

Safia Costes

List of Publications by Year in descending order

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Version: 2024-02-01

31
papers

9,552
citations

304368

22
h-index

500791

28
g-index

31
all docs

31
docs citations

31
times ranked

21990
citing authors

#	ARTICLE	IF	CITATIONS
1	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). <i>Autophagy</i> , 2016, 12, 1-222.	4.3	4,701
2	Guidelines for the use and interpretation of assays for monitoring autophagy. <i>Autophagy</i> , 2012, 8, 445-544.	4.3	3,122
3	Autophagy defends pancreatic β cells from human islet amyloid polypeptide-induced toxicity. <i>Journal of Clinical Investigation</i> , 2014, 124, 3489-3500.	3.9	188
4	GLP-1 Mediates Antiapoptotic Effect by Phosphorylating Bad through a β -Arrestin 1-mediated ERK1/2 Activation in Pancreatic β -Cells. <i>Journal of Biological Chemistry</i> , 2010, 285, 1989-2002.	1.6	156
5	Human-IAPP disrupts the autophagy/lysosomal pathway in pancreatic β -cells: protective role of p62-positive cytoplasmic inclusions. <i>Cell Death and Differentiation</i> , 2011, 18, 415-426.	5.0	119
6	The CDK4/pRB/E2F1 pathway controls insulin secretion. <i>Nature Cell Biology</i> , 2009, 11, 1017-1023.	4.6	118
7	Extracellularly Regulated Kinases 1/2 (p44/42 Mitogen-Activated Protein Kinases) Phosphorylate Synapsin I and Regulate Insulin Secretion in the MIN6 β -Cell Line and Islets of Langerhans. <i>Endocrinology</i> , 2005, 146, 643-654.	1.4	103
8	β -Cell Dysfunctional ERAD/Ubiquitin/Proteasome System in Type 2 Diabetes Mediated by Islet Amyloid Polypeptide-Induced UCH-L1 Deficiency. <i>Diabetes</i> , 2011, 60, 227-238.	0.3	103
9	β -Cell Failure in Type 2 Diabetes: A Case of Asking Too Much of Too Few?. <i>Diabetes</i> , 2013, 62, 327-335.	0.3	103
10	ERK1/2 Control Phosphorylation and Protein Level of cAMP-Responsive Element-Binding Protein: A Key Role in Glucose-Mediated Pancreatic β -Cell Survival. <i>Diabetes</i> , 2006, 55, 2220-2230.	0.3	89
11	The effect of curcumin on human islet amyloid polypeptide misfolding and toxicity. <i>Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis</i> , 2010, 17, 118-128.	1.4	83
12	Calcium-activated Calpain-2 Is a Mediator of Beta Cell Dysfunction and Apoptosis in Type 2 Diabetes. <i>Journal of Biological Chemistry</i> , 2010, 285, 339-348.	1.6	79
13	Roles and Regulation of the Transcription Factor CREB in Pancreatic β -Cells. <i>Current Molecular Pharmacology</i> , 2011, 4, 187-195.	0.7	72
14	Glucagon Promotes cAMP-response Element-binding Protein Phosphorylation via Activation of ERK1/2 in MIN6 Cell Line and Isolated Islets of Langerhans. <i>Journal of Biological Chemistry</i> , 2004, 279, 20345-20355.	1.6	62
15	Activation of Melatonin Signaling Promotes β -Cell Survival and Function. <i>Molecular Endocrinology</i> , 2015, 29, 682-692.	3.7	62
16	UCHL1 deficiency exacerbates human islet amyloid polypeptide toxicity in β -cells. <i>Autophagy</i> , 2014, 10, 1004-1014.	4.3	54
17	Degradation of cAMP-Responsive Element-Binding Protein by the Ubiquitin-Proteasome Pathway Contributes to Glucotoxicity in β -Cells and Human Pancreatic Islets. <i>Diabetes</i> , 2009, 58, 1105-1115.	0.3	53
18	Cyclin-Dependent Kinase 5 Promotes Pancreatic β -Cell Survival via Fak-Akt Signaling Pathways. <i>Diabetes</i> , 2011, 60, 1186-1197.	0.3	44

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19	Î ² -Arrestin 1 Is Required for PAC1 Receptor-mediated Potentiation of Long-lasting ERK1/2 Activation by Glucose in Pancreatic Î ² -Cells. <i>Journal of Biological Chemistry</i> , 2009, 284, 4332-4342.	1.6	40
20	CHOP Contributes to, But Is Not the Only Mediator of, IAPP Induced Î ² -Cell Apoptosis. <i>Molecular Endocrinology</i> , 2016, 30, 446-454.	3.7	39
21	Insulin-Degrading Enzyme Inhibition, a Novel Therapy for Type 2 Diabetes?. <i>Cell Metabolism</i> , 2014, 20, 201-203.	7.2	25
22	Targeting protein misfolding to protect pancreatic beta-cells in type 2 diabetes. <i>Current Opinion in Pharmacology</i> , 2018, 43, 104-110.	1.7	25
23	Mechanisms of Beta-Cell Apoptosis in Type 2 Diabetes-Prone Situations and Potential Protection by GLP-1-Based Therapies. <i>International Journal of Molecular Sciences</i> , 2021, 22, 5303.	1.8	25
24	ERK1 is dispensable for mouse pancreatic beta cell function but is necessary for glucose-induced full activation of MSK1 and CREB. <i>Diabetologia</i> , 2017, 60, 1999-2010.	2.9	21
25	Cooperative Effects between Protein Kinase A and p44/p42 Mitogen-Activated Protein Kinase to Promote cAMP-Responsive Element Binding Protein Activation after Î ² Cell Stimulation by Glucose and Its Alteration Due to Glucotoxicity. <i>Annals of the New York Academy of Sciences</i> , 2004, 1030, 230-242.	1.8	19
26	Î ² Cellâ€™specific increased expression of calpastatin prevents diabetes induced by islet amyloid polypeptide toxicity. <i>JCI Insight</i> , 2016, 1, e89590.	2.3	17
27	Proteasomal degradation of the histone acetyl transferase p300 contributes to beta-cell injury in a diabetes environment. <i>Cell Death and Disease</i> , 2018, 9, 600.	2.7	16
28	The Glucagon-Miniglucagon Interplay: A New Level in the Metabolic Regulation. <i>Annals of the New York Academy of Sciences</i> , 2006, 1070, 161-166.	1.8	11
29	The nuclear receptor REV-ERBÎ± is implicated in the alteration of Î ² -cell autophagy and survival under diabetogenic conditions. <i>Cell Death and Disease</i> , 2022, 13, 353.	2.7	3
30	Signaling Pathways Involved in Physiopathology of Pancreatic Î ² -Cells. <i>Recent Patents on Endocrine, Metabolic & Immune Drug Discovery</i> , 2007, 1, 180-192.	0.7	0
31	Methods to Study Roles of Î ² -Arrestins in the Regulation of Pancreatic Î ² -Cell Function. <i>Methods in Molecular Biology</i> , 2019, 1957, 345-364.	0.4	0