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Biotechnological Tools in Genetics for Primary Prophylaxis of Essential Arterial Hypertension

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***Abstract*— hypertension is one of the most spread cardiological diseases, due to recent biotechnological tools it is discovered that arterial hypertension is a polygenic disease. Another possible explanation might be the variety of the studied populations. The current results of genetic analyses of essential hypertension highlight the need for a more differentiated approach to the understanding of complex, polygenetic traits that implements gene-gene- and gene-environment interactions or differentiated functional testing of thoroughly phenotyped cohorts under standardised environmental conditions. The advancement of molecular genetics now permits the establishment of a connection between high blood pressure and certain traits. The aim of this work is to analyse how genetic testing for primary prophylaxis of arterial hypertension can be applied.**

Keywords—hypertension; gene mutation; polygenic; epigenetics; biotechnology

I. INTRODUCTION

Hypertension, or high blood pressure, is a dangerous medical condition that raises the risk of heart, brain, kidney, and other ailments dramatically. [1] In Western nations, arterial hypertension is one of the primary cardiovascular risk factors [2]. Globally, an estimated 1.28 billion persons aged 30-79 have hypertension, with the majority [two-thirds] residing in low- and middle-income nations [3]. Genetic testing for example pcr screening, dna sequencing methods, Southern blot is already an option to identify of appearance of arterial hypertension and treat this before it appears [4]. In this illness, essential hypertension is the most prevalent diagnosis, indicating that a single cause has not been found. Nonetheless, other risk factors for EH have been discovered, including age, gender, demographic, environmental, genetic, and vascular variables [5] Arterial hypertension is genetically complicated, which explains why the discovery of the underlying genes for hypertension has been less effective than for other disorders [6]. Significant advances have been made in the area as a result of genetic analysis of well-defined endocrine types of hypertension in which classification of individuals into homogenous cohorts is achievable [7]. The aim is to analyse how genetic testing for primary prophylaxis of arterial hypertension can be applied.

II. MATERIAL AND METHODS

This is a systematic review study. It represents the results of anlysis of 200 surces wich were selected according to the key words in web of science and google scholar. After filtering out remained 10 sources.

Atrial hypertension

Systemic Hypertension is elevated blood pressure in the systemic arteries, which deliver blood from the heart to the tissues of the body. Usually, the constriction of the tiny arteries causes high systemic blood pressure[2]. This increases peripheral blood flow resistance, which in turn increases the heart's workload and arterial pressure.It is measured at its peak and lowest points. Normal systolic blood pressure varies with age, but a maximum normal adult reading is around 140 mm Hg. Around 90 mm Hg is the top range for normal diastolic blood pressure. [3] Pulse Pressure refers to the difference between systolic and diastolic blood pressure. In 95% of instances, the etiology of primary systemic hypertension is unknown. Secondary hypertension is systemic hypertension that is caused by another aliment or disease. The advancement of molecular genetics has

made it possible to now demonstrate a connection between high blood pressure and certain traits. [2]

Gene mutation that lead to hypertension

The identification of susceptibility genes involved in multifactorial hypertension is based on linkage and gene association studies, which aim to establish a significant association between a specific chromosomal region or allelic variant and the disease and are based on the hypothesis of "common disease–common variant"[4]. This hypothesis begins with the premise that the major susceptibility alleles can be identified in all patients with the same condition. Families of patients underwent chain tests on the idea that patients would carry a particular allele [of a candidate gene] more frequently than healthy family members [5]. Association studies are conducted in a population to compare the incidence of a particular gene polymorphism with its incidence in a control group;[6] if a specific allele of the polymorphic locus is significantly more prevalent in diseased individuals than in unaffected individuals, then the allele is assumed to be involved in the pathogenesis of the disease. Several gene variations that confer vulnerability to hypertension were found during the "pregenomic" period, including genes. [5]encoding different components of the renin–angiotensin system, ion channels, and enzymes involved in the manufacture of aldosterone. Following items were identified: the ADD1 gene [-bringing, hydrostatic pressure change sensor, located on chromosome 4p16]; the AGT gene [for angiotensinogen, located on chromosome 1q42-q43]; the REN gene (for renin, located on chromosome 1q32]; the ACE gene [for the angiotensin converting enzyme, located on chromosome 17q23[; the AT1R gene (for the angiotensin receptor. [3]

Primary prophylaxis

It is possible to prevent hypertension by using measures that target both the general public and individuals and groups at a greater risk for developing high blood pressure [2]. Lifestyle treatments are more likely to be effective, and absolute reductions in the risk of hypertension are likely to be larger, when focused at older individuals and those with a higher risk of developing hypertension, compared to younger individuals or those with a lower risk. However, early life preventive methods have the greatest long term promise for preventing the precursors that lead to hypertension and increased blood pressure levels and for minimizing the total burden of blood pressure-related diseases on the population.[8]

Genetic approach

- Arterial hypertension is presently the leading cause of worldwide mortality and illness burden, with a substantial link between high blood pressure and cardiovascular disease. Therefore, a greater knowledge of the genetic basis of a polygenic illness, such as hypertension, might bring antihypertensive treatment one step closer to achieving the suggested therapeutic targets[7]. Individual risk and prognosis stratifications based on the identification of particular genetic variations may also benefit from the use of a genetic profile. A targeted genetic analysis for hypertensive patients would enable the identification of disease-causing genes and the precise characterization of genomic regions associated with arterial hypertension. The study of genetic variations has progressed in the direction of pharmacogenomics, the study of genomic variation that affects individual reactions to medication [5, 8]. This idea is important for clarifying antihypertensive pharmacological therapeutic activities and for comprehending why some patients react to regular therapy while others do not. [9]Different metabolic profiles and genetic variations of metabolizing enzymes, the genetic variability of sodium sensitivity, and proteins from renal tubules [responsible for the regulation of ion transport or variability of response to diuretics] appear to be associated with the genetic individual variability of antihypertensive treatment response[7]. The Genome Wide Association Study (GWAS) has been an important tool for uncovering the genetic implications of essential arterial hypertension from this perspective. A GWAS allows for the broad-scale typing of a significant number of Single Nucleotide Polymorphisms (SNP), and in the context of major consortia, several research were published that led to the discovery of more than 100 SNPs associated with high blood pressure. [4] the mutotion in the foloowinh genes wich are importat for the develompent of arterial hypertension were discovered- systolic BP ATP2B1, CYP17A1, PLEKHA7, SH2B3 (Levy et al., 2009), Diastolic BP ATP2B1, CACNB2, CSK-ULK3, SH2B3, TBX3-TBX5, ULK4 (Levy et al., 2009), Systolic or diastolic BP CYP17A1, CYP1A2, FGF5, SH2B3, MTHFR, c10orf107, PLCD3 (Newton-Cheh et al., 2009), Pulse pressure CHIC2/PDGFRA, PIK3CG, NOV, ADAMTS8 (Wain et al., 2011), Mean arterial pressure CHIC2/PDGFRA, PIK3CG, NOV, ADAMTS8 (Wain et al., 2011)

Biotechnological tools for genetic screening of arterial hypertension

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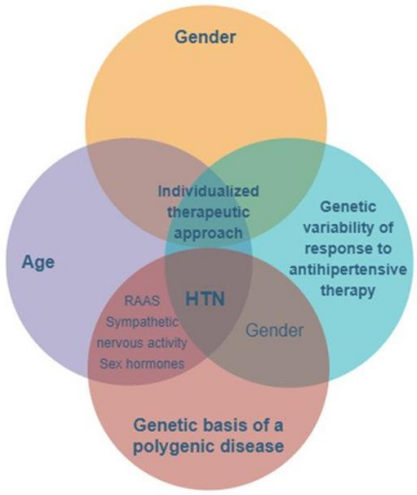

Figure 1. The main features and key factors for personalizing treatment in hypertension., https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8229802/#B6-pharmaceutics-13-00856

III. *Conclusions*

1. A complicated interaction of genetic, epigenetic, and environmental variables causes essential arterial hypertension.
2. New biotechnologies permit genetic screening of genetic variants leading to essential arterial hypertension to ensure its primary prophylaxis in relatives predisposed to this disease.
3. Application of genetic testing is important for personalized medicine and may enhance the design of randomized controlled trials.

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