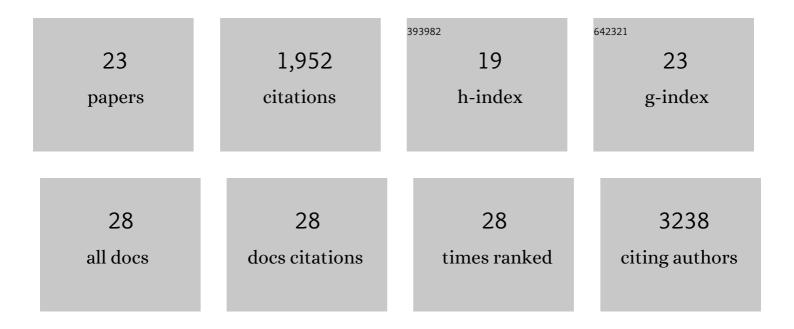
Jing Zhao

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	A central mechanism of analgesia in mice and humans lacking the sodium channel NaV1.7. Neuron, 2021, 109, 1497-1512.e6.	3.8	42
2	Tools for analysis and conditional deletion of subsets of sensory neurons. Wellcome Open Research, 2021, 6, 250.	0.9	8
3	Sensory neuron–derived Na _V 1.7 contributes to dorsal horn neuron excitability. Science Advances, 2020, 6, eaax4568.	4.7	22
4	Brain-derived neurotrophic factor derived from sensory neurons plays a critical role in chronic pain. Brain, 2018, 141, 1028-1039.	3.7	116
5	Mapping protein interactions of sodium channel Na _V 1.7 using epitopeâ€ŧagged geneâ€ŧargeted mice. EMBO Journal, 2018, 37, 427-445.	3.5	54
6	A novel human pain insensitivity disorder caused by a point mutation in ZFHX2. Brain, 2018, 141, 365-376.	3.7	32
7	The Genetics of Pain: Implications for Therapeutics. Annual Review of Pharmacology and Toxicology, 2018, 58, 123-142.	4.2	49
8	Distinct transcriptional responses of mouse sensory neurons in models of human chronic pain conditions. Wellcome Open Research, 2018, 3, 78.	0.9	34
9	MicroRNA-1-associated effects of neuron-specific brain-derived neurotrophic factor gene deletion in dorsal root ganglia. Molecular and Cellular Neurosciences, 2016, 75, 36-43.	1.0	19
10	Endogenous opioids contribute to insensitivity to pain in humans and mice lacking sodium channel Nav1.7. Nature Communications, 2015, 6, 8967.	5.8	150
11	Glycine at the Gate—from Model to Mechanism. Neuron, 2015, 85, 1152-1154.	3.8	1
12	Regulation of Nav1.7: A Conserved SCN9A Natural Antisense Transcript Expressed in Dorsal Root Ganglia. PLoS ONE, 2015, 10, e0128830.	1.1	28
13	Nav1.8 channels in ganglionated plexi modulate atrial fibrillation inducibility. Cardiovascular Research, 2014, 102, 480-486.	1.8	36
14	TRPC3 and TRPC6 are essential for normal mechanotransduction in subsets of sensory neurons and cochlear hair cells. Open Biology, 2012, 2, 120068.	1.5	135
15	Temporal Control of Gene Deletion in Sensory Ganglia Using a Tamoxifen-Inducible <i>Advillin-CreERT2</i> Recombinase Mouse. Molecular Pain, 2011, 7, 1744-8069-7-100.	1.0	84
16	Pain channelopathies. Journal of Physiology, 2010, 588, 1897-1904.	1.3	72
17	Genetic variation in SCN10A influences cardiac conduction. Nature Genetics, 2010, 42, 149-152.	9.4	248
18	Small RNAs Control Sodium Channel Expression, Nociceptor Excitability, and Pain Thresholds. Journal of Neuroscience, 2010, 30, 10860-10871.	1.7	152

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#	Article	IF	CITATIONS
19	Nociceptor-Expressed Ephrin-B2 Regulates Inflammatory and Neuropathic Pain. Molecular Pain, 2010, 6, 1744-8069-6-77.	1.0	43
20	The Cell and Molecular Basis of Mechanical, Cold, and Inflammatory Pain. Science, 2008, 321, 702-705.	6.0	419
21	Ion Channel Activities Implicated in Pathological Pain. Novartis Foundation Symposium, 2008, , 32-46.	1.2	29
22	Nociceptor-derived brain-derived neurotrophic factor regulates acute and inflammatory but not neuropathic pain. Molecular and Cellular Neurosciences, 2006, 31, 539-548.	1.0	148
23	Tamoxifen-inducible NaV1.8-CreERT2 recombinase activity in nociceptive neurons of dorsal root ganglia. Genesis, 2006, 44, 364-371.	0.8	25