Annik Prat

List of Publications by Year in descending order

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Version: 2024-02-01

83 12,011 43 88 papers citations h-index g-index

90 90 90 10947 all docs docs citations times ranked citing authors

#	Article	IF	CITATIONS
1	The Multifaceted Biology of PCSK9. Endocrine Reviews, 2022, 43, 558-582.	20.1	75
2	PCSK9 Contributes to the Cholesterol, Glucose, and Insulin2 Homeostasis in Seminiferous Tubules and Maintenance of Immunotolerance in Testis. Frontiers in Cell and Developmental Biology, 2022, 10, 889972.	3.7	2
3	The loss-of-function PCSK9Q152H variant increases ER chaperones GRP78 and GRP94 and protects against liver injury. Journal of Clinical Investigation, 2021, 131, .	8.2	29
4	Substantial PCSK9 inactivation in \hat{l}^2 -cells does not modify glucose homeostasis or insulin secretion in mice. Biochimica Et Biophysica Acta - Molecular and Cell Biology of Lipids, 2021, 1866, 158968.	2.4	24
5	PCSK9 is not secreted from mature differentiated intestinalÂcells. Journal of Lipid Research, 2021, 62, 100096.	4.2	4
6	PCSK9 regulates the NODAL signaling pathway and cellular proliferation in hiPSCs. Stem Cell Reports, 2021, 16, 2958-2972.	4.8	7
7	Circulating Rather Than Intestinal PCSK9 (Proprotein Convertase Subtilisin Kexin Type 9) Regulates Postprandial Lipemia in Mice. Arteriosclerosis, Thrombosis, and Vascular Biology, 2020, 40, 2084-2094.	2.4	18
8	Circulating PCSK9 is associated with liver biomarkers and hepatic steatosis. Clinical Biochemistry, 2020, 77, 20-25.	1.9	26
9	Proprotein convertase 7 (PCSK7) reduces apoAâ€V levels. FEBS Journal, 2020, 287, 3565-3578.	4.7	13
10	Novel strategies to target proprotein convertase subtilisin kexin 9: beyond monoclonal antibodies. Cardiovascular Research, 2019, 115, 510-518.	3.8	63
11	Pcsk9 knockout exacerbates diet-induced non-alcoholic steatohepatitis, fibrosis and liver injury in mice. JHEP Reports, 2019, 1, 418-429.	4.9	51
12	Transcriptome Analysis Reveals Nonfoamy Rather Than Foamy Plaque Macrophages Are Proinflammatory in Atherosclerotic Murine Models. Circulation Research, 2018, 123, 1127-1142.	4.5	275
13	A single domain antibody against the Cys- and His-rich domain of PCSK9 and evolocumab exhibit different inhibition mechanisms in humanized PCSK9 mice. Biological Chemistry, 2018, 399, 1363-1374.	2.5	10
14	Low-density lipoprotein (LDL)-dependent uptake of Gram-positive lipoteichoic acid and Gram-negative lipopolysaccharide occurs through LDL receptor. Scientific Reports, 2018, 8, 10496.	3.3	47
15	Thrombin activation of protein C requires prior processing by a liver proprotein convertase. Journal of Biological Chemistry, 2017, 292, 10564-10573.	3.4	10
16	Endoplasmic Reticulum Stress and Ca2+ Depletion Differentially Modulate the Sterol Regulatory Protein PCSK9 to Control Lipid Metabolism. Journal of Biological Chemistry, 2017, 292, 1510-1523.	3.4	31
17	The Proprotein Convertases in Hypercholesterolemia and Cardiovascular Diseases: Emphasis on Proprotein Convertase Subtilisin/Kexin 9. Pharmacological Reviews, 2017, 69, 33-52.	16.0	90
18	Association Between Plasma Proprotein Convertase Subtilisin/Kexin Type 9 and the Presence of Metabolic Syndrome in a Predominantly Rural-Based Sub-Saharan African Population. Metabolic Syndrome and Related Disorders, 2017, 15, 423-429.	1.3	13

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19	Pcsk5 is required in the early cranio-cardiac mesoderm for heart development. BMC Developmental Biology, 2017, 17, 6.	2.1	10
20	Estrogen Signals Through Peroxisome Proliferator-Activated Receptorâ [~] γ Coactivator 1α to Reduce Oxidative Damage Associated With Diet-Induced Fatty Liver Disease. Gastroenterology, 2017, 152, 243-256.	1.3	132
21	Proprotein Convertase Subtilisin/Kexin type 9 affects insulin but not lipid metabolism in cystic fibrosis. Clinical and Investigative Medicine, 2017, 40, 59.	0.6	1
22	Differential Expression of PCSK9 Modulates Infection, Inflammation, and Coagulation in a Murine Model of Sepsis. Shock, 2016, 46, 672-680.	2.1	110
23	Proprotein Convertase Subtilisin/Kexin Type 9 (PCSK9) Single Domain Antibodies Are Potent Inhibitors of Low Density Lipoprotein Receptor Degradation. Journal of Biological Chemistry, 2016, 291, 16659-16671.	3.4	28
24	Liver-Specific Inactivation of the Proprotein Convertase FURIN Leads to Increased Hepatocellular Carcinoma Growth. BioMed Research International, 2015, 2015, 1-8.	1.9	15
25	Amyloid Precursor-like Protein 2 and Sortilin Do Not Regulate the PCSK9 Convertase-mediated Low Density Lipoprotein Receptor Degradation but Interact with Each Other. Journal of Biological Chemistry, 2015, 290, 18609-18620.	3.4	47
26	PCSK9 deficiency unmasks a sex- and tissue-specific subcellular distribution of the LDL and VLDL receptors in mice. Journal of Lipid Research, 2015, 56, 2133-2142.	4.2	45
27	Implication of the proprotein convertases in iron homeostasis: Proprotein convertase 7 sheds human transferrin receptor 1 and furin activates hepcidin. Hepatology, 2013, 57, 2514-2524.	7.3	57
28	Beyond LDL-C lowering: Distinct molecular sphingolipids are good indicators of proprotein convertase subtilisin/kexin type 9 (PCSK9) deficiency. Atherosclerosis, 2013, 228, 380-385.	0.8	34
29	Decreased APOE-containing HDL subfractions and cholesterol efflux capacity of serum in mice lacking Pcsk9. Lipids in Health and Disease, 2013, 12, 112.	3.0	22
30	Furin Is the Primary in Vivo Convertase of Angiopoietin-like 3 and Endothelial Lipase in Hepatocytes. Journal of Biological Chemistry, 2013, 288, 26410-26418.	3.4	43
31	Disruption of the expression of the proprotein convertase PC7 reduces BDNF production and affects learning and memory in mice. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 17362-17367.	7.1	74
32	In utero Measurement of Heart Rate in Mouse by Noninvasive M-mode Echocardiography. Journal of Visualized Experiments, 2013, , e50994.	0.3	4
33	Modulation of Protease Activated Receptor 1 Influences Human Metapneumovirus Disease Severity in a Mouse Model. PLoS ONE, 2013, 8, e72529.	2.5	33
34	Proprotein Convertase Subtilisin/Kexin Type 9 (PCSK9) Can Mediate Degradation of the Low Density Lipoprotein Receptor-Related Protein 1 (LRP-1). PLoS ONE, 2013, 8, e64145.	2.5	183
35	Identification and characterization of new gain-of-function mutations in the PCSK9 gene responsible for autosomal dominant hypercholesterolemia. Atherosclerosis, 2012, 223, 394-400.	0.8	92
36	Automated design of ligands to polypharmacological profiles. Nature, 2012, 492, 215-220.	27.8	698

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37	Proprotein Convertase Subtilisin/Kexin Type 9 Deficiency Reduces Melanoma Metastasis in Liver. Neoplasia, 2012, 14, 1122-IN5.	5.3	94
38	The biology and therapeutic targeting of the proprotein convertases. Nature Reviews Drug Discovery, 2012, 11, 367-383.	46.4	647
39	Loss of Endothelial Furin Leads to Cardiac Malformation and Early Postnatal Death. Molecular and Cellular Biology, 2012, 32, 3382-3391.	2.3	43
40	Gene Inactivation of Proprotein Convertase Subtilisin/Kexin Type 9 Reduces Atherosclerosis in Mice. Circulation, 2012, 125, 894-901.	1.6	193
41	Annexin A2 Is a Natural Extrahepatic Inhibitor of the PCSK9-Induced LDL Receptor Degradation. PLoS ONE, 2012, 7, e41865.	2.5	98
42	Quantitative Proteomic Analysis of PCSK9 Gain of Function in Human Hepatic HuH7 Cells. Journal of Proteome Research, 2011, 10, 2011-2026.	3.7	15
43	The LDLR deficient mouse as a model for aortic calcification and quantification by micro-computed tomography. Atherosclerosis, 2011, 219, 455-462.	0.8	54
44	Antigen processing by nardilysin and thimet oligopeptidase generates cytotoxic T cell epitopes. Nature Immunology, 2011, 12, 45-53.	14.5	94
45	Inactivation of endothelial proprotein convertase 5/6 decreases collagen deposition in the cardiovascular system: role of fibroblast autophagy. Journal of Molecular Medicine, 2011, 89, 1103-1111.	3.9	25
46	PCSK9 reduces the protein levels of the LDL receptor in mouse brain during development and after ischemic stroke. Journal of Lipid Research, 2011, 52, 1383-1391.	4.2	77
47	Furin Is the Major Processing Enzyme of the Cardiac-specific Growth Factor Bone Morphogenetic Protein 10. Journal of Biological Chemistry, 2011, 286, 22785-22794.	3.4	52
48	In Vivo Evidence That Furin from Hepatocytes Inactivates PCSK9. Journal of Biological Chemistry, 2011, 286, 4257-4263.	3.4	132
49	A Novel Mouse Model of Alzheimer's Disease with Chronic Estrogen Deficiency Leads to Glial Cell Activation and Hypertrophy. Journal of Aging Research, 2011, 2011, 1-12.	0.9	21
50	Circulating Proprotein Convertase Subtilisin/Kexin 9 (PCSK9) Regulates VLDLR Protein and Triglyceride Accumulation in Visceral Adipose Tissue. Arteriosclerosis, Thrombosis, and Vascular Biology, 2011, 31, 785-791.	2.4	220
51	Latent Transforming Growth Factor \hat{l}^2 -Binding Proteins-2 and -3 Inhibit the Proprotein Convertase 5/6A. Journal of Biological Chemistry, 2011, 286, 29063-29073.	3.4	20
52	PCSK9â€deficient mice exhibit impaired glucose tolerance and pancreatic islet abnormalities. FEBS Letters, 2010, 584, 701-706.	2.8	165
53	Dissection of the Endogenous Cellular Pathways of PCSK9-induced Low Density Lipoprotein Receptor Degradation. Journal of Biological Chemistry, 2009, 284, 28856-28864.	3.4	228
54	Genetic Variation at the Proprotein Convertase Subtilisin/Kexin Type 5 Gene Modulates High-Density Lipoprotein Cholesterol Levels. Circulation: Cardiovascular Genetics, 2009, 2, 467-475.	5.1	33

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55	PCSK9 impedes hepatitis C virus infection <i>in vitro</i> and modulates liver CD81 expression. Hepatology, 2009, 50, 17-24.	7.3	129
56	The proprotein convertase PC5/6 is protective against intestinal tumorigenesis: in vivo mouse model. Molecular Cancer, 2009, 8, 73.	19.2	29
57	Proprotein convertase subtilisin/kexin type 9 (PCSK9): Hepatocyte-specific low-density lipoprotein receptor degradation and critical role in mouse liver regeneration. Hepatology, 2008, 48, 646-654.	7.3	354
58	The activation and physiological functions of the proprotein convertases. International Journal of Biochemistry and Cell Biology, 2008, 40, 1111-1125.	2.8	285
59	The Proprotein Convertase PCSK9 Induces the Degradation of Low Density Lipoprotein Receptor (LDLR) and Its Closest Family Members VLDLR and ApoER2. Journal of Biological Chemistry, 2008, 283, 2363-2372.	3.4	402
60	<i>In vivo</i> functions of the proprotein convertase PC5/6 during mouse development: Gdf11 is a likely substrate. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 5750-5755.	7.1	99
61	VACTERL/caudal regression/Currarino syndrome-like malformations in mice with mutation in the proprotein convertase <i>Pcsk5</i> . Genes and Development, 2008, 22, 1465-1477.	5.9	110
62	The Cellular Trafficking of the Secretory Proprotein Convertase PCSK9 and Its Dependence on the LDLR. Traffic, 2007, 8, 718-732.	2.7	213
63	The proprotein convertases are potential targets in the treatment of dyslipidemia. Journal of Molecular Medicine, 2007, 85, 685-696.	3.9	145
64	Implication of the proprotein convertase NARC-1/PCSK9 in the development of the nervous system. Journal of Neurochemistry, 2006, 98, 838-850.	3.9	99
65	The proprotein convertases and their implication in sterol and/or lipid metabolism. Biological Chemistry, 2006, 387, 871-7.	2.5	88
66	Deletion of the Gene Encoding Proprotein Convertase 5/6 Causes Early Embryonic Lethality in the Mouse. Molecular and Cellular Biology, 2006, 26, 354-361.	2.3	73
67	Miniglucagon (MG)-Generating Endopeptidase, which Processes Glucagon into MG, Is Composed of N-Arginine Dibasic Convertase and Aminopeptidase B. Endocrinology, 2005, 146, 702-712.	2.8	38
68	Statins UpregulatePCSK9, the Gene Encoding the Proprotein Convertase Neural Apoptosis-Regulated Convertase-1 Implicated in Familial Hypercholesterolemia. Arteriosclerosis, Thrombosis, and Vascular Biology, 2004, 24, 1454-1459.	2.4	557
69	NARC-1/PCSK9 and Its Natural Mutants. Journal of Biological Chemistry, 2004, 279, 48865-48875.	3.4	544
70	Nardilysin, A Basic Residues Specific Metallopeptidase That Mediates Cell Migration and Proliferation. Protein and Peptide Letters, 2004, 11, 501-508.	0.9	18
71	Mutations in PCSK9 cause autosomal dominant hypercholesterolemia. Nature Genetics, 2003, 34, 154-156.	21.4	2,532
72	The secretory proprotein convertase neural apoptosis-regulated convertase 1 (NARC-1): Liver regeneration and neuronal differentiation. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 928-933.	7.1	1,012

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73	Down-regulation of alphav/beta3 integrin via misrouting to lysosomes by overexpression of a beta3Lamp1 fusion protein. Biochemical Journal, 2003, 370, 703-711.	3.7	12
74	The metalloendopeptidase nardilysin (NRDc) is potently inhibited by heparin-binding epidermal growth factor-like growth factor (HB-EGF). Biochemical Journal, 2002, 367, 229-238.	3.7	24
75	Precursor convertases in the secretory pathway, cytosol and extracellular milieu. Essays in Biochemistry, 2002, 38, 79-94.	4.7	190
76	N-arginine dibasic convertase is a specific receptor for heparin-binding EGF-like growth factor that mediates cell migration. EMBO Journal, 2001, 20, 3342-3350.	7.8	115
77	Inhibition of Proprotein Convertases Is Associated with Loss of Growth and Tumorigenicity of HT-29 Human Colon Carcinoma Cells. Journal of Biological Chemistry, 2001, 276, 30686-30693.	3.4	156
78	N-arginine dibasic convertase (nardilysin) isoforms are soluble dibasic-specific metalloendopeptidases that localize in the cytoplasm and at the cell surface. Biochemical Journal, 2000, 349, 587.	3.7	29
79	N-arginine dibasic convertase (nardilysin) isoforms are soluble dibasic-specific metalloendopeptidases that localize in the cytoplasm and at the cell surface. Biochemical Journal, 2000, 349, 587-597.	3.7	34
80	The Kex2p Proregion Is Essential for the Biosynthesis of an Active Enzyme and Requires a C-terminal Basic Residue for Its Function. Molecular Biology of the Cell, 2000, 11, 1947-1957.	2.1	26
81	Human and rat testis express two mRNA species encoding variants of NRD convertase, a metalloendopeptidase of the insulinase family. Biochemical Journal, 1997, 327, 773-779.	3.7	27
82	Expression and retinoid modulation of N-arginine dibasic convertase and an aminopeptidase-B in human neuroblastoma cell lines. Journal of Neuro-Oncology, 1997, 31, 99-106.	2.9	15
83	[45] N-arginine dibasic convertase. Methods in Enzymology, 1995, 248, 703-716.	1.0	20