Philippe P Juin

List of Publications by Year
in descending order

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83	9,886	94269 37	56606 83
papers	citations	h-index	g-index

92	92	92	14531
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Keeping Cell Death Alive: An Introduction into the French Cell Death Research Network. Biomolecules, 2022, 12, 901.	1.8	2
2	Development of an absolute assignment predictor for triple-negative breast cancer subtyping using machine learning approaches. Computers in Biology and Medicine, 2021, 129, 104171.	3.9	8
3	bc-GenExMiner 4.5: new mining module computes breast cancer differential gene expression analyses. Database: the Journal of Biological Databases and Curation, 2021, 2021, .	1.4	81
4	Random forest of perfect trees: concept, performance, applications and perspectives. Bioinformatics, 2021, 37, 2165-2174.	1.8	20
5	Synthesis and biological studies of new piperidino-1,2,3-triazole hybrids with 3-aryl isoxazole side chains. Bioorganic and Medicinal Chemistry Letters, 2021, 52, 128390.	1.0	3
6	An EMTâ€“primary ciliumâ€“GLIS2 signaling axis regulates mammogenesis and claudin-low breast tumorigenesis. Science Advances, 2021, 7, eabf6063.	4.7	14
7	Mitochondria at Center of Exchanges between Cancer Cells and Cancer-Associated Fibroblasts during Tumor Progression. Cancers, 2020, 12, 3017.	1.7	16
8	Targeting of BCL-2 Family Members during Anticancer Treatment: A Necessary Compromise between Individual Cell and Ecosystemic Responses?. Biomolecules, 2020, 10, 1109.	1.8	4
9	RE: Immune Checkpoint Profiles in Luminal B Breast Cancer (Alliance). Journal of the National Cancer Institute, 2020, 112, 863-864.	3.0	1
10	Mitotic stress-induced secretome primes cancer cells to apoptosis and maximizes paclitaxel response in breast tumors when combined with BCL-xL-targeting BH3 mimetics. Molecular and Cellular Oncology, 2020, 7, 1735912.	0.3	1
11	STING-dependent paracriny shapes apoptotic priming of breast tumors in response to anti-mitotic treatment. Nature Communications, 2020, 11, 259.	5.8	65
12	Abstract P4-06-11: High immune response identified as a good prognostic factor by proteomic SWATH-MS approach in 157 ER+/HER2- early breast cancer. , 2020, , .		0
13	Interactions between cancer-associated fibroblasts and tumor cells promote MCL-1 dependency in estrogen receptor-positive breast cancers. Oncogene, 2019, 38, 3261-3273.	2.6	41
14	Targeting PUMA/Bcl-xL interaction by new specific compounds to unleash apoptotic process in cancer cells. European Journal of Medicinal Chemistry, 2019, 162, 334-347.	2.6	3
15	Synthesis and cytotoxicity studies of newly designed benzyl-hydroquinone derivatives. Medicinal Chemistry Research, 2018, 27, 1050-1065.	1.1	4
16	The oncogenic tyrosine kinase Lyn impairs the pro-apoptotic function of Bim. Oncogene, 2018, 37, 2122-2136.	2.6	8
17	Molecular mechanisms of cell death: recommendations of the Nomenclature Committee on Cell Death 2018. Cell Death and Differentiation, 2018, 25, 486-541.	5.0	4,036
18	E2F1 interacts with <scp>BCL</scp> â€•<scp>xL</scp> and regulates its subcellular localization dynamics to trigger cell death. EMBO Reports, 2018, 19, 234-243.	2.0	7

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19	Structure-guided design of pyridoclax derivatives based on Noxa / Mcl-1 interaction mode. European Journal of Medicinal Chemistry, 2018, 159, 357-380.	2.6	12
20	Abstract 3996: Pyridoclax and its derivatives from oligopyridine family directly inhibit Mcl-1 and exert potent antitumor effects on ovarian cancer in vitro and in vivo. , 2018, , .		1
21	DNA methylation signal has a major role in the response of human breast cancer cells to the microenvironment. Oncogenesis, 2017, 6, e390-e390.	2.1	27
22	BCL-XL directly modulates RAS signalling to favour cancer cell stemness. Nature Communications, 2017, 8, 1123.	5.8	43
23	Tight Sequestration of BH3 Proteins by BCL-xL at Subcellular Membranes Contributes to Apoptotic Resistance. Cell Reports, 2016, 17, 3347-3358.	2.9	44
24	Novel 1,6-naphthyridin-2(1H)-ones as potential anticancer agents targeting Hsp90. European Journal of Medicinal Chemistry, 2016, 119, 17-33.	2.6	18
25	TCTP contains a BH3-like domain, which instead of inhibiting, activates Bcl-xL. Scientific Reports, 2016, 6, 19725.	1.6	39
26	Survivin contributes to DNA repair by homologous recombination in breast cancer cells. Breast Cancer Research and Treatment, 2016, 155, 53-63.	1.1	48
27	Constitutive p53 heightens mitochondrial apoptotic priming and favors cell death induction by BH3 mimetic inhibitors of BCL-xL. Cell Death and Disease, 2016, 7, e2083-e2083.	2.7	34
28	Preliminary Studies on the Activity of Mixed Polyphenol-Heterocyclic Systems Against B16-F10 Melanoma Cancer Cells. Medicinal Chemistry, 2016, 12, 419-425.	0.7	4
29	First Evidence That Oligopyridines, Î±-Helix Foldamers, Inhibit Mcl-1 and Sensitize Ovarian Carcinoma Cells to Bcl-x_L-Targeting Strategies. Journal of Medicinal Chemistry, 2015, 58, 1644-1668.	2.9	40
30	A combination of in silico and SAR studies to identify binding hot spots of Bcl-xL inhibitors. Bioorganic and Medicinal Chemistry, 2015, 23, 1747-1757.	1.4	6
31	Irinotecan treatment and senescence failure promote the emergence of more transformed and invasive cells that depend on anti-apoptotic Mcl-1. Oncotarget, 2015, 6, 409-426.	0.8	42
32	YM155 potently triggers cell death in breast cancer cells through an autophagy-NF-kB network. Oncotarget, 2015, 6, 13476-13486.	0.8	43
33	miR-491-5p-induced apoptosis in ovarian carcinoma depends on the direct inhibition of both BCL-XL and EGFR leading to BIM activation. Cell Death and Disease, 2014, 5, e1445-e1445.	2.7	91
34	Bcl-xL controls a switch between cell death modes during mitotic arrest. Cell Death and Disease, 2014, 5, e1291-e1291.	2.7	52
35	Design, synthesis and biological evaluation of new inhibitors of Bax/Bcl-xL interaction in cancer cells. Bioorganic and Medicinal Chemistry Letters, 2014, 24, 1758-1761.	1.0	5
36	Synthesis of new mixed phenol/heterocyclic derivatives and studies of their activity as inhibitors of Bax/Bcl-xL interaction. Tetrahedron, 2014, 70, 301-311.	1.0	5

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37	Decoding and unlocking the BCL-2 dependency of cancer cells. Nature Reviews Cancer, 2013, 13, 455-465.	12.8	194
38	pRb/E2F-1-mediated caspase-dependent induction of Noxa amplifies the apoptotic effects of the Bcl-2/Bcl-xL inhibitor ABT-737. Cell Death and Differentiation, 2013, 20, 755-764.	5.0	19
39	Protect and serve: Bcl-2 proteins as guardians and rulers of cancer cell survival. Cell Cycle, 2013, 12, 2937-2947.	1.3	25
40	Hemisynthesis of Selected Embelin Analogs and Investigation of their Proapoptotic Activity Against Cancer Cells. Medicinal Chemistry, 2013, 9, 1028-1034.	0.7	10
41	Î³-Secretase inhibition promotes cell death, Noxa upregulation, and sensitization to BH3 mimetic ABT-737 in human breast cancer cells. Breast Cancer Research, 2012, 14, R96.	2.2	37
42	Structureâ€“activity relationship of selected polyphenol derivatives as inhibitors of Bax/Bcl-xL interaction. European Journal of Medicinal Chemistry, 2012, 51, 286-293.	2.6	12
43	Regulation of cancer cell survival by BCL2 family members upon prolonged mitotic arrest: opportunities for anticancer therapy. Anticancer Research, 2012, 32, 4225-33.	0.5	39
44	Noxa controls Mule-dependent Mcl-1 ubiquitination through the regulation of the Mcl-1/USP9X interaction. Biochemical and Biophysical Research Communications, 2011, 413, 460-464.	1.0	71
45	Serum-Nutrient Starvation Induces Cell Death Mediated by Bax and Puma That Is Counteracted by p21 and Unmasked by Bcl-xL Inhibition. PLoS ONE, 2011, 6, e23577.	1.1	60
46	Mitochondrial targeting by use of lipid nanocapsules loaded with SV30, an analogue of the small-molecule Bcl-2 inhibitor HA14-1. Journal of Controlled Release, 2011, 151, 74-82.	4.8	31
47	ABT-737 and/or folate reverse the PDGF-induced alterations in the mitochondrial apoptotic pathway in low-grade glioma patients. Clinical Epigenetics, 2011, 2, 369-381.	1.8	8
48	c-Myc dependent expression of pro-apoptotic Bim renders HER2-overexpressing breast cancer cells dependent on anti-apoptotic Mcl-1. Molecular Cancer, 2011, 10, 110.	7.9	38
49	Resistance to HER2 inhibitors: Is addition better than substitution? Rationale for the hypothetical concept of drug sedimentation. Critical Reviews in Oncology/Hematology, 2011, 78, 195-205.	2.0	22
50	Escape from p21-mediated Oncogene-induced Senescence Leads to Cell Dedifferentiation and Dependence on Anti-apoptotic Bcl-xL and MCL1 Proteins. Journal of Biological Chemistry, 2011, 286, 12825-12838.	1.6	44
51	Bax Activation by Engagement with, Then Release from, the BH3 Binding Site of Bcl-x_L. Molecular and Cellular Biology, 2011, 31, 832-844.	1.1	32
52	Bax activation by the BH3-only protein Puma promotes cell dependence on antiapoptotic Bcl-2 family members. Journal of Cell Biology, 2009, 185, 279-290.	2.3	132
53	P53 and PTEN expression contribute to the inhibition of EGFR downstream signaling pathway by cetuximab. Cancer Gene Therapy, 2009, 16, 498-507.	2.2	19
54	Bax activation by the BH3-only protein Puma promotes cell dependence on antiapoptotic Bcl-2 family members. Journal of Experimental Medicine, 2009, 206, i8-i8.	4.2	0

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55	The synthesis of new, selected analogues of the pro-apoptotic and anticancer molecule HA 14-1. Tetrahedron Letters, 2008, 49, 3276-3278.	0.7	44
56	HA14-1, a small molecule inhibitor of Bcl-2, bypasses chemoresistance in leukaemia cells. Leukemia Research, 2007, 31, 859-863.	0.4	33
57	Sensitization of osteosarcoma cells to apoptosis by oncostatin M depends on STAT5 and p53. Oncogene, 2007, 26, 6653-6664.	2.6	45
58	TOM22, a core component of the mitochondria outer membrane protein translocation pore, is a mitochondrial receptor for the proapoptotic protein Bax. Cell Death and Differentiation, 2007, 14, 785-794.	5.0	142
59	Functional and physical interaction between Bcl-XL and a BH3-like domain in Beclin-1. EMBO Journal, 2007, 26, 2527-2539.	3.5	1,003
60	Bax activation and mitochondrial insertion during apoptosis. Apoptosis: an International Journal on Programmed Cell Death, 2007, 12, 887-896.	2.2	278
61	Mitochondria as the target of the pro-apoptotic protein Bax. Biochimica Et Biophysica Acta - Bioenergetics, 2006, 1757, 1301-1311.	0.5	210
62	The Small Organic Compound HA14-1 Prevents Bcl-2 Interaction with Bax to Sensitize Malignant Glioma Cells to Induction of Cell Death. Cancer Research, 2006, 66, 2757-2764.	0.4	127
63	Activation of Bax by BH3 Domains during Apoptosis:The zunfolding of a Deadly Plot. Cell Cycle, 2005, 4, 637-642.	1.3	12
64	Differentiation-dependent Sensitivity to Apoptogenic Factors in PC12 Cells. Journal of Biological Chemistry, 2004, 279, 30983-30993.	1.6	36
65	The p18 Truncated Form of Bax Behaves Like a Bcl-2 Homology Domain 3-only Protein. Journal of Biological Chemistry, 2004, 279, 11503-11512.	1.6	38
66	Shooting at survivors: Bcl-2 family members as drug targets for cancer. Biochimica Et Biophysica Acta - Molecular Cell Research, 2004, 1644, 251-260.	1.9	54
67	The First Î± Helix of Bax Plays a Necessary Role in Its Ligand-Induced Activation by the BH3-Only Proteins Bid and PUMA. Molecular Cell, 2004, 16, 807-818.	4.5	235
68	Minimal BH3 Peptides Promote Cell Death by Antagonizing Anti-apoptotic Proteins. Journal of Biological Chemistry, 2003, 278, 19426-19435.	1.6	80
69	Impact of proapoptotic proteins Bax and Bak in tumor progression and response to treatment. Expert Review of Anticancer Therapy, 2003, 3, 563-570.	1.1	28
70	Nonredundant Role of Bax and Bak in Bid-Mediated Apoptosis. Molecular and Cellular Biology, 2003, 23, 4701-4712.	1.1	102
71	c-Myc Functionally Cooperates with Bax To Induce Apoptosis. Molecular and Cellular Biology, 2002, 22, 6158-6169.	1.1	128
72	Petites molÃ©cules antagonistes de Bcl-2 : des grains de sable pour enrayer la carcinogenÃ¨se ?. Medecine/Sciences, 2001, 17, 655.	0.0	0

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73	Caspase 8: The killer you can't live without. Nature Medicine, 2000, 6, 498-500.	15.2	16
74	The coordinate release of cytochrome c during apoptosis is rapid, complete and kinetically invariant. Nature Cell Biology, 2000, 2, 156-162.	4.6	973
75	Modifications de la permÃ©abilitÃ© membranaire mitochondriale au cours de l'apoptose : ouverture ou rupture ?. Medecine/Sciences, 2000, 16, 261.	0.0	1
76	The C-Terminus of bax Is Not a Membrane Addressing/Anchoring Signal. Biochemical and Biophysical Research Communications, 1999, 260, 582-591.	1.0	48
77	c-Myc-induced sensitization to apoptosis is mediated through cytochrome c release. Genes and Development, 1999, 13, 1367-1381.	2.7	294
78	Potentiation of Apoptosis by Mitochondria in a Cell-Free System. Biochemical and Biophysical Research Communications, 1998, 253, 185-191.	1.0	9
79	The Isolated Complex of the Translocase of the Outer Membrane of Mitochondria. Journal of Biological Chemistry, 1998, 273, 31032-31039.	1.6	97
80	Induction of a Caspase-3-like Activity by Calcium in Normal Cytosolic Extracts Triggers Nuclear Apoptosis in a Cell-free System. Journal of Biological Chemistry, 1998, 273, 17559-17564.	1.6	106
81	Relationship between the Peptide-sensitive Channel and the Mitochondrial Outer Membrane Protein Translocation Machinery. Journal of Biological Chemistry, 1997, 272, 6044-6050.	1.6	36
82	Characterization and function of the mitochondrial outer membrane peptide-sensitive channel. Journal of Bioenergetics and Biomembranes, 1996, 28, 101-108.	1.0	29
83	Involvement of the Peptide-Sensitive Channel in the Translocation of Basic Peptides into Mitochondria. Biochemical and Biophysical Research Communications, 1995, 211, 92-99.	1.0	13