Hyunbum Jang

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/2108659/publications.pdf

Version: 2024-02-01

99	4,490	94433	128289
papers	citations	37 h-index	60 g-index

103	103	103	4052
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Novel MAPK/AKT-impairing germline NRAS variant identified in a melanoma-prone family. Familial Cancer, 2022, 21, 347-355.	1.9	1
2	Mechanism of activation and the rewired network: New drug design concepts. Medicinal Research Reviews, 2022, 42, 770-799.	10.5	15
3	How can same-gene mutations promote both cancer and developmental disorders?. Science Advances, 2022, 8, eabm2059.	10.3	29
4	Allostery, and how to define and measure signal transduction. Biophysical Chemistry, 2022, 283, 106766.	2.8	24
5	Allostery: Allosteric Cancer Drivers and Innovative Allosteric Drugs. Journal of Molecular Biology, 2022, 434, 167569.	4.2	26
6	The mechanism of activation of MEK1 by B-Raf and KSR1. Cellular and Molecular Life Sciences, 2022, 79, 281.	5.4	7
7	Open Structural Data in Precision Medicine. Annual Review of Biomedical Data Science, 2022, 5, 95-117.	6.5	7
8	The structural basis of BCR-ABL recruitment of GRB2 in chronic myelogenous leukemia. Biophysical Journal, 2022, 121, 2251-2265.	0.5	9
9	Neurodevelopmental disorders, immunity, and cancer are connected. IScience, 2022, 25, 104492.	4.1	10
10	Conformational Dynamics Allows Sampling of an â€œActive-likeâ€•State by Oncogenic K-Ras-GDP. Journal of Molecular Biology, 2022, 434, 167695.	4.2	13
11	PI3K Driver Mutations: A Biophysical Membrane-Centric Perspective. Cancer Research, 2021, 81, 237-247.	0.9	26
12	Phosphorylation and Driver Mutations in PI3KÎ± and PTEN Autoinhibition. Molecular Cancer Research, 2021, 19, 543-548.	3.4	23
13	A new precision medicine initiative at the dawn of exascale computing. Signal Transduction and Targeted Therapy, 2021, 6, 3.	17.1	31
14	The mechanism of activation of monomeric B-Raf V600E. Computational and Structural Biotechnology Journal, 2021, 19, 3349-3363.	4.1	38
15	Active and Inactive Cdc42 Differ in Their Insert Region Conformational Dynamics. Biophysical Journal, 2021, 120, 306-318.	0.5	20
16	Inhibition of Nonfunctional Ras. Cell Chemical Biology, 2021, 28, 121-133.	5.2	23
17	Drugging multiple same-allele driver mutations in cancer. Expert Opinion on Drug Discovery, 2021, 16, 1-6.	5.0	10
18	Mechanistic Differences of Activation of Rac1^{P29S} and Rac1^{A159V}. Journal of Physical Chemistry B, 2021, 125, 3790-3802.	2.6	9

#	Article	IF	Citations
19	The mechanism of full activation of tumor suppressor PTEN at the phosphoinositide-enriched membrane. IScience, 2021, 24, 102438.	4.1	30
20	The structural basis of Akt PH domain interaction with calmodulin. Biophysical Journal, 2021, 120, 1994-2008.	0.5	10
21	Normal Mode Analysis of KRas4B Reveals Partner Specific Dynamics. Journal of Physical Chemistry B, 2021, 125, 5210-5221.	2.6	7
22	Ras isoform-specific expression, chromatin accessibility, and signaling. Biophysical Reviews, 2021, 13, 489-505.	3.2	14
23	B-Raf autoinhibition in the presence and absence of 14-3-3. Structure, 2021, 29, 768-777.e2.	3.3	26
24	Signaling in the crowded cell. Current Opinion in Structural Biology, 2021, 71, 43-50.	5.7	8
25	The dynamic nature of the K-Ras/calmodulin complex can be altered by oncogenic mutations. Current Opinion in Structural Biology, 2021, 71, 164-170.	5.7	8
26	The mechanism of Raf activation through dimerization. Chemical Science, 2021, 12, 15609-15619.	7.4	15
27	Anticancer drug resistance: An update and perspective. Drug Resistance Updates, 2021, 59, 100796.	14.4	122
28	Autoinhibition can identify rare driver mutations and advise pharmacology. FASEB Journal, 2020, 34, 16-29.	0.5	23
29	Structural Features that Distinguish Inactive and Active PI3K Lipid Kinases. Journal of Molecular Biology, 2020, 432, 5849-5859.	4.2	28
30	Medin Oligomer Membrane Pore Formation: A Potential Mechanism of Vascular Dysfunction. Biophysical Journal, 2020, 118, 2769-2782.	0.5	9
31	The Mystery of Rap1 Suppression of Oncogenic Ras. Trends in Cancer, 2020, 6, 369-379.	7.4	23
32	Nucleotide-Specific Autoinhibition of Full-Length K-Ras4B Identified by Extensive Conformational Sampling. Frontiers in Molecular Biosciences, 2020, 7, 145.	3.5	11
33	SOS1 interacts with Grb2 through regions that induce closed nSH3 conformations. Journal of Chemical Physics, 2020, 153, 045106.	3.0	14
34	PI3K inhibitors: review and new strategies. Chemical Science, 2020, 11, 5855-5865.	7.4	106
35	Are Parallel Proliferation Pathways Redundant?. Trends in Biochemical Sciences, 2020, 45, 554-563.	7.5	21
36	Ras assemblies and signaling at the membrane. Current Opinion in Structural Biology, 2020, 62, 140-148.	5.7	26

#	Article	IF	Citations
37	Oncogenic K-Ras4B Dimerization Enhances Downstream Mitogen-activated Protein Kinase Signaling. Journal of Molecular Biology, 2020, 432, 1199-1215.	4.2	16
38	High-Affinity Interactions of the nSH3/cSH3 Domains of Grb2 with the C-Terminal Proline-Rich Domain of SOS1. Journal of the American Chemical Society, 2020, 142, 3401-3411.	13.7	25
39	The quaternary assembly of KRas4B with Raf-1 at the membrane. Computational and Structural Biotechnology Journal, 2020, 18, 737-748.	4.1	50
40	Protein ensembles link genotype to phenotype. PLoS Computational Biology, 2019, 15, e1006648.	3.2	58
41	The Structural Basis of the Farnesylated and Methylated KRas4B Interaction with Calmodulin. Structure, 2019, 27, 1647-1659.e4.	3.3	30
42	Does Ras Activate Raf and PI3K Allosterically?. Frontiers in Oncology, 2019, 9, 1231.	2.8	41
43	Ca²⁺-Dependent Switch of Calmodulin Interaction Mode with Tandem IQ Motifs in the Scaffolding Protein IQGAP1. Biochemistry, 2019, 58, 4903-4911.	2.5	12
44	The structural basis for Ras activation of PI3KÎ± lipid kinase. Physical Chemistry Chemical Physics, 2019, 21, 12021-12028.	2.8	43
45	The mechanism of PI3KÎ± activation at the atomic level. Chemical Science, 2019, 10, 3671-3680.	7.4	75
46	Review: Precision medicine and driver mutations: Computational methods, functional assays and conformational principles for interpreting cancer drivers. PLoS Computational Biology, 2019, 15, e1006658.	3.2	83
47	Computational Structural Biology: Successes, Future Directions, and Challenges. Molecules, 2019, 24, 637.	3.8	16
48	Why Are Some Driver Mutations Rare?. Trends in Pharmacological Sciences, 2019, 40, 919-929.	8.7	29
49	Precision medicine review: rare driver mutations and their biophysical classification. Biophysical Reviews, 2019, 11, 5-19.	3.2	43
50	Oncogenic KRas mobility in the membrane and signaling response. Seminars in Cancer Biology, 2019, 54, 109-113.	9.6	20
51	Is Nanoclustering essential for all oncogenic KRas pathways? Can it explain why wild-type KRas can inhibit its oncogenic variant?. Seminars in Cancer Biology, 2019, 54, 114-120.	9.6	35
52	Dynamic Protein Allosteric Regulation and Disease. Advances in Experimental Medicine and Biology, 2019, 1163, 25-43.	1.6	13
53	Unraveling the molecular mechanism of interactions of the Rho GTPases Cdc42 and Rac1 with the scaffolding protein IQGAP2. Journal of Biological Chemistry, 2018, 293, 3685-3699.	3.4	36
54	Interaction of Calmodulin with the cSH2 Domain of the p85 Regulatory Subunit. Biochemistry, 2018, 57, 1917-1928.	2.5	10

#	Article	IF	Citations
55	Raf-1 Cysteine-Rich Domain Increases the Affinity of K-Ras/Raf at the Membrane, Promoting MAPK Signaling. Structure, 2018, 26, 513-525.e2.	3.3	60
56	Oncogenic Ras Isoforms Signaling Specificity at the Membrane. Cancer Research, 2018, 78, 593-602.	0.9	96
57	Calmodulin and IQGAP1 activation of PI3KÎ± and Akt in KRAS, HRAS and NRAS-driven cancers. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2018, 1864, 2304-2314.	3.8	16
58	Mutations in LZTR1 drive human disease by dysregulating RAS ubiquitination. Science, 2018, 362, 1177-1182.	12.6	133
59	Autoinhibition in Ras effectors Raf, PI3KÎ±, and RASSF5: a comprehensive review underscoring the challenges in pharmacological intervention. Biophysical Reviews, 2018, 10, 1263-1282.	3.2	40
60	Calmodulin (CaM) Activates PI3KÎ± by Targeting the â€œSoftâ€•CaM-Binding Motifs in Both the nSH2 and cSH2 Domains of p85Î±. Journal of Physical Chemistry B, 2018, 122, 11137-11146.	2.6	15
61	Arl2-Mediated Allosteric Release of Farnesylated KRas4B from Shuttling Factor PDEÎ´. Journal of Physical Chemistry B, 2018, 122, 7503-7513.	2.6	12
62	Allosteric KRas4B Can Modulate SOS1 Fast and Slow Ras Activation Cycles. Biophysical Journal, 2018, 115, 629-641.	0.5	24
63	Calmodulin and PI3K Signaling in KRAS Cancers. Trends in Cancer, 2017, 3, 214-224.	7.4	58
64	A New View of Pathway-Driven Drug Resistance in Tumor Proliferation. Trends in Pharmacological Sciences, 2017, 38, 427-437.	8.7	68
65	The dynamic mechanism of RASSF5 and MST kinase activation by Ras. Physical Chemistry Chemical Physics, 2017, 19, 6470-6480.	2.8	22
66	Flexible-body motions of calmodulin and the farnesylated hypervariable region yield a high-affinity interaction enabling K-Ras4B membrane extraction. Journal of Biological Chemistry, 2017, 292, 12544-12559.	3.4	40
67	Graphite-Templated Amyloid Nanostructures Formed by a Potential Pentapeptide Inhibitor for Alzheimerâ€™s Disease: A Combined Study of Real-Time Atomic Force Microscopy and Molecular Dynamics Simulations. Langmuir, 2017, 33, 6647-6656.	3.5	16
68	Intrinsic protein disorder in oncogenic KRAS signaling. Cellular and Molecular Life Sciences, 2017, 74, 3245-3261.	5.4	45
69	PDEÎ´ Binding to Ras Isoforms Provides a Route to Proper Membrane Localization. Journal of Physical Chemistry B, 2017, 121, 5917-5927.	2.6	26
70	Phosphorylated Calmodulin Promotes PI3K Activation by Binding to the SH2 Domains. Biophysical Journal, 2017, 113, 1956-1967.	0.5	51
71	Drugging Ras GTPase: a comprehensive mechanistic and signaling structural view. Chemical Society Reviews, 2016, 45, 4929-4952.	38.1	150
72	Independent and core pathways in oncogenic KRAS signaling. Expert Review of Proteomics, 2016, 13, 711-716.	3.0	16

#	Article	IF	Citations
73	Inhibitors of Rasâ€“SOS Interactions. ChemMedChem, 2016, 11, 814-821.	3.2	62
74	The Structural Basis of Oncogenic Mutations G12, G13 and Q61 in Small GTPase K-Ras4B. Scientific Reports, 2016, 6, 21949.	3.3	149
75	The higher level of complexity of Kâ€Ras4B activation at the membrane. FASEB Journal, 2016, 30, 1643-1655.	0.5	73
76	Oncogenic KRAS signaling and YAP1/Î²-catenin: Similar cell cycle control in tumor initiation. Seminars in Cell and Developmental Biology, 2016, 58, 79-85.	5.0	54
77	RASSF5: An MST activator and tumor suppressor in vivo but opposite in vitro. Current Opinion in Structural Biology, 2016, 41, 217-224.	5.7	29
78	Membrane-associated Ras dimers are isoform-specific: K-Ras dimers differ from H-Ras dimers. Biochemical Journal, 2016, 473, 1719-1732.	3.7	92
79	Ras Conformational Ensembles, Allostery, and Signaling. Chemical Reviews, 2016, 116, 6607-6665.	47.7	290
80	Comparison of the Conformations of KRAS Isoforms, K-Ras4A and K-Ras4B, Points to Similarities and Significant Differences. Journal of Physical Chemistry B, 2016, 120, 667-679.	2.6	45
81	A New View of Ras Isoforms in Cancers. Cancer Research, 2016, 76, 18-23.	0.9	87
82	K-Ras4B/calmodulin/PI3KÎ±: A promising new adenocarcinoma-specific drug target?. Expert Opinion on Therapeutic Targets, 2016, 20, 831-842.	3.4	29
83	The disordered hypervariable region and the folded catalytic domain of oncogenic K-Ras4B partner in phospholipid binding. Current Opinion in Structural Biology, 2016, 36, 10-17.	5.7	38
84	Computational Methods for Structural and Functional Studies of Alzheimerâ€™s Amyloid Ion Channels. Methods in Molecular Biology, 2016, 1345, 251-268.	0.9	7
85	GTP Binding and Oncogenic Mutations May Attenuate Hypervariable Region (HVR)-Catalytic Domain Interactions in Small GTPase K-Ras4B, Exposing the Effector Binding Site. Journal of Biological Chemistry, 2015, 290, 28887-28900.	3.4	73
86	Plasma membrane regulates Ras signaling networks. Cellular Logistics, 2015, 5, e1136374.	0.9	35
87	High-Affinity Interaction of the K-Ras4B Hypervariable Region with the Ras Active Site. Biophysical Journal, 2015, 109, 2602-2613.	0.5	67
88	Oligomerization and nanocluster organization render specificity. Biological Reviews, 2015, 90, 587-598.	10.4	42
89	GTP-Dependent K-Ras Dimerization. Structure, 2015, 23, 1325-1335.	3.3	187
90	The Key Role of Calmodulin in KRAS-Driven Adenocarcinomas. Molecular Cancer Research, 2015, 13, 1265-1273.	3.4	72

#	Article	IF	Citations
91	Mechanisms of Membrane Binding of Small GTPase K-Ras4B Farnesylated Hypervariable Region. Journal of Biological Chemistry, 2015, 290, 9465-9477.	3.4	98
92	Principles of K-Ras effector organization and the role of oncogenic K-Ras in cancer initiation through G1 cell cycle deregulation. Expert Review of Proteomics, 2015, 12, 669-682.	3.0	37
93	Dynamic multiprotein assemblies shape the spatial structure of cell signaling. Progress in Biophysics and Molecular Biology, 2014, 116, 158-164.	2.9	27
94	Disordered amyloidogenic peptides may insert into the membrane and assemble into common cyclic structural motifs. Chemical Society Reviews, 2014, 43, 6750-6764.	38.1	80
95	The structural basis for cancer treatment decisions. Oncotarget, 2014, 5, 7285-7302.	1.8	43
96	Mechanisms for the Insertion of Toxic, Fibril-like Î²-Amyloid Oligomers into the Membrane. Journal of Chemical Theory and Computation, 2013, 9, 822-833.	5.3	126
97	Familial Alzheimerâ€™s Disease Osaka Mutant (Î”E22) Î²-Barrels Suggest an Explanation for the Different Aβ_{1â€“40/42} Preferred Conformational States Observed by Experiment. Journal of Physical Chemistry B, 2013, 117, 11518-11529.	2.6	29
98	Polymorphism of amyloid Î² peptide in different environments: implications for membrane insertion and pore formation. Soft Matter, 2011, 7, 5267.	2.7	61
99	Truncated Î²-amyloid peptide channels provide an alternative mechanism for Alzheimerâ€™s Disease and Down syndrome. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 6538-6543.	7.1	210