

Tudor Moldoveanu

List of Publications by Year in descending order

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Version: 2024-02-01

42
papers

4,925
citations

159585

30
h-index

276875

41
g-index

46
all docs

46
docs citations

46
times ranked

6353
citing authors

#	ARTICLE	IF	CITATIONS
1	Structural basis of BAK activation in mitochondrial apoptosis initiation. <i>Nature Communications</i> , 2022, 13, 250.	12.8	19
2	Protein-protein and protein-lipid interactions of pore-forming BCL-2 family proteins in apoptosis initiation. <i>Biochemical Society Transactions</i> , 2022, , .	3.4	12
3	A killer metamorphosis: catching BAK in action at the membrane. <i>EMBO Journal</i> , 2021, 40, e109529.	7.8	2
4	BAX, BAK, and BOK: A Coming of Age for the BCL-2 Family Effector Proteins. <i>Cold Spring Harbor Perspectives in Biology</i> , 2020, 12, a036319.	5.5	106
5	Linker Histone H1.2 Directly Activates BAK through the K/RVVKP Motif on the C-Terminal Domain. <i>Biochemistry</i> , 2020, 59, 3332-3346.	2.5	3
6	Uncovering human mixed lineage kinase domain-like activation in necroptosis. <i>Future Medicinal Chemistry</i> , 2019, 11, 2831-2844.	2.3	2
7	Direct Activation of Human MLKL by a Select Repertoire of Inositol Phosphate Metabolites. <i>Cell Chemical Biology</i> , 2019, 26, 863-877.e7.	5.2	38
8	Methods to Probe Conformational Activation and Mitochondrial Activity of Proapoptotic BAK. <i>Methods in Molecular Biology</i> , 2019, 1877, 185-200.	0.9	3
9	Intrinsic Instability of BOK Enables Membrane Permeabilization in Apoptosis. <i>Cell Reports</i> , 2018, 23, 2083-2094.e6.	6.4	41
10	Characterization of MLKL-mediated Plasma Membrane Rupture in Necroptosis. <i>Journal of Visualized Experiments</i> , 2018, , .	0.3	14
11	MLKL Requires the Inositol Phosphate Code to Execute Necroptosis. <i>Molecular Cell</i> , 2018, 70, 936-948.e7.	9.7	111
12	Metastability, an emerging concept governing BOK-mediated apoptosis initiation. <i>Oncotarget</i> , 2018, 9, 30944-30945.	1.8	5
13	MP57-03 IDENTIFICATION AND CHARACTERIZATION OF SELECTIVE ANDROGEN RECEPTOR DEGRADERS (SARDS) FOR THE TREATMENT OF ENZALUTAMIDE UNRESPONSIVE AND/OR RESISTANT PROSTATE CANCER. <i>Journal of Urology</i> , 2017, 197, .	0.4	0
14	Novel Selective Agents for the Degradation of Androgen Receptor Variants to Treat Castration-Resistant Prostate Cancer. <i>Cancer Research</i> , 2017, 77, 6282-6298.	0.9	62
15	Extra-mitochondrial prosurvival BCL-2 proteins regulate gene transcription by inhibiting the SUFU tumour suppressor. <i>Nature Cell Biology</i> , 2017, 19, 1226-1236.	10.3	38
16	Discoveries and controversies in BCL-2 protein-mediated apoptosis. <i>FEBS Journal</i> , 2016, 283, 2690-2700.	4.7	176
17	Sequential Engagement of Distinct MLKL Phosphatidylinositol-Binding Sites Executes Necroptosis. <i>Molecular Cell</i> , 2016, 61, 589-601.	9.7	183
18	BOK Is a Non-canonical BCL-2 Family Effector of Apoptosis Regulated by ER-Associated Degradation. <i>Cell</i> , 2016, 165, 421-433.	28.9	197

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19	Many players in BCL-2 family affairs. Trends in Biochemical Sciences, 2014, 39, 101-111.	7.5	352
20	Metabolic Activation of CaMKII by Coenzyme A. Molecular Cell, 2013, 52, 325-339.	9.7	35
21	Metabolic Activation of CaMKII by Coenzyme A. Molecular Cell, 2013, 52, 468.	9.7	1
22	BID-induced structural changes in BAK promote apoptosis. Nature Structural and Molecular Biology, 2013, 20, 589-597.	8.2	181
23	A Unified Model of Mammalian BCL-2 Protein Family Interactions at the Mitochondria. Molecular Cell, 2011, 44, 517-531.	9.7	502
24	BH3 Domains other than Bim and Bid Can Directly Activate Bax/Bak. Journal of Biological Chemistry, 2011, 286, 491-501.	3.4	139
25	Diversifying selection and functional analysis of interleukin-4 suggests antagonism-driven evolution at receptor-binding interfaces. BMC Evolutionary Biology, 2010, 10, 223.	3.2	19
26	Apoptotic Regulation by MCL-1 through Heterodimerization. Journal of Biological Chemistry, 2010, 285, 19615-19624.	3.4	64
27	The BCL-2 Family Reunion. Molecular Cell, 2010, 37, 299-310.	9.7	1,295
28	Concerted multi-pronged attack by calpastatin to occlude the catalytic cleft of heterodimeric calpains. Nature, 2008, 456, 404-408.	27.8	131
29	Development of Calpain-specific Inactivators by Screening of Positional Scanning Epoxide Libraries. Journal of Biological Chemistry, 2007, 282, 9600-9611.	3.4	36
30	Structural Basis for UBA-mediated Dimerization of c-Cbl Ubiquitin Ligase. Journal of Biological Chemistry, 2007, 282, 27547-27555.	3.4	37
31	Structural Model of the BCL-w~BID Peptide Complex and Its Interactions with Phospholipid Micelles,. Biochemistry, 2006, 45, 2250-2256.	2.5	54
32	Calpain Inhibition by Î±-Ketoamide and Cyclic Hemiacetal Inhibitors Revealed by X-ray Crystallography,. Biochemistry, 2006, 45, 7446-7452.	2.5	52
33	The X-Ray Structure of a BAK Homodimer Reveals an Inhibitory Zinc Binding Site. Molecular Cell, 2006, 24, 677-688.	9.7	213
34	Determination of Peptide Substrate Specificity for Î¼-Calpain by a Peptide Library-based Approach. Journal of Biological Chemistry, 2005, 280, 40632-40641.	3.4	115
35	Calpain Activation by Cooperative Ca ²⁺ Binding at Two Non-EF-hand Sites. Journal of Biological Chemistry, 2004, 279, 6106-6114.	3.4	48
36	Insertion Sequence 1 of Muscle-specific Calpain, p94, Acts as an Internal Propeptide. Journal of Biological Chemistry, 2004, 279, 27656-27666.	3.4	48

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37	Crystal Structures of Calpain ^ε E64 and ^ε Leupeptin Inhibitor Complexes Reveal Mobile Loops Gating the Active Site. <i>Journal of Molecular Biology</i> , 2004, 343, 1313-1326.	4.2	80
38	Calpain silencing by a reversible intrinsic mechanism. <i>Nature Structural and Molecular Biology</i> , 2003, 10, 371-378.	8.2	72
39	A Ca ²⁺ Switch Aligns the Active Site of Calpain. <i>Cell</i> , 2002, 108, 649-660.	28.9	311
40	Mutations in Calpain 3 Associated with Limb Girdle Muscular Dystrophy: Analysis by Molecular Modeling and by Mutation in m-Calpain. <i>Biophysical Journal</i> , 2001, 80, 2590-2596.	0.5	58
41	Ca ²⁺ -induced structural changes in rat m-calpain revealed by partial proteolysis. <i>BBA - Proteins and Proteomics</i> , 2001, 1545, 245-254.	2.1	25
42	Calpain Mutants with Increased Ca ²⁺ Sensitivity and Implications for the Role of the C2-like Domain. <i>Journal of Biological Chemistry</i> , 2001, 276, 7404-7407.	3.4	45