Jing Yi

List of Publications by Year in descending order

59 papers	3,758 citations	136950 32 h-index	144013 57 g-index
59 all docs	59 docs citations	59 times ranked	6369 citing authors

#	Article	IF	Citations
1	SENP3 loss promotes M2 macrophage polarization and breast cancer progression. Molecular Oncology, 2022, 16, 1026-1044.	4.6	29
2	SENP3 senses oxidative stress to facilitate STING-dependent dendritic cell antitumor function. Molecular Cell, 2021, 81, 940-952.e5.	9.7	48
3	Redox-sensitive enzyme SENP3 mediates vascular remodeling via de-SUMOylation of Î²-catenin and regulation of its stability. EBioMedicine, 2021, 67, 103386.	6.1	25
4	SUMO proteases SENP3 and SENP5 spatiotemporally regulate the kinase activity of Aurora A. Journal of Cell Science, 2021, 134, .	2.0	4
5	Assessment of SENP3-interacting proteins in hepatocytes treated with diethylnitrosamine by BioID assay. Acta Biochimica Et Biophysica Sinica, 2021, 53, 1237-1246.	2.0	5
6	A fine-tuning mechanism underlying self-control for autophagy: deSUMOylation of BECN1 by SENP3. Autophagy, 2020, 16, 975-990.	9.1	49
7	The correlation between plasma total homocysteine level and gestational diabetes mellitus in a Chinese Han population. Scientific Reports, 2020, 10, 18679.	3.3	9
8	P53 suppresses SENP3 phosphorylation to mediate G2 checkpoint. Cell Discovery, 2020, 6, 21.	6.7	15
9	SENP3 in monocytes/macrophages upâ€regulates tissue factor and mediates lipopolysaccharideâ€induced acute lung injury by enhancing JNK phosphorylation. Journal of Cellular and Molecular Medicine, 2020, 24, 5454-5462.	3.6	12
10	SENP3 Suppresses Osteoclastogenesis by De-conjugating SUMO2/3 from IRF8 in Bone Marrow-Derived Monocytes. Cell Reports, 2020, 30, 1951-1963.e4.	6.4	16
11	hCINAP regulates the DNA-damage response and mediates the resistance of acute myelocytic leukemia cells to therapy. Nature Communications, 2019, 10, 3812.	12.8	31
12	SENP1-Sirt3 Signaling Controls Mitochondrial Protein Acetylation and Metabolism. Molecular Cell, 2019, 75, 823-834.e5.	9.7	119
13	Tumor suppressor HIC1 is synergistically compromised by cancer-associated fibroblasts and tumor cells through the IL-6/pSTAT3 axis in breast cancer. BMC Cancer, 2019, 19, 1180.	2.6	20
14	DeSUMOylation of MKK7 kinase by the SUMO2/3 protease SENP3 potentiates lipopolysaccharide-induced inflammatory signaling in macrophages. Journal of Biological Chemistry, 2018, 293, 3965-3980.	3.4	32
15	SENP3 maintains the stability and function of regulatory T cells via BACH2 deSUMOylation. Nature Communications, 2018, 9, 3157.	12.8	87
16	Nucleolar Stress: hallmarks, sensing mechanism and diseases. Cell Stress, 2018, 2, 125-140.	3.2	172
17	A Small-Molecule Inhibitor of Bax and Bak Oligomerization Prevents Genotoxic Cell Death and Promotes Neuroprotection. Cell Chemical Biology, 2017, 24, 493-506.e5.	5.2	76
18	Cleavage of Ku80 by caspase-2 promotes non-homologous end joining-mediated DNA repair. DNA Repair, 2017, 60, 18-28.	2.8	9

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19	A redox mechanism underlying nucleolar stress sensing by nucleophosmin. Nature Communications, 2016, 7, 13599.	12.8	94
20	SENP3 regulates the global protein turnover and the Sp1 level via antagonizing SUMO2/3-targeted ubiquitination and degradation. Protein and Cell, 2016, 7, 63-77.	11.0	21
21	Orphan Nuclear Receptor Nur77 Inhibits Angiotensin IIâ€“Induced Vascular Remodeling via Downregulation of Î²-Catenin. Hypertension, 2016, 67, 153-162.	2.7	51
22	Phenylethyl isothiocyanate reverses cisplatin resistance in biliary tract cancer cells via glutathionylation-dependent degradation of Mcl-1. Oncotarget, 2016, 7, 10271-10282.	1.8	29
23	The orphan nuclear receptor Nur77 inhibits low shear stress-induced carotid artery remodeling in mice. International Journal of Molecular Medicine, 2015, 36, 1547-1555.	4.0	16
24	SoNar, a Highly Responsive NAD+/NADH Sensor, Allows High-Throughput Metabolic Screening of Anti-tumor Agents. Cell Metabolism, 2015, 21, 777-789.	16.2	311
25	Atorvastatin Improves Plaque Stability in ApoE-Knockout Mice by Regulating Chemokines and Chemokine Receptors. PLoS ONE, 2014, 9, e97009.	2.5	53
26	De-SUMOylation of FOXC2 by SENP3 promotes the epithelial-mesenchymal transition in gastric cancer cells. Oncotarget, 2014, 5, 7093-7104.	1.8	55
27	Expressions of farnesoid X receptor and myeloid cell leukemia sequence 1 protein are associated with poor prognosis in patients with gallbladder cancer. Chinese Medical Journal, 2014, 127, 2637-42.	2.3	5
28	Emodin As an Effective Agent in Targeting Cancer Stem-Like Side Population Cells of Gallbladder Carcinoma. Stem Cells and Development, 2013, 22, 554-566.	2.1	59
29	Mechanisms of verapamil-enhanced chemosensitivity of gallbladder cancer cells to platinum drugs: Glutathione reduction and MRP1 downregulation. Oncology Reports, 2013, 29, 676-684.	2.6	32
30	The biphasic redox sensing of SENP3 accounts for the HIF-1 transcriptional activity shift by oxidative stress. Acta Pharmacologica Sinica, 2012, 33, 953-963.	6.1	34
31	Redox Sensing by Proteins: Oxidative Modifications on Cysteines and the Consequent Events. Antioxidants and Redox Signaling, 2012, 16, 649-657.	5.4	156
32	Synthesis and antitumour activity of Î²-hydroxyisovalerylshikonin analogues. European Journal of Medicinal Chemistry, 2011, 46, 3934-3941.	5.5	32
33	SUMO2 and SUMO3 transcription is differentially regulated by oxidative stress in an Sp1-dependent manner. Biochemical Journal, 2011, 435, 489-498.	3.7	30
34	Emodin potentiates the anticancer effect of cisplatin on gallbladder cancer cells through the generation of reactive oxygen species and the inhibition of survivin expression. Oncology Reports, 2011, 26, 1143-8.	2.6	22
35	Emodin enhances sensitivity of gallbladder cancer cells to platinum drugs via glutathion depletion and MRP1 downregulation. Biochemical Pharmacology, 2010, 79, 1134-1140.	4.4	64
36	Redox regulation of the stability of the SUMO protease SENP3 via interactions with CHIP and Hsp90. EMBO Journal, 2010, 29, 3773-3786.	7.8	107

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37	SENP3-mediated De-conjugation of SUMO2/3 from Promyelocytic Leukemia Is Correlated with Accelerated Cell Proliferation under Mild Oxidative Stress. Journal of Biological Chemistry, 2010, 285, 12906-12915.	3.4	92
38	Autografts and Xenografts of Skin Fibroblasts Delivering BMPâ€2 Effectively Promote Orthotopic and Ectopic Osteogenesis. Anatomical Record, 2009, 292, 777-786.	1.4	17
39	Oxidative modification of caspase-9 facilitates its activation via disulfide-mediated interaction with Apaf-1. Cell Research, 2009, 19, 449-457.	12.0	101
40	SENP3 is responsible for HIF-1 transactivation under mild oxidative stress via p300 de-SUMOylation. EMBO Journal, 2009, 28, 2748-2762.	7.8	172
41	PML/RARÎ± fusion protein mediates the unique sensitivity to arsenic cytotoxicity in acute promyelocytic leukemia cells: Mechanisms involve the impairment of cAMP signaling and the aberrant regulation of NADPH oxidase. Journal of Cellular Physiology, 2008, 217, 486-493.	4.1	17
42	NADPH oxidase-derived reactive oxygen species are responsible for the high susceptibility to arsenic cytotoxicity in acute promyelocytic leukemia cells. Leukemia Research, 2008, 32, 429-436.	0.8	41
43	Emodin-Induced Generation of Reactive Oxygen Species Inhibits RhoA Activation to Sensitize Gastric Carcinoma Cells to Anoikis. Neoplasia, 2008, 10, 41-IN19.	5.3	74
44	Emodin enhances cytotoxicity of chemotherapeutic drugs in prostate cancer cells: The mechanisms involve ROS-mediated suppression of multidrug resistance and hypoxia inducible factor-1. Cancer Biology and Therapy, 2008, 7, 468-475.	3.4	93
45	Cancer cell killing via ROS: To increase or decrease, that is the question. Cancer Biology and Therapy, 2008, 7, 1875-1884.	3.4	694
46	The endogenous reactive oxygen species promote NF-ÎºB activation by targeting on activation of NF-ÎºB-inducing kinase in oral squamous carcinoma cells. Free Radical Research, 2007, 41, 963-971.	3.3	43
47	Alteration of subcellular redox equilibrium and the consequent oxidative modification of nuclear factor ÎºB are critical for anticancer cytotoxicity by emodin, a reactive oxygen species-producing agent. Free Radical Biology and Medicine, 2006, 40, 2183-2197.	2.9	53
48	Gene expression alteration during redox-dependent enhancement of arsenic cytotoxicity by emodin in HeLa cells. Cell Research, 2005, 15, 511-522.	12.0	16
49	Dicoumarol Alters Cellular Redox State and Inhibits Nuclear Factor Kappa B to Enhance Arsenic Trioxide-Induced Apoptosis. Acta Biochimica Et Biophysica Sinica, 2004, 36, 235-242.	2.0	13
50	The cell cycle related apoptotic susceptibility to arsenic trioxide is associated with the level of reactive oxygen species. Cell Research, 2004, 14, 81-85.	12.0	54
51	Anthraquinones sensitize tumor cells to arsenic cytotoxicity in vitro and in vivo via reactive oxygen species-mediated dual regulation of apoptosis. Free Radical Biology and Medicine, 2004, 37, 2027-2041.	2.9	70
52	Emodin Enhances Arsenic Trioxide-Induced Apoptosis via Generation of Reactive Oxygen Species and Inhibition of Survival Signaling. Cancer Research, 2004, 64, 108-116.	0.9	148
53	The sensitivity of digestive tract tumor cells to As2O3 is associated with the inherent cellular level of reactive oxygen species. World Journal of Gastroenterology, 2002, 8, 36.	3.3	23
54	Apoptosis susceptibility of tumor cells to arsenic trioxide and the inherent cellular level of reactive oxygen species. Chinese Medical Journal, 2002, 115, 603-6.	2.3	1

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55	P16 methylation of the colorectal cancer and association with dukes stages. Chinese Journal of Cancer Research: Official Journal of China Anti-Cancer Association, Beijing Institute for Cancer Research, 2001, 13, 288-290.	2.2	0
56	p16 gene methylation in colorectal cancers associated with Dukeâ€™s staging. World Journal of Gastroenterology, 2001, 7, 722.	3.3	38
57	In situ PCR and immunohistochemical studies on p16 gene in pituitary adenomas. Chinese Journal of Cancer Research: Official Journal of China Anti-Cancer Association, Beijing Institute for Cancer Research, 2000, 12, 10-15.	2.2	0
58	The convergent point of the endocytic and autophagic pathways in leydig cells. Cell Research, 1999, 9, 243-253.	12.0	32
59	Functional implication of autophagy in steroid-secreting cells of the rat. The Anatomical Record, 1995, 242, 137-146.	1.8	37