Bente Vilsen

List of Publications by Year
in descending order

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		201575	206029
49	3,522	27	48
papers	citations	h-index	g-index

49	49	49	3166
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Cryoelectron microscopy of Na ⁺ ,K ⁺ -ATPase in the two E2P states with and without cardiotonic steroids. Proceedings of the National Academy of Sciences of the United States of America, 2022, 119, e2123226119.	3.3	10
2	Role of a conserved ion-binding site tyrosine in ion selectivity of the Na+/K+ pump. Journal of General Physiology, 2022, 154, .	0.9	7
3	Binding of cardiotonic steroids to Na ⁺ ,K ⁺ -ATPase in the E2P state. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	3.3	35
4	ATP1A2- and ATP1A3-associated early profound epileptic encephalopathy and polymicrogyria. Brain, 2021, 144, 1435-1450.	3.7	35
5	Distinct effects of Q925 mutation on intracellular and extracellular Na+ and K+ binding to the Na+, K+-ATPase. Scientific Reports, 2019, 9, 13344.	1.6	10
6	Asparagine 905 of the mammalian phospholipid flippase ATP8A2 is essential for lipid substrateâ€“induced activation of ATP8A2 dephosphorylation. Journal of Biological Chemistry, 2019, 294, 5970-5979.	1.6	14
7	Functional consequences of the CAPOS mutation E818K of Na+,K+-ATPase. Journal of Biological Chemistry, 2019, 294, 269-280.	1.6	14
8	Arginine substitution of a cysteine in transmembrane helix M8 converts Na ⁺ ,K ⁺ -ATPase to an electroneutral pump similar to H ⁺ ,K ⁺ -ATPase. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 316-321.	3.3	18
9	Importance of a Potential Protein Kinase A Phosphorylation Site of Na+,K+-ATPase and Its Interaction Network for Na+ Binding. Journal of Biological Chemistry, 2016, 291, 10934-10947.	1.6	15
10	Neurological disease mutations of Î±3 Na+,K+-ATPase: Structural and functional perspectives and rescue of compromised function. Biochimica Et Biophysica Acta - Bioenergetics, 2016, 1857, 1807-1828.	0.5	37
11	Glutamate transporter activity promotes enhanced Na⁺/K⁺â€ATPaseâ€mediated extracellular K⁺ management during neuronal activity. Journal of Physiology, 2016, 594, 6627-6641.	1.3	24
12	Rescue of Na+ Affinity in Aspartate 928 Mutants of Na+,K+-ATPase by Secondary Mutation of Glutamate 314. Journal of Biological Chemistry, 2015, 290, 9801-9811.	1.6	14
13	Relationship between Intracellular Na+ Concentration and Reduced Na+ Affinity in Na+,K+-ATPase Mutants Causing Neurological Disease. Journal of Biological Chemistry, 2014, 289, 3186-3197.	1.6	38
14	Critical roles of isoleucine-364 and adjacent residues in a hydrophobic gate control of phospholipid transport by the mammalian P4-ATPase ATP8A2. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, E1334-43.	3.3	103
15	Distinct neurological disorders with ATP1A3 mutations. Lancet Neurology, The, 2014, 13, 503-514.	4.9	206
16	Crystal structure of a Na+-bound Na+,K+-ATPase preceding the E1P state. Nature, 2013, 502, 201-206.	13.7	271
17	Somatic mutations in ATP1A1 and ATP2B3 lead to aldosterone-producing adenomas and secondary hypertension. Nature Genetics, 2013, 45, 440-444.	9.4	460
18	Inhibition of Phosphorylation of Na+,K+-ATPase by Mutations Causing Familial Hemiplegic Migraine. Journal of Biological Chemistry, 2012, 287, 2191-2202.	1.6	21

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19	Critical role of a transmembrane lysine in aminophospholipid transport by mammalian photoreceptor P ₄ -ATPase ATP8A2. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 1449-1454.	3.3	88
20	Mania-like behavior induced by genetic dysfunction of the neuron-specific Na ⁺ ,K ⁺ -ATPase Î±3 sodium pump. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 18144-18149.	3.3	127
21	The Rapid-onset Dystonia Parkinsonism Mutation D923N of the Na+,K+-ATPase Î±3 Isoform Disrupts Na+ Interaction at the Third Na+ Site. Journal of Biological Chemistry, 2010, 285, 26245-26254.	1.6	53
22	The C Terminus of Na+,K+-ATPase Controls Na+ Affinity on Both Sides of the Membrane through Arg935. Journal of Biological Chemistry, 2009, 284, 18715-18725.	1.6	49
23	Mutation I810N in the Î±3 isoform of Na ⁺ ,K ⁺ -ATPase causes impairments in the sodium pump and hyperexcitability in the CNS. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 14085-14090.	3.3	128
24	A C-terminal mutation of ATP1A3 underscores the crucial role of sodium affinity in the pathophysiology of rapid-onset dystonia-parkinsonism. Human Molecular Genetics, 2009, 18, 2370-2377.	1.4	59
25	Crystal structure of the sodiumâ€“potassium pump. Nature, 2007, 450, 1043-1049.	13.7	789
26	Mutations Phe785Leu and Thr618Met in Na+,K+-ATPase, Associated with Familial Rapid-onset Dystonia Parkinsonism, Interfere with Na+ Interaction by Distinct Mechanisms. Journal of Biological Chemistry, 2006, 281, 18539-18548.	1.6	66
27	Mutation of Gly-94 in transmembrane segment M1 of Na+,K+-ATPase interferes with Na+ and K+ binding in E2P conformation. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 11254-11259.	3.3	25
28	Glutamate-183 in the conserved TGES motif of domain A of sarcoplasmic reticulum Ca2+-ATPase assists in catalysis of E2/E2P partial reactions. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 2776-2781.	3.3	71
29	Importance of Transmembrane Segment M3 of Na⁺,K⁺â€ATPase for Control of Conformational Changes and the Cytoplasmic Entry Pathway for Na⁺. Annals of the New York Academy of Sciences, 2003, 986, 50-57.	1.8	3
30	Functional Consequences of Alterations to Ile279, Ile283, Glu284, His285, Phe286, and His288 in the NH2-terminal Part of Transmembrane Helix M3 of the Na+,K+-ATPase. Journal of Biological Chemistry, 2003, 278, 38653-38664.	1.6	10
31	Importance of Glu282 in Transmembrane Segment M3 of the Na+,K+-ATPase for Control of Cation Interaction and Conformational Changes. Journal of Biological Chemistry, 2002, 277, 38607-38617.	1.6	23
32	Mutational Effects on Conformational Changes of the Dephospho- and Phospho-forms of the Na+,K+-ATPaseâ€. Biochemistry, 2001, 40, 5521-5532.	1.2	30
33	Mutant Phe788 â†' Leu of the Na+,K+-ATPase Is Inhibited by Micromolar Concentrations of Potassium and Exhibits High Na+-ATPase Activity at Low Sodium Concentrationsâ€. Biochemistry, 1999, 38, 11389-11400.	1.2	27
34	Mutation to the Glutamate in the Fourth Membrane Segment of Na+,K+-ATPase and Ca2+-ATPase Affects Cation Binding from Both Sides of the Membrane and Destabilizes the Occluded Enzyme Formsâ€. Biochemistry, 1998, 37, 10961-10971.	1.2	88
35	Leucine 332 at the Boundary Between the Fourth Transmembrane Segment and the Cytoplasmic Domain of Na+,K+-ATPase Plays a Pivotal Role in the Ion Translocating Conformational Changes. Biochemistry, 1997, 36, 13312-13324.	1.2	50
36	Functional Consequences of Mutations in the Transmembrane Core Region for Cation Translocation and Energy Transduction in the Na+, K+-ATPase and the SR Ca2+-ATPase. Annals of the New York Academy of Sciences, 1997, 834, 297-309.	1.8	34

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37	Mutant Glu781 .fwdarw. Ala of the rat kidney Na+,K+-ATPase displays low cation affinity and catalyzes ATP hydrolysis at a high rate in the absence of potassium ions. Biochemistry, 1995, 34, 1455-1463.	1.2	91
38	Structure-function relationships of cation translocation by Ca2+- and Na+,K+-ATPases studied by site-directed mutagenesis. FEBS Letters, 1995, 359, 101-106.	1.3	101
39	Functional consequences of mutation Asn326â†'Leu in the 4th transmembrane segment of the a-subunit of the rat kidney Na+,K+-ATPase. FEBS Letters, 1995, 363, 179-183.	1.3	20
40	A Glu329â†' Gln variant of the Î±-subunit of the rat kidney Na+K+-ATPase can sustain active transport of Na+and K+and Na+K+-activated ATP hydrolysis with normal turnover number. FEBS Letters, 1993, 333, 44-50.	1.3	14
41	Chimeric Ca2+-ATPase/Na+,K+-ATPase molecules. FEBS Letters, 1993, 336, 248-254.	1.3	17
42	Glutamate 329 located in the fourth transmembrane segment of the .alpha.-subunit of the rat kidney sodium-potassium-ATPase is not an essential residue for active transport of sodium and potassium ions. Biochemistry, 1993, 32, 13340-13349.	1.2	69
43	Deduced amino acid sequence and E1-E2equilibrium of the sarcoplasmic reticulum Ca2+-ATPase of frog skeletal muscle Comparison with the Ca2+-ATPase of rabbit fast twitch muscle. FEBS Letters, 1992, 306, 213-218.	1.3	29
44	Mutational analysis of the role of Glu309in the sarcoplasmic reticulum Ca2+-ATPase of frog skeletal muscle. FEBS Letters, 1992, 306, 247-250.	1.3	28
45	Functional consequences of alterations to Pro328and Leu332located in the 4th transmembrane segment of the Î±-subunit of the rat kidney Na+,K+-ATPase. FEBS Letters, 1992, 314, 301-307.	1.3	52
46	Radiation inactivation analysis of sarcoplasmic reticulum Ca-ATPase in membrane-bound form and in detergent-solubilized monomeric states. FEBS Letters, 1988, 234, 120-126.	1.3	4
47	Effect of phospholipid, detergent and protein-protein interaction on stability and phosphoenzyme isomerization of soluble sarcoplasmic reticulum Ca-ATPase. FEBS Journal, 1987, 170, 421-429.	0.2	20
48	Equilibrium between monomers and oligomers of soluble Ca2+ -ATPase during the functional cycle. FEBS Letters, 1985, 189, 13-17.	1.3	22
49	Cryoâ€electron microscopy of Na ⁺ ,K ⁺ â€•<scp>ATPase</scp> reveals how the extracellular gate locks in the <scp>E2</scp> Â· <scp>2K</scp> ⁺ state. FEBS Letters, 0, , .	1.3	3