## Ole Holger Petersen

List of Publications by Year in descending order

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36271 29127 13,991 111 51 104 citations h-index g-index papers 113 113 113 12059 docs citations times ranked citing authors all docs

#	Article	IF	CITATIONS
1	The ARRIVE guidelines 2.0: Updated guidelines for reporting animal research. PLoS Biology, 2020, 18, e3000410.	2.6	2,209
2	The ARRIVE guidelines 2.0: Updated guidelines for reporting animal research. Experimental Physiology, 2020, 105, 1459-1466.	0.9	1,300
3	Reporting animal research: Explanation and elaboration for the ARRIVE guidelines 2.0. PLoS Biology, 2020, 18, e3000411.	2.6	1,069
4	Calcium-activated potassium channels and their role in secretion. Nature, 1984, 307, 693-696.	13.7	685
5	Local and global cytosolic Ca2+ oscillations in exocrine cells evoked by agonists and inositol trisphosphate. Cell, 1993, 74, 661-668.	13.5	496
6	ATP-dependent accumulation and inositol trisphosphate- or cyclic ADP-ribose-mediated release of Ca2+ from the nuclear envelope. Cell, 1995, 80, 439-444.	13.5	367
7	Pulsatile intracellular calcium release does not depend on fluctuations in inositol trisphosphate concentration. Nature, 1989, 339, 317-320.	13.7	354
8	Spatial dynamics of second messengers: IP3 and cAMP as long-range and associative messengers. Trends in Neurosciences, 1994, 17, 95-101.	4.2	289
9	Receptor-activated cytoplasmic Ca2+ spiking mediated by inositol trisphosphate is due to Ca2+-induced Ca2+ release. Cell, 1990, 63, 1025-1032.	13.5	268
10	Ca2+ Flow via Tunnels in Polarized Cells: Recharging of Apical Ca2+ Stores by Focal Ca2+ Entry through Basal Membrane Patch. Cell, 1997, 88, 49-55.	13.5	268
11	Polarized Calcium Signaling in Exocrine Gland Cells. Annual Review of Physiology, 2008, 70, 273-299.	5.6	266
12	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
13	Inositol Trisphosphate and Cyclic ADP-Ribose–Mediated Release of Ca2+ from Single Isolated Pancreatic Zymogen Granules. Cell, 1996, 84, 473-480.	13.5	233
14	Calcium uptake via endocytosis with rapid release from acidifying endosomes. Current Biology, 1998, 8, 1335-1338.	1.8	227
15	Calcium Elevation in Mitochondria Is the Main Ca2+ Requirement for Mitochondrial Permeability Transition Pore (mPTP) Opening. Journal of Biological Chemistry, 2009, 284, 20796-20803.	1.6	217
16	Intraluminal calcium as a primary regulator of endoplasmic reticulum function. Cell Calcium, 2005, 38, 303-310.	1.1	214
17	NAADP mobilizes Ca2+ from a thapsigargin-sensitive store in the nuclear envelope by activating ryanodine receptors. Journal of Cell Biology, 2003, 163, 271-282.	2.3	209
18	Calcium signalling: Past, present and future. Cell Calcium, 2005, 38, 161-169.	1.1	206

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19	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
20	The ARRIVE guidelines 2.0: updated guidelines for reporting animal research. Journal of Physiology, 2020, 598, 3793-3801.	1.3	177
21	Transformation of local Ca2+ spikes to global Ca2+ transients: the combinatorial roles of multiple Ca2+ releasing messengers. EMBO Journal, 2002, 21, 909-919.	3.5	166
22	Mechanism of mitochondrial permeability transition pore induction and damage in the pancreas: inhibition prevents acute pancreatitis by protecting production of ATP. Gut, 2016, 65, 1333-1346.	6.1	159
23	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
24	Ca <sup>2+</sup> release-activated Ca <sup>2+</sup> channel blockade as a potential tool in antipancreatitis therapy. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 13186-13191.	3.3	154
25	The endoplasmic reticulum: one continuous or several separate Ca2+ stores?. Trends in Neurosciences, 2001, 24, 271-276.	4.2	151
26	Bile acids induce calcium signals in mouse pancreatic acinar cells: implications for bileâ€induced pancreatic pathology. Journal of Physiology, 2002, 540, 49-55.	1.3	149
27	NAADP, cADPR and IP3 all release Ca2+ from the endoplasmic reticulum and an acidic store in the secretory granule area. Journal of Cell Science, 2006, 119, 226-238.	1.2	149
28	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
29	Direct Activation of Cytosolic Ca2+ Signaling and Enzyme Secretion by Cholecystokinin in Human Pancreatic Acinar Cells. Gastroenterology, 2008, 135, 632-641.	0.6	139
30	Ca2+ signalling and pancreatitis: effects of alcohol, bile and coffee. Trends in Pharmacological Sciences, 2006, 27, 113-120.	4.0	138
31	Fatty acid ethyl ester synthase inhibition ameliorates ethanol-induced Ca <sup>2+</sup> -dependent mitochondrial dysfunction and acute pancreatitis. Gut, 2014, 63, 1313-1324.	6.1	135
32	Bile Acids Induce Ca2+ 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
33	Menadione-induced apoptosis: roles of cytosolic Ca(2+) elevations and the mitochondrial permeability transition pore. Journal of Cell Science, 2002, 115, 485-97.	1.2	123
34	Dynamic Changes in Cytosolic and Mitochondrial ATP Levels in Pancreatic Acinar Cells. Gastroenterology, 2010, 138, 1976-1987.e5.	0.6	120
35	The role of Ca <sup>2+</sup> in the pathophysiology of pancreatitis. Journal of Physiology, 2014, 592, 269-280.	1.3	116
36	Ribosome-free Terminals of Rough ER Allow Formation of STIM1 Puncta and Segregation of STIM1 from IP3 Receptors. Current Biology, 2009, 19, 1648-1653.	1.8	114

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37	Pancreatic protease activation by alcohol metabolite depends on Ca <sup>2+</sup> release via acid store IP <sub>3</sub> receptors. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 10758-10763.	3.3	97
38	The Exocrine Pancreas: The Acinar-Ductal Tango in Physiology and Pathophysiology. Reviews of Physiology, Biochemistry and Pharmacology, 2013, 165, 1-30.	0.9	97
39	Ca2+ signalling and Ca2+-activated ion channels in exocrine acinar cells. Cell Calcium, 2005, 38, 171-200.	1.1	94
40	Calcium binding capacity of the cytosol and endoplasmic reticulum of mouse pancreatic acinar cells. Journal of Physiology, 1999, 518, 463-467.	1.3	81
41	From Galvani to patch clamp: the development of electrophysiology. Pflugers Archiv European Journal of Physiology, 2006, 453, 233-247.	1.3	81
42	Generation of Specific Ca2+ Signals from Ca2+ Stores and Endocytosis by Differential Coupling to Messengers. Current Biology, 2006, 16, 1931-1937.	1.8	79
43	Localization of Ca2+ Extrusion Sites in Pancreatic Acinar Cells. Journal of Biological Chemistry, 1996, 271, 7615-7619.	1.6	78
44	Caffeine protects against experimental acute pancreatitis by inhibition of inositol 1,4,5-trisphosphate receptor-mediated Ca <sup>2+</sup> release. Gut, 2017, 66, 301-313.	6.1	74
45	Localized Ca2+ uncaging reveals polarized distribution of Ca2+-sensitive Ca2+ release sites. Journal of Cell Biology, 2002, 158, 283-292.	2.3	69
46	The roles of calcium and ATP in the physiology and pathology of the exocrine pancreas. Physiological Reviews, 2021, 101, 1691-1744.	13.1	69
47	Region-specific Activity of the Plasma Membrane Ca2+Pump and Delayed Activation of Ca2+Entry Characterize the Polarized, Agonist-evoked Ca2+Signals in Exocrine Cells. Journal of Biological Chemistry, 1995, 270, 8528-8535.	1.6	62
48	Human pancreatic acinar cells: Studies of stimulus-secretion coupling. Gastroenterology, 1985, 89, 109-117.	0.6	56
49	Localization and regulation of Ca2+ entry and exit pathways in exocrine gland cells. Cell Calcium, 2003, 33, 337-344.	1.1	54
50	Ca <sup>2+</sup> signals mediated by bradykinin type 2 receptors in normal pancreatic stellate cells can be inhibited by specific Ca <sup>2+</sup> channel blockade. Journal of Physiology, 2016, 594, 281-293.	1.3	53
51	Short pulses of acetylcholine stimulation induce cytosolic Ca2+ signals that are excluded from the nuclear region in pancreatic acinar cells. Pflugers Archiv European Journal of Physiology, 1996, 432, 1055-1061.	1.3	52
52	Both RyRs and TPCs are required for NAADP-induced intracellular Ca2+ release. Cell Calcium, 2015, 58, 237-245.	1.1	50
53	Academia Europaea Position Paper on Translational Medicine: The Cycle Model for Translating Scientific Results into Community Benefits. Journal of Clinical Medicine, 2020, 9, 1532.	1.0	50
54	Non-uniform distribution of mitochondria in pancreatic acinar cells. Cell and Tissue Research, 2003, 313, 37-45.	1.5	49

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55	Control of K+ conductance by cholecystokinin and Ca2+ in single pancreatic acinar cells studied by the patch-clamp technique. Journal of Membrane Biology, 1984, 79, 293-298.	1.0	48
56	Calcium-dependent release of NO from intracellular S-nitrosothiols. EMBO Journal, 2006, 25, 3024-3032.	3.5	48
57	Ca <sup>2+</sup> tunnelling through the ER lumen as a mechanism for delivering Ca <sup>2+</sup> entering via storeâ€operated Ca <sup>2+</sup> channels to specific target sites. Journal of Physiology, 2017, 595, 2999-3014.	1.3	48
58	Calmodulin protects against alcohol-induced pancreatic trypsinogen activation elicited via Ca <sup>2+</sup> release through IP <sub>3</sub> receptors. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 5873-5878.	3.3	47
59	Long Distance Communication between Muscarinic Receptors and Ca2+ Release Channels Revealed by Carbachol Uncaging in Cell-attached Patch Pipette. Journal of Biological Chemistry, 2003, 278, 20860-20864.	1.6	46
60	Nitric oxide signals are interlinked with calcium signals in normal pancreatic stellate cells upon oxidative stress and inflammation. Open Biology, 2016, 6, 160149.	1.5	41
61	Calcium signalling in the acinar environment of the exocrine pancreas: physiology and pathophysiology. Journal of Physiology, 2018, 596, 2663-2678.	1.3	40
62	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
63	Calcium and ATP control multiple vital functions. Philosophical Transactions of the Royal Society B: Biological Sciences, 2016, 371, 20150418.	1.8	39
64	Bile acids induce necrosis in pancreatic stellate cells dependent on calcium entry and sodiumâ€driven bile uptake. Journal of Physiology, 2016, 594, 6147-6164.	1.3	38
65	A Novel Role for Bcl-2 in Regulation of Cellular Calcium Extrusion. Current Biology, 2012, 22, 1241-1246.	1.8	37
66	Revision of the ARRIVE guidelines: rationale and scope. BMJ Open Science, 2018, 2, e000002.	0.8	36
67	Cation Channels: Homing in on the Elusive CAN Channels. Current Biology, 2002, 12, R520-R522.	1.8	35
68	THE EFFECT OF Na+AND Cl-REMOVAL AND OF LOOP DIURETICS ON ACETYLCHOLINE-EVOKED MEMBRANE POTENTIAL CHANGES IN MOUSE LACRIMAL ACINAR CELLS. Quarterly Journal of Experimental Physiology (Cambridge, England), 1985, 70, 437-445.	1.0	34
69	Stimulus-excitation coupling in plasma membranes of pancreatic acinar cells. BBA - Biomembranes, 1982, 694, 163-184.	7.9	33
70	Calcium and adenosine triphosphate control of cellular pathology: asparaginase-induced pancreatitis elicited via protease-activated receptor 2. Philosophical Transactions of the Royal Society B: Biological Sciences, 2016, 371, 20150423.	1.8	33
71	Galactose protects against cell damage in mouse models of acute pancreatitis. Journal of Clinical Investigation, 2018, 128, 3769-3778.	3.9	31
72	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

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73	High versus low energy administration in the early phase of acute pancreatitis (GOULASH trial): protocol of a multicentre randomised double-blind clinical trial. BMJ Open, 2017, 7, e015874.	0.8	30
74	Endocytic uptake of SARS-CoV-2: the critical roles of pH, Ca2+, and NAADP. Function, 2020, 1, .	1.1	30
75	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
76	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
77	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
78	Calcium signalling in pancreatic stellate cells: Mechanisms and potential roles. Cell Calcium, 2016, 59, 140-144.	1.1	22
79	Pathobiology of acute pancreatitis: focus on intracellular calcium and calmodulin. F1000 Medicine Reports, 2011, 3, 15.	2.9	21
80	Ca2+ signalling in the endoplasmic reticulum/secretory granule microdomain. Cell Calcium, 2015, 58, 397-404.	1.1	19
81	ABTâ€199 (Venetoclax), a BH3â€mimetic Bclâ€2 inhibitor, does not cause Ca 2+ â€signalling dysregulation or toxicity in pancreatic acinar cells. British Journal of Pharmacology, 2019, 176, 4402-4415.	2.7	18
82	SARS-CoV-2 S Protein Subunit 1 Elicits Ca2+ Influx – Dependent Ca2+ Signals in Pancreatic Stellate Cells and Macrophages <i>In Situ</i> . Function, 2022, 3, zqac002.	1.1	16
83	Calcium Signaling in Pancreatic Immune Cells <i>In situ</i> . Function, 2020, 2, zqaa026.	1.1	14
84	BH3 mimetic-elicited Ca2+ signals in pancreatic acinar cells are dependent on Bax and can be reduced by Ca2+-like peptides. Cell Death and Disease, 2017, 8, e2640-e2640.	2.7	9
85	BH4 domain peptides derived from Bcl-2/Bcl-XL as novel tools against acute pancreatitis. Cell Death Discovery, 2018, 4, 58.	2.0	9
86	<b>Re</b> current acute pancreatitis prevention by the elimination of alcohol and ciga <b>r</b> ette smoking (REAPPEAR): protocol of a randomised controlled trial and a cohort study. BMJ Open, 2022, 12, e050821.	0.8	8
87	Inequality of Research Funding between Different Countries and Regions is a Serious Problem for Global Science. Function, 2021, 2, zqab060.	1.1	7
88	Monitoring of intraâ€ <scp>ER</scp> free Ca <sup>2+</sup> . Environmental Sciences Europe, 2014, 3, 63-71.	2.6	6
89	When a Discovery Is a Rediscovery: Do We Know the History of Our Own Subject?. Function, 2021, 2, zqab030.	1.1	6
90	Ups and Downs of Science during a Tumultuous Period of History: A Personal Perspective. European Review, 2022, 30, 591-626.	0.4	5

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91	Can specific calcium channel blockade be the basis for a drug-based treatment of acute pancreatitis?. Expert Review of Gastroenterology and Hepatology, 2014, 8, 339-341.	1.4	4
92	Different Effects of Alcohol on the Liver and the Pancreas. Function, 2021, 2, zqab008.	1.1	4
93	Is CD38 involved in Ca2+ signalling elicited by activation of T cell receptors?. Cell Calcium, 2022, 101, 102524.	1.1	4
94	The effects of Ca <sup>2+</sup> buffers on cytosolic Ca <sup>2+</sup> signalling. Journal of Physiology, 2017, 595, 3107-3108.	1.3	3
95	Reproducibility – again. Journal of Physiology, 2019, 597, 657-658.	1.3	3
96	One or Two Ca2+ Stores in the Neuronal Endoplasmic Reticulum?. Trends in Neurosciences, 2019, 42, 755-757.	4.2	2
97	FUNCTION is now functional. Function, 2020, 1, zqaa001.	1.1	2
98	In Memoriam Sir Michael Berridge 1938 – 2020. Cell Calcium, 2020, 88, 102209.	1.1	2
99	The responsibility of scientists in a time of war. Function, 0, , .	1.1	2
100	The International Union of Physiological Sciences. IUPS Editorial VIII. Physiology, 2009, 24, 320-321.	1.6	1
101	â€~Now We Have to Use the Skills We Have Developed in Cell Physiological Studies to Attack the Most Crucial Problems in Pancreatic Pathology'. Pancreatology, 2009, 9, 323-326.	0.5	1
102	Bernd Nilius: The Bard of ion channels. Congratulations on 65th birthday. Pflugers Archiv European Journal of Physiology, 2010, 460, 691-694.	1.3	1
103	Bradykinin, COVID-19, and Pancreatitis, a Personal Perspective. Function, 2021, 2, zqab046.	1.1	1
104	Electrophysiology of Exocrine Gland Cells. Bioelectricity, 2022, 4, 48-58.	0.6	1
105	A Special Issue on the cell-specific roles of mitochondrial Ca2+ handling. Pflugers Archiv European Journal of Physiology, 2012, 464, 1-2.	1.3	0
106	Science and Scientific Advice in a Time of Crisis. Function, 2020, 1, zqaa025.	1.1	0
107	Kafka and Asking the Right Question at the Right Time. Function, 2021, 2, zqab013.	1.1	0
108	FUNCTION Is One Year Old: How Did We Do?. Function, 2021, 2, zqab023.	1.1	0

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109	Acid Tests and the Hope for Adequate Oxygen Intake in 2021. Function, 2020, 2, zqaa035.	1.1	0
110	Do We Need a Different Debate About How to Manage Pandemics?. Function, 2022, 3, 29ab075.	1.1	0
111	Editorial Statement. Function, 2022, 3, zqac014.	1.1	O