Bente Vilsen

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/4659002/publications.pdf

Version: 2024-02-01

		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	Crystal structure of the sodiumâ€“potassium pump. Nature, 2007, 450, 1043-1049.	13.7	789
2	Somatic mutations in ATP1A1 and ATP2B3 lead to aldosterone-producing adenomas and secondary hypertension. Nature Genetics, 2013, 45, 440-444.	9.4	460
3	Crystal structure of a Na+-bound Na+,K+-ATPase preceding the E1P state. Nature, 2013, 502, 201-206.	13.7	271
4	Distinct neurological disorders with ATP1A3 mutations. Lancet Neurology, The, 2014, 13, 503-514.	4.9	206
5	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
6	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
7	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
8	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
9	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
10	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
11	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
12	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
13	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
14	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
15	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
16	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
17	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
18	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

#	Article	IF	Citations
19	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
20	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
21	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
22	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
23	ATP1A2- and ATP1A3-associated early profound epileptic encephalopathy and polymicrogyria. Brain, 2021, 144, 1435-1450.	3.7	35
24	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
25	Mutational Effects on Conformational Changes of the Dephospho- and Phospho-forms of the Na+,K+-ATPaseâ€. Biochemistry, 2001, 40, 5521-5532.	1.2	30
26	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
27	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
28	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
29	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
30	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
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	Equilibrium between monomers and oligomers of soluble Ca2+ -ATPase during the functional cycle. FEBS Letters, 1985, 189, 13-17.	1.3	22
33	Inhibition of Phosphorylation of Na+,K+-ATPase by Mutations Causing Familial Hemiplegic Migraine. Journal of Biological Chemistry, 2012, 287, 2191-2202.	1.6	21
34	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
35	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
36	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

#	Article	IF	Citations
37	Chimeric Ca2+-ATPase/Na+,K+-ATPase molecules. FEBS Letters, 1993, 336, 248-254.	1.3	17
38	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
39	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
40	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
41	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
42	Functional consequences of the CAPOS mutation E818K of Na+,K+-ATPase. Journal of Biological Chemistry, 2019, 294, 269-280.	1.6	14
43	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
44	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
45	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
46	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
47	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
48	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
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