

Gary Yellen

List of Publications by Year in descending order

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Version: 2024-02-01

82
papers

12,614
citations

41344

49
h-index

62596

80
g-index

118
all docs

118
docs citations

118
times ranked

9349
citing authors

#	ARTICLE	IF	CITATIONS
1	Neurophotonic Tools for Microscopic Measurements and Manipulation: Status Report. <i>Neurophotonics</i> , 2022, 9, 013001.	3.3	17
2	A high-throughput multiparameter screen for accelerated development and optimization of soluble genetically encoded fluorescent biosensors. <i>Nature Communications</i> , 2022, 13, .	12.8	39
3	The distinct roles of calcium in rapid control of neuronal glycolysis and the tricarboxylic acid cycle. <i>ELife</i> , 2021, 10, .	6.0	51
4	Metabolism-based therapies for epilepsy: new directions for future cures. <i>Annals of Clinical and Translational Neurology</i> , 2021, 8, 1730-1737.	3.7	6
5	Delivery of AAV for Expression of Fluorescent Biosensors in Juvenile Mouse Hippocampus. <i>Bio-protocol</i> , 2021, 11, e4259.	0.4	3
6	Hepatic NADH reductive stress underlies common variation in metabolic traits. <i>Nature</i> , 2020, 583, 122-126.	27.8	108
7	Fluorescent Biosensors for Neuronal Metabolism and the Challenges of Quantitation. <i>Current Opinion in Neurobiology</i> , 2020, 63, 111-121.	4.2	32
8	Quantitative <i>in vivo</i> imaging of neuronal glucose concentrations with a genetically encoded fluorescence lifetime sensor. <i>Journal of Neuroscience Research</i> , 2019, 97, 946-960.	2.9	67
9	Neurons rely on glucose rather than astrocytic lactate during stimulation. <i>Journal of Neuroscience Research</i> , 2019, 97, 883-889.	2.9	90
10	Live cell imaging of cytosolic NADH/NAD ⁺ ratio in hepatocytes and liver slices. <i>American Journal of Physiology - Renal Physiology</i> , 2018, 314, G97-G108.	3.4	20
11	<i>BAD</i> knockout provides metabolic seizure resistance in a genetic model of epilepsy with sudden unexplained death in epilepsy. <i>Epilepsia</i> , 2018, 59, e1-e4.	5.1	14
12	<i>BAD</i> and <i>KATP</i> channels regulate neuron excitability and epileptiform activity. <i>ELife</i> , 2018, 7, .	6.0	35
13	Fueling thought: Management of glycolysis and oxidative phosphorylation in neuronal metabolism. <i>Journal of Cell Biology</i> , 2018, 217, 2235-2246.	5.2	248
14	Neuronal Stimulation Triggers Neuronal Glycolysis and Not Lactate Uptake. <i>Cell Metabolism</i> , 2017, 26, 361-374.e4.	16.2	327
15	<i>Akt</i> regulation of glycolysis mediates bioenergetic stability in epithelial cells. <i>ELife</i> , 2017, 6, .	6.0	55
16	Cytosolic NADH/NAD ⁺ Redox Visualized in Brain Slices by Two-Photon Fluorescence Lifetime Biosensor Imaging. <i>Antioxidants and Redox Signaling</i> , 2016, 25, 553-563.	5.4	77
17	The leak channel <i>NALCN</i> controls tonic firing and glycolytic sensitivity of substantia nigra pars reticulata neurons. <i>ELife</i> , 2016, 5, .	6.0	63
18	Variants in <i>KCNJ11</i> and <i>BAD</i> do not predict response to ketogenic dietary therapies for epilepsy. <i>Epilepsy Research</i> , 2015, 118, 22-28.	1.6	6

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19	The inward rectifier potassium channel Kir2.1 is expressed in mouse neutrophils from bone marrow and liver. <i>American Journal of Physiology - Cell Physiology</i> , 2015, 308, C264-C276.	4.6	36
20	Quantitative two-photon imaging of fluorescent biosensors. <i>Current Opinion in Chemical Biology</i> , 2015, 27, 24-30.	6.1	69
21	Quantitative determinants of aerobic glycolysis identify flux through the enzyme GAPDH as a limiting step. <i>ELife</i> , 2014, 3, .	6.0	222
22	A PKA activity sensor for quantitative analysis of endogenous GPCR signaling via 2-photon FRET-FLIM imaging. <i>Frontiers in Pharmacology</i> , 2014, 5, 56.	3.5	76
23	Metabolism Regulates the Spontaneous Firing of Substantia Nigra Pars Reticulata Neurons via K_{ATP} and Nonselective Cation Channels. <i>Journal of Neuroscience</i> , 2014, 34, 16336-16347.	3.6	49
24	Imaging Changes in the Cytosolic ATP-to-ADP Ratio. <i>Methods in Enzymology</i> , 2014, 547, 355-371.	1.0	23
25	Live-Cell Imaging of Cytosolic NADH/NAD ⁺ Redox State Using a Genetically Encoded Fluorescent Biosensor. <i>Methods in Molecular Biology</i> , 2014, 1071, 83-95.	0.9	47
26	Imaging energy status in live cells with a fluorescent biosensor of the intracellular ATP-to-ADP ratio. <i>Nature Communications</i> , 2013, 4, 2550.	12.8	364
27	The ketogenic diet: metabolic influences on brain excitability and epilepsy. <i>Trends in Neurosciences</i> , 2013, 36, 32-40.	8.6	271
28	Native Currents in Hepatocytes with Characteristic Properties of Kir2 Channels. <i>Biophysical Journal</i> , 2013, 104, 129a.	0.5	0
29	Charge movement in gating-locked HCN channels reveals weak coupling of voltage sensors and gate. <i>Journal of General Physiology</i> , 2012, 140, 469-479.	1.9	30
30	Structural changes during HCN channel gating defined by high affinity metal bridges. <i>Journal of General Physiology</i> , 2012, 140, 279-291.	1.9	36
31	BAD-Dependent Regulation of Fuel Metabolism and KATP Channel Activity Confers Resistance to Epileptic Seizures. <i>Neuron</i> , 2012, 74, 719-730.	8.1	145
32	Optogenetic reporters. <i>Progress in Brain Research</i> , 2012, 196, 235-263.	1.4	54
33	Imaging Cytosolic NADH/NAD ⁺ Redox State with a Genetically Encoded Fluorescent Biosensor. <i>Cell Metabolism</i> , 2011, 14, 545-554.	16.2	431
34	Imaging Intracellular pH in Live Cells with a Genetically Encoded Red Fluorescent Protein Sensor. <i>Journal of the American Chemical Society</i> , 2011, 133, 10034-10037.	13.7	375
35	Single KATP Channel Opening in Response to Action Potential Firing in Mouse Dentate Granule Neurons. <i>Journal of Neuroscience</i> , 2011, 31, 8689-8696.	3.6	133
36	A genetically encoded fluorescent reporter of ATP:ADP ratio. <i>Nature Methods</i> , 2009, 6, 161-166.	19.0	416

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37	Ketone bodies, glycolysis, and K ⁺ ATP channels in the mechanism of the ketogenic diet. <i>Epilepsia</i> , 2008, 49, 80-82.	5.1	89
38	Ketogenic Diet Metabolites Reduce Firing in Central Neurons by Opening KATP Channels. <i>Journal of Neuroscience</i> , 2007, 27, 3618-3625.	3.6	261
39	Reversal of HCN Channel Voltage Dependence via Bridging of the S4-S5 Linker and Post-S6. <i>Journal of General Physiology</i> , 2006, 128, 273-282.	1.9	56
40	Distinct Populations of HCN Pacemaker Channels Produce Voltage-dependent and Voltage-independent Currents. <i>Journal of General Physiology</i> , 2006, 127, 183-190.	1.9	47
41	Cooperative Gating between Single HCN Pacemaker Channels. <i>Journal of General Physiology</i> , 2006, 128, 561-567.	1.9	41
42	Status of the Intracellular Gate in the Activated-not-open State of Shaker K ⁺ Channels. <i>Journal of General Physiology</i> , 2005, 126, 419-428.	1.9	57
43	Intracellular gate opening in Shaker K ⁺ channels defined by high-affinity metal bridges. <i>Nature</i> , 2004, 428, 864-868.	27.8	203
44	Inactivation in HCN Channels Results from Reclosure of the Activation Gate. <i>Neuron</i> , 2004, 41, 737-744.	8.1	110
45	Movements near the Gate of a Hyperpolarization-activated Cation Channel. <i>Journal of General Physiology</i> , 2003, 122, 501-510.	1.9	47
46	Fast and Slow Voltage Sensor Movements in HERG Potassium Channels. <i>Journal of General Physiology</i> , 2002, 119, 275-293.	1.9	107
47	Voltage-Controlled Gating at the Intracellular Entrance to a Hyperpolarization-Activated Cation Channel. <i>Journal of General Physiology</i> , 2002, 119, 83-91.	1.9	98
48	The voltage-gated potassium channels and their relatives. <i>Nature</i> , 2002, 419, 35-42.	27.8	630
49	Dimers among friends: ion channel regulation by dimerization of tail domains. <i>Trends in Pharmacological Sciences</i> , 2001, 22, 439-441.	8.7	7
50	Tight Steric Closure at the Intracellular Activation Gate of a Voltage-Gated K ⁺ Channel. <i>Neuron</i> , 2001, 32, 649-656.	8.1	257
51	Keeping K ⁺ completely comfortable. <i>Nature Structural Biology</i> , 2001, 8, 1011-1013.	9.7	15
52	Blocker State Dependence and Trapping in Hyperpolarization-Activated Cation Channels. <i>Journal of General Physiology</i> , 2001, 117, 91-102.	1.9	162
53	Blocker protection in the pore of a voltage-gated K ⁺ channel and its structural implications. <i>Nature</i> , 2000, 403, 321-325.	27.8	342
54	The bacterial K ⁺ channel structure and its implications for neuronal channels. <i>Current Opinion in Neurobiology</i> , 1999, 9, 267-273.	4.2	25

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55	Premonitions of ion channel gating. <i>Nature Structural Biology</i> , 1998, 5, 421-421.	9.7	6
56	The Activation Gate of a Voltage-Gated K ⁺ Channel Can Be Trapped in the Open State by an Intersubunit Metal Bridge. <i>Neuron</i> , 1998, 21, 617-621.	8.1	194
57	The moving parts of voltage-gated ion channels. <i>Quarterly Reviews of Biophysics</i> , 1998, 31, 239-295.	5.7	434
58	Single Channel Seeks Permeant Ion for Brief but Intimate Relationship. <i>Journal of General Physiology</i> , 1997, 110, 83-85.	1.9	21
59	Trapping of Organic Blockers by Closing of Voltage-dependent K ⁺ Channels. <i>Journal of General Physiology</i> , 1997, 109, 527-535.	1.9	206
60	Defective "pacemaker" current (I _h) in a zebrafish mutant with a slow heart rate. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 1997, 94, 4554-4559.	7.1	215
61	Gated Access to the Pore of a Voltage-Dependent K ⁺ Channel. <i>Neuron</i> , 1997, 19, 175-184.	8.1	475
62	On the Use of Thiol-modifying Agents to Determine Channel Topology. <i>Neuropharmacology</i> , 1996, 35, 797-804.	4.1	218
63	Dynamic Rearrangement of the Outer Mouth of a K ⁺ Channel during Gating. <i>Neuron</i> , 1996, 16, 859-867.	8.1	433
64	Use-Dependent Blockers and Exit Rate of the Last Ion from the Multi-Ion Pore of a K ⁺ Channel. <i>Science</i> , 1996, 271, 653-656.	12.6	217
65	Two functionally distinct subsites for the binding of internal blockers to the pore of voltage-activated K ⁺ channels. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 1996, 93, 13357-13361.	7.1	57
66	Alternative mechanism for pathogenesis of an inherited epilepsy by a nicotinic AChR mutation. <i>Nature Genetics</i> , 1996, 13, 396-397.	21.4	14
67	The inward rectification mechanism of the HERG cardiac potassium channel. <i>Nature</i> , 1996, 379, 833-836.	27.8	736
68	N-type inactivation and the S4-S5 region of the Shaker K ⁺ channel. <i>Journal of General Physiology</i> , 1996, 108, 195-206.	1.9	99
69	Modulation of K ⁺ current by frequency and external [K ⁺]: A tale of two inactivation mechanisms. <i>Neuron</i> , 1995, 15, 951-960.	8.1	362
70	Structure and selectivity. <i>Nature</i> , 1993, 366, 109-110.	27.8	17
71	The internal quaternary ammonium receptor site of Shaker potassium channels. <i>Neuron</i> , 1993, 10, 533-541.	8.1	258
72	A novel K ⁺ channel with unique localizations in mammalian brain: Molecular cloning and characterization. <i>Neuron</i> , 1992, 8, 473-481.	8.1	122

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73	Ion effects on gating of the Ca(2+)-activated K+ channel correlate with occupancy of the pore. Biophysical Journal, 1992, 61, 639-648.	0.5	88
74	The inactivation gate of the Shaker K+ channel behaves like an open-channel blocker. Neuron, 1991, 7, 743-753.	8.1	293
75	Tetraethylammonium blockade distinguishes two inactivation mechanisms in voltage-activated K+ channels.. Proceedings of the National Academy of Sciences of the United States of America, 1991, 88, 5092-5095.	7.1	423
76	Expression of Torpedo nicotinic acetylcholine receptor subunits in yeast is enhanced by use of yeast signal sequences. Gene, 1990, 86, 145-152.	2.2	13
77	Ionic permeation and blockade in Ca2+-activated K+ channels of bovine chromaffin cells.. Journal of General Physiology, 1984, 84, 157-186.	1.9	484
78	Relief of Na+ block of Ca2+-activated K+ channels by external cations.. Journal of General Physiology, 1984, 84, 187-199.	1.9	135
79	The immune system uses ion channels, too. Trends in Neurosciences, 1984, 7, 179-181.	8.6	5
80	Channels from genes: The oocyte as an expression system. Trends in Neurosciences, 1984, 7, 457-458.	8.6	17
81	Analysis of Nonstationary Channel Kinetics. , 1983, , 287-299.		17
82	Single Ca2+-activated nonselective cation channels in neuroblastoma. Nature, 1982, 296, 357-359.	27.8	450