Review Article
Volume 16:10, 2022

Journal of Molecular and Genetic Medicine

ISSN: 1747-0862 Open Access

The Components of Lifestyle Adjustments in Hypertension Guidelines Which Consider Gene and Environmental Interactions

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Abstract

Lifestyle choices and genetics are two types of risk factors for hypertension. Heritability estimates for BP range from 34–67%, according to studies of twins and family history. About 20% of the population variation in BP has been explained by the most recent paper on BP GWAS. Due to violations of shared environment assumptions, poor phenotyping practices in control cohorts, failure to account for epistasis, gene-gene and gene-environment interactions and other non-genetic sources of phenotype modulation, twin studies may have overestimated heritability. GWAS may have underestimated heritability. The proposals of hypertension rules in significant nations comprise of the accompanying components: Dietary sodium reduction, weight loss, increased physical activity, quitting smoking and moderate alcohol consumption are some of the other strategies. Beyond race and culture, the guidelines for hypertension are generally the same in every country or region. By describing lifestyle changes based on the hypertension guidelines, we present a synopsis of gene-environment interactions associated with hypertension in this review. In the age of precision medicine, doctors who are in charge of managing hypertension should think about how genes interact with the environment and the right aspects of a healthy lifestyle can help prevent and treat hypertension. We briefly discussed how genetic and environmental factors interact with the components of the guidelines for hypertension; however, there is not yet sufficient evidence and the results of genetic factors frequently varied from study to study.

Keywords: Hypertension • Physical activity • Lifestyle adjustments

Introduction

The most significant risk factor for cardiovascular disease (CVD) is hypertension. Recent research suggests that hypertension is also linked to common non-CVD conditions like renal dysfunction and dementia. Lifestyle choices and genetics are two types of risk factors for hypertension. Blood pressure (BP) estimates based on family history and twin studies range from 34% to 67%. The aggregate impact of all BP loci recognized through far reaching affiliation studies (GWAS) represented just 3.5% of BP changeability. The most recent BP GWAS paper identified 901 SNPs that are associated with BP and explained approximately 20% of the BP population variation. Due to violations of shared environment assumptions, poor phenotyping practices in control cohorts, failure to account for epistasis, gene-gene (GG) and gene-environment (GE) interactions and other non-genetic sources of phenotype modulation, twin studies may have overestimated heritability. GWAS may have underestimated heritability.

Description

The following components make up the recommendations of major international guidelines for hypertension: weight loss, a healthy diet (high in fruits, vegetables, whole grains, legumes, seeds, nuts, fish, low-fat dairy, low in meat and sweets and low in sodium), increased physical activity, quitting smoking (including avoiding passive smoking) and moderate alcohol consumption. Beyond race and culture, the guidelines for hypertension are generally the same in every country or region. By describing lifestyle changes based on the hypertension guidelines, we present a synopsis of gene-environment interactions associated with hypertension in this review. According to the INTERSALT study, excessive salt intake is linked to high blood pressure. According to the Dietary Approaches to Stop Hypertension (DASH) study, systolic blood pressure (DBP) and diastolic blood pressure (SBP) were both reduced when sodium intake was restricted from a high level to an intermediate level and from an intermediate level to a low level. According to a pooled analysis of the data, the best people to target for lowering sodium intake are people with hypertension who consume high-sodium diets [1].

Guidelines for managing hypertension base their recommendations on these outcomes: salt intakes of less than 5 g per day in Europe and 6 g per day in Japan and sodium intakes of less than 1500 mg per day (salt intake of less than 3) 81 g/day in the United States. Salt sensitivity is caused by genetic and environmental factors and manifests as an increase in blood pressure in response to excessive salt consumption in the diet. Salt sensitivity is more common in hypertensive people than in normotensive people, in people of colour than in Caucasians and in older people than in younger people. The subjects' race and age group must be taken into account when studying gene-sodium interactions. According to the findings of a cross-sectional study conducted in Korea, the mutant alleles of CSK rs1378942 and CSK-MIR4513 rs3784789 provided the subjects in the middle group of the 24-hour estimated urinary sodium-potassium excretion ratio with the strongest protection against hypertension [2].

Through the examination of an SNP, demonstrated that the interaction for CLGN rs2567241 was associated with the effects of sodium intake on SBP, DBP and mean blood pressure (MBP), the effects of UST rs13211840 on DBP and the effects of LOC105369882 rs11104632 on SBP. MKNK1, C2orf80, EPHA6, SCOC-AS1, SCOC, CLGN, MGAT4D, ARHGAP42, CASP4 and LINC01478 were also found to be linked to at least one BP variable through genome-wide gene-based interactions with sodium. Salt intake and

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Received: 01 October, 2022, Manuscript No: jmgm-22-82807; **Editor Assigned:** 03 October, 2022, Pre-QC No. P-82807; **Reviewed:** 15 October, 2022, QC No. Q-82807; **Revised:** 20 October, 2022, Manuscript No: R-82807; **Published:** 28 October, 2022, DOI: 10.37421/1747-0862.2022.16.581

ACE genotype were found to interact with hypertension in Chinese Kazakh women. East Asian men consume alcohol at a higher rate than Western men, but Western women consume alcohol at a higher rate than East Asian women. The so-called "Oriental flushing syndrome" can be attributed to the fact that aldehyde dehydrogenase (ALDH) deficiency affects approximately half of East Asians. ALDH inadequacy represents an expanded gamble of high BP [3].

The apolipoprotein E phenotype had a significant impact on the BP-raising effects of alcohol consumption in a study of middle-aged Finnish men. A cross-sectional investigation of a Chinese populace showed a huge communication between the CYP11B2 genotype and DNA methylation (CpG1 methylation) of the ADD1 quality advertiser and liquor utilization on the gamble of hypertension. In addition, the Stanford Asia-Pacific Program for Hypertension and Insulin Resistance (SAPPHIRe) study demonstrated that in a prospective Chinese cohort, ALDH2 genetic variants were linked to the development of hypertension. In a case-control study of 532 Japanese patients, there was no significant interaction between the ALDH2 genotype and alcohol consumption overall or in Japanese male patients: in a cross-sectional study of 5724 Japanese participants, ALDH2 rs671 significantly and synergistically influenced the subjects' drinking behavior and influenced the level of BP independently of the amount of alcohol consumed. It's possible that this study didn't have enough power to find the interaction [4].

The 2015 Global Burden of Disease Study found that men in central and Eastern Europe and Southeast Asia smoked more than the global average, while women in western and central Europe smoked more than the global average. Men and women in the East Asian region had higher population-attributable fractions of coronary heart disease caused by smoking than in the Western Pacific region. The cigarette smoking index and the ACE gene had a low exposure-gene effect on essential hypertension with interaction indices in a rural Chinese population. An increased risk of essential hypertension was linked to the gene-environment interactions between rs1126742 and smoking in an eastern Chinese Han population. In nonsmoking Chinese hypertensive patients, a case-control study demonstrated that KCNJ11 gene polymorphisms were associated with BP response to the antihypertensive drug irbesartan. An analysis of 6889 participants in the Framingham Heart Study revealed 7 significant and 21 suggestive BP loci through gene-smoking interactions. This was a genome-wide study.

Adult obesity rates worldwide rose from 28.8% and 29.8% in 1980 to 36.9% and 38.0% in 2013 for men and women, respectively, in both developed and developing nations. In developing nations, the prevalence of overweight and obesity among children and adolescents is also rising, rising from 8.1% and 8.4% in 1980 to 12.9% and 13.4% in 2013. SBP and DBP decreased by 1.05 mmHg and 0.92 mmHg, respectively, when body weight decreased by 1 kg, according to a meta-analysis of 25 studies. As a result, lowering blood pressure requires obese people to lose weight. According to the Atherosclerosis Risk in Communities Study, there was a significant interaction between the GNB3 C825T polymorphism, obesity status and physical activity in predicting hypertension in African American subjects.

Those who were both obese and had a low level of activity with the T allele were 2.7 times more likely to be hypertensive than those who were not obese and had active C homozygotes. The FTO and MC4-R loci contain the representative SNPs associated with BMI. In various ethnic groups, SNPs in FTO were associated with hypertension. Despite having the highest obesity rate in the world, the Pima Indians of Arizona have a relatively low prevalence of hypertension and atherosclerotic disease. Although the Pima Indian population has a high prevalence of obesity and hyperinsulinemia, it is unknown why this population has a low tendency for hypertension. The lack of increase in muscle sympathetic nerve activity with increasing adiposity and insulinemia may partially explain this [5].

Conclusion

We provide a summary of the gene-environment interactions that are associated with hypertension by describing common lifestyle modifications that are recommended by major country hypertension guidelines and consist of the following components: Dietary sodium reduction, weight loss, increased physical activity, quitting smoking and moderate alcohol consumption are some of the other strategies. We briefly discussed how genetic and environmental factors interact with the components of the guidelines for hypertension; however, there is not yet sufficient evidence and the results of genetic factors frequently varied from study to study.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Ritchie, Krista. "The Components of Lifestyle Adjustments in Hypertension Guidelines Which Consider Gene and Environmental Interactions." J Mol Genet Med 16 (2022): 581.