Stephen W Fesik

List of Publications by Year
in descending order

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69 papers	22,086 citations	34493 54 h-index	104191 69 g-index
72 all docs	72 docs citations	72 times ranked	23665 citing authors

#	Article	IF	Citations
1	Discovery of Sulfonamide-Derived Agonists of SOS1-Mediated Nucleotide Exchange on RAS Using Fragment-Based Methods. Journal of Medicinal Chemistry, 2020, 63, 8325-8337.	2.9	20
2	Drugging an undruggable pocket on KRAS. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 15823-15829.	3.3	280
3	High-throughput screening identifies small molecules that bind to the RAS:SOS:RAS complex and perturb RAS signaling. Analytical Biochemistry, 2018, 548, 44-52.	1.1	48
4	A Novel MCL1 Inhibitor Combined with Venetoclax Rescues Venetoclax-Resistant Acute Myelogenous Leukemia. Cancer Discovery, 2018, 8, 1566-1581.	7.7	250
5	Discovery and Structure-Based Optimization of Benzimidazole-Derived Activators of SOS1-Mediated Nucleotide Exchange on RAS. Journal of Medicinal Chemistry, 2018, 61, 8875-8894.	2.9	41
6	Discovery of Aminopiperidine Indoles That Activate the Guanine Nucleotide Exchange Factor SOS1 and Modulate RAS Signaling. Journal of Medicinal Chemistry, 2018, 61, 6002-6017.	2.9	33
7	Twenty years on: the impact of fragments on drug discovery. Nature Reviews Drug Discovery, 2016, 15, 605-619.	21.5	711
8	Discovery of 2-Indole-acylsulfonamide Myeloid Cell Leukemia 1 (Mcl-1) Inhibitors Using Fragment-Based Methods. Journal of Medicinal Chemistry, 2016, 59, 2054-2066.	2.9	114
9	Small molecule Mcl-1 inhibitors for the treatment of cancer. , 2015, 145, 76-84.		145
10	Approach for targeting Ras with small molecules that activate SOS-mediated nucleotide exchange. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 3401-3406.	3.3	165
11	Drugging the undruggable RAS: Mission Possible?. Nature Reviews Drug Discovery, 2014, 13, 828-851.	21.5	1,484
12	A method for the second-site screening of K-Ras in the presence of a covalently attached first-site ligand. Journal of Biomolecular NMR, 2014, 60, 11-14.	1.6	32
13	Discovery of a Potent Inhibitor of Replication Protein A Proteinâ€“Protein Interactions Using a Fragment-Linking Approach. Journal of Medicinal Chemistry, 2013, 56, 9242-9250.	2.9	59
14	Discovery of Potent Myeloid Cell Leukemia 1 (Mcl-1) Inhibitors Using Fragment-Based Methods and Structure-Based Design. Journal of Medicinal Chemistry, 2013, 56, 15-30.	2.9	248
15	Fragment-based drug discovery using NMR spectroscopy. Journal of Biomolecular NMR, 2013, 56, 65-75.	1.6	179
16	Discovery of Small Molecules that Bind to Kâ€Ras and Inhibit Sosâ€Mediated Activation. Angewandte Chemie - International Edition, 2012, 51, 6140-6143.	7.2	419
17	Discovery of a potent and selective Bcl-2 inhibitor using SAR by NMR. Bioorganic and Medicinal Chemistry Letters, 2010, 20, 6587-6591.	1.0	68
18	Discovery of an Orally Bioavailable Small Molecule Inhibitor of Prosurvival B-Cell Lymphoma 2 Proteins. Journal of Medicinal Chemistry, 2008, 51, 6902-6915.	2.9	267

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19	Activity of the Bcl-2 Family Inhibitor ABT-263 in a Panel of Small Cell Lung Cancer Xenograft Models. Clinical Cancer Research, 2008, 14, 3268-3277.	3.2	182
20	Influence of Bcl-2 Family Members on the Cellular Response of Small-Cell Lung Cancer Cell Lines to ABT-737. Cancer Research, 2007, 67, 1176-1183.	0.4	283
21	Studies Leading to Potent, Dual Inhibitors of Bcl-2 and Bcl-xL. Journal of Medicinal Chemistry, 2007, 50, 641-662.	2.9	281
22	Discovery and Structureâ˜'Activity Relationship of Antagonists of B-Cell Lymphoma 2 Family Proteins with Chemopotentiation Activity in Vitro and in Vivo. Journal of Medicinal Chemistry, 2006, 49, 1165-1181.	2.9	126
23	Discovery of a Potent Inhibitor of the Antiapoptotic Protein Bcl-xLfrom NMR and Parallel Synthesis. Journal of Medicinal Chemistry, 2006, 49, 656-663.	2.9	289
24	A Small-Molecule Inhibitor of Bcl-XL Potentiates the Activity of Cytotoxic Drugs In vitro and In vivo. Cancer Research, 2006, 66, 8731-8739.	0.4	141
25	Non-peptidic small molecule inhibitors of XIAP. Bioorganic and Medicinal Chemistry Letters, 2005, 15, 771-775.	1.0	60
26	Promoting apoptosis as a strategy for cancer drug discovery. Nature Reviews Cancer, 2005, 5, 876-885.	12.8	1,006
27	An inhibitor of Bcl-2 family proteins induces regression of solid tumours. Nature, 2005, 435, 677-681.	13.7	3,157
28	1H,13C and15N Resonance Assignments of a Bcl-xL/Bad Peptide Complex. Journal of Biomolecular NMR, 2005, 32, 260-260.	1.6	6
29	Structural biology of the Bcl-2 family of proteins. Biochimica Et Biophysica Acta - Molecular Cell Research, 2004, 1644, 83-94.	1.9	602
30	Discovery of Potent Antagonists of the Antiapoptotic Protein XIAP for the Treatment of Cancer. Journal of Medicinal Chemistry, 2004, 47, 4417-4426.	2.9	356
31	Defining the p53 DNA-binding domain/Bcl-xL-binding interface using NMR. FEBS Letters, 2004, 559, 171-174.	1.3	94
32	Solution Structure of the BHRF1 Protein From Epstein-Barr Virus, a Homolog of Human Bcl-2. Journal of Molecular Biology, 2003, 332, 1123-1130.	2.0	77
33	Solution structure of a Bcl-2 homolog from Kaposi sarcoma virus. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 3428-3433.	3.3	121
34	Structural Basis for the Inhibition of Caspase-3 by XIAP. Cell, 2001, 104, 791-800.	13.5	717
35	BCL-2 AND IAP Proteins As Potential Drug Targets. Scientific World Journal, The, 2001, 1, 105-105.	0.8	1
36	Rational Design of Diflunisal Analogues with Reduced Affinity for Human Serum Albumin. Journal of the American Chemical Society, 2001, 123, 10429-10435.	6.6	87

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37	Structural basis for binding of Smac/DIABLO to the XIAP BIR3 domain. Nature, 2000, 408, 1004-1008.	13.7	624
38	Rationale for Bclâ€X_L/Bad peptide complex formation from structure, mutagenesis, and biophysical studies. Protein Science, 2000, 9, 2528-2534.	3.1	365
39	An approach for high-throughput structure determination of proteins by NMR spectroscopy. Journal of Biomolecular NMR, 2000, 18, 229-238.	1.6	58
40	NMR Structure and Mutagenesis of the Third Bir Domain of the Inhibitor of Apoptosis Protein XIAP. Journal of Biological Chemistry, 2000, 275, 33777-33781.	1.6	224
41	Expression, Refolding, and Isotopic Labeling of Human Serum Albumin Domains for NMR Spectroscopy. Protein Expression and Purification, 2000, 20, 492-499.	0.6	20
42	14-3-3 Proteins and Survival Kinases Cooperate to Inactivate BAD by BH3 Domain Phosphorylation. Molecular Cell, 2000, 6, 41-51.	4.5	571
43	Insights into Programmed Cell Death through Structural Biology. Cell, 2000, 103, 273-282.	13.5	282
44	NMR Studies of the Anti-Apoptotic Protein Bcl-xL in Micelles. Biochemistry, 2000, 39, 11024-11033.	1.2	96
45	NMR-Based Screening of Proteins Containing13C-Labeled Methyl Groups. Journal of the American Chemical Society, 2000, 122, 7898-7904.	6.6	207
46	The Use of Differential Chemical Shifts for Determining the Binding Site Location and Orientation of Protein-Bound Ligands. Journal of the American Chemical Society, 2000, 122, 1241-1242.	6.6	113
47	Privileged Molecules for Protein Binding Identified from NMR-Based Screening. Journal of Medicinal Chemistry, 2000, 43, 3443-3447.	2.9	360
48	Role of Bcl-2 family members in apoptotic cell death. Kidney International, 1999, 56, 1192.	2.6	0
49	NMR structure and mutagenesis of the inhibitor-of-apoptosis protein XIAP. Nature, 1999, 401, 818-822.	13.7	332
50	The BH3 Domain of Bcl-x_S Is Required for Inhibition of the Antiapoptotic Function of Bcl-x_L. Molecular and Cellular Biology, 1999, 19, 6673-6681.	1.1	43
51	NMR structure and mutagenesis of the FADD (Mort1) death-effector domain. Nature, 1998, 392, 941-945.	13.7	225
52	NMR Structure and Mutagenesis of the N-Terminal Dbl Homology Domain of the Nucleotide Exchange Factor Trio. Cell, 1998, 95, 269-277.	13.5	168
53	Changes in the NMR-Derived Motional Parameters of the Insulin Receptor Substrate 1 Phosphotyrosine Binding Domain upon Binding to an Interleukin 4 Receptor Phosphopeptide. Biochemistry, 1997, 36, 4118-4124.	1.2	55
54	Three-dimensional structures of proteins involved in programmed cell death. Journal of Molecular Biology, 1997, 274, 291-302.	2.0	63

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55	The lymphoproliferation mutation in Fas locally unfolds the Fas death domain. Nature Structural Biology, 1997, 4, 983-985.	9.7	38
56	Bcl-xL forms an ion channel in synthetic lipid membranes. Nature, 1997, 385, 353-357.	13.7	810
57	Structure of Bcl-xL-Bak Peptide Complex: Recognition Between Regulators of Apoptosis. Science, 1997, 275, 983-986.	6.0	1,394
58	Use of deuterium labeling in NMR: overcoming a sizeable problem. Structure, 1996, 4, 1245-1249.	1.6	118
59	Structural basis for IL-4 receptor phosphopeptide recognition by thelRS-1 PTB domain. Nature Structural and Molecular Biology, 1996, 3, 388-393.	3.6	142
60	X-ray and NMR structure of human Bcl-xL, an inhibitor of programmed cell death. Nature, 1996, 381, 335-341.	13.7	1,427
61	NMR structure and mutagenesis of the Fas (APO-1/CD95) death domain. Nature, 1996, 384, 638-641.	13.7	366
62	Phosphatidylinositol 4,5-Bisphosphate Binding to the Pleckstrin Homology Domain of Phospholipase C-Î´1 Enhances Enzyme Activity. Journal of Biological Chemistry, 1996, 271, 25316-25326.	1.6	109
63	Structure and function of the phosphotyrosine binding (PTB) domain. Progress in Biophysics and Molecular Biology, 1995, 64, 221-235.	1.4	20
64	Structure and ligand recognition of the phosphotyrosine binding domain of Shc. Nature, 1995, 378, 584-592.	13.7	370
65	Binding Affinities of Tyrosine-phosphorylated Peptides to the COOH-terminal SH2 and NH2-terminal Phosphotyrosine Binding Domains of Shc. Journal of Biological Chemistry, 1995, 270, 31119-31123.	1.6	53
66	Structural Characterization of the Interaction between a Pleckstrin Homology Domain and Phosphatidylinositol 4,5-Bisphosphate. Biochemistry, 1995, 34, 9859-9864.	1.2	125
67	Solution structure of a pleckstrin-homology domain. Nature, 1994, 369, 672-675.	13.7	240
68	Pleckstrin homology domains bind to phosphatidylinositol-4,5-bisphosphate. Nature, 1994, 371, 168-170.	13.7	782
69	Side chain and backbone assignments in isotopically labeled proteins from two heteronuclear triple resonance experiments. FEBS Letters, 1992, 314, 413-418.	1.3	126