Shengyi Sun

List of Publications by Year
in descending order

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25 papers	1,862 citations	411340 20 h-index	651938 25 g-index
28 all docs	28 docs citations	28 times ranked	3821 citing authors

#	Article	IF	Citations
1	Cellular abundance of sodium phosphate cotransporter SLC20A1/PiT1 and phosphate uptake are controlled post-transcriptionally by ESCRT. Journal of Biological Chemistry, 2022, 298, 101945.	1.6	4
2	Endoplasmic reticulumâ€“associated degradation is required for nephrin maturation and kidney glomerular filtration function. Journal of Clinical Investigation, 2021, 131, .	3.9	21
3	Notch-induced endoplasmic reticulum-associated degradation governs mouse thymocyte Î²â^'selection. ELife, 2021, 10, .	2.8	13
4	Protein Aggregation in the ER: Calm behind the Storm. Cells, 2021, 10, 3337.	1.8	18
5	Protein quality control through endoplasmic reticulum-associated degradation maintains haematopoietic stem cell identity and niche interactions. Nature Cell Biology, 2020, 22, 1162-1169.	4.6	32
6	ER-associated degradation preserves hematopoietic stem cell quiescence and self-renewal by restricting mTOR activity. Blood, 2020, 136, 2975-2986.	0.6	40
7	The orphan nuclear receptor SHP regulates ER stress response by inhibiting XBP1s degradation. Genes and Development, 2019, 33, 1083-1094.	2.7	14
8	Toll-like receptors TLR2 and TLR4 block the replication of pancreatic Î² cells in diet-induced obesity. Nature Immunology, 2019, 20, 677-686.	7.0	48
9	Hepatic Sel1Lâ€Hrd1 ERâ€associated degradation (ERAD) manages FGF21 levels and systemic metabolism via CREBH. EMBO Journal, 2018, 37, .	3.5	55
10	FGF21 Is an Exocrine Pancreas Secretagogue. Cell Metabolism, 2017, 25, 472-480.	7.2	92
11	ER-associated degradation is required for vasopressin prohormone processing and systemic water homeostasis. Journal of Clinical Investigation, 2017, 127, 3897-3912.	3.9	63
12	Epithelial Sel1L is required for the maintenance of intestinal homeostasis. Molecular Biology of the Cell, 2016, 27, 483-490.	0.9	36
13	High-Resolution Metabolomics with Acyl-CoA Profiling Reveals Widespread Remodeling in Response to Diet*. Molecular and Cellular Proteomics, 2015, 14, 1489-1500.	2.5	95
14	IRE1Î± is an endogenous substrate of endoplasmic-reticulum-associated degradation. Nature Cell Biology, 2015, 17, 1546-1555.	4.6	173
15	Sel1L is indispensable for mammalian endoplasmic reticulum-associated degradation, endoplasmic reticulum homeostasis, and survival. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, E582-91.	3.3	148
16	Diet-Induced Alterations in Gut Microflora Contribute to Lethal Pulmonary Damage in TLR2/TLR4-Deficient Mice. Cell Reports, 2014, 8, 137-149.	2.9	43
17	The ER-Associated Degradation Adaptor Protein Sel1L Regulates LPL Secretion and Lipid Metabolism. Cell Metabolism, 2014, 20, 458-470.	7.2	92
18	The ATP-P2X7 Signaling Axis Is Dispensable for Obesity-Associated Inflammasome Activation in Adipose Tissue. Diabetes, 2012, 61, 1471-1478.	0.3	62

#	Article	IF	Citations
19	Activation of Natural Killer T Cells Promotes M2 Macrophage Polarization in Adipose Tissue and Improves Systemic Glucose Tolerance via Interleukin-4 (IL-4)/STAT6 Protein Signaling Axis in Obesity. Journal of Biological Chemistry, 2012, 287, 13561-13571.	1.6	182
20	Short Term High Fat Diet Challenge Promotes Alternative Macrophage Polarization in Adipose Tissue via Natural Killer T Cells and Interleukin-4. Journal of Biological Chemistry, 2012, 287, 24378-24386.	1.6	128
21	Mechanisms of Inflammatory Responses in Obese Adipose Tissue. Annual Review of Nutrition, 2012, 32, 261-286.	4.3	242
22	Emerging Roles for XBP1, a sUPeR Transcription Factor. Gene Expression, 2010, 15, 13-25.	0.5	93
23	A Phos-Tag-Based Approach Reveals the Extent of Physiological Endoplasmic Reticulum Stress. PLoS ONE, 2010, 5, e11621.	1.1	67
24	14-3-3 Protein Regulates Cell Adhesion in the Seminiferous Epithelium of Rat Testes. Endocrinology, 2009, 150, 4713-4723.	1.4	57
25	14-3-3 and its binding partners are regulators of proteinâ€“protein interactions during spermatogenesis. Journal of Endocrinology, 2009, 202, 327-336.	1.2	39