

Tip W Loo

List of Publications by Year in Descending Order

Source: <https://exaly.com/author-pdf/8190195/tip-w-loo-publications-by-year.pdf>

Version: 2024-04-27

This document has been generated based on the publications and citations recorded by exaly.com. For the latest version of this publication list, visit the link given above.

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

114
papers

7,046
citations

52
h-index

81
g-index

114
ext. papers

7,284
ext. citations

4.9
avg, IF

5.97
L-index

#	Paper	IF	Citations
114	Thiol-reactive drug substrates of human P-glycoprotein label the same sites to activate ATPase activity in membranes or dodecyl maltoside detergent micelles. <i>Biochemical and Biophysical Research Communications</i> , 2017 , 488, 573-577	3.4	4
113	Corrector VX-809 promotes interactions between cytoplasmic loop one and the first nucleotide-binding domain of CFTR. <i>Biochemical Pharmacology</i> , 2017 , 136, 24-31	6	31
112	Attachment of a molecular spring restores drug-stimulated ATPase activity to P-glycoprotein lacking both Q loop glutamines. <i>Biochemical and Biophysical Research Communications</i> , 2017 , 483, 366-370	3.4	12
111	A short cross-linker activates human P-glycoprotein missing a catalytic carboxylate. <i>Biochemical Pharmacology</i> , 2017 , 145, 27-33	6	2
110	P-glycoprotein ATPase activity requires lipids to activate a switch at the first transmission interface. <i>Biochemical and Biophysical Research Communications</i> , 2016 , 472, 379-83	3.4	18
109	Drugs Modulate Interactions between the First Nucleotide-Binding Domain and the Fourth Cytoplasmic Loop of Human P-Glycoprotein. <i>Biochemistry</i> , 2016 , 55, 2817-20	3.2	5
108	The Transmission Interfaces Contribute Asymmetrically to the Assembly and Activity of Human P-glycoprotein. <i>Journal of Biological Chemistry</i> , 2015 , 290, 16954-63	5.4	21
107	Mapping the Binding Site of the Inhibitor Tariquidar That Stabilizes the First Transmembrane Domain of P-glycoprotein. <i>Journal of Biological Chemistry</i> , 2015 , 290, 29389-401	5.4	32
106	Locking intracellular helices 2 and 3 together inactivates human P-glycoprotein. <i>Journal of Biological Chemistry</i> , 2014 , 289, 229-36	5.4	18
105	Cysteines introduced into extracellular loops 1 and 4 of human P-glycoprotein that are close only in the open conformation spontaneously form a disulfide bond that inhibits drug efflux and ATPase activity. <i>Journal of Biological Chemistry</i> , 2014 , 289, 24749-58	5.4	10
104	Tariquidar inhibits P-glycoprotein drug efflux but activates ATPase activity by blocking transition to an open conformation. <i>Biochemical Pharmacology</i> , 2014 , 92, 558-66	6	35
103	Identification of the distance between the homologous halves of P-glycoprotein that triggers the high/low ATPase activity switch. <i>Journal of Biological Chemistry</i> , 2014 , 289, 8484-92	5.4	19
102	The cystic fibrosis V232D mutation inhibits CFTR maturation by disrupting a hydrophobic pocket rather than formation of aberrant interhelical hydrogen bonds. <i>Biochemical Pharmacology</i> , 2014 , 88, 46-57	6	8
101	Corrector VX-809 stabilizes the first transmembrane domain of CFTR. <i>Biochemical Pharmacology</i> , 2013 , 86, 612-9	6	73
100	A salt bridge in intracellular loop 2 is essential for folding of human p-glycoprotein. <i>Biochemistry</i> , 2013 , 52, 3194-6	3.2	20
99	Niemann-Pick NPC1: sterols to the rescue and beyond. <i>Chemistry and Biology</i> , 2013 , 20, 297-8		3
98	Bithiazole correctors rescue CFTR mutants by two different mechanisms. <i>Biochemistry</i> , 2013 , 52, 5161-3	3.2	13

97	Drug rescue distinguishes between different structural models of human P-glycoprotein. <i>Biochemistry</i> , 2013 , 52, 7167-9	3.2	18
96	Human P-glycoprotein contains a greasy ball-and-socket joint at the second transmission interface. <i>Journal of Biological Chemistry</i> , 2013 , 288, 20326-33	5.4	36
95	Corrector-mediated rescue of misprocessed CFTR mutants can be reduced by the P-glycoprotein drug pump. <i>Biochemical Pharmacology</i> , 2012 , 83, 345-54	6	15
94	Thiorhodamines containing amide and thioamide functionality as inhibitors of the ATP-binding cassette drug transporter P-glycoprotein (ABCB1). <i>Bioorganic and Medicinal Chemistry</i> , 2012 , 20, 4290-3024	3.4	8
93	Chalcogenopyrylium compounds as modulators of the ATP-binding cassette transporters P-glycoprotein (P-gp/ABCB1) and multidrug resistance protein 1 (MRP1/ABCC1). <i>Journal of Medicinal Chemistry</i> , 2012 , 55, 4683-99	8.3	38
92	The ATPase activity of the P-glycoprotein drug pump is highly activated when the N-terminal and central regions of the nucleotide-binding domains are linked closely together. <i>Journal of Biological Chemistry</i> , 2012 , 287, 26806-16	5.4	48
91	Predicting P-glycoprotein-mediated drug transport based on support vector machine and three-dimensional crystal structure of P-glycoprotein. <i>PLoS ONE</i> , 2011 , 6, e25815	3.7	89
90	The W232R suppressor mutation promotes maturation of a truncation mutant lacking both nucleotide-binding domains and restores interdomain assembly and activity of P-glycoprotein processing mutants. <i>Biochemistry</i> , 2011 , 50, 672-85	3.2	6
89	Benzbromarone stabilizes Δ508 CFTR at the cell surface. <i>Biochemistry</i> , 2011 , 50, 4393-5	3.2	10
88	Repair of CFTR folding defects with correctors that function as pharmacological chaperones. <i>Methods in Molecular Biology</i> , 2011 , 741, 23-37	1.4	6
87	The V510D suppressor mutation stabilizes ΔF508-CFTR at the cell surface. <i>Biochemistry</i> , 2010 , 49, 6352-7	3.2	48
86	Human P-glycoprotein is active when the two halves are clamped together in the closed conformation. <i>Biochemical and Biophysical Research Communications</i> , 2010 , 395, 436-40	3.4	52
85	Correctors enhance maturation of ΔF508 CFTR by promoting interactions between the two halves of the molecule. <i>Biochemistry</i> , 2009 , 48, 9882-90	3.2	31
84	Rhodamine inhibitors of P-glycoprotein: an amide/thioamide "switch" for ATPase activity. <i>Journal of Medicinal Chemistry</i> , 2009 , 52, 3328-41	8.3	50
83	Identification of residues in the drug translocation pathway of the human multidrug resistance P-glycoprotein by arginine mutagenesis. <i>Journal of Biological Chemistry</i> , 2009 , 284, 24074-87	5.4	69
82	Mutational analysis of ABC proteins. <i>Archives of Biochemistry and Biophysics</i> , 2008 , 476, 51-64	4.1	69
81	Processing mutations disrupt interactions between the nucleotide binding and transmembrane domains of P-glycoprotein and the cystic fibrosis transmembrane conductance regulator (CFTR). <i>Journal of Biological Chemistry</i> , 2008 , 283, 28190-7	5.4	66
80	Arginines in the first transmembrane segment promote maturation of a P-glycoprotein processing mutant by hydrogen bond interactions with tyrosines in transmembrane segment 11. <i>Journal of Biological Chemistry</i> , 2008 , 283, 24860-70	5.4	24

79	Correctors promote folding of the CFTR in the endoplasmic reticulum. <i>Biochemical Journal</i> , 2008 , 413, 29-36	3.8	43
78	Correctors promote maturation of cystic fibrosis transmembrane conductance regulator (CFTR)-processing mutants by binding to the protein. <i>Journal of Biological Chemistry</i> , 2007 , 282, 33247-33251	5.4	106
77	Modulating the folding of P-glycoprotein and cystic fibrosis transmembrane conductance regulator truncation mutants with pharmacological chaperones. <i>Molecular Pharmacology</i> , 2007 , 71, 751-8	4.3	66
76	Chemical and pharmacological chaperones as new therapeutic agents. <i>Expert Reviews in Molecular Medicine</i> , 2007 , 9, 1-18	6.7	83
75	Additive effect of multiple pharmacological chaperones on maturation of CFTR processing mutants. <i>Biochemical Journal</i> , 2007 , 406, 257-63	3.8	53
74	Suppressor mutations in the transmembrane segments of P-glycoprotein promote maturation of processing mutants and disrupt a subset of drug-binding sites. <i>Journal of Biological Chemistry</i> , 2007 , 282, 32043-52	5.4	38
73	Nucleotide binding, ATP hydrolysis, and mutation of the catalytic carboxylates of human P-glycoprotein cause distinct conformational changes in the transmembrane segments. <i>Biochemistry</i> , 2007 , 46, 9328-36	3.2	23
72	Specific rescue of cystic fibrosis transmembrane conductance regulator processing mutants using pharmacological chaperones. <i>Molecular Pharmacology</i> , 2006 , 70, 297-302	4.3	85
71	Insertion of an arginine residue into the transmembrane segments corrects protein misfolding. <i>Journal of Biological Chemistry</i> , 2006 , 281, 29436-40	5.4	16
70	Transmembrane segment 1 of human P-glycoprotein contributes to the drug-binding pocket. <i>Biochemical Journal</i> , 2006 , 396, 537-45	3.8	76
69	Transmembrane segment 7 of human P-glycoprotein forms part of the drug-binding pocket. <i>Biochemical Journal</i> , 2006 , 399, 351-9	3.8	88
68	The chemical chaperone CFcor-325 repairs folding defects in the transmembrane domains of CFTR-processing mutants. <i>Biochemical Journal</i> , 2006 , 395, 537-42	3.8	40
67	Using a cysteine-less mutant to provide insight into the structure and mechanism of CFTR. <i>Journal of Physiology</i> , 2006 , 572, 312	3.9	7
66	Rescue of DeltaF508 and other misprocessed CFTR mutants by a novel quinazoline compound. <i>Molecular Pharmaceutics</i> , 2005 , 2, 407-13	5.6	67
65	Do drug substrates enter the common drug-binding pocket of P-glycoprotein through "gates"?. <i>Biochemical and Biophysical Research Communications</i> , 2005 , 329, 419-22	3.4	66
64	ATP hydrolysis promotes interactions between the extracellular ends of transmembrane segments 1 and 11 of human multidrug resistance P-glycoprotein. <i>Biochemistry</i> , 2005 , 44, 10250-8	3.2	40
63	Recent progress in understanding the mechanism of P-glycoprotein-mediated drug efflux. <i>Journal of Membrane Biology</i> , 2005 , 206, 173-85	2.3	162
62	Rescue of folding defects in ABC transporters using pharmacological chaperones. <i>Journal of Bioenergetics and Biomembranes</i> , 2005 , 37, 501-7	3.7	50

61	The dileucine motif at the COOH terminus of human multidrug resistance P-glycoprotein is important for folding but not activity. <i>Journal of Biological Chemistry</i> , 2005 , 280, 2522-8	5.4	21
60	Processing mutations located throughout the human multidrug resistance P-glycoprotein disrupt interactions between the nucleotide binding domains. <i>Journal of Biological Chemistry</i> , 2004 , 279, 38395-401	5.4	22
59	The DeltaF508 mutation disrupts packing of the transmembrane segments of the cystic fibrosis transmembrane conductance regulator. <i>Journal of Biological Chemistry</i> , 2004 , 279, 39620-7	5.4	76
58	Disulfiram metabolites permanently inactivate the human multidrug resistance P-glycoprotein. <i>Molecular Pharmaceutics</i> , 2004 , 1, 426-33	5.6	51
57	The drug-binding pocket of the human multidrug resistance P-glycoprotein is accessible to the aqueous medium. <i>Biochemistry</i> , 2004 , 43, 12081-9	3.2	50
56	Thapsigargin or curcumin does not promote maturation of processing mutants of the ABC transporters, CFTR, and P-glycoprotein. <i>Biochemical and Biophysical Research Communications</i> , 2004 , 325, 580-5	3.4	48
55	Val133 and Cys137 in transmembrane segment 2 are close to Arg935 and Gly939 in transmembrane segment 11 of human P-glycoprotein. <i>Journal of Biological Chemistry</i> , 2004 , 279, 18232-8	5.4	50
54	Disulfide cross-linking analysis shows that transmembrane segments 5 and 8 of human P-glycoprotein are close together on the cytoplasmic side of the membrane. <i>Journal of Biological Chemistry</i> , 2004 , 279, 7692-7	5.4	61
53	Drug binding in human P-glycoprotein causes conformational changes in both nucleotide-binding domains. <i>Journal of Biological Chemistry</i> , 2003 , 278, 1575-8	5.4	96
52	Methanethiosulfonate derivatives of rhodamine and verapamil activate human P-glycoprotein at different sites. <i>Journal of Biological Chemistry</i> , 2003 , 278, 50136-41	5.4	67
51	Simultaneous binding of two different drugs in the binding pocket of the human multidrug resistance P-glycoprotein. <i>Journal of Biological Chemistry</i> , 2003 , 278, 39706-10	5.4	146
50	Permanent activation of the human P-glycoprotein by covalent modification of a residue in the drug-binding site. <i>Journal of Biological Chemistry</i> , 2003 , 278, 20449-52	5.4	47
49	Application of chemical chaperones to the rescue of folding defects. <i>Methods in Molecular Biology</i> , 2003 , 232, 231-43	1.4	3
48	Substrate-induced conformational changes in the transmembrane segments of human P-glycoprotein. Direct evidence for the substrate-induced fit mechanism for drug binding. <i>Journal of Biological Chemistry</i> , 2003 , 278, 13603-6	5.4	139
47	Location of the rhodamine-binding site in the human multidrug resistance P-glycoprotein. <i>Journal of Biological Chemistry</i> , 2002 , 277, 44332-8	5.4	169
46	The "LSGGQ" motif in each nucleotide-binding domain of human P-glycoprotein is adjacent to the opposing walker A sequence. <i>Journal of Biological Chemistry</i> , 2002 , 277, 41303-6	5.4	120
45	Introduction of the most common cystic fibrosis mutation (Delta F508) into human P-glycoprotein disrupts packing of the transmembrane segments. <i>Journal of Biological Chemistry</i> , 2002 , 277, 27585-8	5.4	31
44	Vanadate trapping of nucleotide at the ATP-binding sites of human multidrug resistance P-glycoprotein exposes different residues to the drug-binding site. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2002 , 99, 3511-6	11.5	76

43	Defining the drug-binding site in the human multidrug resistance P-glycoprotein using a methanethiosulfonate analog of verapamil, MTS-verapamil. <i>Journal of Biological Chemistry</i> , 2001 , 276, 14972-9	5.4	153
42	Cross-linking of human multidrug resistance P-glycoprotein by the substrate, tris-(2-maleimidoethyl)amine, is altered by ATP hydrolysis. Evidence for rotation of a transmembrane helix. <i>Journal of Biological Chemistry</i> , 2001 , 276, 31800-5	5.4	60
41	Determining the dimensions of the drug-binding domain of human P-glycoprotein using thiol cross-linking compounds as molecular rulers. <i>Journal of Biological Chemistry</i> , 2001 , 276, 36877-80	5.4	151
40	The packing of the transmembrane segments of human multidrug resistance P-glycoprotein is revealed by disulfide cross-linking analysis. <i>Journal of Biological Chemistry</i> , 2000 , 275, 5253-6	5.4	77
39	Identification of residues within the drug-binding domain of the human multidrug resistance P-glycoprotein by cysteine-scanning mutagenesis and reaction with dibromobimane. <i>Journal of Biological Chemistry</i> , 2000 , 275, 39272-8	5.4	106
38	Drug-stimulated ATPase activity of human P-glycoprotein is blocked by disulfide cross-linking between the nucleotide-binding sites. <i>Journal of Biological Chemistry</i> , 2000 , 275, 19435-8	5.4	52
37	The human multidrug resistance P-glycoprotein is inactive when its maturation is inhibited: potential for a role in cancer chemotherapy. <i>FASEB Journal</i> , 1999 , 13, 1724-32	0.9	80
36	The transmembrane domains of the human multidrug resistance P-glycoprotein are sufficient to mediate drug binding and trafficking to the cell surface. <i>Journal of Biological Chemistry</i> , 1999 , 274, 24759-65	5.4	110
35	Identification of residues in the drug-binding domain of human P-glycoprotein. Analysis of transmembrane segment 11 by cysteine-scanning mutagenesis and inhibition by dibromobimane. <i>Journal of Biological Chemistry</i> , 1999 , 274, 35388-92	5.4	95
34	Molecular dissection of the human multidrug resistance P-glycoprotein. <i>Biochemistry and Cell Biology</i> , 1999 , 77, 11-23	3.6	60
33	Determining the structure and mechanism of the human multidrug resistance P-glycoprotein using cysteine-scanning mutagenesis and thiol-modification techniques. <i>Biochimica Et Biophysica Acta - Biomembranes</i> , 1999 , 1461, 315-25	3.8	69
32	The glycosylation and orientation in the membrane of the third cytoplasmic loop of human P-glycoprotein is affected by mutations and substrates. <i>Biochemistry</i> , 1999 , 38, 5124-9	3.2	17
31	Nonylphenol ethoxylates, but not nonylphenol, are substrates of the human multidrug resistance P-glycoprotein. <i>Biochemical and Biophysical Research Communications</i> , 1998 , 247, 478-80	3.4	18
30	Quality control by proteases in the endoplasmic reticulum. Removal of a protease-sensitive site enhances expression of human P-glycoprotein. <i>Journal of Biological Chemistry</i> , 1998 , 273, 32373-6	5.4	45
29	Superfolding of the partially unfolded core-glycosylated intermediate of human P-glycoprotein into the mature enzyme is promoted by substrate-induced transmembrane domain interactions. <i>Journal of Biological Chemistry</i> , 1998 , 273, 14671-4	5.4	82
28	Mutational analysis of human P-glycoprotein. <i>Methods in Enzymology</i> , 1998 , 292, 480-92	1.7	16
27	Identification of residues in the drug-binding site of human P-glycoprotein using a thiol-reactive substrate. <i>Journal of Biological Chemistry</i> , 1997 , 272, 31945-8	5.4	117
26	Correction of defective protein kinesis of human P-glycoprotein mutants by substrates and modulators. <i>Journal of Biological Chemistry</i> , 1997 , 272, 709-12	5.4	192

25	Drug-stimulated ATPase activity of human P-glycoprotein requires movement between transmembrane segments 6 and 12. <i>Journal of Biological Chemistry</i> , 1997 , 272, 20986-9	5.4	84
24	Disease-associated mutations in cytoplasmic loops 1 and 2 of cystic fibrosis transmembrane conductance regulator impede processing or opening of the channel. <i>Biochemistry</i> , 1997 , 36, 11966-74	3.2	67
23	Cystic fibrosis: channel, catalytic, and folding properties of the CFTR protein. <i>Journal of Bioenergetics and Biomembranes</i> , 1997 , 29, 429-42	3.7	38
22	The minimum functional unit of human P-glycoprotein appears to be a monomer. <i>Journal of Biological Chemistry</i> , 1996 , 271, 27488-92	5.4	49
21	Inhibition of oxidative cross-linking between engineered cysteine residues at positions 332 in predicted transmembrane segments (TM) 6 and 975 in predicted TM12 of human P-glycoprotein by drug substrates. <i>Journal of Biological Chemistry</i> , 1996 , 271, 27482-7	5.4	69
20	Disease-associated mutations in the fourth cytoplasmic loop of cystic fibrosis transmembrane conductance regulator compromise biosynthetic processing and chloride channel activity. <i>Journal of Biological Chemistry</i> , 1996 , 271, 15139-45	5.4	93
19	Cytoplasmic loop three of cystic fibrosis transmembrane conductance regulator contributes to regulation of chloride channel activity. <i>Journal of Biological Chemistry</i> , 1996 , 271, 27493-9	5.4	80
18	Mutational analysis of the predicted first transmembrane segment of each homologous half of human P-glycoprotein suggests that they are symmetrically arranged in the membrane. <i>Journal of Biological Chemistry</i> , 1996 , 271, 15414-9	5.4	29
17	Expression of a functionally active human renal sodium-calcium exchanger lacking a signal sequence. <i>Journal of Biological Chemistry</i> , 1995 , 270, 19345-50	5.4	20
16	Membrane topology of a cysteine-less mutant of human P-glycoprotein. <i>Journal of Biological Chemistry</i> , 1995 , 270, 843-8	5.4	207
15	Covalent modification of human P-glycoprotein mutants containing a single cysteine in either nucleotide-binding fold abolishes drug-stimulated ATPase activity. <i>Journal of Biological Chemistry</i> , 1995 , 270, 22957-61	5.4	130
14	P-glycoprotein. Associations between domains and between domains and molecular chaperones. <i>Journal of Biological Chemistry</i> , 1995 , 270, 21839-44	5.4	103
13	Rapid purification of human P-glycoprotein mutants expressed transiently in HEK 293 cells by nickel-chelate chromatography and characterization of their drug-stimulated ATPase activities. <i>Journal of Biological Chemistry</i> , 1995 , 270, 21449-52	5.4	150
12	Mutations to amino acids located in predicted transmembrane segment 6 (TM6) modulate the activity and substrate specificity of human P-glycoprotein. <i>Biochemistry</i> , 1994 , 33, 14049-57	3.2	122
11	Deletion of NH ₂ - and COOH-terminal sequences destroys function of the Ca ²⁺ ATPase of rabbit fast-twitch skeletal muscle sarcoplasmic reticulum. <i>FEBS Letters</i> , 1993 , 336, 168-70	3.8	15
10	Expression and mutation of Ca ²⁺ ATPases of the sarcoplasmic reticulum. <i>Cytoskeleton</i> , 1989 , 14, 26-34		17
9	Location of high affinity Ca ²⁺ -binding sites within the predicted transmembrane domain of the sarcoplasmic reticulum Ca ²⁺ -ATPase. <i>Nature</i> , 1989 , 339, 476-8	5.4	57.0
8	Expression of rubella virus cDNA coding for the structural proteins. <i>Gene</i> , 1988 , 65, 23-30	3.8	27

7	Nucleotide sequence and in vitro expression of rubella virus 24S subgenomic messenger RNA encoding the structural proteins E1, E2 and C. <i>Nucleic Acids Research</i> , 1987 , 15, 3041-57	20.1	95
6	Nucleotide sequence of the pntA and pntB genes encoding the pyridine nucleotide transhydrogenase of Escherichia coli. <i>FEBS Journal</i> , 1986 , 158, 647-53		91
5	Detection of antibodies to individual proteins of rubella virus. <i>Journal of Virological Methods</i> , 1986 , 13, 149-59	2.6	15
4	Structural analysis of a new GC-specific insertion element IS186. <i>FEBS Letters</i> , 1985 , 192, 47-52	3.8	21
3	Interaction of Escherichia coli F1-ATPase with dicyclohexylcarbodiimide-binding polypeptide. <i>Biochimica Et Biophysica Acta - Biomembranes</i> , 1983 , 733, 274-82	3.8	17
2	The DCCD-binding polypeptide is close to the F1 ATPase-binding site on the cytoplasmic surface of the cell membrane of Escherichia coli. <i>Biochemical and Biophysical Research Communications</i> , 1982 , 106, 400-6	3.4	23
1	The DCCD-binding polypeptide alone is insufficient for proton translocation through F0 in membranes of Escherichia coli. <i>Biochemical and Biophysical Research Communications</i> , 1981 , 103, 52-9	3.4	25