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Progress of Piezo1 Ion Channels in Hypertension Research: From Molecular Mechanisms to Therapeutic Potential

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Abstract: ***Hypertension, a globally prevalent chronic disease and a common cardiovascular disorder in China, significantly increases the risk of complications such as stroke and coronary heart disease. Mechanosensitive ion channels are capable of sensing mechanical changes in the cell membrane and responding rapidly. Among them, piezo1, a novel mechanosensitive ion channel protein, is widely distributed in the cardiovascular system and participates in the pathological progression of hypertension by regulating processes such as baroreceptor reflex, vascular tone, vascular remodeling, and macrophage polarization. This article systematically reviews the mechanisms underlying the role of piezo1 in hypertension and explores its potential as a therapeutic target, aiming to provide a theoretical foundation for precision treatment of hypertension.***

Keywords: Hypertension, Mechanical sensitive ion channel, Piezo1, Traditional Chinese medicine**.**

1. Introduction

Hypertension, a globally prevalent chronic condition, ranks among China's most common cardiovascular diseases. Not only does it exhibit high prevalence rates, but it also serves as a core risk factor for severe complications such as stroke, coronary heart disease, and end-stage renal disease. Prolonged blood pressure elevation can cause irreversible damage to multiple organs including the heart, brain, and kidneys, severely compromising patients' quality of life [1]. By 2019, the number of patients with hypertension is 1.3 billion. However, there is still a significant gap in the diagnosis rate, treatment rate and control rate of patients with hypertension in the world at the present stage: only 54% of patients are diagnosed, 42% receive antihypertensive drug treatment, and less than 21% of them achieve blood pressure target [2]. The situation in China is particularly severe. The 2018 China chronic disease surveillance data shows that the number of adults with hypertension has reached 245 million, but the awareness rate (41%), treatment rate (34.9%), and control rate (11%) remain far below ideal levels [3], indicating that the prevention and control system urgently needs optimization.

In recent years, the role of mechanically sensitive ion channels in the pathogenesis of hypertension has attracted much attention. Such channels can directly sense the changes of mechanical forces on the cell membrane (such as blood flow shear force, vascular wall tension, etc.), and participate in cardiovascular stability regulation through electrical signals or chemical signal transduction [4]. This paper reviews the progress of molecular mechanisms of piezo1 channel in hypertension, and discusses its translational potential as a novel therapeutic target, so as to provide theoretical basis for precise intervention strategies [5-9].

2. Overview of Hypertension

Definition of hypertension: Clinical criteria include measuring office blood pressure on non-consecutive days without antihypertensive drugs, with systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, or having a history of hypertension or taking antihypertensive medications [10].

2.2 Pathogenesis of Hypertension

Blood pressure (Bp) refers to the lateral pressure exerted by blood on a unit area of the vascular wall during blood flow, serving as the driving force for blood circulation. The commonly referred blood pressure specifically denotes systemic arterial blood pressure [11]. Arterial vessel walls consist of multiple cellular components including endothelial cells, vascular smooth muscle cell layers (VSMCs), and extracellular matrix, which can be subdivided into three distinct layers or membranes: the intima, media, and adventitia. The innermost lining is composed of a single layer of endothelial cells, while the outer membrane is the outermost layer of the vascular wall, forming a connective tissue sheath mainly composed of collagen and elastin fibers [12]. The pathogenesis of hypertension is generally believed to have the following causes: endothelial injury: vascular endothelial cell damage can reduce the production of vasodilator substances, such as nitric oxide (NO), and increase the expression of vasoconstrictor substances, leading to vascular constriction and blood pressure elevation [13]. Matrix deposition: The accumulation of collagen and glycoproteins in blood vessel walls leads to stiffness and reduced elasticity, which is a key factor contributing to elevated blood pressure. Vascular remodeling: This refers to adaptive structural and functional changes in blood vessels, which may result in thickened vessel walls and narrowed lumens, thereby increasing vascular resistance [14].

3. Structural Features of Piezo1

Piezo1 is the first eukaryotic mechanically sensitive ion channel identified. Its three-dimensional structure is trilobite propeller, including a central ionic pore, peripheral propeller blades and a central cap [15]. Mechanical stimulation (such as shear force or membrane deformation) can induce piezo1 conformational changes, open ion channels to mediate Ca^{2+} and Na^{+} influx, and regulate cell proliferation, migration and inflammatory response [16]. Low-temperature electron microscopy studies reveal that piezo1 is a propeller-shaped homodimer composed of three subunits, measuring approximately 185 A in diameter and 140 A in height. Each subunit contains 38 transmembrane helices (TM), forming a distinctive "three-leaf" structure where each leaf consists of nine repeating transmembrane helical units (THUs), each containing four transmembrane helices. The central region contains an ionic conduction pore module comprising a C-terminal domain (CTD), anchor domain, and outer/hydrophobic (OH)/inner/hydrophilic (IH) helices. Key structural components include: Transmembrane blades (Blades): Composed of THU1-THU9 subunits in a highly curved configuration, which may sense mechanical forces by inducing membrane curvature changes. Intracellular beam structures: Approximately 90 A long, connecting distal THUs to the central pore module, serving as a "lever" in mechanical transduction. Ionic channel pore: The hydrophobic channel formed by the inner helix (IH) comprises three intracellular side portals and a narrow intramembrane cavity (with V2476 as the constriction point), which is presumed to remain closed [17]. Zhao et al. experimentally confirmed that mutations in fulcrum residues (L1342A/L1345A) significantly impair mechanical sensitivity. This model system elucidates the molecular mechanism of mechanical energy cascade transmission through the blade-lever-pore cascade, while identifying functional sites of extracellular loops during mechanical activation, providing a structural framework for mechanotransduction structure-function relationship studies.

The YANG team [18] achieved a groundbreaking resolution of the piezo1 ion channel's bistable conformations in lipid membranes through cryo-electron microscopy: the curvature state (3.46 Å) and flat state (6.81 Å), elucidating the structural basis of its mechanical sensitivity. They proposed a dual-gating mechanism: lateral bolt gates regulate intracellular opening, while transmembrane helical reorganization mediates central pore dilation. This study systematically explains how piezo1 perceives mechanical forces through membrane curvature and achieves ion channel gating at the molecular level.

4. The Role of 3piezo1 in Hypertension

4.1 Endothelial Cell Shear Stress Sensing and Vascular Tension Regulation

In physiological states, endothelial cells (EC) in blood vessels are exposed to hemodynamic forces over time. These cells sense hemodynamic stimulation through mechanosensing mechanism, and then trigger physiological regulatory responses such as vascular diameter changes [4]. Wang [18] demonstrated that activation of the endothelium mechanically sensitive ion channel piezo1 is crucial for downstream p2Y2/Gq/G11-mediated signaling following ATP release, which subsequently triggers AKT/pI3K-mediated phosphorylation and activation of nitric oxide synthase (eNOS), ultimately leading to nitric oxide (NO) production. In a mouse model with endothelial-specific piezo1 gene knockout, researchers observed significant impairment in vasodilation function due to the inability to stimulate NO synthesis through blood flow stimulation, resulting in hypertension. Their findings confirm that piezo1 serves as a key molecule maintaining nitric oxide generation, regulating vascular tone, and stabilizing blood pressure homeostasis. The team of Iring [19] further revealed that the mechanosensitive cation channel piezo1 in endothelial cells senses fluid shear stress, triggers the release of adrenomedullin, and activates its downstream Gs protein coupled receptor. Notably, the Gs/pKa-mediated signaling pathway synergizes with the AKT pathway to jointly promote phosphorylation at the 1177th serine site of eNOS. Animal experiments further revealed that endothelial cell-specific knockout models of adrenomedullin, its receptor, or Gas exhibited significantly reduced blood flow-stimulated eNOS activation and vasodilation capacity, ultimately developing into hypertension phenotypes.

The above evidence shows that piezo1, as the core molecule of shear stress sensing, forms a key mechanical conduction pathway to maintain basic endothelial NO generation, vascular tension homeostasis and blood pressure balance by regulating the adrenal medullary Gs-cAMp signaling axis.

4.2 Piezo1 and Vascular Remodeling

Hypertension is the most common cause of death worldwide. Vascular remodeling is one of the important pathological links in this process. In the process of vascular remodeling, endothelial cell dysfunction and smooth muscle cell proliferation, migration and fibrosis play an important role [20].

In pulmonary artery smooth muscle cells (pASMC), the upregulation of piezo1 in proliferative pASMC may require sufficient Ca2+ to ensure nuclear/cell division and pASMC proliferation, thereby promoting the occurrence and progression of pulmonary vascular remodeling under pH [21]. Experimental data reveal: (1) Piezo1 is specifically overexpressed in small artery smooth muscle cells, and its gene deletion completely inhibits the activity of the stretch-activated channel (SAC); (2) Although this channel does not participate in arterial myogenic tension regulation, it plays a critical role in pressure-induced structural remodeling; (3) Pathological conditions with increased Piezo1 channel opening can induce vascular trophic changes, manifested as reduced diameter and thickened walls of resistance arteries in hypertension; (4) Mechanistic studies demonstrate that Piezo1 mediates intracellular calcium ion elevation, thereby activating the activity of —transglutaminase—a key enzyme in vascular remodeling—ultimately driving arterial structural reorganization.

This study for the first time elucidated the molecular mechanism link between piezo1 mediated mechanical signal transduction process and clinical arterial remodeling in smooth muscle cells, which is related to vascular damage associated with hypertension process. Therefore, intervention of Piezo1 may become a new method for future hypertension vascular treatment.

4.3 Pressure Receptor Reflex Regulation

Arterial pressure receptors (located in carotid sinus and aortic arch) can rapidly regulate blood pressure through feedback mechanism, especially in the case of body position change, bleeding or acute stress. Blood pressure can be maintained stable within seconds to minutes by regulating cardiac output and peripheral vascular resistance [22]. ZENG [6] and colleagues elucidated the core role of piezo1/piezo2 ion channels in blood pressure regulation through analyzing mechanosensing mechanisms in baroreceptors. Using fluorescent cholera toxin retrograde labeling of mouse carotid afferent neurons, they demonstrated that baroreceptor neurons co-express both piezo1 and piezo1 transcripts. Double knockout of piezo1/piezo2 caused increased blood pressure fluctuations (greater variability), dysregulation of arterial pressure reflexes, and unstable hypertension in mice, while single-gene knockout showed no significant impact on the baroreceptor reflex. These findings suggest functional redundancy in baroreceptor mechanosensing mechanisms. This study establishes for the first time that the piezo family serves as the molecular basis for long-unknown pressure reflex mechanical sensing.

4.4 Piezo1 Participates in the Immune Inflammatory Mechanism of Hypertension by Regulating Macrophage Polarization

Macrophages, as key components of the innate immune system, have been extensively documented to correlate with hypertension pathogenesis. Evidence indicates that peripheral blood monocytes in hypertensive patients not only exhibit pronounced pro-inflammatory phenotypic characteristics but also demonstrate significantly elevated serum inflammatory cytokine levels, suggesting that immune inflammatory responses play a crucial role in hypertension development [23]. Immune-infiltrating monocytes transform into pro-inflammatory macrophages, releasing pro-inflammatory cytokines (IL-6, IL-1β, TNF-α). These cytokines induce endothelial dysfunction, thereby reducing nitric oxide (NO) secretion, weakening its inhibition of renal NKCC2 channels, and decreasing sodium excretion – ultimately leading to hypertension. Macrophages constitute a functionally heterogeneous immune cell population: M1 type (activated by lipopolysaccharides and interferons) induces inflammatory responses and tissue damage, while M2 type (activated by IL-4/IL-13) mediates anti-inflammatory and tissue repair functions. In Dahl salt-sensitive rat models, high-salt intake elevates renal perfusion pressure, triggering macrophage infiltration. Activated macrophages release pro-inflammatory factors that cause renal injury, subsequently inducing water and sodium retention and blood pressure elevation. This process creates a vicious cycle of “macrophage infiltration, inflammatory damage, and elevated blood pressure,” continuously exacerbating hypertension progression [24]. The absence of piezo1 inhibits M1 polarization (iNOS↓), reduces TNF-α/IL-6 secretion (NF-κB activity↓), and weakens the inflammatory response to IFN-γ/LpS. Promote IL-4/IL-13 induced M2 polarization (Arg1↑), promote phenotype repair by enhancing STAT6 activation [25]. The high expression of piezo1 is directly associated with M2-like phenotypes. In traditionally IL-4-induced M2-type BMDMs, piezo1 expression showed no significant upregulation. However, M2-like polarization induced by apoptosis cell phagocytosis was accompanied by elevated piezo1 expression, revealing that piezo1 regulates M2 polarization through phagocytosis-dependent non-classical pathways. Piezo1 not only serves as a result of phagocytosis-induced M2 polarization but may also reinforce this phenotype via calcium signal feedback [26].

5. Current Drugs Targeting Piezo1 Channel Protein

5.1 Chemical Drug Regulation Targeting the Protein Piezo1

In addition to physical mechanical forces, piezo1 can also be gated chemically. SYEDA [27] et al. screened more than 3 million low molecular weight compounds and identified the first chemical activator of the piezo1 channel, which was named Yoda1. It activates the piezo1 channel protein c intracellular region without mechanical stimulation. and has a faster explosion and shorter decay than Yodal-mediated current, where Jedi1 and Yoda1 synergistically activate different piezo1 sites [8].

At present, the known non-specific inhibitors of piezo1 mainly include gadolinium (Gd3+) in the lanthanide family, ruthenium red (RR), and spider venom peptide GsMTx-4 isolated from tarantula toxin [28]. Gusmtx-4, a tarantula venom extracted from the venom of the spider Grammostola spatate. GsMTx4 is an amphipathic peptide. Current GsMTx4 inhibition models suggest that it works by altering local membrane tension rather than directly binding to the gating element of the piezo1 channel [29].

5.2 Potential Targets of Traditional Chinese Medicine for Protein Piezo1

Traditional Chinese medicine (TCM) has a long history of treating hypertension and demonstrates unique advantages in enhancing clinical efficacy and improving patients ‘quality of life. Li Jing’s [30] research team screened 92 TCM molecules that inhibit Yoda1-induced abnormal activation of the piezo1 channel, with results showing multiple herbs demonstrated inhibitory effects. Notably, Tumuboguosidin-1 achieved a 90% inhibition rate and showed dual inhibitory effects on both endothelial cells and macrophages’ piezo1 channels. Liu Lu [31] summarized the Chinese medicines that could inhibit the abnormally activated piezo1 channel, and found that a variety of Chinese medicines could inhibit the Ca2+ influx caused by the abnormally activated piezo1 channel, such as dicoumarol B, saponins, menthol, etc., with an inhibition rate of more than 90%.Zhang Meng [32] et al. experimentally showed that the serum of astragalus and Danshen had a protective effect on the inflammation and dysfunction of HUVEC induced by low FSS, and the mechanism was related to the regulation of mechanically sensitive cation channel piezo1.Molecular docking results suggest that piezo1 may be a potential pharmacological target of LA. Further experimental data indicate that LA inhibits the activation and expression of piezo1 in macrophages. Berberine (Jat), an isoquinoline alkaloid found in traditional Chinese medicines like Coptis chinensis and Phellodendron amurense, has been shown to inhibit LA-mediated effects. In the study of HOUNG et al.

[34], it was revealed that the component Jat of TCM inhibited vascular inflammation by targeting and regulating piezo1 channel. This study confirmed that Jat, as a piazo1 channel inhibitor, has potential application value in the treatment of vascular inflammatory diseases [33].

These results highlight the potential of highly specific components such as Tumuboside 1, Danshen and Jat to intervene in vascular diseases, and provide a theoretical basis and candidate molecules for the development of new antihypertensive and anti-vasculitis Chinese medicine based on piezo1 target. This discovery provides a molecular probe and research paradigm for the analysis of potential piezo1 regulating components in existing multi-target antihypertensive compounds (such as gastrodia and hook vine drink, liver calming and wind quenching soup) [35-36].

6. Summary

As the core molecule of mechanosensitive ion channels, piezo1 mediates a mechanochronic signaling network that demonstrates multidimensional regulatory characteristics in hypertension pathogenesis. In vascular homeostasis imbalance mechanisms, piezo1 dynamically modulates calcium signaling pathways by sensing shear stress and vascular wall stretch stimuli from endothelial cells, precisely regulating nitric oxide production, vascular smooth muscle tone, and immune-inflammatory mechanisms. Although small molecule modulators (such as Yoda1 agonists) have shown potential for targeted therapy in basic research, their off-target effects and pharmacokinetic limitations hinder clinical translation [37]. While some traditional Chinese medicines have been found to act on this channel, the specific coupling mechanism between their multi-component synergistic effects and the piezo1 signaling network remains an enigmatic black box system.

Future research breakthroughs should focus on: 1) Mapping spatiotemporal interactions between piezo1 and vascular endothelial dysfunction, angiogenic pathways, and immune cell infiltration-related chemokine networks to elucidate the interface regulation of mechanosignal-biochemical signal transduction; 2) Designing endothelium-selective allosteric modulators based on cryo-electron microscopy (cryo-EM) analysis of piezo1's trilevel helical domain conformational changes, and developing biomimetic nanocarrier systems for targeted lesion delivery; 3) Systematically deconstructing dynamic regulatory patterns of piezo1 heterodimers in vascular endothelial/smooth muscle cells using spatial transcriptomics and organoid microarray technologies, establishing a multi-scale interaction model from "component clusters-target modules-pathological phenotypes". These explorations will not only deepen mechanobiology's theoretical framework in cardiovascular diseases but also provide integrated Chinese-Western intervention strategies combining biological precision and systemic regulation to overcome current therapeutic bottlenecks in antihypertensive drugs. 4) Several critical scientific questions remain unresolved in the piezo1 molecular domain: Is the flattened state fully open? This requires validation through functional experiments (e.g., electrophysiology). The dynamic behavior of pore lipids and their specific gating mechanisms need investigation. The potential role of N-terminal unlocalized regions (THU1-THU3) in mechanical transduction requires further exploration. These findings aim to provide therapeutic insights for hypertension management.

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