

Tzyh-Chang Hwang

List of Publications by Year in descending order

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Version: 2024-02-01

35
papers

1,251
citations

331670

21
h-index

414414

32
g-index

35
all docs

35
docs citations

35
times ranked

854
citing authors

#	ARTICLE	IF	CITATIONS
1	Gating of the CFTR Cl ⁻ channel by ATP-driven nucleotide-binding domain dimerisation. <i>Journal of Physiology</i> , 2009, 587, 2151-2161.	2.9	150
2	Deletion of phenylalanine 508 causes attenuated phosphorylation-dependent activation of CFTR chloride channels. <i>Journal of Physiology</i> , 2000, 524, 637-648.	2.9	93
3	Structural mechanisms of CFTR function and dysfunction. <i>Journal of General Physiology</i> , 2018, 150, 539-570.	1.9	90
4	Stable ATP binding mediated by a partial NBD dimer of the CFTR chloride channel. <i>Journal of General Physiology</i> , 2010, 135, 399-414.	1.9	79
5	The Two ATP Binding Sites of Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) Play Distinct Roles in Gating Kinetics and Energetics. <i>Journal of General Physiology</i> , 2006, 128, 413-422.	1.9	71
6	CFTR Gating I. <i>Journal of General Physiology</i> , 2005, 125, 361-375.	1.9	58
7	Identifying the molecular target sites for CFTR potentiators GLPG1837 and VX-770. <i>Journal of General Physiology</i> , 2019, 151, 912-928.	1.9	57
8	Potential of Disease-associated Cystic Fibrosis Transmembrane Conductance Regulator Mutants by Hydrolyzable ATP Analogs. <i>Journal of Biological Chemistry</i> , 2010, 285, 19967-19975.	3.4	54
9	Voltage-dependent flickery block of an open cystic fibrosis transmembrane conductance regulator (CFTR) channel pore. <i>Journal of Physiology</i> , 2001, 532, 435-448.	2.9	50
10	A common mechanism for CFTR potentiators. <i>Journal of General Physiology</i> , 2017, 149, 1105-1118.	1.9	50
11	State-dependent modulation of CFTR gating by pyrophosphate. <i>Journal of General Physiology</i> , 2009, 133, 405-419.	1.9	49
12	The most common cystic fibrosis-associated mutation destabilizes the dimeric state of the nucleotide-binding domains of CFTR. <i>Journal of Physiology</i> , 2011, 589, 2719-2731.	2.9	45
13	Modulation of CFTR gating by permeant ions. <i>Journal of General Physiology</i> , 2015, 145, 47-60.	1.9	44
14	On the mechanism of gating defects caused by the R117H mutation in cystic fibrosis transmembrane conductance regulator. <i>Journal of Physiology</i> , 2016, 594, 3227-3244.	2.9	35
15	Nonequilibrium Gating of CFTR on an Equilibrium Theme. <i>Physiology</i> , 2012, 27, 351-361.	3.1	34
16	CFTR potentiators: from bench to bedside. <i>Current Opinion in Pharmacology</i> , 2017, 34, 98-104.	3.5	33
17	Identification and Characterization of Novel CFTR Potentiators. <i>Frontiers in Pharmacology</i> , 2018, 9, 1221.	3.5	32
18	Synergistic Potentiation of Cystic Fibrosis Transmembrane Conductance Regulator Gating by Two Chemically Distinct Potentiators, Ivacaftor (VX-770) and 5-Nitro-2-(3-Phenylpropylamino) Benzoate. <i>Molecular Pharmacology</i> , 2016, 90, 275-285.	2.3	31

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19	Identification of GLPG/ABBV-2737, a Novel Class of Corrector, Which Exerts Functional Synergy With Other CFTR Modulators. <i>Frontiers in Pharmacology</i> , 2019, 10, 514.	3.5	29
20	Physiological and pharmacological characterization of the N1303K mutant CFTR. <i>Journal of Cystic Fibrosis</i> , 2018, 17, 573-581.	0.7	26
21	A single amino acid substitution in CFTR converts ATP to an inhibitory ligand. <i>Journal of General Physiology</i> , 2014, 144, 311-320.	1.9	24
22	Biological Characterization of F508delCFTR Protein Processing by the CFTR Corrector ABBV-2222/GLPG2222. <i>Journal of Pharmacology and Experimental Therapeutics</i> , 2020, 372, 107-118.	2.5	21
23	Identification of a novel post-hydrolytic state in CFTR gating. <i>Journal of General Physiology</i> , 2012, 139, 359-370.	1.9	19
24	Optimization of the Degenerated Interfacial ATP Binding Site Improves the Function of Disease-related Mutant Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) Channels. <i>Journal of Biological Chemistry</i> , 2010, 285, 37663-37671.	3.4	18
25	CFTR: New insights into structure and function and implications for modulation by small molecules. <i>Journal of Cystic Fibrosis</i> , 2020, 19, S19-S24.	0.7	16
26	Spatial positioning of CFTR's pore-lining residues affirms an asymmetrical contribution of transmembrane segments to the anion permeation pathway. <i>Journal of General Physiology</i> , 2016, 147, 407-422.	1.9	13
27	Functional stability of CFTR depends on tight binding of ATP at its degenerate ATP-binding site. <i>Journal of Physiology</i> , 2021, 599, 4625-4642.	2.9	9
28	Organoids as a personalized medicine tool for ultra-rare mutations in cystic fibrosis: The case of S955P and 1717-2A>G. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2020, 1866, 165905.	3.8	7
29	Cystic fibrosis research topics featured at the 14th ECFS Basic Science Conference: Chairman's summary. <i>Journal of Cystic Fibrosis</i> , 2018, 17, S1-S4.	0.7	5
30	Characterization of I ⁹⁷⁰ (G970-T1122)-CFTR, the most frequent CFTR mutant identified in Japanese cystic fibrosis patients. <i>Journal of Physiological Sciences</i> , 2019, 69, 103-112.	2.1	5
31	The physiology of anion transport: tales of the bizarre and unexpected. <i>Experimental Physiology</i> , 2006, 91, 121-122.	2.0	1
32	CFTR: a missing link between exocrine and endocrine pancreas?. <i>Science China Life Sciences</i> , 2014, 57, 1044-1045.	4.9	1
33	Generation of human induced pluripotent stem cells from cystic fibrosis patient carrying nonsense mutation (p.S308X) in CFTR gene. <i>Stem Cell Research</i> , 2022, 60, 102683.	0.7	1
34	Pharmacological Responses of the G542X-CFTR to CFTR Modulators. <i>Frontiers in Molecular Biosciences</i> , 0, 9, .	3.5	1
35	<i>In vitro</i> assessment of triple combination therapy for the most common disease-associated mutation in cystic fibrosis. <i>European Respiratory Journal</i> , 2022, 59, 2102380.	6.7	0