

Ole Holger Petersen

List of Publications by Year in descending order

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Version: 2024-02-01

111
papers

13,991
citations

36271

51
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29127

104
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113
all docs

113
docs citations

113
times ranked

12059
citing authors

#	ARTICLE	IF	CITATIONS
1	Is CD38 involved in Ca ²⁺ signalling elicited by activation of T cell receptors?. Cell Calcium, 2022, 101, 102524.	1.1	4
2	Recurrent acute pancreatitis prevention by the elimination of alcohol and cigarette smoking (REAPPEAR): protocol of a randomised controlled trial and a cohort study. BMJ Open, 2022, 12, e050821.	0.8	8
3	Do We Need a Different Debate About How to Manage Pandemics?. Function, 2022, 3, zqab075.	1.1	0
4	SARS-CoV-2 S Protein Subunit 1 Elicits Ca ²⁺ Influx-Dependent Ca ²⁺ Signals in Pancreatic Stellate Cells and Macrophages <i>In Situ</i> . Function, 2022, 3, zqac002.	1.1	16
5	Editorial Statement. Function, 2022, 3, zqac014.	1.1	0
6	Electrophysiology of Exocrine Gland Cells. Bioelectricity, 2022, 4, 48-58.	0.6	1
7	Ups and Downs of Science during a Tumultuous Period of History: A Personal Perspective. European Review, 2022, 30, 591-626.	0.4	5
8	Bradykinin, COVID-19, and Pancreatitis, a Personal Perspective. Function, 2021, 2, zqab046.	1.1	1
9	When a Discovery Is a Rediscovery: Do We Know the History of Our Own Subject?. Function, 2021, 2, zqab030.	1.1	6
10	Kafka and Asking the Right Question at the Right Time. Function, 2021, 2, zqab013.	1.1	0
11	Different Effects of Alcohol on the Liver and the Pancreas. Function, 2021, 2, zqab008.	1.1	4
12	FUNCTION Is One Year Old: How Did We Do?. Function, 2021, 2, zqab023.	1.1	0
13	The roles of calcium and ATP in the physiology and pathology of the exocrine pancreas. Physiological Reviews, 2021, 101, 1691-1744.	13.1	69
14	Inequality of Research Funding between Different Countries and Regions is a Serious Problem for Global Science. Function, 2021, 2, zqab060.	1.1	7
15	Early Elimination of Fatty Acids in hypertriglyceridemia-induced acute pancreatitis (ELEFANT trial): Protocol of an open-label, multicenter, adaptive randomized clinical trial. Pancreatology, 2020, 20, 369-376.	0.5	27
16	FUNCTION is now functional. Function, 2020, 1, zqaa001.	1.1	2
17	The ARRIVE guidelines 2.0: Updated guidelines for reporting animal research. PLoS Biology, 2020, 18, e3000410.	2.6	2,209
18	Reporting animal research: Explanation and elaboration for the ARRIVE guidelines 2.0. PLoS Biology, 2020, 18, e3000411.	2.6	1,069

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19	The ARRIVE guidelines 2.0: updated guidelines for reporting animal research. <i>Journal of Physiology</i> , 2020, 598, 3793-3801.	1.3	177
20	The ARRIVE guidelines 2.0: Updated guidelines for reporting animal research. <i>Experimental Physiology</i> , 2020, 105, 1459-1466.	0.9	1,300
21	Endocytic uptake of SARS-CoV-2: the critical roles of pH, Ca ²⁺ , and NAADP. <i>Function</i> , 2020, 1, .	1.1	30
22	Calcium Signaling in Pancreatic Immune Cells<i>In situ</i>. <i>Function</i> , 2020, 2, zqaa026.	1.1	14
23	Science and Scientific Advice in a Time of Crisis. <i>Function</i> , 2020, 1, zqaa025.	1.1	0
24	In Memoriam Sir Michael Berridge 1938 – 2020. <i>Cell Calcium</i> , 2020, 88, 102209.	1.1	2
25	Academia Europaea Position Paper on Translational Medicine: The Cycle Model for Translating Scientific Results into Community Benefits. <i>Journal of Clinical Medicine</i> , 2020, 9, 1532.	1.0	50
26	Acid Tests and the Hope for Adequate Oxygen Intake in 2021. <i>Function</i> , 2020, 2, zqaa035.	1.1	0
27	One or Two Ca ²⁺ Stores in the Neuronal Endoplasmic Reticulum?. <i>Trends in Neurosciences</i> , 2019, 42, 755-757.	4.2	2
28	Reproducibility – again. <i>Journal of Physiology</i> , 2019, 597, 657-658.	1.3	3
29	ABT-199 (Venetoclax), a BH3-mimetic Bcl-2 inhibitor, does not cause Ca ²⁺ signalling dysregulation or toxicity in pancreatic acinar cells. <i>British Journal of Pharmacology</i> , 2019, 176, 4402-4415.	2.7	18
30	Calcium signalling in the acinar environment of the exocrine pancreas: physiology and pathophysiology. <i>Journal of Physiology</i> , 2018, 596, 2663-2678.	1.3	40
31	Revision of the ARRIVE guidelines: rationale and scope. <i>BMJ Open Science</i> , 2018, 2, e000002.	0.8	36
32	BH4 domain peptides derived from Bcl-2/Bcl-XL as novel tools against acute pancreatitis. <i>Cell Death Discovery</i> , 2018, 4, 58.	2.0	9
33	Galactose protects against cell damage in mouse models of acute pancreatitis. <i>Journal of Clinical Investigation</i> , 2018, 128, 3769-3778.	3.9	31
34	The effects of Ca ²⁺ buffers on cytosolic Ca ²⁺ signalling. <i>Journal of Physiology</i> , 2017, 595, 3107-3108.	1.3	3
35	Ca ²⁺ tunnelling through the ER lumen as a mechanism for delivering Ca ²⁺ entering via store-operated Ca ²⁺ channels to specific target sites. <i>Journal of Physiology</i> , 2017, 595, 2999-3014.	1.3	48
36	BH3 mimetic-elicited Ca ²⁺ signals in pancreatic acinar cells are dependent on Bax and can be reduced by Ca ²⁺ -like peptides. <i>Cell Death and Disease</i> , 2017, 8, e2640-e2640.	2.7	9

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37	Caffeine protects against experimental acute pancreatitis by inhibition of inositol 1,4,5-trisphosphate receptor-mediated Ca ²⁺ release. <i>Gut</i> , 2017, 66, 301-313.	6.1	74
38	High versus low energy administration in the early phase of acute pancreatitis (GOULASH trial): protocol of a multicentre randomised double-blind clinical trial. <i>BMJ Open</i> , 2017, 7, e015874.	0.8	30
39	Calcium and ATP control multiple vital functions. <i>Philosophical Transactions of the Royal Society B: Biological Sciences</i> , 2016, 371, 20150418.	1.8	39
40	Calcium and adenosine triphosphate control of cellular pathology: asparaginase-induced pancreatitis elicited via protease-activated receptor 2. <i>Philosophical Transactions of the Royal Society B: Biological Sciences</i> , 2016, 371, 20150423.	1.8	33
41	Ca ²⁺ signals mediated by bradykinin type 2 receptors in normal pancreatic stellate cells can be inhibited by specific Ca ²⁺ channel blockade. <i>Journal of Physiology</i> , 2016, 594, 281-293.	1.3	53
42	Nitric oxide signals are interlinked with calcium signals in normal pancreatic stellate cells upon oxidative stress and inflammation. <i>Open Biology</i> , 2016, 6, 160149.	1.5	41
43	Bile acids induce necrosis in pancreatic stellate cells dependent on calcium entry and sodium-driven bile uptake. <i>Journal of Physiology</i> , 2016, 594, 6147-6164.	1.3	38
44	Calcium signalling in pancreatic stellate cells: Mechanisms and potential roles. <i>Cell Calcium</i> , 2016, 59, 140-144.	1.1	22
45	Mechanism of mitochondrial permeability transition pore induction and damage in the pancreas: inhibition prevents acute pancreatitis by protecting production of ATP. <i>Gut</i> , 2016, 65, 1333-1346.	6.1	159
46	Ca ²⁺ signalling in the endoplasmic reticulum/secretory granule microdomain. <i>Cell Calcium</i> , 2015, 58, 397-404.	1.1	19
47	Both RyRs and TPCs are required for NAADP-induced intracellular Ca ²⁺ release. <i>Cell Calcium</i> , 2015, 58, 237-245.	1.1	50
48	Fatty acid ethyl ester synthase inhibition ameliorates ethanol-induced Ca ²⁺ -dependent mitochondrial dysfunction and acute pancreatitis. <i>Gut</i> , 2014, 63, 1313-1324.	6.1	135
49	Can specific calcium channel blockade be the basis for a drug-based treatment of acute pancreatitis?. <i>Expert Review of Gastroenterology and Hepatology</i> , 2014, 8, 339-341.	1.4	4
50	The role of Ca ²⁺ in the pathophysiology of pancreatitis. <i>Journal of Physiology</i> , 2014, 592, 269-280.	1.3	116
51	Monitoring of intracellular free Ca ²⁺ . <i>Environmental Sciences Europe</i> , 2014, 3, 63-71.	2.6	6
52	Ca ²⁺ release-activated Ca ²⁺ channel blockade as a potential tool in antipancreatitis therapy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2013, 110, 13186-13191.	3.3	154
53	The Exocrine Pancreas: The Acinar-Ductal Tango in Physiology and Pathophysiology. <i>Reviews of Physiology, Biochemistry and Pharmacology</i> , 2013, 165, 1-30.	0.9	97
54	A Novel Role for Bcl-2 in Regulation of Cellular Calcium Extrusion. <i>Current Biology</i> , 2012, 22, 1241-1246.	1.8	37

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55	Specific mitochondrial functions in separate sub-cellular domains of pancreatic acinar cells. Pflugers Archiv European Journal of Physiology, 2012, 464, 77-87.	1.3	22
56	A Special Issue on the cell-specific roles of mitochondrial Ca ²⁺ handling. Pflugers Archiv European Journal of Physiology, 2012, 464, 1-2.	1.3	0
57	Reactive Oxygen Species Induced by Bile Acid Induce Apoptosis and Protect Against Necrosis in Pancreatic Acinar Cells. Gastroenterology, 2011, 140, 2116-2125.	0.6	157
58	Calmodulin protects against alcohol-induced pancreatic trypsinogen activation elicited via Ca ²⁺ release through IP ₃ receptors. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 5873-5878.	3.3	47
59	Pathobiology of acute pancreatitis: focus on intracellular calcium and calmodulin. F1000 Medicine Reports, 2011, 3, 15.	2.9	21
60	Bernd Nilius: The Bard of ion channels. Congratulations on 65th birthday. Pflugers Archiv European Journal of Physiology, 2010, 460, 691-694.	1.3	1
61	Dynamic Changes in Cytosolic and Mitochondrial ATP Levels in Pancreatic Acinar Cells. Gastroenterology, 2010, 138, 1976-1987.e5.	0.6	120
62	The International Union of Physiological Sciences. IUPS Editorial VIII. Physiology, 2009, 24, 320-321.	1.6	1
63	Pancreatic protease activation by alcohol metabolite depends on Ca ²⁺ release via acid store IP ₃ receptors. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 10758-10763.	3.3	97
64	Cholecystokinin-58 and cholecystokinin-8 exhibit similar actions on calcium signaling, zymogen secretion, and cell fate in murine pancreatic acinar cells. American Journal of Physiology - Renal Physiology, 2009, 297, G1085-G1092.	1.6	30
65	Calcium Elevation in Mitochondria Is the Main Ca ²⁺ Requirement for Mitochondrial Permeability Transition Pore (mPTP) Opening. Journal of Biological Chemistry, 2009, 284, 20796-20803.	1.6	217
66	Ribosome-free Terminals of Rough ER Allow Formation of STIM1 Puncta and Segregation of STIM1 from IP ₃ Receptors. Current Biology, 2009, 19, 1648-1653.	1.8	114
67	â€œNow We Have to Use the Skills We Have Developed in Cell Physiological Studies to Attack the Most Crucial Problems in Pancreatic Pathologyâ€™. Pancreatology, 2009, 9, 323-326.	0.5	1
68	Direct Activation of Cytosolic Ca ²⁺ Signaling and Enzyme Secretion by Cholecystokinin in Human Pancreatic Acinar Cells. Gastroenterology, 2008, 135, 632-641.	0.6	139
69	Polarized Calcium Signaling in Exocrine Gland Cells. Annual Review of Physiology, 2008, 70, 273-299.	5.6	266
70	Activation of trypsinogen in large endocytic vacuoles of pancreatic acinar cells. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 5674-5679.	3.3	145
71	Fatty Acid Ethyl Esters Cause Pancreatic Calcium Toxicity via Inositol Trisphosphate Receptors and Loss of ATP Synthesis. Gastroenterology, 2006, 130, 781-793.	0.6	234
72	Ca ²⁺ signalling and pancreatitis: effects of alcohol, bile and coffee. Trends in Pharmacological Sciences, 2006, 27, 113-120.	4.0	138

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73	Calcium-dependent release of NO from intracellular S-nitrosothiols. EMBO Journal, 2006, 25, 3024-3032.	3.5	48
74	From Galvani to patch clamp: the development of electrophysiology. Pflugers Archiv European Journal of Physiology, 2006, 453, 233-247.	1.3	81
75	Generation of Specific Ca ²⁺ Signals from Ca ²⁺ Stores and Endocytosis by Differential Coupling to Messengers. Current Biology, 2006, 16, 1931-1937.	1.8	79
76	NAADP, cADPR and IP ₃ all release Ca ²⁺ from the endoplasmic reticulum and an acidic store in the secretory granule area. Journal of Cell Science, 2006, 119, 226-238.	1.2	149
77	Bile Acids Induce Ca ²⁺ Release from Both the Endoplasmic Reticulum and Acidic Intracellular Calcium Stores through Activation of Inositol Trisphosphate Receptors and Ryanodine Receptors. Journal of Biological Chemistry, 2006, 281, 40154-40163.	1.6	124
78	Intraluminal calcium as a primary regulator of endoplasmic reticulum function. Cell Calcium, 2005, 38, 303-310.	1.1	214
79	Calcium signalling: Past, present and future. Cell Calcium, 2005, 38, 161-169.	1.1	206
80	Ca ²⁺ signalling and Ca ²⁺ -activated ion channels in exocrine acinar cells. Cell Calcium, 2005, 38, 171-200.	1.1	94
81	Bile Acids Induce a Cationic Current, Depolarizing Pancreatic Acinar Cells and Increasing the Intracellular Na ⁺ Concentration. Journal of Biological Chemistry, 2005, 280, 1764-1770.	1.6	39
82	Morphological and functional changes of dissociated single pancreatic acinar cells: testing the suitability of the single cell as a model for exocytosis and calcium signaling. Cell Calcium, 2004, 35, 367-379.	1.1	29
83	Ethanol toxicity in pancreatic acinar cells: Mediation by nonoxidative fatty acid metabolites. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 10738-10743.	3.3	183
84	Non-uniform distribution of mitochondria in pancreatic acinar cells. Cell and Tissue Research, 2003, 313, 37-45.	1.5	49
85	Localization and regulation of Ca ²⁺ entry and exit pathways in exocrine gland cells. Cell Calcium, 2003, 33, 337-344.	1.1	54
86	Long Distance Communication between Muscarinic Receptors and Ca ²⁺ Release Channels Revealed by Carbachol Uncaging in Cell-attached Patch Pipette. Journal of Biological Chemistry, 2003, 278, 20860-20864.	1.6	46
87	NAADP mobilizes Ca ²⁺ from a thapsigargin-sensitive store in the nuclear envelope by activating ryanodine receptors. Journal of Cell Biology, 2003, 163, 271-282.	2.3	209
88	Localized Ca ²⁺ uncaging reveals polarized distribution of Ca ²⁺ -sensitive Ca ²⁺ release sites. Journal of Cell Biology, 2002, 158, 283-292.	2.3	69
89	Cation Channels: Homing in on the Elusive CAN Channels. Current Biology, 2002, 12, R520-R522.	1.8	35
90	Bile acids induce calcium signals in mouse pancreatic acinar cells: implications for bile-induced pancreatic pathology. Journal of Physiology, 2002, 540, 49-55.	1.3	149

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91	Transformation of local Ca ²⁺ spikes to global Ca ²⁺ transients: the combinatorial roles of multiple Ca ²⁺ releasing messengers. <i>EMBO Journal</i> , 2002, 21, 909-919.	3.5	166
92	Menadione-induced apoptosis: roles of cytosolic Ca(2+) elevations and the mitochondrial permeability transition pore. <i>Journal of Cell Science</i> , 2002, 115, 485-97.	1.2	123
93	The endoplasmic reticulum: one continuous or several separate Ca ²⁺ stores?. <i>Trends in Neurosciences</i> , 2001, 24, 271-276.	4.2	151
94	Calcium binding capacity of the cytosol and endoplasmic reticulum of mouse pancreatic acinar cells. <i>Journal of Physiology</i> , 1999, 518, 463-467.	1.3	81
95	Calcium uptake via endocytosis with rapid release from acidifying endosomes. <i>Current Biology</i> , 1998, 8, 1335-1338.	1.8	227
96	Ca ²⁺ Flow via Tunnels in Polarized Cells: Recharging of Apical Ca ²⁺ Stores by Focal Ca ²⁺ Entry through Basal Membrane Patch. <i>Cell</i> , 1997, 88, 49-55.	13.5	268
97	Inositol Trisphosphate and Cyclic ADP-Ribose Mediated Release of Ca ²⁺ from Single Isolated Pancreatic Zymogen Granules. <i>Cell</i> , 1996, 84, 473-480.	13.5	233
98	Short pulses of acetylcholine stimulation induce cytosolic Ca ²⁺ signals that are excluded from the nuclear region in pancreatic acinar cells. <i>Pflugers Archiv European Journal of Physiology</i> , 1996, 432, 1055-1061.	1.3	52
99	Localization of Ca ²⁺ Extrusion Sites in Pancreatic Acinar Cells. <i>Journal of Biological Chemistry</i> , 1996, 271, 7615-7619.	1.6	78
100	Region-specific Activity of the Plasma Membrane Ca ²⁺ Pump and Delayed Activation of Ca ²⁺ Entry Characterize the Polarized, Agonist-evoked Ca ²⁺ Signals in Exocrine Cells. <i>Journal of Biological Chemistry</i> , 1995, 270, 8528-8535.	1.6	62
101	ATP-dependent accumulation and inositol trisphosphate- or cyclic ADP-ribose-mediated release of Ca ²⁺ from the nuclear envelope. <i>Cell</i> , 1995, 80, 439-444.	13.5	367
102	Spatial dynamics of second messengers: IP ₃ and cAMP as long-range and associative messengers. <i>Trends in Neurosciences</i> , 1994, 17, 95-101.	4.2	289
103	Local and global cytosolic Ca ²⁺ oscillations in exocrine cells evoked by agonists and inositol trisphosphate. <i>Cell</i> , 1993, 74, 661-668.	13.5	496
104	Receptor-activated cytoplasmic Ca ²⁺ spiking mediated by inositol trisphosphate is due to Ca ²⁺ -induced Ca ²⁺ release. <i>Cell</i> , 1990, 63, 1025-1032.	13.5	268
105	Pulsatile intracellular calcium release does not depend on fluctuations in inositol trisphosphate concentration. <i>Nature</i> , 1989, 339, 317-320.	13.7	354
106	Human pancreatic acinar cells: Studies of stimulus-secretion coupling. <i>Gastroenterology</i> , 1985, 89, 109-117.	0.6	56
107	THE EFFECT OF Na+AND Cl-REMOVAL AND OF LOOP DIURETICS ON ACETYLCHOLINE-EVOKED MEMBRANE POTENTIAL CHANGES IN MOUSE LACRIMAL ACINAR CELLS. <i>Quarterly Journal of Experimental Physiology (Cambridge, England)</i> , 1985, 70, 437-445.	1.0	34
108	Calcium-activated potassium channels and their role in secretion. <i>Nature</i> , 1984, 307, 693-696.	13.7	685

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109	Control of K ⁺ conductance by cholecystokinin and Ca ²⁺ in single pancreatic acinar cells studied by the patch-clamp technique. <i>Journal of Membrane Biology</i> , 1984, 79, 293-298.	1.0	48
110	Stimulus-excitation coupling in plasma membranes of pancreatic acinar cells. <i>BBA - Biomembranes</i> , 1982, 694, 163-184.	7.9	33
111	The responsibility of scientists in a time of war. <i>Function</i> , 0, , .	1.1	2