Simon J Cook

List of Publications by Year
in descending order

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70 papers	6,111 citations	81900 39 h-index	98798 67 g-index
71 all docs	71 docs citations	71 times ranked	10366 citing authors

#	Article	IF	Citations
1	IKKÎ± plays a major role in canonical NF-ÎºB signalling in colorectal cells. Biochemical Journal, 2022, 479, 305-325.	3.7	7
2	Parallel Optimization of Potency and Pharmacokinetics Leading to the Discovery of a Pyrrole Carboxamide ERK5 Kinase Domain Inhibitor. Journal of Medicinal Chemistry, 2022, 65, 6513-6540.	6.4	3
3	CDK1, the Other â€˜Master Regulatorâ€™ of Autophagy. Trends in Cell Biology, 2021, 31, 95-107.	7.9	30
4	Inhibitory feedback control of NF-ÎºB signalling in health and disease. Biochemical Journal, 2021, 478, 2619-2664.	3.7	84
5	Inhibition of RAF dimers: it takes two to tango. Biochemical Society Transactions, 2021, 49, 237-251.	3.4	35
6	An mTORC1-to-CDK1 Switch Maintains Autophagy Suppression during Mitosis. Molecular Cell, 2020, 77, 228-240.e7.	9.7	74
7	Paradoxical activation of the protein kinase-transcription factor ERK5 by ERK5 kinase inhibitors. Nature Communications, 2020, 11, 1383.	12.8	30
8	Macroautophagy is repressed during mitosis â€“ seeing is believing. Autophagy, 2020, 16, 775-776.	9.1	5
9	Dual-Mechanism ERK1/2 Inhibitors Exploit a Distinct Binding Mode to Block Phosphorylation and Nuclear Accumulation of ERK1/2. Molecular Cancer Therapeutics, 2020, 19, 525-539.	4.1	14
10	Small molecule ERK5 kinase inhibitors paradoxically activate ERK5 signalling: be careful what you wish forâ€¦. Biochemical Society Transactions, 2020, 48, 1859-1875.	3.4	22
11	Targeting melanomaâ€™s MCL1 bias unleashes the apoptotic potential of BRAF and ERK1/2 pathway inhibitors. Nature Communications, 2019, 10, 5167.	12.8	52
12	Identification of a novel orally bioavailable ERK5 inhibitor with selectivity over p38Î± and BRD4. European Journal of Medicinal Chemistry, 2019, 178, 530-543.	5.5	15
13	MEK1/2 inhibitor withdrawal reverses acquired resistance driven by BRAFV600E amplification whereas KRASG13D amplification promotes EMT-chemoresistance. Nature Communications, 2019, 10, 2030.	12.8	39
14	Resistance to ERK1/2 pathway inhibitors; sweet spots, fitness deficits and drug addiction. , 2019, 2, 365-380.		3
15	ERK1/2 inhibitors: New weapons to inhibit the RAS-regulated RAF-MEK1/2-ERK1/2 pathway. , 2018, 187, 45-60.		123
16	Deâ€RSKing ERK â€“ regulation of ERK1/2â€RSK dissociation by phosphorylation within a disordered motif. FEBS Journal, 2018, 285, 42-45.	4.7	1
17	Calcium phosphate particles stimulate interleukin-1Î² release from human vascular smooth muscle cells: A role for spleen tyrosine kinase and exosome release. Journal of Molecular and Cellular Cardiology, 2018, 115, 82-93.	1.9	35
18	Over-expressed, N-terminally truncated BRAF is detected in the nucleus of cells with nuclear phosphorylated MEK and ERK. Heliyon, 2018, 4, e01065.	3.2	1

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19	Targeting IKKÎ² in Cancer: Challenges and Opportunities for the Therapeutic Utilisation of IKKÎ² Inhibitors. Cells, 2018, 7, 115.	4.1	91
20	Visualization of Endogenous ERK1/2 in Cells with a Bioorthogonal Covalent Probe. Bioconjugate Chemistry, 2017, 28, 1677-1683.	3.6	10
21	Control of cell death and mitochondrial fission by <scp>ERK</scp>1/2 <scp>MAP</scp> kinase signalling. FEBS Journal, 2017, 284, 4177-4195.	4.7	147
22	ERK1/2 signalling protects against apoptosis following endoplasmic reticulum stress but cannot provide long-term protection against BAX/BAK-independent cell death. PLoS ONE, 2017, 12, e0184907.	2.5	20
23	RNA-binding proteins ZFP36L1 and ZFP36L2 promote cell quiescence. Science, 2016, 352, 453-459.	12.6	142
24	Tumor cells with KRAS or BRAF mutations or ERK5/MAPK7 amplification are not addicted to ERK5 activity for cell proliferation. Cell Cycle, 2016, 15, 506-518.	2.6	43
25	Maternal DNA Methylation Regulates Early Trophoblast Development. Developmental Cell, 2016, 36, 152-163.	7.0	107
26	Identification of DYRK1B as a substrate of ERK1/2 and characterisation of the kinase activity of DYRK1B mutants from cancer and metabolic syndrome. Cellular and Molecular Life Sciences, 2016, 73, 883-900.	5.4	25
27	DYRK1A-mediated Cyclin D1 Degradation in Neural Stem Cells Contributes to the Neurogenic Cortical Defects in Down Syndrome. EBioMedicine, 2015, 2, 120-134.	6.1	62
28	MEK1 and MEK2 inhibitors and cancer therapy: the long and winding road. Nature Reviews Cancer, 2015, 15, 577-592.	28.4	461
29	Modeling Signaling Networks to Advance New Cancer Therapies. Annual Review of Biomedical Engineering, 2015, 17, 143-163.	12.3	34
30	Adaptation to mTOR kinase inhibitors by amplification of eIF4E to maintain cap-dependent translation. Journal of Cell Science, 2014, 127, 788-800.	2.0	70
31	Epigenetic memory of the first cell fate decision prevents complete ES cell reprogramming into trophoblast. Nature Communications, 2014, 5, 5538.	12.8	68
32	The increase in BIK expression following ERK1/2 pathway inhibition is a consequence of G1 cell-cycle arrest and not a direct effect on BIK protein stability. Biochemical Journal, 2014, 459, 513-524.	3.7	4
33	The role of MAPK signalling pathways in the response to endoplasmic reticulum stress. Biochimica Et Biophysica Acta - Molecular Cell Research, 2014, 1843, 2150-2163.	4.1	322
34	A novel DYRK1B inhibitor AZ191 demonstrates that DYRK1B acts independently of GSK3Î² to phosphorylate cyclin D1 at Thr286, not Thr288. Biochemical Journal, 2014, 457, 43-56.	3.7	54
35	Oncogenic K-Ras suppresses IP3-dependent Ca2+ release through remodeling of IP3Rs isoform composition and ER luminal Ca2+ levels in colorectal cancer cell lines. Journal of Cell Science, 2014, 127, 1607-19.	2.0	63
36	Intrinsic and acquired resistance to MEK1/2 inhibitors in cancer. Biochemical Society Transactions, 2014, 42, 776-783.	3.4	28

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37	FGF Signaling Inhibition in ESCs Drives Rapid Genome-wide Demethylation to the Epigenetic Ground State of Pluripotency. Cell Stem Cell, 2013, 13, 351-359.	11.1	371
38	Adaptation to chronic mTOR inhibition in cancer and in aging. Biochemical Society Transactions, 2013, 41, 956-961.	3.4	12
39	The BH3 mimetic ABT-263 synergizes with the MEK1/2 inhibitor selumetinib/AZD6244 to promote BIM-dependent tumour cell death and inhibit acquired resistance. Biochemical Journal, 2013, 450, 285-294.	3.7	53
40	That which does not kill me makes me stronger; combining <scp>ERK</scp>1/2 pathway inhibitors and <scp>BH</scp>3 mimetics to kill tumour cells and prevent acquired resistance. British Journal of Pharmacology, 2013, 169, 1708-1722.	5.4	19
41	MEK Inhibitor U0126 Reverses Protection of Axons from Wallerian Degeneration Independently of MEKâ€“ERK Signaling. PLoS ONE, 2013, 8, e76505.	2.5	8
42	Tumour cell responses to MEK1/2 inhibitors: acquired resistance and pathway remodelling. Biochemical Society Transactions, 2012, 40, 73-78.	3.4	21
43	Mechanisms and clinical significance of BIM phosphorylation in chronic lymphocytic leukemia. Blood, 2012, 119, 1726-1736.	1.4	52
44	Regulation of MEK/ERK pathway output by subcellular localization of B-Raf. Biochemical Society Transactions, 2012, 40, 67-72.	3.4	20
45	ERK5 and its role in tumour development. Biochemical Society Transactions, 2012, 40, 251-256.	3.4	66
46	CDK1, not ERK1/2 or ERK5, is required for mitotic phosphorylation of BIMEL. Cellular Signalling, 2012, 24, 170-180.	3.6	17
47	Amplification of the Driving Oncogene, KRAS or BRAF , Underpins Acquired Resistance to MEK1/2 Inhibitors in Colorectal Cancer Cells. Science Signaling, 2011, 4, ra17.	3.6	186
48	BIMEL, an intrinsically disordered protein, is degraded by 20S proteasomes in the absence of poly-ubiquitylation. Journal of Cell Science, 2011, 124, 969-977.	2.0	65
49	Refining the minimal sequence required for ERK1/2-dependent poly-ubiquitination and proteasome-dependent turnover of BIM. Cellular Signalling, 2010, 22, 801-808.	3.6	9
50	^{V600E} Braf induces gastrointestinal crypt senescence and promotes tumour progression through enhanced CpG methylation of p16 ^{INK4a}. EMBO Molecular Medicine, 2010, 2, 458-471.	6.9	128
51	ERK1/2, but not ERK5, is necessary and sufficient for phosphorylation and activation of c-Fos. Cellular Signalling, 2009, 21, 969-977.	3.6	47
52	Intrinsic resistance to the MEK1/2 inhibitor AZD6244 (ARRYâ€142886) is associated with weak ERK1/2 signalling and/or strong PI3K signalling in colorectal cancer cell lines. International Journal of Cancer, 2009, 125, 2332-2341.	5.1	125
53	Apoptosis and autophagy: BIM as a mediator of tumour cell death in response to oncogeneâ€targeted therapeutics. FEBS Journal, 2009, 276, 6050-6062.	4.7	90
54	Ca2+ signalling checkpoints in cancer: remodelling Ca2+ for cancer cell proliferation and survival. Nature Reviews Cancer, 2008, 8, 361-375.	28.4	600

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55	ERK1/2-dependent phosphorylation of BimEL promotes its rapid dissociation from Mcl-1 and Bcl-xL. EMBO Journal, 2007, 26, 2856-2867.	7.8	157
56	The duration of ERK1/2 activity determines the activation of c-Fos and Fra-1 and the composition and quantitative transcriptional output of AP-1. Cellular Signalling, 2007, 19, 695-704.	3.6	54
57	c-Cbl is not required for ERK1/2-dependent degradation of BimEL. Cellular Signalling, 2007, 19, 2605-2611.	3.6	26
58	Recent advances in Ca2+-dependent Ras regulation and cell proliferation. Cell Calcium, 2006, 39, 101-112.	2.4	68
59	The conditional kinase Î”MEKK1:ER* selectively activates the JNK pathway and protects against serum withdrawal-induced cell death. Cellular Signalling, 2005, 17, 1412-1422.	3.6	3
60	Identification of a DEF-type Docking Domain for Extracellular Signal-regulated Kinases 1/2 That Directs Phosphorylation and Turnover of the BH3-only Protein BimEL. Journal of Biological Chemistry, 2005, 280, 17657-17663.	3.4	42
61	ERK1/2 and p38 cooperate to induce a p21CIP1-dependent G1 cell cycle arrest. Oncogene, 2004, 23, 3284-3295.	5.9	84
62	Extracellular Signal-regulated Kinases 1/2 Are Serum-stimulated â€œBimEL Kinasesâ€•That Bind to the BH3-only Protein BimEL Causing Its Phosphorylation and Turnover. Journal of Biological Chemistry, 2004, 279, 8837-8847.	3.4	172
63	Activation of ERK1/2 by Î”Raf-1â€‰:â€‰ER* represses Bim expression independently of the JNK or PI3K pathways. Oncogene, 2003, 22, 1281-1293.	5.9	161
64	Selective activation of the c-Jun N-terminal kinase (JNK) pathway fails to elicit Bax activation or apoptosis unless the phosphoinositide 3â€²-kinase (PI3K) pathway is inhibited. Oncogene, 2003, 22, 4690-4701.	5.9	47
65	Activation of the ERK1/2 Signaling Pathway Promotes Phosphorylation and Proteasome-dependent Degradation of the BH3-only Protein, Bim. Journal of Biological Chemistry, 2003, 278, 18811-18816.	3.4	539
66	Cell-cycle arrest by PD184352 requires inhibition of extracellular signal-regulated kinases (ERK) 1/2 but not ERK5/BMK1. Biochemical Journal, 2002, 366, 673-680.	3.7	94
67	Î”MEKK3:ER* activation induces a p38Î±/Î²2-dependent cell cycle arrest at the G2 checkpoint. Oncogene, 2002, 21, 8089-8104.	5.9	53
68	Sustained MAP kinase activation is required for the expression of cyclin D1, p21Cip1 and a subset of AP-1 proteins in CCL39 cells. Oncogene, 1999, 18, 3085-3097.	5.9	215
69	The Repertoire of Fos and Jun Proteins Expressed during the G₁ Phase of the Cell Cycle Is Determined by the Duration of Mitogen-Activated Protein Kinase Activation. Molecular and Cellular Biology, 1999, 19, 330-341.	2.3	174
70	ERK5 Signalling and Resistance to ERK1/2 Pathway Therapeutics: The Path Less Travelled?. Frontiers in Cell and Developmental Biology, 0, 10, .	3.7	9