REVIEW

Piezo1: A Novel Factor in Regulating the Growth of Skeletal Muscle

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Summary

Piezo type mechanosensitive ion channel component 1 (Piezo1) plays a crucial role in skeletal muscle development, regeneration, and myovascular physiological regulation. Piezo1 regulates the assembly of myosin, providing a foundation for skeletal muscle injury repair. Activation of Piezo1 induces proliferation, differentiation, and myotube formation in skeletal muscle satellite cells (MuSCs), while its knockout leads to reduced MuSC numbers, disrupted cell cycle, and abnormal myotube assembly. Additionally, Piezo1 participates in skeletal muscle vascular endothelial cell signaling, inducing nitric oxide (NO) release, regulating vascular smooth muscle tone, increasing skeletal muscle blood flow perfusion, and promoting skeletal muscle injury repair. This paper systematically reviews the structural characteristics, biological mechanisms, and mechanisms of action of Piezo1 in skeletal muscle, providing theoretical basis for the therapeutic application of Piezo1 in skeletal muscle-related diseases.

Key words

Piezo1 • Skeletal muscle • Muscle satellite cells

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Introduction

The Piezo1 ion channel, discovered in 2010, senses mechanical stimuli in various cell types and converts these stimuli into electrochemical signals, thereby participating in the regulation of multiple physiological processes [1]. Piezo1 is widely expressed in skeletal muscle tissue and is involved in muscle cell growth and development [2], regulating muscle cell proliferation and apoptosis, inducing myotube formation through Ca^{2+} influx [3,4], and participating in MuSC differentiation [5]. During myocyte differentiation, Piezo1 promotes increased mRNA and protein levels, and its expression in MuSCs induces myotube formation [3]. Piezo1 inhibits MuSC senescence to maintain the MuSC pool. Various mechanical forces, such as shear force, tensile force, and cell indentation, activate the Piezo1 channel [6], leading to Ca^{2+} influx across the cell membrane and regulating actin assembly [7]. Piezo1 promotes the activation of mitogen-activated protein kinase (MAPK)/extracellular signal-regulated kinase 1/2 (ERK1/2) [8]. Activation of ERK1/2 leads to increased expression of cell cycle-related proteins, accelerating the cell proliferation cycle; ERK1/2 activation increases actin assembly and reduces disassembly. In skeletal muscle vascular endothelial cells, Piezo1 channels are activated by shear stress from blood flow and membrane stretching force from elevated blood pressure, leading to vascular remodeling and blood pressure regulation [5].

This article will review the mechanisms by which Piezo1 regulates skeletal muscle growth, aiming to elucidate its role in skeletal muscle mechanotransduction and provide important theoretical basis for exploring therapeutic strategies to maintain skeletal muscle function under disease conditions.

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Introduction to the Piezo1 channel

Structure of Piezo1

The human Piezo1 gene is located on chromosome 16, contains 51 exons, and encodes 2520 amino acid residues [5]. The mouse Piezo1 gene is located on chromosome 8, consists of 53 exons, and encodes 2,547 amino acid residues [9]. The Piezo1 channel is a large trimeric transmembrane protein with a three-leaflet, trimeric propeller-like structure, featuring an extracellular cap domain at the top of the central ion conduction pore module. Each subunit contains one central ion conduction pore and two peripheral modules. The central ion conduction pore consists of an outer helix, an inner helix, an intracellular C-terminal domain, and an extracellular C-terminal domain. The peripheral mechanical transduction module includes a long beam-like structure and an anchoring domain; the peripheral propeller blades contain 38 transmembrane helices, functioning as a mechanical sensing module [3]. The peripheral propeller blades form the mechanical sensing module, while the mechanical transduction module consists of an intracellular bundle, an anchor, and a C-terminal domain. The two transmembrane helices, the extracellular C-terminal domain, and the intracellular C-terminal domain of Piezo1 form a pore module through trimerization. The C-terminal domains form the cap of the ion conduction pathway, located between the outer and inner helices. Each propeller blade has a 36-transmembrane helix topology, divided into nine repeating transmembrane helix units. The C-terminal transmembrane helix units are connected to the outer helix *via* triangular α-helix arrangements, forming the anchor domain [2]. Each subunit of the Piezo1 protein has 38 transmembrane helices, with a total of 114 transmembrane helices in the homotrimer. Transmembrane helices 37 and 38 are close to the central domain [10]. The central pore serves as a cation channel, composed of the extracellular vestibule, intracellular vestibule, and membrane vestibule within the central cap [5]. The central ion conduction pore module is responsible for ion selectivity and passage. The inner and outer transmembrane helices of Piezo1 are close to the central axis, and the surrounding 12 transmembrane helices are divided into three bundles of four transmembrane helices each, with identical topological structures. The piezoelectric protein of Piezo1 serves as the non-conductive subunit of the ion channel, enabling proper expression or regulation of channel properties [11] (Fig. 1).

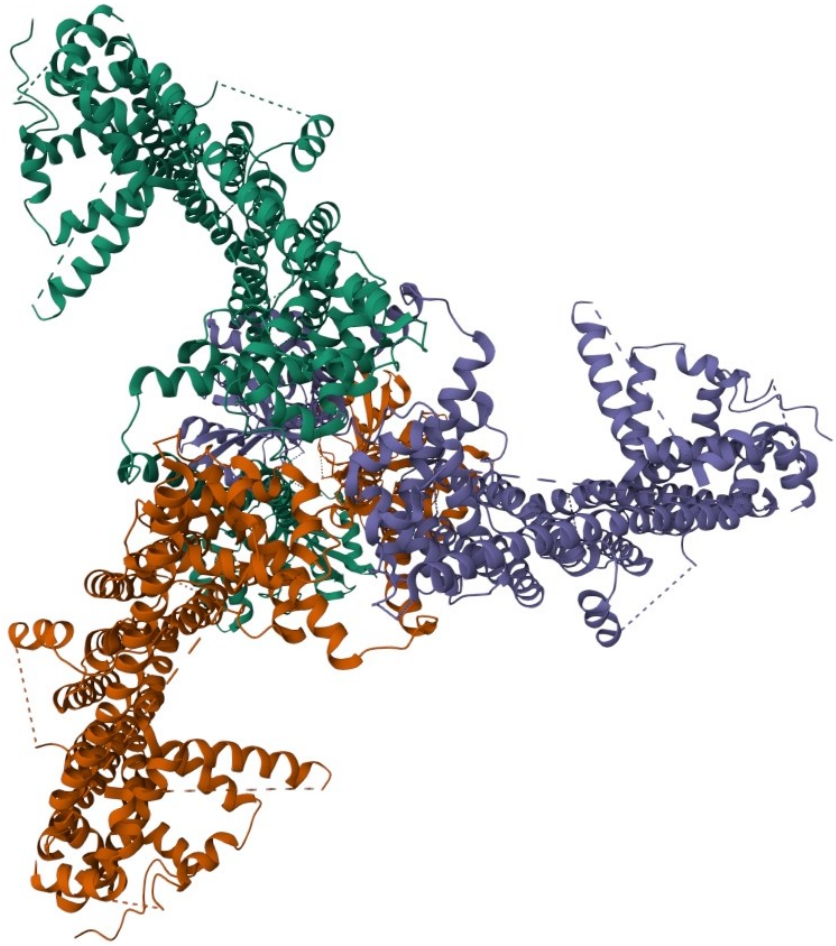


Fig. 1. Structure of the mechanically activated ion channel Piezo1 (Electron microscopy 3.8 Å).

Piezo1 channel kinetic properties

The Piezo1 channel exhibits rapid activation and voltage-dependent inactivation properties [5]. The Piezo1 channel can be activated by stimuli such as stretching, compression, shear force, membrane torsion force, bilayer indentation, and osmotic force [12]. A long lever-shaped beam transmits mechanical forces from the leaflet to the ion conduction module. The leaflet, composed of highly curved and non-planar transmembrane helical units, serves as a mechanical receptor, sensing changes in membrane curvature and tension. The lever pivot is located near the central pore. This conduction mechanism enables the Piezo1 channel to convert the large conformational changes in the distal leaflet into relatively minor openings in the central pore, facilitating cation-selective transmembrane transport [13]. Membrane stretching induced by tension, shear force, cell compression, changes in matrix stiffness, and voltage changes causes deformation in the leaflet region. The beam region acts as a lever arm, converting deformation into minute movements within the pore, activating the Piezo1 channel and causing cation influx into the cell. Piezo1 activity can be regulated by interactions with the ECM or cytoskeletal structures. In the absence of the ECM, Piezo1 becomes less sensitive to mechanical stimuli. Cell relaxin D inhibits actin polymerization, resulting in reduced ion flow through the Piezo1 channel in response to mechanical stimuli [14]. Piezo1 is expressed in vascular endothelial cells. Piezo1-dependent Ca^{2+} influx activates downstream signaling pathways that influence vascular remodelling. Piezo1 deficiency leads to impaired vascular development in mouse embryos and impaired endothelial cell response to shear stress [15].

Effects of Piezo1 on the skeletal muscle cell cytoskeleton

In 2018, Tsuchiya [14] first identified Piezo1 channels in skeletal muscle cells. Activation of Piezo1 promotes myoblast proliferation and facilitates the fusion of myoblasts to form multinucleated myotubes [16]. After 72 h of myoblast differentiation, the mRNA expression level of Piezo1 significantly increased. Knocking out Piezo1 reduced myoblast fusion and myotube maturation, decreased Piezo1 mRNA expression levels, and caused a reduction in myosin synthesis [14]. Inactivation of Piezo1 in MuSCs leads to a significant decrease in the number of paired box 7 (PAX7) cells in skeletal muscle [17]. Compensatory increases in calcium influx through T-type calcium channels, activation of protein kinase C, upregulation of nicotinamide adenine dinucleotide phosphate oxidase 4 (NADPH oxidase 4, NOX4), increased reactive oxygen species (ROS) production, and elevated expression of the tumor protein p53 (P53) [14]. ROS causes DNA breaks, activates DNA damage-related pathways, and induces the upregulation of the p53-mediated apoptosis pathway, leading to MuSC cell apoptosis. In C2C12 cells, Piezo1 regulates the assembly of myosin required for myotube formation, and Piezo1 deficiency results in reduced myosin synthesis [18]. Stress-activated Piezo1 channels promote Ca^{2+} influx, leading to the activation of Yes-associated protein (YAP), and increased YAP expression promotes increased skeletal muscle protein synthesis. Piezo1 increases the promoter activity of the Myc gene (cellular Myc, c-Myc) and myogenic differentiation factor (MyoD) in myocytes, while inhibiting the promoter activity of muscle-specific RING Finger 1 (MuRF-1), thereby promoting skeletal muscle growth [14]. Piezo1-mediated Ca^{2+} influx promotes RhoA/Rock-mediated myotube actin assembly, facilitating myotube formation [19].

Piezo1 senses changes in cell membrane tension [20]. Changes in membrane tension are caused by the adhesion of myofibrillar components to the lipid bilayer [21]. The actin cytoskeleton acts as a key mediator in generating intracellular mechanical forces. Activation of Piezo1 triggers the sliding of actin complexes, leading to an increase in cell membrane tension [22]. Mechanical stimulation activates Piezo1, and Ca^{2+} transmits external stimuli to the cytoskeleton, triggering actin contractility, which, through actin complex collaboration, initiates cytoskeletal tension. Actin activation promotes the mammalian target of rapamycin complex 1 (mTORC1)/p70 ribosomal protein S6 kinase (p70S6K) signaling pathway, which facilitates skeletal muscle protein synthesis [14]. Piezo1 activation induces Ca^{2+} influx, activating the Rho/RocK/actin pathway to control actin assembly. Piezo1 silencing leads to reduced actin synthesis and defective cell fusion [16]. When the cytoskeleton is disrupted, even minimal mechanical force can induce a significant increase in Piezo1 activity [23]. Piezo1 channels mediate adhesion between cell-cell and cell-ECM complexes [24]. Piezo1 regulates F-actin filament-mediated cell migration in human embryonic kidney cells [25]. Transfection with siRNA to inhibit Piezo1 causes a decrease in Piezo1 expression levels by over 60 % and significantly inhibits cell migration speed. When Piezo1 channels are inactivated, normal directed cell migration is significantly inhibited [23] (Fig. 2).

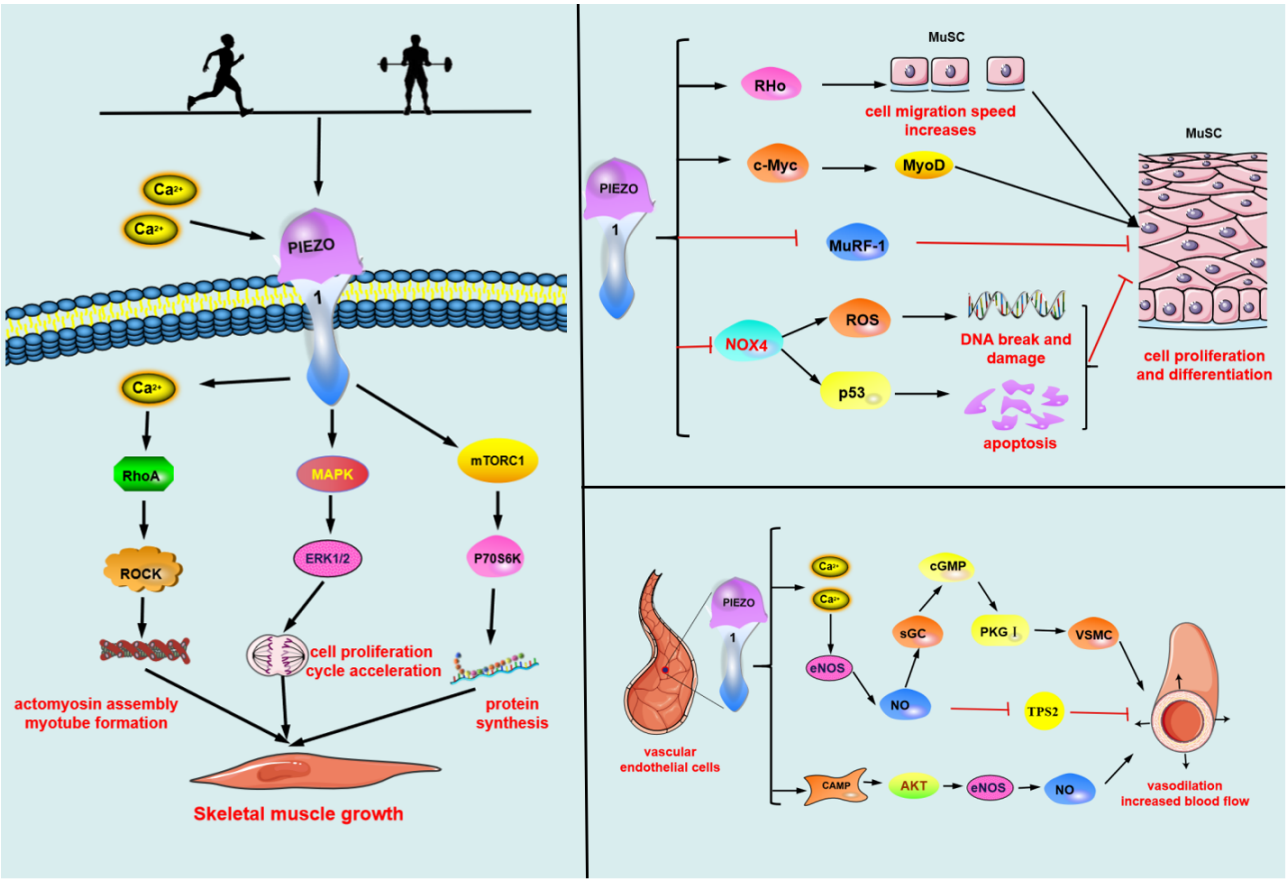


Fig 2. Mechanical gravitational stimulation activates the Piezo1 pathway to promote skeletal muscle growth mechanism.

The effect of Piezo1 on MuSC

Piezo1 plays an important role in regulating MuSC activation, proliferation, and preventing skeletal muscle atrophy. After skeletal muscle injury, quiescent MuSC are activated to enter the cell cycle and proliferate. Activated MuSC proliferate and differentiate into myoblasts that fuse with existing muscle fibers or generate new muscle fibers [19]. During exercise, activation of Piezo1 channels in muscle cells leads to extensive proliferation of MuSCs, resulting in increased expression of Pax7+/Myf5+/MyoD+ [16]. Activation of Rho by Piezo1 promotes the transition of MuSCs from a quiescent state to proliferation and migration, enabling them to rapidly aggregate at the injury site and facilitate the formation and maturation of new skeletal muscle fibers. A decrease in Piezo1/Rho activity disrupts cytoplasmic division in MuSCs [19].

Piezo1 knockout reduces MuSC numbers by 80 % [12], and Piezo1 deficiency in MuSCs causes cell cycle arrest, reduced proliferative capacity, delayed mitosis, and abnormal skeletal muscle regeneration.Piezo1 deficiency induces MuSC premature maturation, weakened proliferation, and impaired differentiation. Dienes *et al.* [9] found that treating freshly isolated muscle satellite cells derived from flexor digitorum brevis fibers with Yoda1 increased myotube formation. Piezo1 regulates MuSC cell morphology, and using the Piezo1 activator Yoda1 can restore abnormal MuSC state distribution, alter stem cell shortened protrusion length, and simultaneously promote skeletal muscle regeneration [26]. Inactivation of the Piezo1 channel upregulates Ca^{2+} influx through the T-type Ca^{2+} channel genes Cacna1g and Cacna1h, inducing NOX4 expression *via* protein kinase C (PKC). Activation of NOX4 leads to increased ROS levels, causing DNA damage and inducing muscle cell senescence and death [27] (Fig. 2).

Effects of Piezo1 on skeletal muscle vasculature

Piezo1 is involved in skeletal muscle vasculature development [28], regulates vascular tone [29], and controls blood pressure [30]. Stress-activated Piezo1 increases Ca^{2+} permeability in vascular endothelial cells, leading to increased Ca^{2+} influx, which promotes the activation of endothelial nitric oxide synthase (eNOS)

and results in Ca^{2+}-dependent NO release [17], which, through the soluble guanylate cyclase (SGC)/cyclic guanosine monophosphate (cGMP)/cGMP-dependent protein kinase 1 (PKGI) pathway, causes vascular smooth muscle relaxation and increased skeletal muscle blood flow [31,32]. During exercise, shear stress generated by blood flow activates Piezo1 in endothelial cells, promoting an increase in eNOS within endothelial cells. The resulting rise in local NO concentration diffuses to adjacent pericytes, inhibiting the expression of thrombospondin-2 (TSP2) gene, thereby weakening TSP2 and its pro-apoptotic effects, leading to vasodilation of skeletal muscle microvessels and increased perfusion [33]. Inactivation of Piezo1 in endothelial cells reduces capillary density by approximately 20 % [34].

Shear force-induced activation of Piezo1 stimulates renin, which further activates calcitonin receptor-like receptors and receptor-active modulator 2 receptors, activating G proteins to increase cyclic adenosine monophosphate (cAMP), phosphorylate AKT and eNOS, and ultimately produce NO [35], leading to microvascular dilation and improved skeletal muscle perfusion [1]. In vascular smooth muscle cells, activation of Piezo1 channels promotes Ca^{2+} influx, leading to the expression of PKC and aquaporin-1 on the plasma membrane [36]. Activated Piezo1 redirects blood flow from the intestines back to skeletal muscle [37] (Fig. 2).

Discussion and possible direction of future studies

Piezo1 plays a multidimensional regulatory role in skeletal muscle development, regeneration, and myovascular regulation. In skeletal muscle cells, Piezo1 promotes mTORC1/p70S6K activation to participate in protein synthesis, upregulates c-Myc and MyoD promoter activity, inhibits MuRF-1 expression, and forms a positive regulatory network for skeletal muscle growth.Piezo1 activation promotes the transition of myogenic stem cells (MuSCs) from a quiescent to a proliferative state, increases the proportion of Pax7+/Myf5+/MyoD+ cells, and accelerates myotube formation. Its absence leads to reduced MuSC numbers, disrupted cell cycle, elevated reactive oxygen species (ROS) levels, and p53-induced senescence, significantly impairing skeletal muscle regenerative capacity. Piezo1 senses blood flow shear stress to regulate endothelial cell function, modulating vascular smooth muscle tone *via* the eNOS/NO pathway, thereby influencing skeletal muscle blood flow perfusion. Endothelial cell Piezo1 expands microvessels through the adrenomedullin signaling axis, redirecting blood flow toward skeletal muscle to provide adequate nutrients to muscle cells.

Current research lays the theoretical foundation for Piezo1-targeted therapy for skeletal muscle diseases, but several issues remain to be addressed. The mechanisms underlying the interactions between Piezo1 and the cytoskeleton and extracellular matrix remain incompletely understood. For instance, the molecular mechanisms by which actin dynamics reversely regulate Piezo1 activity are unclear, and the differences in channel conformation changes induced by various mechanical stimuli, as well as their selective activation effects on downstream signaling pathways, require further elucidation. For diseases such as muscle atrophy and muscular dystrophy, the therapeutic molecular mechanisms of Piezo1 activators like Yoda1 could be explored.

In summary, as a key node in mechanical signal transduction, the central role of Piezo1 in skeletal muscle physiological and pathological processes is gradually becoming clear. Through interdisciplinary research, it is hoped that Piezo1 will transition from basic mechanism research to clinical application, providing new targets and strategies for the treatment of skeletal muscle-related diseases.

Conflict of Interest

There is no conflict of interest.

References

1. Zhu H, He W, Ye P, Chen JT, Wu XQ, Mu XR, et al. Piezo1 in skin wound healing and related diseases: Mechanotransduction and therapeutic implications. Int Immunopharmacol 2023;123:110779. https://doi.org/10.1016/j.intimp.2023.110779
2. Thien ND, Hai-Nam N, Anh DT, Baecker D. Piezo1 and its inhibitors: Overview and perspectives. Eur J Med Chem 2024;273:116502. https://doi.org/10.1016/j.ejmech.2024.116502
3. Lei L, Wen Z, Cao M, Zhang HZ, Ling SKK, Fu BSC, et al. The emerging role of Piezo1 in the Musculoskeletal system and disease. Theranostics 2024;14:3963-3983. https://doi.org/10.7150/thno.96959

4. Qian W, Hadi T, Silvestro M, Ma X, Rivera CF, Bajpai A, et al. Microskeletal stiffness promotes aortic aneurysm by sustaining pathological vascular smooth Muscle cell mechanosensation via Piezo1. Nat Commun 2022;13:512. https://doi.org/10.1038/s41467-021-27874-5
5. Tang H, Zeng R, He E, Zhang I, Ding CY, Zhang A. Piezo-Type mechanosensitive ion channel component 1 (Piezo1): a promising therapeutic target and its modulators. J Med Chem 2022;65:6441-6453. https://doi.org/10.1021/acs.jmedchem.2c00085
6. Wijerathne TD, Ozkan AD, Lacroix JJ. Microscopic mechanism of Piezo1 activation by pressure-induced membrane stretch. J Gen Physiol 2023;155:e202213260. https://doi.org/10.1085/jgp.202213260
7. Syeda R, Xu J, Dubin AE, Coste B, Mathur J, Huynh T, et al. Chemical activation of the mechanotransduction channel Piezo1. Elife 2015;4:e07369. https://doi.org/10.7554/eLife.07369
8. Pardo-Pastor C, Rosenblatt J. Piezo1 activates noncanonical EGFR endocytosis and signaling. Sci Adv 2023;9:eadi1328. https://doi.org/10.1126/sciadv.adi1328
9. Dienes B, Bazsó T, Szabó L, László C. The role of the Piezo1 mechanosensitive channel in the Musculoskeletal system. Int J Mol Sci 2023;24:6513. https://doi.org/10.3390/ijms24076513
10. Saotome K, Murthy SE, Kefauver JM, Whitwam T, Patapoutian A, Ward AB. Structure of the mechanically activated ion channel Piezo1. Nature 2017;554:481-486. https://doi.org/10.1038/nature25453
11. Coste B, Mathur J, Schmidt M, Earley TJ, Ranade S, Petrus MJ, et al. Piezo1 and Piezo2 are essential components of distinct mechanically activated cation channels. Science 2010;330:55-60. https://doi.org/10.1126/science.1193270
12. Savadipour A, Palmer D, Ely EV, Collins KH, Garcia-Castorena JM, Harissa Z, et al. The role of PIEZO ion channels in the musculoskeletal system. Am J Physiol Cell Physiol 2023;324:C728-C740. https://doi.org/10.1152/ajpcell.00544.2022
13. Gnanasambandam R, Bae C, Gottlieb PA, Sachs F. Ionic selectivity and permeation properties of human Piezo1 channels. PLoS One 2015;10:e0125503. https://doi.org/10.1371/journal.pone.0125503
14. Mirzoev TM. The emerging role of Piezo1 channels in skeletal Muscle physiology. Biophys Rev 2023;15:1171-1184. https://doi.org/10.1007/s12551-023-01154-6
15. Ranade SS, Qiu Z, Woo SH, Hur SS, Murthy SE, Cahalan SM, et al. Piezo1, a mechanically activated ion channel, is required for vascular development in mice. Proc Natl Acad Sci U S A 2014;111:10347-10352. https://doi.org/10.1073/pnas.1409233111
16. Bernareggi A, Bosutti A, Massaria G, Giniatullin R, Malm T, Sciancalepore M. The state of the art of Piezo1 channels in skeletal muscle regeneration. Int J Mol Sci 2022;23:6616. https://doi.org/10.3390/ijms23126616
17. Bosutti A, Giniatullin A, Odnoshivkina Y, Giudice L, Malm T, Sciancalepore M, et al. "Time window" effect of Yoda1-evoked Piezo1 channel activity during mouse skeletal muscle differentiation. Acta Physiol (Oxf) 2021;233:e13702. https://doi.org/10.1111/apha.13702
18. Tsuchiya M, Hara Y, Okuda M, Itoh K, Nishioka R, Shiomi A, et al. Cell surface flip-flop of phosphatidylserine is critical for Piezo1-mediated myotube formation. Nat Commun 2018;9:2049. https://doi.org/10.1038/s41467-018-04436-w
19. Hirano K, Tsuchiya M, Shiomi A, Takabayashi S, Suzuki M, Ishikawa Y, et al. The mechanosensitive ion channel Piezo1 promotes satellite cell function in muscle regeneration. Life Sci Alliance 2022;6:e202201783. https://doi.org/10.26508/lsa.202201783
20. Cox CD, Bae C, Ziegler L, Hartley S, Nikolova-Krstevski V, Rohde PR, et al. Removal of the mechanoprotective influence of the cytoskeleton reveals Piezo1 is gated by bilayer tension. Nat Commun 2016;7:10366. https://doi.org/10.1038/ncomms10366
21. Wang HJ, Wang Y, Mirjavadi SS, Andersen T, Moldovan L, Vatankhah P, et al. Microscale geometrical modulation of Piezo1 mediated mechanosensing through cytoskeletal redistribution. Nat Commun 2024;15:5521. https://doi.org/10.1038/s41467-024-49833-6
22. Luu N, Bajpai A, Li R, Park S, Noor M, Ma X. Aging-associated decline in vascular smooth muscle cell mechanosensation is mediated by Piezo1 channel. Aging Cell 2023;23:e14036. https://doi.org/10.1111/acel.14036
23. Li Y, Li L, Li B, Liao WX, Liu TT, Shen FJ. Mechanical stretching induces fibroblasts apoptosis through activating Piezo1 and then destroying actin cytoskeleton. Int J Med Sci 2023;20:771-780. https://doi.org/10.7150/ijms.81666

24. Ouyang M, Zhang Q, Zhu Y, Luo MZ, Bu B, Deng LH. α-catenin and Piezo1 mediate cell mechanical communication via cell adhesions. Biology(Basel) 2024;13:357. https://doi.org/10.3390/biology13050357
25. Morena F, Argentati C, Caponi S, Lüchtefeld I, Emiliani C, Vassalli M. Piezo1-Serine/threonine-protein phosphatase 2A - Cofilin1 biochemical mechanotransduction axis controls F-actin dynamics and cell migration. Heliyon 2024;10:e32458. https://doi.org/10.1016/j.heliyon.2024.e32458
26. Ma N, Chen D, Lee JH, Kuri P, Hernandez EB, Kocan J, et al. Piezo1 regulates the regenerative capacity of skeletal Muscles via orchestration of stem cell morphological states. Sci Adv 2022;8:eabn0485. https://doi.org/10.1126/sciadv.abn0485
27. Peng Y, Du J, Günther S, Guo XY, Wang SP, Schneider A, et al. Mechano-signaling via Piezo1 prevents activation and p53-mediated senescence of muscle stem cells. Redox Biol 2022;52:102309. https://doi.org/10.1016/j.redox.2022.102309
28. Jiang F, Yin K, Wu K, Zhang MM, Wang SQ, Cheng HP, et al. The mechanosensitive Piezo1 channel mediates heart mechano-chemo transduction. Nat Commun 2021;12:869. https://doi.org/10.1038/s41467-021-21178-4
29. Lacroix JJ, Botello-Smith WM, Luo Y. Probing the gating mechanism of the mechanosensitive channel Piezo1 with the small molecule Yoda1. Nat Commun 2018;9:2029. https://doi.org/10.1038/s41467-018-04405-3
30. Lewis AH, Grandl J. Inactivation kinetics and mechanical gating of Piezo1 ion channels depend on subdomains within the cap. Cell Rep 2020;30:870-880.e2. https://doi.org/10.1016/j.celrep.2019.12.040
31. Catacuzzeno L, Michelucci A. Emerging connections between Piezo1 and BK channels in vascular smooth Muscle cells. Pflugers Arch 2024;476:1475-1477. https://doi.org/10.1007/s00424-024-03001-3
32. Lai A, Zhou Y, Thurgood P, Chheang C, Sekar NC, Nguyen N, et al. Endothelial response to the combined biomechanics of vessel stiffness and shear stress is regulated via Piezo1. ACS Appl Mater Interfaces 2023;15:59103-59116. https://doi.org/10.1021/acsami.3c07756
33. Li J, Hou B, Beech DJ. Endothelial Piezo1: life depends on it. Channels (Austin) 2015;9:1-2. https://doi.org/10.4161/19336950.2014.986623
34. Bartoli F, Debant M, Chuntharpursat-Bon E, Evans EL, Musialowski KE, Parsonage G, et al. Endothelial Piezo1 sustains muscle capillary density and contributes to physical activity. J Clin Invest 2022;132:e141775. https://doi.org/10.1172/JCI141775
35. Lai A, Cox CD, Chandra Sekar N, Thurgood P, Jaworowski A, Peter K, et al. Mechanosensing by Piezo1 and its implications for physiology and various pathologies. Biol Rev Camb Philos Soc 2021;97:604-614. https://doi.org/10.1111/brv.12814
36. Michelucci A, Catacuzzeno L. Piezo1, the new actor in cell volume regulation. Pflugers Arch 2024;476:1023-1039. https://doi.org/10.1007/s00424-024-02951-y
37. Beech DJ. Endothelial Piezo1 channels as sensors of exercise. J Physiol 2018;596:979-984. https://doi.org/10.1113/JP274396