Cai J. Burden of hypertension in China over the past decades: systematic analysis of prevalence, treatment and control of hypertension. *Eur J Prev Cardiol*. 2016;23:792–800. doi: 10.1177/2047487315617105
226. Ma YQ, Mei WH, Yin P, Yang XH, Rastegar SK, Yan JD. Prevalence of hypertension in Chinese cities: a meta-analysis of published studies. *PLoS One*. 2013;8:e58302. doi: 10.1371/journal.pone.0058302
227. Chow ZY, Jun SM, Ching SM, Tan CH, Lee KW, Devaraj NK, Syahida H, Ramachandran V, Hoo FK, Cheong AT, et al. Prevalence, awareness and control of hypertension in malaysia 1980 – 2017: a systematic review and meta-analysis. *bioRxiv*. 2019:625004.
228. Chowdhury MZI, Rahman M, Akter T, Akhter T, Ahmed A, Shovon MA, Farhana Z, Chowdhury N, Turin TC. Hypertension prevalence and its trend in Bangladesh: evidence from a systematic review and meta-analysis. *Clin Hypertens*. 2020;26:10. doi: 10.1186/s40885-020-00143-1
229. Naing C, Aung K. Prevalence and risk factors of hypertension in myanmar: a systematic review and meta-analysis. *Medicine (Baltimore)*. 2014;93:e100. doi: 10.1097/MD.0000000000000100
230. Rizwan SA, Kumar R, Singh AK, Kusuma YS, Yadav K, Pandav CS. Prevalence of hypertension in Indian tribes: a systematic review and meta-analysis of observational studies. *PLoS One*. 2014;9:e95896. doi: 10.1371/journal.pone.0095896
231. Soo MJ, Chow ZY, Ching SM, Tan CH, Lee KW, Devaraj NK, Salim HS, Ramachandran V, Lim PY, Sivaratnam D. Prevalence, awareness and control of hypertension in malaysia from 1980-2018: a systematic review and meta-analysis. *World J Meta Anal*. 2020;8:320–344
232. Haghdoost AA, Sadeghirad B, Rezazadehkermani M. Epidemiology and heterogeneity of hypertension in Iran: a systematic review. *Arch Iran Med*. 2008;11:444–452. doi: 08114/AIM.0017
233. Mirzaei M, Moayedallaie S, Jabbari L, Mohammadi M. Prevalence of hypertension in Iran 1980-2012: a systematic review. *J Tehran Heart Cent*. 2016;11:159–167.

234. Oori MJ, Mohammadi F, Norozi K, Fallahi-Khoshknab M, Ebadi A, Gheshlagh RG. Prevalence of HTN in Iran: meta-analysis of published studies in 2004-2018. *Curr Hypertens Rev*. 2019;15:113–122. doi: 10.2174/1573402115666190118142818
235. Afsargharehbagh R, Rezaie-Keikhaie K, Rafiemanesh H, Balouchi A, Bouya S, Dehghan B. Hypertension and pre-hypertension among Iranian adults population: a meta-analysis of prevalence, awareness, treatment, and control. *Curr Hypertens Rep*. 2019;21:27. doi: 10.1007/s11906-019-0933-z
236. Hörnsten C, Weidung B, Littbrand H, Carlberg B, Nordström P, Lövheim H, Gustafson Y. High blood pressure as a risk factor for incident stroke among very old people: a population-based cohort study. *J Hypertens*. 2016;34:2059–2065. doi: 10.1097/HJH.0000000000001048
237. Jung MH, Yi SW, An SJ, Yi JJ. Age-specific associations between systolic blood pressure and cardiovascular mortality. *Heart*. 2019;105:1070–1077. doi: 10.1136/heartjnl-2019-314697
238. Kim H, Lee S, Ha E, Kwon SH, Jeon JS, Noh H, Han DC, Oh HJ, Ryu DR. Age and sex specific target of blood pressure for the prevention of cardiovascular event among the treatment naive hypertensive patients. *Sci Rep*. 2020;10:21538. doi: 10.1038/s41598-020-78641-3
239. Komi R, Tanaka F, Omama S, Ishibashi Y, Tanno K, Onoda T, Ohsawa M, Tanaka K, Okayama A, Nakamura M; Iwate-Kenco Study Group. Burden of high blood pressure as a contributing factor to stroke in the Japanese community-based diabetic population. *Hypertens Res*. 2018;41:531–538. doi: 10.1038/s41440-018-0042-4
240. Lai YJ, Chen HC, Chou P. Gender difference in the interaction effects of diabetes and hypertension on stroke among the elderly in the Shih-Pai Study, Taiwan. *PLoS One*. 2015;10:e0136634. doi: 10.1371/journal.pone.0136634
241. Sobieraj P, Lewandowski J, Siński M, Symonides B, Gaciong Z. Low diastolic blood pressure is not related to risk of first episode of stroke in a high-risk population: a secondary analysis of SPRINT. *J Am Heart Assoc*. 2019;8:e010811. doi: 10.1161/JAHA.118.010811
242. Wang Y, Dai Y, Zheng J, Xie Y, Guo R, Guo X, Sun G, Sun Z, Sun Y, Zheng L. Sex difference in the incidence of stroke and its corresponding influence factors: results from a follow-up 8.4 years of rural China hypertensive prospective cohort study. *Lipids Health Dis*. 2019;18:72. doi: 10.1186/s12944-019-1010-y
243. Yu Y, Liu L, Huang J, Shen G, Chen C, Huang Y, Zhang B, Tang S, Feng Y. Association between systolic blood pressure and first ischemic stroke in the chinese older hypertensive population. *J Int Med Res*. 2020;48:0300060520920091
244. Cushman WC, Evans GW, Byington RP, Goff DC, Jr., Grimm RH, Jr., Cutler JA, Simons-Morton DG, Basile JN, Corson MA, Probstfield JL, et al. Effects of intensive blood-pressure control in type 2 diabetes mellitus. *N Engl J Med*. 2010;362:1575–1585. doi: 10.1056/NEJMoa1001286
245. Verdecchia P, Staessen JA, Angeli F, de Simone G, Achilli A, Ganau A, Mureddu G, Pede S, Maggioni AP, Lucci D, et al; Cardio-Sis investigators. Usual versus tight control of systolic blood pressure in non-diabetic patients with hypertension (Cardio-Sis): an open-label randomised trial. *Lancet*. 2009;374:525–533. doi: 10.1016/S0140-6736(09)61340-4
246. Five-year findings of the hypertension detection and follow-up program: Iii. Reduction in stroke incidence among persons with high blood pressure. *JAMA*. 1982;247:633–638.
247. Wright JT, Jr., Williamson JD, Whelton PK, Snyder JK, Sink KM, Rocco MV, Reboussin DM, Rahman M, Oparil S, Lewis CE, et al. A randomized trial of intensive versus standard blood-pressure control. *N Engl J Med*. 2015;373:2103–2116. doi: 10.1056/NEJMoa1511939
248. Benavente OR, Coffey CS, Conwit R, Hart RG, McClure LA, Pearce LA, Pergola PE, Szychowski JM. Blood-pressure targets in patients with recent lacunar stroke: The sps3 randomised trial. *Lancet*. 2013;382:507–515. doi: 10.1016/S0140-6736(13)60852-1
249. Ogihara T, Saruta T, Rakugi H, Matsuoka H, Shimamoto K, Shimada K, Imai Y, Kikuchi K, Ito S, Eto T, et al; Valsartan in Elderly Isolated Systolic Hypertension Study Group. Target blood pressure for treatment of isolated systolic hypertension in the elderly: valsartan in elderly isolated systolic hypertension study. *Hypertension*. 2010;56:196–202. doi: 10.1161/HYPERTENSIONAHA.109.146035
250. Campos CL, Rodriguez CJ. High blood pressure in Hispanics in the United States: a review. *Curr Opin Cardiol*. 2019;34:350–358. doi: 10.1097/HCO.0000000000000636
251. Delgado J, Jacobs EA, Lackland DT, Evans DA, de Leon CF. Differences in blood pressure control in a large population-based sample of older African Americans and non-Hispanic whites. *J Gerontol A Biol Sci Med Sci*. 2012;67:1253–1258. doi: 10.1093/gerona/gls106
252. Holmes L, Hossain J, Ward D, Opara F. Racial/ethnic variability in hypertension prevalence and risk factors in national health interview survey. *ISRN Hypertension*. 2013;2013:257842.
253. Thorpe RJ Jr, Bowie JV, Smolen JR, Bell CN, Jenkins ML Jr, Jackson J, LaVeist TA. Racial disparities in hypertension awareness and management: are there differences among African Americans and Whites living under similar social conditions? *Ethn Dis*. 2014;24:269–275.
254. Yi S, Elfassy T, Gupta L, Myers C, Kerker B. Nativity, language spoken at home, length of time in the United States, and race/ethnicity: associations with self-reported hypertension. *Am J Hypertens*. 2014;27:237–244. doi: 10.1093/ajh/hpt209
255. Zhao B, Jose PO, Pu J, Chung S, Ancheta IB, Fortmann SP, Palaniappan LP. Racial/ethnic differences in hypertension prevalence, treatment, and control for outpatients in northern California 2010-2012. *Am J Hypertens*. 2015;28:631–639. doi: 10.1093/ajh/hpu189
256. Howard G, Cushman M, Moy CS, Oparil S, Muntner P, Lackland DT, Manly JJ, Flaherty ML, Judd SE, Wadley VG, et al. Association of clinical and social factors with excess hypertension risk in black compared with White US Adults. *JAMA*. 2018;320:1338–1348. doi: 10.1001/jama.2018.13467
257. Jung MY, Lee S, Thomas SB, Juon HS. Hypertension prevalence, treatment, and related behaviors among asian americans: an examination by method of measurement and disaggregated subgroups. *J Racial Ethn Health Disparities*. 2019;6:584–593. doi: 10.1007/s40615-018-00557-6
258. Lane D, Beevers DG, Lip GY. Ethnic differences in blood pressure and the prevalence of hypertension in England. *J Hum Hypertens*. 2002;16:267–273. doi: 10.1038/sj.jhh.1001371
259. Barrera L, Gómez F, Ortega-Lenis D, Corchuelo Ojeda J, Méndez F. Prevalence, awareness, treatment and control of high blood pressure in the elderly according to the ethnic group. Colombian survey. *Colomb Med (Cali)*. 2019;50:115–127. doi: 10.25100/cm.v50i2.4124
260. Abu-Saad K, Chetrit A, Eilat-Adar S, Alpert G, Atamna A, Gillon-Keren M, Rogowski O, Ziv A, Kalter-Leibovici O. Blood pressure level and hypertension awareness and control differ by marital status, sex, and ethnicity: a population-based study. *Am J Hypertens*. 2014;27:1511–1520. doi: 10.1093/ajh/hpu081
261. Chen SC, Lo TC, Chang JH, Kuo HW. Ethnic disparities in blood pressure: a population-based study. *J Immigr Minor Health*. 2017;19:1427–1433. doi: 10.1007/s10903-016-0434-y
262. Willey JZ, Moon YP, Kahn E, Rodriguez CJ, Rundek T, Cheung K, Sacco RL, Elkind MS. Population attributable risks of hypertension and diabetes for cardiovascular disease and stroke in the northern Manhattan study. *J Am Heart Assoc*. 2014;3:e001106. doi: 10.1161/JAHA.114.001106
263. Goff DC Jr, Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Gibbons R, Greenland P, Lackland DT, Levy D, O'Donnell CJ, et al; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;129(25 Suppl 2):S49–S73. doi: 10.1161/01.cir.0000437741.48606.98
264. Ministry of health sierra leone non communicable diseases: Diagnosis and treatment desk guide. 2017. Accessed March 16, 2022. http://intranet.qmu.ac.uk/media/6507/nihr-ncd-clinical-deskguide-sierraleone.pdf.
265. Ministry of health rwanda. National guideline for management of Non-Communicable Diseases (NCDS). 2016. Accessed March 16, 2022. http://ncdsynergies.org/wp-content/uploads/2018/02/IMB-NCDs-guideline.pdf.
266. Government of tanzania. Standard treatment guidelines & national essential medicines list tanzania mainland. 2017. Accessed March 16, 2022. https://www.who.int/selection_medicines/country_lists/Tanzania_STG_052013.pdf.
267. Dagnaw WW, Yadeta D, Feleke Y, Kebede T. Ethiopian national guideline on major ncds 2016 guidelines on clinical and programmatic management of major non communicable diseases; 2016. Accessed March 16, 2022. https://extranet.who.int/ncdccs/Data/ETH_D1_National%20NCD%20Guideline%20June%2010,%202016%20for%20print.pdf.
268. Ministry of Health Kenya. Ministry of health kenya national guidelines for cardiovascular diseases management division of non-communicable diseases Republic of Kenya Kenya National Guidelines for cardiovascular diseases management division of non-communicable diseases. 2018. Accessed March 16, 2022. https://www.health.go.ke/wp-content/uploads/2018/06/Cardiovascular-guidelines-2018_A4_Final.pdf.
269. Republic of Zambia, Ministry of Health. Standard treatment guidelines, essential medicines list and essential laboratory supplies list for Zambia. 2017. Accessed March 16, 2022. https://www.medbox.org/preview/5d07a18a-d840-411c-a736-599c1fcc7b87/doc.pdf.

270. Government of Zimbabwe. 7th essential medicines list and standard treatment guidelines for zimbabwe. 2015. Accessed March 16, 2022. https://extranet.who.int/ncdccs/Data/ZWE_D1_7th%20edition%20EDLIZ%202015%20_%20Final%20Version%20with%20Signatures.docx.
271. Ogihara T, Matsuoka H, Rakugi H. Practitioner's trial on the efficacy of antihypertensive treatment in elderly patients with hypertension II (PATE-hypertension II study) in Japan. *Geriatr Gerontol Int.* 2011;11:414–421. doi: 10.1111/j.1447-0594.2011.00690.x
272. Muramatsu T, Matsushita K, Yamashita K, Kondo T, Maeda K, Shintani S, Ichimiya S, Ohno M, Sone T, Ikeda N, et al; NAGOYA HEART Study Investigators. Comparison between valsartan and amlodipine regarding cardiovascular morbidity and mortality in hypertensive patients with glucose intolerance: NAGOYA HEART Study. *Hypertension.* 2012;59:580–586. doi: 10.1161/HYPERTENSIONAHA.111.184226
273. Narumi H, Takano H, Shindo S, Fujita M, Mizuma H, Kuwabara Y, Komuro I; Valsartan Amlodipine Randomized Trial Investigators. Effects of valsartan and amlodipine on cardiorenal protection in Japanese hypertensive patients: the Valsartan Amlodipine Randomized Trial. *Hypertens Res.* 2011;34:62–69. doi: 10.1038/hr.2010.186
274. Yui Y, Sumiyoshi T, Kodama K, Hirayama A, Nonogi H, Kanmatsuse K, Origasa H, Iimura O, Ishii M, Saruta T, et al; Japan Multicenter Investigation for Cardiovascular Diseases-B Study Group. Comparison of nifedipine retard with angiotensin converting enzyme inhibitors in Japanese hypertensive patients with coronary artery disease: the Japan Multicenter Investigation for Cardiovascular Diseases-B (JMIC-B) randomized trial. *Hypertens Res.* 2004;27:181–191. doi: 10.1291/hypres.27.181
275. Nakamura T, Kanno Y, Takenaka T, Suzuki H; Efficacy of Candesartan on Outcome in Saitama Trial Group. An angiotensin receptor blocker reduces the risk of congestive heart failure in elderly hypertensive patients with renal insufficiency. *Hypertens Res.* 2005;28:415–423. doi: 10.1291/hypres.28.415
276. Chan JC, Wat NM, So WY, Lam KS, Chua CT, Wong KS, Morad Z, Dickson TZ, Hille D, Zhang Z, et al; Asian RENAAL Study Investigators. Renin angiotensin aldosterone system blockade and renal disease in patients with type 2 diabetes. An Asian perspective from the RENAAL Study. *Diabetes Care.* 2004;27:874–879. doi: 10.2337/diacare.27.4.874
277. Kasanuki H, Hagiwara N, Hosoda S, Sumiyoshi T, Honda T, Haze K, Nagashima M, Yamaguchi J, Origasa H, Urashima M, et al; HIJ-CREATE Investigators. Angiotensin II receptor blocker-based vs. non-angiotensin II receptor blocker-based therapy in patients with angiographically documented coronary artery disease and hypertension: the Heart Institute of Japan Candesartan Randomized Trial for Evaluation in Coronary Artery Disease (HIJ-CREATE). *Eur Heart J.* 2009;30:1203–1212. doi: 10.1093/eurheartj/ehp101
278. Sakata Y, Shiba N, Takahashi J, Miyata S, Nochioka K, Miura M, Takada T, Saga C, Shinozaki T, Sugi M, et al; SUPPORT Trial Investigators; SUPPORT Trial Investigators. Clinical impacts of additive use of olmesartan in hypertensive patients with chronic heart failure: the supplemental benefit of an angiotensin receptor blocker in hypertensive patients with stable heart failure using olmesartan (SUPPORT) trial. *Eur Heart J.* 2015;36:915–923. doi: 10.1093/eurheartj/ehu504
279. Imai E, Chan JC, Ito S, Yamasaki T, Kobayashi F, Haneda M, Makino H; ORIENT study investigators. Effects of olmesartan on renal and cardiovascular outcomes in type 2 diabetes with overt nephropathy: a multicentre, randomised, placebo-controlled study. *Diabetologia.* 2011;54:2978–2986. doi: 10.1007/s00125-011-2325-z
280. Iseki K, Arima H, Kohagura K, Komiya I, Ueda S, Tokuyama K, Shiohira Y, Uehara H, Toma S; Olmesartan Clinical Trial in Okinawan Patients Under OKIDS (OCTOPUS) Group. Effects of Angiotensin Receptor Blockade (ARB) on mortality and cardiovascular outcomes in patients with long-term haemodialysis: a randomized controlled trial. *Nephrol Dial Transplant.* 2013;28:1579–1589. doi: 10.1093/ndt/gfs590
281. Liu L, Zhang Y, Liu G, Li W, Zhang X, Zanchetti A; FEVER Study Group. The Felodipine Event Reduction (FEVER) Study: a randomized long-term placebo-controlled trial in Chinese hypertensive patients. *J Hypertens.* 2005;23:2157–2172. doi: 10.1097/01.hjh.0000194120.42722.ac
282. Kim-Mitsuyama S, Ogawa H, Matsui K, Jinnouchi T, Jinnouchi H, Arakawa K. An angiotensin II receptor blocker-calcium channel blocker combination prevents cardiovascular events in elderly high-risk hypertensive patients with chronic kidney disease better than high-dose angiotensin II receptor blockade alone. *Kidney Int.* 2013;83:167–176. doi: 10.1038/ki.2012.326
283. Gong L, Zhang W, Zhu Y, Zhu J, Kong D, Pagé V, Ghadirian P, LeLorier J, Hamet P. Shanghai trial of nifedipine in the elderly (STONE). *J Hypertens.* 1996;14:1237–1245. doi: 10.1097/00004872-199610000-00013
284. Matsuzaki M, Ogihara T, Umemoto S, Rakugi H, Matsuoka H, Shimada K, Abe K, Suzuki N, Eto T, Higaki J, et al; Combination Therapy of Hypertension to Prevent Cardiovascular Events Trial Group. Prevention of cardiovascular events with calcium channel blocker-based combination therapies in patients with hypertension: a randomized controlled trial. *J Hypertens.* 2011;29:1649–1659. doi: 10.1097/HJH.0b013e328348345d
285. Liu L, Wang Z, Gong L, Zhang Y, Thijs L, Staessen JA, Wang J. Blood pressure reduction for the secondary prevention of stroke: a Chinese trial and a systematic review of the literature. *Hypertens Res.* 2009;32:1032–1040. doi: 10.1038/hr.2009.139
286. Wang JG, Staessen JA, Gong L, Liu L. Chinese trial on isolated systolic hypertension in the elderly. Systolic Hypertension in China (Syst-China) Collaborative Group. *Arch Intern Med.* 2000;160:211–220. doi: 10.1001/archinte.160.2.211
287. Liu L, Wang JG, Gong L, Liu G, Staessen JA. Comparison of active treatment and placebo in older Chinese patients with isolated systolic hypertension. Systolic Hypertension in China (Syst-China) Collaborative Group. *J Hypertens.* 1998;16(12 Pt 1):1823–1829. doi: 10.1097/00004872-199816120-00016
288. Liu LS, Gong LS, Wang W; Blood Pressure Lowering to Prevent Recurrent Stroke Study Group. [Effects of blood pressure lowering treatment on stroke recurrence in patients with cerebrovascular diseases-a large-scale, randomized, placebo controlled trial]. *Zhonghua Xin Xue Guan Bing Za Zhi.* 2005;33:613–617.
289. Chiu M, Austin PC, Manuel DG, Tu JV. Comparison of cardiovascular risk profiles among ethnic groups using population health surveys between 1996 and 2007. *CMAJ.* 2010;182:E301–E310. doi: 10.1503/cmaj.091676
290. Miranda ML, Swamy GK, Edwards S, Maxson P, Gelfand A, James S. Disparities in maternal hypertension and pregnancy outcomes: evidence from North Carolina, 1994-2003. *Public Health Rep.* 2010;125:579–587. doi: 10.1177/003335491012500413
291. Cameron NA, Molsberry R, Pierce JB, Perak AM, Grobman WA, Allen NB, Greenland P, Lloyd-Jones DM, Khan SS. Pre-pregnancy hypertension among women in rural and urban areas of the United States. *J Am Coll Cardiol.* 2020;76:2611–2619. doi: 10.1016/j.jacc.2020.09.601
292. Mogos MF, Jones LM, Robinson NS, Whitehead AO, Piscotty R, Goba GK. Prevalence, correlates, and outcomes of co-occurring depression and hypertensive disorders of pregnancy. *J Womens Health (Larchmt).* 2019;28:1460–1467. doi: 10.1089/jwh.2018.7144
293. Nakagawa K, Lim E, Harvey S, Miyamura J, Juarez DT. Racial/Ethnic disparities in the association between preeclampsia risk factors and preeclampsia among women residing in Hawaii. *Matern Child Health J.* 2016;20:1814–1824. doi: 10.1007/s10995-016-1984-2
294. O'Connor HD, Hehir MP, Kent EM, Foley ME, Fitzpatrick C, Geary MP, Malone FD. Eclampsia: trends in incidence and outcomes over 30 years. *Am J Perinatol.* 2013;30:661–664. doi: 10.1055/s-0032-1331026
295. Vousden N, Lawley E, Seed PT, Gidiri MF, Goudar S, Sandall J, Chappell LC, Shennan AH; CRADLE Trial Collaborative Group. Incidence of eclampsia and related complications across 10 low- and middle-resource geographical regions: Secondary analysis of a cluster randomised controlled trial. *PLoS Med.* 2019;16:e1002775. doi: 10.1371/journal.pmed.1002775
296. Carr A, Kershaw T, Brown H, Allen T, Small M. Hypertensive disease in pregnancy: an examination of ethnic differences and the Hispanic paradox. *J Neonatal Perinatal Med.* 2013;6:11–15. doi: 10.3233/NPM-1356111
297. Xiao J, Shen F, Xue Q, Chen G, Zeng K, Stone P, Zhao M, Chen Q. Is ethnicity a risk factor for developing preeclampsia? An analysis of the prevalence of preeclampsia in China. *J Hum Hypertens.* 2014;28:694–698. doi: 10.1038/jhh.2013.148
298. Lanska DJ, Kryscio RJ. Risk factors for peripartum and postpartum stroke and intracranial venous thrombosis. *Stroke.* 2000;31:1274–1282. doi: 10.1161/01.str.31.6.1274
299. Irgens HU, Reisaeter L, Irgens LM, Lie RT. Long term mortality of mothers and fathers after pre-eclampsia: population based cohort study. *BMJ.* 2001;323:1213–1217. doi: 10.1136/bmj.323.7323.1213
300. Ros HS, Lichtenstein P, Bellocco R, Petersson G, Cnattingius S. Pulmonary embolism and stroke in relation to pregnancy: how can high-risk women be identified? *Am J Obstet Gynecol.* 2002;186:198–203. doi: 10.1067/mob.2002.119177
301. Wilson BJ, Watson MS, Prescott GJ, Sunderland S, Campbell DM, Hannaford P, Smith WC. Hypertensive diseases of pregnancy and risk of hypertension and stroke in later life: results from cohort study. *BMJ.* 2003;326:845. doi: 10.1136/bmj.326.7394.845
302. James AH, Bushnell CD, Jamison MG, Myers ER. Incidence and risk factors for stroke in pregnancy and the puerperium. *Obstet Gynecol.* 2005;106:509–516. doi: 10.1097/01.AOG.0000172428.78411.b0

303. Bateman BT, Schumacher HC, Bushnell CD, Pile-Spellman J, Simpson LL, Sacco RL, Berman MF. Intracerebral hemorrhage in pregnancy: frequency, risk factors, and outcome. *Neurology*. 2006;67:424–429. doi: 10.1212/01.wnl.0000228277.84760.a2
304. Brown DW, Dueker N, Jamieson DJ, Cole JW, Wozniak MA, Stern BJ, Giles WH, Kittner SJ. Preeclampsia and the risk of ischemic stroke among young women: results from the Stroke Prevention in Young Women Study. *Stroke*. 2006;37:1055–1059. doi: 10.1161/01.STR.0000206284.96739.ee
305. Lykke JA, Langhoff-Roos J, Sibai BM, Funai EF, Triche EW, Paidas MJ. Hypertensive pregnancy disorders and subsequent cardiovascular morbidity and type 2 diabetes mellitus in the mother. *Hypertension*. 2009;53:944–951. doi: 10.1161/HYPERTENSIONAHA.109.130765
306. Tang CH, Wu CS, Lee TH, Hung ST, Yang CY, Lee CH, Chu PH. Preeclampsia-eclampsia and the risk of stroke among peripartum in Taiwan. *Stroke*. 2009;40:1162–1168. doi: 10.1161/STROKEAHA.108.540880
307. Garovic VD, Bailey KR, Boerwinkle E, Hunt SC, Weder AB, Curb D, Mosley TH Jr, Wiste HJ, Turner ST. Hypertension in pregnancy as a risk factor for cardiovascular disease later in life. *J Hypertens*. 2010;28:826–833. doi: 10.1097/HJH.0b013e328335c29a
308. Lin YS, Tang CH, Yang CY, Wu LS, Hung ST, Hwa HL, Chu PH. Effect of pre-eclampsia-eclampsia on major cardiovascular events among peripartum women in Taiwan. *Am J Cardiol*. 2011;107:325–330. doi: 10.1016/j.amjcard.2010.08.073
309. Wang IK, Chang SN, Liao CC, Liang CC, Chang CT, Lin HH, Liu JH, Liu YL, Chuang FR, Hsu CY, et al. Hypertensive disorders in pregnancy and preterm delivery and subsequent stroke in Asian women: a retrospective cohort study. *Stroke*. 2011;42:716–721. doi: 10.1161/STROKEAHA.110.594523
310. Stuart JJ, Rimm EB, Missmer SA, Spiegelman D, Hibert EN, Rexrode KM, Mukamal KJ, Rich-Edwards JW. Hypertensive disorders in pregnancy and risk of myocardial infarction and stroke. *Am J Epidemiol*. 2013;177:S41–S41
311. Männistö T, Mendola P, Vääräsmäki M, Järvelin MR, Hartikainen AL, Pouta A, Suvanto E. Elevated blood pressure in pregnancy and subsequent chronic disease risk. *Circulation*. 2013;127:681–690. doi: 10.1161/CIRCULATIONAHA.112.128751
312. Hovsepian DA, Sriram N, Kamel H, Fink ME, Navi BB. Acute cerebrovascular disease occurring after hospital discharge for labor and delivery. *Stroke*. 2014;45:1947–1950. doi: 10.1161/STROKEAHA.114.005129
313. Savitz DA, Danilack VA, Elston B, Lipkind HS. Pregnancy-induced hypertension and diabetes and the risk of cardiovascular disease, stroke, and diabetes hospitalization in the year following delivery. *Am J Epidemiol*. 2014;180:41–44. doi: 10.1093/aje/kwu118
314. Heida KY, Franx A, van Rijn BB, Eijkemans MJ, Boer JM, Verschuren MW, Oudijk MA, Bots ML, van der Schouw YT. Earlier age of onset of chronic hypertension and Type 2 diabetes mellitus after a hypertensive disorder of pregnancy or gestational diabetes mellitus. *Hypertension*. 2015;66:1116–1122. doi: 10.1161/HYPERTENSIONAHA.115.06005
315. Canoy D, Cairns BJ, Balkwill A, Wright FL, Khalil A, Beral V, Green J, Reeves G; Million Women Study Collaborators. Hypertension in pregnancy and risk of coronary heart disease and stroke: a prospective study in a large UK cohort. *Int J Cardiol*. 2016;222:1012–1018. doi: 10.1016/j.ijcard.2016.07.170
316. Lin LT, Tsui KH, Cheng JT, Cheng JS, Huang WC, Liou WS, Tang PL. Increased Risk of intracranial hemorrhage in patients with pregnancy-induced hypertension: a nationwide population-based retrospective cohort study. *Medicine (Baltimore)*. 2016;95:e3732. doi: 10.1097/MD.0000000000003732
317. Nelander M, Cnattingius S, Åkerud H, Wikström J, Pedersen NL, Wikström AK. Pregnancy hypertensive disease and risk of dementia and cardiovascular disease in women aged 65 years or older: a cohort study. *BMJ Open*. 2016;6:e009880. doi: 10.1136/bmjopen-2015-009880
318. Tooher J, Thornton C, Makris A, Ogle R, Korda A, Hennessy A. All Hypertensive disorders of pregnancy increase the risk of future cardiovascular disease. *Hypertension*. 2017;70:798–803. doi: 10.1161/HYPERTENSIONAHA.117.09246
319. Riise HKR, Sulo G, Tell GS, Igland J, Nygård O, Iversen AC, Daltveit AK. Association between gestational hypertension and risk of cardiovascular disease among 617 589 Norwegian Women. *J Am Heart Assoc*. 2018;7:e008337. doi: 10.1161/JAHA.117.008337
320. Andolf EG, Sydsjö GC, Bladh MK, Berg G, Sharma S. Hypertensive disorders in pregnancy and later dementia: a Swedish National Register Study. *Acta Obstet Gynecol Scand*. 2017;96:464–471. doi: 10.1111/aogs.13096
321. Honigberg MC, Zekavat SM, Aragam K, Klarin D, Bhatt DL, Scott NS, Peloso GM, Natarajan P. Long-term cardiovascular risk in women with hypertension during pregnancy. *J Am Coll Cardiol*. 2019;74:2743–2754. doi: 10.1016/j.jacc.2019.09.052
322. Leon LJ, McCarthy FP, Direk K, Gonzalez-Izquierdo A, Prieto-Merino D, Casas JP, Chappell L. Preeclampsia and cardiovascular disease in a large uk pregnancy cohort of linked electronic health records: A CALIBER Study. *Circulation*. 2019;140:1050–1060. doi: 10.1161/CIRCULATIONAHA.118.038080
323. Garovic VD, White WM, Vaughan L, Saiki M, Parashuram S, Garcia-Valencia O, Weissgerber TL, Milic N, Weaver A, Mielke MM. Incidence and long-term outcomes of hypertensive disorders of pregnancy. *J Am Coll Cardiol*. 2020;75:2323–2334. doi: 10.1016/j.jacc.2020.03.028
324. Theilen LH, Fraser A, Hollingshaus MS, Schliep KC, Varner MW, Smith KR, Esplin MS. All-cause and cause-specific mortality after hypertensive disease of pregnancy. *Obstet Gynecol*. 2016;128:238–244. doi: 10.1097/AOG.0000000000001534
325. Bhattacharya S, Prescott GJ, Iversen L, Campbell DM, Smith WC, Hannaford PC. Hypertensive disorders of pregnancy and future health and mortality: a record linkage study. *Pregnancy Hypertens*. 2012;2:1–7. doi: 10.1016/j.preghy.2011.08.116
326. Hannaford P, Ferry S, Hirsch S. Cardiovascular sequelae of toxaemia of pregnancy. *Heart*. 1997;77:154–158. doi: 10.1136/hrt.77.2.154
327. Ray JG, Vermeulen MJ, Schull MJ, Redelmeier DA. Cardiovascular health after maternal placental syndromes (CHAMPS): population-based retrospective cohort study. *Lancet*. 2005;366:1797–1803. doi: 10.1016/S0140-6736(05)67726-4