Frank McCormick

List of Publications by Year
in descending order

96 papers	25,119 citations	50276 46 h-index	37204 96 g-index
103 all docs	103 docs citations	103 times ranked	27110 citing authors

#	Article	IF	Citations
1	Machine learningâ€“driven multiscale modeling reveals lipid-dependent dynamics of RAS signaling proteins. Proceedings of the National Academy of Sciences of the United States of America, 2022, 119, .	7.1	44
2	A brief history of RAS and the RAS Initiative. Advances in Cancer Research, 2022, 153, 1-27.	5.0	6
3	Classical RAS proteins are not essential for paradoxical ERK activation induced by RAF inhibitors. Proceedings of the National Academy of Sciences of the United States of America, 2022, 119, .	7.1	8
4	RAS at 40: Update from the RAS Initiative. Cancer Discovery, 2022, 12, 895-898.	9.4	12
5	Targeting CD70 in cutaneous T-cell lymphoma using an antibody-drug conjugate in patient-derived xenograft models. Blood Advances, 2022, 6, 2290-2302.	5.2	6
6	Insights into the Cross Talk between Effector and Allosteric Lobes of KRAS from Methyl Conformational Dynamics. Journal of the American Chemical Society, 2022, 144, 4196-4205.	13.7	14
7	Cross-species analysis of LZTR1 loss-of-function mutants demonstrates dependency to RIT1 orthologs. ELife, 2022, 11, .	6.0	8
8	More to the RAS Story: KRAS^{G12C} Inhibition, Resistance Mechanisms, and Moving Beyond KRAS^{G12C}. American Society of Clinical Oncology Educational Book / ASCO American Society of Clinical Oncology Meeting, 2022, 42, 205-217.	3.8	13
9	ARAF protein kinase activates RAS by antagonizing its binding to RASGAP NF1. Molecular Cell, 2022, 82, 2443-2457.e7.	9.7	9
10	Structureâ€“function analysis of the SHOC2â€“MRASâ€“PP1C holophosphatase complex. Nature, 2022, 609, 408-415.	27.8	28
11	Cutaneous T-Cell Lymphoma PDX Drug Screening Platform Identifies Cooperation between Inhibitions of PI3KÎ±/Î´ and HDAC. Journal of Investigative Dermatology, 2021, 141, 364-373.	0.7	17
12	DoMY-Seq: A yeast two-hybridâ€“based technique for precision mapping of proteinâ€“protein interaction motifs. Journal of Biological Chemistry, 2021, 296, 100023.	3.4	5
13	KRAS interaction with RAF1 RAS-binding domain and cysteine-rich domain provides insights into RAS-mediated RAF activation. Nature Communications, 2021, 12, 1176.	12.8	107
14	The metabolic landscape of RAS-driven cancers from biology to therapy. Nature Cancer, 2021, 2, 271-283.	13.2	139
15	Sensitivity of Oncogenic KRAS-Expressing Cells to CDK9 Inhibition. SLAS Discovery, 2021, 26, 922-932.	2.7	1
16	Minor intron retention drives clonal hematopoietic disorders and diverse cancer predisposition. Nature Genetics, 2021, 53, 707-718.	21.4	61
17	RAS-targeted therapies. Nature Reviews Drug Discovery, 2021, , .	46.4	14
18	Targeted mass-spectrometry-based assays enable multiplex quantification of receptor tyrosine kinase, MAP kinase, and AKT signaling. Cell Reports Methods, 2021, 1, 100015.	2.9	10

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19	UDP-glucose pyrophosphorylase 2, a regulator of glycogen synthesis and glycosylation, is critical for pancreatic cancer growth. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, e2103592118.	7.1	14
20	Inhibition of MET Signaling with Ficlatuzumab in Combination with Chemotherapy in Refractory AML: Clinical Outcomes and High-Dimensional Analysis. Blood Cancer Discovery, 2021, 2, 434-449.	5.0	7
21	A Covalent Calmodulin Inhibitor as a Tool to Study Cellular Mechanisms of K-Ras-Driven Stemness. Frontiers in Cell and Developmental Biology, 2021, 9, 665673.	3.7	13
22	RAS interaction with Sin1 is dispensable for mTORC2 assembly and activity. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	7.1	21
23	The RAS GTPase RIT1 compromises mitotic fidelity through spindle assembly checkpoint suppression. Current Biology, 2021, 31, 3915-3924.e9.	3.9	14
24	Sticking it to KRAS: Covalent Inhibitors Enter the Clinic. Cancer Cell, 2020, 37, 3-4.	16.8	41
25	SPRED proteins and their roles in signal transduction, development, and malignancy. Genes and Development, 2020, 34, 1410-1421.	5.9	22
26	Structural Insights into the SPRED1-Neurofibromin-KRAS Complex and Disruption of SPRED1-Neurofibromin Interaction by Oncogenic EGFR. Cell Reports, 2020, 32, 107909.	6.4	41
27	Uncovering a membrane-distal conformation of KRAS available to recruit RAF to the plasma membrane. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 24258-24268.	7.1	34
28	Analysis of RAS protein interactions in living cells reveals a mechanism for pan-RAS depletion by membrane-targeted RAS binders. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 12121-12130.	7.1	19
29	RAS-targeted therapies: is the undruggable drugged?. Nature Reviews Drug Discovery, 2020, 19, 533-552.	46.4	569
30	Biochemical and structural analyses reveal that the tumor suppressor neurofibromin (NF1) forms a high-affinity dimer. Journal of Biological Chemistry, 2020, 295, 1105-1119.	3.4	25
31	ssGSEA score-based Ras dependency indexes derived from gene expression data reveal potential Ras addiction mechanisms with possible clinical implications. Scientific Reports, 2020, 10, 10258.	3.3	105
32	Undermining Glutaminolysis Bolsters Chemotherapy While NRF2 Promotes Chemoresistance in KRAS-Driven Pancreatic Cancers. Cancer Research, 2020, 80, 1630-1643.	0.9	157
33	Romidepsin Plus Liposomal Doxorubicin Is Safe and Effective in Patients with Relapsed or Refractory T-Cell Lymphoma: Results of a Phase I Dose-Escalation Study. Clinical Cancer Research, 2020, 26, 1000-1008.	7.0	26
34	The duality of human oncoproteins: drivers of cancer and congenital disorders. Nature Reviews Cancer, 2020, 20, 383-397.	28.4	44
35	Biochemical and structural analyses reveal that the tumor suppressor neurofibromin (NF1) forms a high-affinity dimer. Journal of Biological Chemistry, 2020, 295, 1105-1119.	3.4	25
36	The molecular functions of RIT1 and its contribution to human disease. Biochemical Journal, 2020, 477, 2755-2770.	3.7	11

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37	Membrane interactions of the globular domain and the hypervariable region of KRAS4b define its unique diffusion behavior. ELife, 2020, 9, .	6.0	23
38	Second harmonic generation detection of Ras conformational changes and discovery of a small molecule binder. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 17290-17297.	7.1	16
39	Blockade of leukemia inhibitory factor as a therapeutic approach to KRAS driven pancreatic cancer. Nature Communications, 2019, 10, 3055.	12.8	81
40	Structures of N-terminally processed KRAS provide insight into the role of N-acetylation. Scientific Reports, 2019, 9, 10512.	3.3	47
41	KRAS G13D sensitivity to neurofibromin-mediated GTP hydrolysis. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 22122-22131.	7.1	85
42	MAP kinase and autophagy pathways cooperate to maintain RAS mutant cancer cell survival. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 4508-4517.	7.1	97
43	Progress in targeting RAS with small molecule drugs. Biochemical Journal, 2019, 476, 365-374.	3.7	53
44	Feasibility of using NF1-GRD and AAV for gene replacement therapy in NF1-associated tumors. Gene Therapy, 2019, 26, 277-286.	4.5	21
45	KRAS Prenylation Is Required for Bivalent Binding with Calmodulin in a Nucleotide-Independent Manner. Biophysical Journal, 2019, 116, 1049-1063.	0.5	41
46	RIT1 oncoproteins escape LZTR1-mediated proteolysis. Science, 2019, 363, 1226-1230.	12.6	66
47	Quantitative biophysical analysis defines key components modulating recruitment of the GTPase KRAS to the plasma membrane. Journal of Biological Chemistry, 2019, 294, 2193-2207.	3.4	38
48	Oncogenic Signaling Pathways in The Cancer Genome Atlas. Cell, 2018, 173, 321-337.e10.	28.9	2,111
49	Differential Effector Engagement by Oncogenic KRAS. Cell Reports, 2018, 22, 1889-1902.	6.4	101
50	c-Raf in KRas Mutant Cancers: A Moving Target. Cancer Cell, 2018, 33, 158-159.	16.8	23
51	Dual gene activation and knockout screen reveals directional dependencies in genetic networks. Nature Biotechnology, 2018, 36, 170-178.	17.5	120
52	SHOC2â€“MRASâ€“PP1 complex positively regulates RAF activity and contributes to Noonan syndrome pathogenesis. Proceedings of the National Academy of Sciences of the United States of America, 2018, 115, E10576-E10585.	7.1	59
53	ETSâ€targeted therapy: can it substitute for MEK inhibitors?. Clinical and Translational Medicine, 2017, 6, 16.	4.0	30
54	Establishment and characterization of an oral tongue squamous cell carcinoma cell line from a never-smoking patient. Oral Oncology, 2017, 69, 1-10.	1.5	8

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55	Drug-tolerant persister cancer cells are vulnerable to GPX4 inhibition. Nature, 2017, 551, 247-250.	27.8	1,043
56	RAS Proteins and Their Regulators in Human Disease. Cell, 2017, 170, 17-33.	28.9	1,262
57	The neurofibromin recruitment factor Spred1 binds to the GAP related domain without affecting Ras inactivation. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 7497-7502.	7.1	50
58	Structural basis of recognition of farnesylated and methylated KRAS4b by PDEÎ´. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, E6766-E6775.	7.1	145
59	K-Ras protein as a drug target. Journal of Molecular Medicine, 2016, 94, 253-258.	3.9	85
60	ETS1 inactivation causes innate drug resistance to EGFR inhibitors. Molecular and Cellular Oncology, 2016, 3, e1078924.	0.7	5
61	Farnesylated and methylated KRAS4b: high yield production of protein suitable for biophysical studies of prenylated protein-lipid interactions. Scientific Reports, 2015, 5, 15916.	3.3	65
62	EGFR inhibition evokes innate drug resistance in lung cancer cells by preventing Akt activity and thus inactivating Ets-1 function. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E3855-63.	7.1	84
63	Enhanced MET Translation and Signaling Sustains K-Rasâ€“Driven Proliferation under Anchorage-Independent Growth Conditions. Cancer Research, 2015, 75, 2851-2862.	0.9	52
64	Ras-GTP dimers activate the Mitogen-Activated Protein Kinase (MAPK) pathway. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, 7996-8001.	7.1	233
65	KRAS as a Therapeutic Target. Clinical Cancer Research, 2015, 21, 1797-1801.	7.0	262
66	Resistance to EGFR-targeted therapy by Ets-1 inactivation. Cell Cycle, 2015, 14, 3211-3212.	2.6	3
67	AKT inactivation causes persistent drug tolerance to EGFR inhibitors. Pharmacological Research, 2015, 102, 132-137.	7.1	29
68	K-Ras Promotes Tumorigenicity through Suppression of Non-canonical Wnt Signaling. Cell, 2015, 163, 1237-1251.	28.9	195
69	Fatty Acid Binding Protein 7 Is a Molecular Marker in Adenoid Cystic Carcinoma of the Salivary Glands: Implications for Clinical Significance. Translational Oncology, 2014, 7, 780-787.	3.7	17
70	Social Interactomes for Enabling Research Communities. Cancer Discovery, 2014, 4, 1265-1268.	9.4	0
71	c-Kit Expression is Rate-Limiting for Stem Cell Factor-Mediated Disease Progression in Adenoid Cystic Carcinoma of the Salivary Glands. Translational Oncology, 2014, 7, 537-545.	3.7	13
72	Adenovirus E4ORF1-Induced MYC Activation Promotes Host Cell Anabolic Glucose Metabolism and Virus Replication. Cell Metabolism, 2014, 19, 694-701.	16.2	209

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73	Development of siRNA Payloads to Target KRAS-Mutant Cancer. Cancer Discovery, 2014, 4, 1182-1197.	9.4	93
74	Targeting RAF kinases for cancer therapy: BRAF-mutated melanoma and beyond. Nature Reviews Cancer, 2014, 14, 455-467.	28.4	683
75	Oncogene Mimicry as a Mechanism of Primary Resistance to BRAF Inhibitors. Cell Reports, 2014, 8, 1037-1048.	6.4	69
76	Dragging Ras Back in the Ring. Cancer Cell, 2014, 25, 272-281.	16.8	707
77	Oncogenic and Wild-type Ras Play Divergent Roles in the Regulation of Mitogen-Activated Protein Kinase Signaling. Cancer Discovery, 2013, 3, 112-123.	9.4	183
78	Single-molecule superresolution imaging allows quantitative analysis of RAF multimer formation and signaling. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 18519-18524.	7.1	153
79	A shared molecular mechanism underlies the human rasopathies Legius syndrome and Neurofibromatosis-1. Genes and Development, 2012, 26, 1421-1426.	5.9	127
80	Basal Subtype and MAPK/ERK Kinase (MEK)-Phosphoinositide 3-Kinase Feedback Signaling Determine Susceptibility of Breast Cancer Cells to MEK Inhibition. Cancer Research, 2009, 69, 565-572.	0.9	340
81	Chapter 1 Ras Signaling and Therapies. Advances in Cancer Research, 2009, 102, 1-17.	5.0	182
82	A Phosphatase Holoenzyme Comprised of Shoc2/Sur8 and the Catalytic Subunit of PP1 Functions as an M-Ras Effector to Modulate Raf Activity. Molecular Cell, 2006, 22, 217-230.	9.7	169
83	Signaling Specificity by Ras Family GTPases Is Determined by the Full Spectrum of Effectors They Regulate. Molecular and Cellular Biology, 2004, 24, 4943-4954.	2.3	287
84	Adenovirus-Mediated p14ARF Gene Transfer in Human Mesothelioma Cells. Journal of the National Cancer Institute, 2000, 92, 636-641.	6.3	120
85	Î²-Catenin regulates expression of cyclin D1 in colon carcinoma cells. Nature, 1999, 398, 422-426.	27.8	3,405
86	An essential role for Rac in Ras transformation. Nature, 1995, 374, 457-459.	27.8	877
87	The 2.2 Ã... crystal structure of the Ras-binding domain of the serine/threonine kinase c-Raf1 in complex with RaplA and a GTP analogue. Nature, 1995, 375, 554-560.	27.8	632
88	Loss of The Normal NF1 Allele from the Bone Marrow of Children with Type 1 Neurofibromatosis and Malignant Myeloid Disorders. New England Journal of Medicine, 1994, 330, 597-601.	27.0	423
89	Somatic mutations in the neurofibromatosis 1 gene in human tumors. Cell, 1992, 69, 275-281.	28.9	365
90	The GTPase superfamily: conserved structure and molecular mechanism. Nature, 1991, 349, 117-127.	27.8	3,349

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91	Differential regulation of rasGAP and neurofibromatosis gene product activities. Nature, 1991, 351, 576-579.	27.8	333
92	Ras and the awd couple. Nature, 1991, 353, 390-391.	27.8	21
93	Suppression of c-ras transformation by GTPase-activating protein. Nature, 1990, 346, 754-756.	27.8	169
94	The GTPase superfamily: a conserved switch for diverse cell functions. Nature, 1990, 348, 125-132.	27.8	2,407
95	The GAP-related domain of the neurofibromatosis type 1 gene product interacts with ras p21. Cell, 1990, 63, 843-849.	28.9	949
96	PDGF Î²-receptor stimulates tyrosine phosphorylation of GAP and association of GAP with a signaling complex. Cell, 1990, 61, 125-133.	28.9	581