

Jiafu Wang

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

39
papers

1,168
citations

16
h-index

34
g-index

41
ext. papers

1,402
ext. citations

5.1
avg, IF

4.24
L-index

#	Paper	IF	Citations
39	IL-20 promotes cutaneous inflammation and peripheral itch sensation in atopic dermatitis.. <i>FASEB Journal</i> , 2022 , 36, e22334	0.9	0
38	Th2 Modulation of Transient Receptor Potential Channels: An Unmet Therapeutic Intervention for Atopic Dermatitis. <i>Frontiers in Immunology</i> , 2021 , 12, 696784	8.4	11
37	Novel insights into the TRPV3-mediated itch in atopic dermatitis. <i>Journal of Allergy and Clinical Immunology</i> , 2021 , 147, 1110-1114.e5	11.5	9
36	Selective Expression of a SNARE-Cleaving Protease in Peripheral Sensory Neurons Attenuates Pain-Related Gene Transcription and Neuropeptide Release. <i>International Journal of Molecular Sciences</i> , 2021 , 22,	6.3	6
35	Innate immune regulates cutaneous sensory IL-13 receptor alpha 2 to promote atopic dermatitis. <i>Brain, Behavior, and Immunity</i> , 2021 , 98, 28-39	16.6	4
34	Interventions in the B-type natriuretic peptide signalling pathway as a means of controlling chronic itch. <i>British Journal of Pharmacology</i> , 2020 , 177, 1025-1040	8.6	9
33	Role of SNAREs in Atopic Dermatitis-Related Cytokine Secretion and Skin-Nerve Communication. <i>Journal of Investigative Dermatology</i> , 2019 , 139, 2324-2333	4.3	8
32	New Engineered-Botulinum Toxins Inhibit the Release of Pain-Related Mediators. <i>International Journal of Molecular Sciences</i> , 2019 , 21,	6.3	8
31	New mechanism underlying IL-31-induced atopic dermatitis. <i>Journal of Allergy and Clinical Immunology</i> , 2018 , 141, 1677-1689.e8	11.5	73
30	A SNAP-25 cleaving chimera of botulinum neurotoxin /A and /E prevents TNF-induced elevation of the activities of native TRP channels on early postnatal rat dorsal root ganglion neurons. <i>Neuropharmacology</i> , 2018 , 138, 257-266	5.5	12
29	Conjugate of an IgG Binding Domain with Botulinum Neurotoxin A Lacking the Acceptor Moiety Targets Its SNARE Protease into TrkA-Expressing Cells When Coupled to Anti-TrkA IgG or Fc-NGF. <i>Bioconjugate Chemistry</i> , 2017 , 28, 1684-1692	6.3	5
28	A novel therapeutic with two SNAP-25 inactivating proteases shows long-lasting anti-hyperalgesic activity in a rat model of neuropathic pain. <i>Neuropharmacology</i> , 2017 , 118, 223-232	5.5	15
27	Neuronal entry and high neurotoxicity of botulinum neurotoxin A require its N-terminal binding sub-domain. <i>Scientific Reports</i> , 2017 , 7, 44474	4.9	5
26	TNF-induced co-trafficking of TRPV1/TRPA1 in VAMP1-containing vesicles to the plasmalemma via Munc18-1/syntaxin1/SNAP-25 mediated fusion. <i>Scientific Reports</i> , 2016 , 6, 21226	4.9	59
25	Role of SNARE proteins in tumorigenesis and their potential as targets for novel anti-cancer therapeutics. <i>Biochimica Et Biophysica Acta: Reviews on Cancer</i> , 2015 , 1856, 1-12	11.2	32
24	Engineering of botulinum neurotoxins as novel therapeutic tools 2015 , 995-1015		2
23	Targeted delivery of a SNARE protease to sensory neurons using a single chain antibody (scFv) against the extracellular domain of P2X(3) inhibits the release of a pain mediator. <i>Biochemical Journal</i> , 2014 , 462, 247-56	3.8	13

22	Fusion of Golgi-derived vesicles mediated by SNAP-25 is essential for sympathetic neuron outgrowth but relatively insensitive to botulinum neurotoxins in vitro. <i>FEBS Journal</i> , 2014 , 281, 3243-60	5.7	3
21	Selective cleavage of SNAREs in sensory neurons unveils protein complexes mediating peptide exocytosis triggered by different stimuli. <i>Molecular Neurobiology</i> , 2014 , 50, 574-88	6.2	14
20	SNAP-23 and VAMP-3 contribute to the release of IL-6 and TNF α from a human synovial sarcoma cell line. <i>FEBS Journal</i> , 2014 , 281, 750-65	5.7	15
19	Molecular components required for resting and stimulated endocytosis of botulinum neurotoxins by glutamatergic and peptidergic neurons. <i>FASEB Journal</i> , 2013 , 27, 3167-80	0.9	16
18	Therapeutic effectiveness of botulinum neurotoxin A: potent blockade of autonomic transmission by targeted cleavage of only the pertinent SNAP-25. <i>Neuropharmacology</i> , 2013 , 70, 287-95	5.5	14
17	Pharmacological characteristics of Kv1.1- and Kv1.2-containing channels are influenced by the stoichiometry and positioning of their β subunits. <i>Biochemical Journal</i> , 2013 , 454, 101-8	3.8	14
16	Targeted delivery into motor nerve terminals of inhibitors for SNARE-cleaving proteases via liposomes coupled to an atoxic botulinum neurotoxin. <i>FEBS Journal</i> , 2012 , 279, 2555-67	5.7	27
15	Extravesicular intraneuronal migration of internalized botulinum neurotoxins without detectable inhibition of distal neurotransmission. <i>Biochemical Journal</i> , 2012 , 441, 443-52	3.8	47
14	Longer-acting and highly potent chimaeric inhibitors of excessive exocytosis created with domains from botulinum neurotoxin A and B. <i>Biochemical Journal</i> , 2012 , 444, 59-67	3.8	33
13	Novel chimeras of botulinum and tetanus neurotoxins yield insights into their distinct sites of neuroparalysis. <i>FASEB Journal</i> , 2012 , 26, 5035-48	0.9	31
12	Position-dependent attenuation by Kv1.6 of N-type inactivation of Kv1.4-containing channels. <i>Biochemical Journal</i> , 2011 , 438, 389-96	3.8	6
11	Novel therapeutics based on recombinant botulinum neurotoxins to normalize the release of transmitters and pain mediators. <i>FEBS Journal</i> , 2011 , 278, 4454-66	5.7	35
10	A dileucine in the protease of botulinum toxin A underlies its long-lived neuroparalysis: transfer of longevity to a novel potential therapeutic. <i>Journal of Biological Chemistry</i> , 2011 , 286, 6375-85	5.4	72
9	Arrangement of Kv1 alpha subunits dictates sensitivity to tetraethylammonium. <i>Journal of General Physiology</i> , 2010 , 136, 273-82	3.4	20
8	Multiple Steps in the Blockade of Exocytosis by Botulinum Neurotoxins 2009 , 1-14.e1		0
7	Activation of TRPV1 mediates calcitonin gene-related peptide release, which excites trigeminal sensory neurons and is attenuated by a retargeted botulinum toxin with anti-nociceptive potential. <i>Journal of Neuroscience</i> , 2009 , 29, 4981-92	6.6	168
6	Neuro-exocytosis: botulinum toxins as inhibitory probes and versatile therapeutics. <i>Current Opinion in Pharmacology</i> , 2009 , 9, 326-35	5.1	65
5	Novel chimeras of botulinum neurotoxins A and E unveil contributions from the binding, translocation, and protease domains to their functional characteristics. <i>Journal of Biological Chemistry</i> , 2008 , 283, 16993-7002	5.4	86

- 4 Synaptobrevin I mediates exocytosis of CGRP from sensory neurons and inhibition by botulinum toxins reflects their anti-nociceptive potential. *Journal of Cell Science*, **2007**, 120, 2864-74 5-3 194
- 3 Two protein trafficking processes at motor nerve endings unveiled by botulinum neurotoxin E. *Journal of Pharmacology and Experimental Therapeutics*, **2007**, 320, 410-8 4-7 28
- 2 Construction of cytopathic PK-15 cell model of classical swine fever virus. *Science Bulletin*, **2003**, 48, 887-896 6
- 1 Construction of cDNA library, nucleotide sequence and analysis of entire genome of classical swine fever virus strain Shimen. *Science Bulletin*, **2000**, 45, 367-369 2