

Takahiro Seki

List of Publications by Year in descending order

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Version: 2024-02-01

16
papers

1,107
citations

623188

14
h-index

940134

16
g-index

16
all docs

16
docs citations

16
times ranked

2080
citing authors

#	ARTICLE	IF	CITATIONS
1	CETSA interaction proteomics define specific RNA-modification pathways as key components of fluorouracil-based cancer drug cytotoxicity. <i>Cell Chemical Biology</i> , 2022, 29, 572-585.e8.	2.5	18
2	Therapeutic paradigm of dual targeting VEGF and PDGF for effectively treating FGF-2 off-target tumors. <i>Nature Communications</i> , 2020, 11, 3704.	5.8	62
3	CETSA-based target engagement of taxanes as biomarkers for efficacy and resistance. <i>Scientific Reports</i> , 2019, 9, 19384.	1.6	22
4	Dual roles of endothelial FGF-2â€“FGFR1â€“PDGF-BB and perivascular FGF-2â€“FGFR2â€“PDGFRÎ² signaling pathways in tumor vascular remodeling. <i>Cell Discovery</i> , 2018, 4, 3.	3.1	42
5	Ablation of endothelial VEGFR1 improves metabolic dysfunction by inducing adipose tissue browning. <i>Journal of Experimental Medicine</i> , 2018, 215, 611-626.	4.2	66
6	Cancer Lipid Metabolism Confers Antiangiogenic Drug Resistance. <i>Cell Metabolism</i> , 2018, 28, 104-117.e5.	7.2	191
7	Off-tumor targets compromise antiangiogenic drug sensitivity by inducing kidney erythropoietin production. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, E9635-E9644.	3.3	12
8	A miR-327â€“FGF10â€“FGFR2-mediated autocrine signaling mechanism controls white fat browning. <i>Nature Communications</i> , 2017, 8, 2079.	5.8	52
9	Switching harmful visceral fat to beneficial energy combustion improves metabolic dysfunctions. <i>JCI Insight</i> , 2017, 2, e89044.	2.3	28
10	Pericyteâ€“fibroblast transition promotes tumor growth and metastasis. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2016, 113, E5618-27.	3.3	246
11	Endothelial PDGF-CC regulates angiogenesis-dependent thermogenesis in beige fat. <i>Nature Communications</i> , 2016, 7, 12152.	5.8	84
12	Discontinuation of anti-VEGF cancer therapy promotes metastasis through a liver revascularization mechanism. <i>Nature Communications</i> , 2016, 7, 12680.	5.8	89
13	PlGF-induced VEGFR1-dependent vascular remodeling determines opposing antitumor effects and drug resistance to Dll4-Notch inhibitors. <i>Science Advances</i> , 2015, 1, e1400244.	4.7	21
14	VEGFR2-Mediated Vascular Dilation as a Mechanism of VEGF-Induced Anemia and Bone Marrow Cell Mobilization. <i>Cell Reports</i> , 2014, 9, 569-580.	2.9	28
15	Modulation of age-related insulin sensitivity by VEGF-dependent vascular plasticity in adipose tissues. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2014, 111, 14906-14911.	3.3	52
16	Tumour PDGF-BB expression levels determine dual effects of anti-PDGF drugs on vascular remodelling and metastasis. <i>Nature Communications</i> , 2013, 4, 2129.	5.8	94