

Grant Dewson

List of Publications by Year in descending order

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87
papers

8,693
citations

66250

44
h-index

62345

84
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94
all docs

94
docs citations

94
times ranked

11705
citing authors

#	ARTICLE	IF	CITATIONS
1	Therapeutic targeting of mitophagy in Parkinson's disease. <i>Biochemical Society Transactions</i> , 2022, 50, 783-797.	1.6	20
2	Activation mechanism of PINK1. <i>Nature</i> , 2022, 602, 328-335.	13.7	59
3	Ubiquitin signalling in neurodegeneration: mechanisms and therapeutic opportunities. <i>Cell Death and Differentiation</i> , 2021, 28, 570-590.	5.0	197
4	A new crystal form of GABARAPL2. <i>Acta Crystallographica Section F, Structural Biology Communications</i> , 2021, 77, 140-147.	0.4	1
5	Too much death can kill you: inhibiting intrinsic apoptosis to treat disease. <i>EMBO Journal</i> , 2021, 40, e107341.	3.5	26
6	Structure of detergent-activated BAK dimers derived from the inert monomer. <i>Molecular Cell</i> , 2021, 81, 2123-2134.e5.	4.5	26
7	Dynamic reconfiguration of pro-apoptotic BAK on membranes. <i>EMBO Journal</i> , 2021, 40, e107237.	3.5	20
8	VDAC2 and the BCL-2 family of proteins. <i>Biochemical Society Transactions</i> , 2021, 49, 2787-2795.	1.6	23
9	EBV BCL-2 homologue BHRF1 drives chemoresistance and lymphomagenesis by inhibiting multiple cellular pro-apoptotic proteins. <i>Cell Death and Differentiation</i> , 2020, 27, 1554-1568.	5.0	35
10	Mechanism and inhibition of the papain-like protease, PLpro, of SARS-CoV-2. <i>EMBO Journal</i> , 2020, 39, e106275.	3.5	330
11	BAK core dimers bind lipids and can be bridged by them. <i>Nature Structural and Molecular Biology</i> , 2020, 27, 1024-1031.	3.6	49
12	MCL-1 is essential for survival but dispensable for metabolic fitness of FOXP3+ regulatory T cells. <i>Cell Death and Differentiation</i> , 2020, 27, 3374-3385.	5.0	2
13	Robust autoactivation for apoptosis by BAK but not BAX highlights BAK as an important therapeutic target. <i>Cell Death and Disease</i> , 2020, 11, 268.	2.7	27
14	A small molecule interacts with VDAC2 to block mouse BAK-driven apoptosis. <i>Nature Chemical Biology</i> , 2019, 15, 1057-1066.	3.9	30
15	BAX Activation: Mutations Near Its Proposed Non-canonical BH3 Binding Site Reveal Allosteric Changes Controlling Mitochondrial Association. <i>Cell Reports</i> , 2019, 27, 359-373.e6.	2.9	31
16	Parkin inhibits BAK and BAX apoptotic function by distinct mechanisms during mitophagy. <i>EMBO Journal</i> , 2019, 38, .	3.5	66
17	Mcl-1 and Bcl-xL sequestration of Bak confers differential resistance to BH3-only proteins. <i>Cell Death and Differentiation</i> , 2018, 25, 721-734.	5.0	44
18	BAK/BAX macropores facilitate mitochondrial herniation and mtDNA efflux during apoptosis. <i>Science</i> , 2018, 359, .	6.0	581

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19	How do thymic epithelial cells die?. <i>Cell Death and Differentiation</i> , 2018, 25, 1002-1004.	5.0	17
20	VDAC2 enables BAX to mediate apoptosis and limit tumor development. <i>Nature Communications</i> , 2018, 9, 4976.	5.8	110
21	Mutant TRP53 exerts a target gene-selective dominant-negative effect to drive tumor development. <i>Genes and Development</i> , 2018, 32, 1420-1429.	2.7	29
22	Humanized Mcl-1 mice enable accurate preclinical evaluation of MCL-1 inhibitors destined for clinical use. <i>Blood</i> , 2018, 132, 1573-1583.	0.6	67
23	The Walrus and the Carpenter: Complex Regulation of Tumor Immunity in Colorectal Cancer. <i>Cell</i> , 2018, 174, 14-16.	13.5	70
24	BCL-2: Long and winding path from discovery to therapeutic target. <i>Biochemical and Biophysical Research Communications</i> , 2017, 482, 459-469.	1.0	55
25	Huntingtin Inclusions Trigger Cellular Quiescence, Deactivate Apoptosis, and Lead to Delayed Necrosis. <i>Cell Reports</i> , 2017, 19, 919-927.	2.9	98
26	A critical epithelial survival axis regulated by MCL-1 maintains thymic function in mice. <i>Blood</i> , 2017, 130, 2504-2515.	0.6	40
27	Synergistic action of the MCL-1 inhibitor S63845 with current therapies in preclinical models of triple-negative and HER2-amplified breast cancer. <i>Science Translational Medicine</i> , 2017, 9, .	5.8	148
28	BAK $\hat{\pm}$ 6 permits activation by BH3-only proteins and homooligomerization via the canonical hydrophobic groove. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, 7629-7634.	3.3	32
29	Conversion of Bim-BH3 from Activator to Inhibitor of Bak through Structure-Based Design. <i>Molecular Cell</i> , 2017, 68, 659-672.e9.	4.5	57
30	Parkin and mitophagy in cancer. <i>Oncogene</i> , 2017, 36, 1315-1327.	2.6	201
31	Disordered clusters of Bak dimers rupture mitochondria during apoptosis. <i>ELife</i> , 2017, 6, .	2.8	60
32	Identification of an activation site in Bak and mitochondrial Bax triggered by antibodies. <i>Nature Communications</i> , 2016, 7, 11734.	5.8	50
33	Doughnuts, daisy chains and crescent moons: the quest for the elusive apoptotic pore. <i>EMBO Journal</i> , 2016, 35, 371-373.	3.5	9
34	Physiological restraint of Bak by Bcl-x _L is essential for cell survival. <i>Genes and Development</i> , 2016, 30, 1240-1250.	2.7	40
35	Characterizing Bcl-2 Family Protein Conformation and Oligomerization Using Cross-Linking and Antibody Gel-Shift in Conjunction with Native PAGE. <i>Methods in Molecular Biology</i> , 2016, 1419, 185-196.	0.4	3
36	Apoptosis regulates endothelial cell number and capillary vessel diameter but not vessel regression during retinal angiogenesis. <i>Development (Cambridge)</i> , 2016, 143, 2973-82.	1.2	34

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37	Endothelial cell survival during angiogenesis requires the pro-survival protein MCL1. <i>Cell Death and Differentiation</i> , 2016, 23, 1371-1379.	5.0	27
38	Mitochondria and apoptosis: emerging concepts. <i>F1000prime Reports</i> , 2015, 7, 42.	5.9	69
39	Bid chimeras indicate that most BH3-only proteins can directly activate Bak and Bax, and show no preference for Bak versus Bax. <i>Cell Death and Disease</i> , 2015, 6, e1735-e1735.	2.7	76
40	Bak apoptotic pores involve a flexible C-terminal region and juxtaposition of the C-terminal transmembrane domains. <i>Cell Death and Differentiation</i> , 2015, 22, 1665-1675.	5.0	51
41	Two roads to death – Bax targets mitochondria by distinct routes before or during apoptotic cell death. <i>Molecular and Cellular Oncology</i> , 2015, 2, e974460.	0.3	2
42	Dissociation of Bak \pm 1 helix from the core and latch domains is required for apoptosis. <i>Nature Communications</i> , 2015, 6, 6841.	5.8	48
43	Investigating Bak/Bax Activating Conformation Change by Immunoprecipitation: Table 1.. <i>Cold Spring Harbor Protocols</i> , 2015, 2015, pdb.prot086454.	0.2	3
44	Blue Native PAGE and Antibody Gel Shift to Assess Bak and Bax Conformation Change and Oligomerization. <i>Cold Spring Harbor Protocols</i> , 2015, 2015, pdb.prot086488.	0.2	6
45	Detection of Bak/Bax Activating Conformation Change by Intracellular Flow Cytometry: Table 1.. <i>Cold Spring Harbor Protocols</i> , 2015, 2015, pdb.prot086462.	0.2	2
46	Investigating the Oligomerization of Bak and Bax during Apoptosis by Cysteine Linkage: Figure 1.. <i>Cold Spring Harbor Protocols</i> , 2015, 2015, pdb.prot086470.	0.2	4
47	Investigating Bax Subcellular Localization and Membrane Integration. <i>Cold Spring Harbor Protocols</i> , 2015, 2015, pdb.prot086447.	0.2	13
48	Crystal structure of Bax bound to the BH3 peptide of Bim identifies important contacts for interaction. <i>Cell Death and Disease</i> , 2015, 6, e1809-e1809.	2.7	54
49	An aspartyl protease defines a novel pathway for export of <i>Toxoplasma</i> proteins into the host cell. <i>ELife</i> , 2015, 4, .	2.8	99
50	The Functional Differences between Pro-survival and Pro-apoptotic B Cell Lymphoma 2 (Bcl-2) Proteins Depend on Structural Differences in Their Bcl-2 Homology 3 (BH3) Domains. <i>Journal of Biological Chemistry</i> , 2014, 289, 36001-36017.	1.6	33
51	Apoptotic pore formation is associated with in-plane insertion of Bak or Bax central helices into the mitochondrial outer membrane. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2014, 111, E4076-85.	3.3	111
52	Building blocks of the apoptotic pore: how Bax and Bak are activated and oligomerize during apoptosis. <i>Cell Death and Differentiation</i> , 2014, 21, 196-205.	5.0	330
53	RIPK1 Regulates RIPK3-MLKL-Driven Systemic Inflammation and Emergency Hematopoiesis. <i>Cell</i> , 2014, 157, 1175-1188.	13.5	492
54	Bax targets mitochondria by distinct mechanisms before or during apoptotic cell death: a requirement for VDAC2 or Bak for efficient Bax apoptotic function. <i>Cell Death and Differentiation</i> , 2014, 21, 1925-1935.	5.0	106

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55	Bak Core and Latch Domains Separate during Activation, and Freed Core Domains Form Symmetric Homodimers. <i>Molecular Cell</i> , 2014, 55, 938-946.	4.5	140
56	Lymphotoxin α induces apoptosis, necroptosis and inflammatory signals with the same potency as tumour necrosis factor. <i>FEBS Journal</i> , 2013, 280, 5283-5297.	2.2	57
57	Bax Crystal Structures Reveal How BH3 Domains Activate Bax and Nucleate Its Oligomerization to Induce Apoptosis. <i>Cell</i> , 2013, 152, 519-531.	13.5	491
58	Bak apoptotic function is not directly regulated by phosphorylation. <i>Cell Death and Disease</i> , 2013, 4, e452-e452.	2.7	12
59	Assembly of the Bak Apoptotic Pore. <i>Journal of Biological Chemistry</i> , 2013, 288, 26027-26038.	1.6	67
60	Bax dimerizes via a symmetric BH3:groove interface during apoptosis. <i>Cell Death and Differentiation</i> , 2012, 19, 661-670.	5.0	161
61	Granzyme B triggers a prolonged pressure to die in Bcl-2 overexpressing cells, defining a window of opportunity for effective treatment with ABT-737. <i>Cell Death and Disease</i> , 2012, 3, e344-e344.	2.7	18
62	Translocation of a Bak C-Terminus Mutant from Cytosol to Mitochondria to Mediate Cytochrome c Release: Implications for Bak and Bax Apoptotic Function. <i>PLoS ONE</i> , 2012, 7, e31510.	1.1	46
63	Crystal Structure of a BCL-W Domain-Swapped Dimer: Implications for the Function of BCL-2 Family Proteins. <i>Structure</i> , 2011, 19, 1467-1476.	1.6	25
64	Molecular biology of Bax and Bak activation and action. <i>Biochimica Et Biophysica Acta - Molecular Cell Research</i> , 2011, 1813, 521-531.	1.9	415
65	Fas-mediated neutrophil apoptosis is accelerated by Bid, Bak, and Bax and inhibited by Bcl-2 and Mcl-1. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2011, 108, 13135-13140.	3.3	98
66	Maximal killing of lymphoma cells by DNA damage α -inducing therapy requires not only the p53 targets Puma and Noxa, but also Bim. <i>Blood</i> , 2010, 116, 5256-5267.	0.6	87
67	Inhibition of Bak Activation by VDAC2 Is Dependent on the Bak Transmembrane Anchor. <i>Journal of Biological Chemistry</i> , 2010, 285, 36876-36883.	1.6	83
68	Mechanisms by which Bak and Bax permeabilise mitochondria during apoptosis. <i>Journal of Cell Science</i> , 2009, 122, 2801-2808.	1.2	283
69	Bak Activation for Apoptosis Involves Oligomerization of Dimers via Their ± 6 Helices. <i>Molecular Cell</i> , 2009, 36, 696-703.	4.5	200
70	To Trigger Apoptosis, Bak Exposes Its BH3 Domain and Homodimerizes via BH3:Groove Interactions. <i>Molecular Cell</i> , 2008, 30, 369-380.	4.5	296
71	Mitochondrial permeabilization relies on BH3 ligands engaging multiple prosurvival Bcl-2 relatives, not Bak. <i>Journal of Cell Biology</i> , 2007, 177, 277-287.	2.3	109
72	Interleukin 15 α -mediated survival of natural killer cells is determined by interactions among Bim, Noxa and Mcl-1. <i>Nature Immunology</i> , 2007, 8, 856-863.	7.0	231

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73	Mitochondrial Release of Pro-apoptotic Proteins. <i>Journal of Biological Chemistry</i> , 2005, 280, 2266-2274.	1.6	154
74	Proapoptotic Bak is sequestered by Mcl-1 and Bcl-xL, but not Bcl-2, until displaced by BH3-only proteins. <i>Genes and Development</i> , 2005, 19, 1294-1305.	2.7	1,071
75	Intermediate Filaments Control the Intracellular Distribution of Caspases During Apoptosis. <i>American Journal of Pathology</i> , 2004, 164, 395-407.	1.9	60
76	Conformational change and mitochondrial translocation of Bax accompany proteasome inhibitor-induced apoptosis of chronic lymphocytic leukemic cells. <i>Oncogene</i> , 2003, 22, 2643-2654.	2.6	102
77	Multigene family isoform profiling from blood cell lineages. <i>BMC Genomics</i> , 2002, 3, 22.	1.2	6
78	Death and survival signalling in neutrophils. <i>Trends in Biochemical Sciences</i> , 2002, 27, 118-119.	3.7	0
79	Controlling phagocytosis: brief encounter or death embrace?. <i>Trends in Biochemical Sciences</i> , 2002, 27, 502.	3.7	0
80	Interleukin-5 inhibits translocation of Bax to the mitochondria, cytochrome c release, and activation of caspases in human eosinophils. <i>Blood</i> , 2001, 98, 2239-2247.	0.6	88
81	Small, but deadly: small-molecule inhibition of Bcl-2 homologue heterodimerization. <i>Trends in Biochemical Sciences</i> , 2001, 26, 218-219.	3.7	2
82	Bax to the wall: Bax- and Bak-induced mitochondrial dysfunction in apoptosis. <i>Trends in Biochemical Sciences</i> , 2001, 26, 353.	3.7	3
83	Return of the living dead. <i>Trends in Biochemical Sciences</i> , 2001, 26, 529-530.	3.7	0
84	Interleukin-13 induces PSGL-1/Pâ€œselectinâ€œdependent adhesion of eosinophils, but not neutrophils, to human umbilical vein endothelial cells under flow. <i>Blood</i> , 2000, 95, 3146-3152.	0.6	5
85	Expression of Bcl-2 and Its Homologues in Human Eosinophils. <i>American Journal of Respiratory Cell and Molecular Biology</i> , 1999, 20, 720-728.	1.4	54
86	A comparative study of different methods for the assessment of apoptosis and necrosis in human eosinophils. <i>Journal of Immunological Methods</i> , 1998, 217, 153-163.	0.6	100
87	Human eosinophils: Apoptosis versus survival in the mediation of inflammation. <i>Apoptosis: an International Journal on Programmed Cell Death</i> , 1996, 1, 111-118.	2.2	6