Paul E Hughes

List of Publications by Year
in descending order

33 papers	3,752 citations	218677 26 h-index	395702 33 g-index
33 all docs	33 docs citations	33 times ranked	4232 citing authors

#	Article	IF	Citations
1	Breaking the Integrin Hinge. Journal of Biological Chemistry, 1996, 271, 6571-6574.	3.4	518
2	Suppression of Integrin Activation: A Novel Function of a Ras/Raf-Initiated MAP Kinase Pathway. Cell, 1997, 88, 521-530.	28.9	480
3	Integrin affinity modulation. Trends in Cell Biology, 1998, 8, 359-364.	7.9	416
4	AMG 176, a Selective MCL1 Inhibitor, Is Effective in Hematologic Cancer Models Alone and in Combination with Established Therapies. Cancer Discovery, 2018, 8, 1582-1597.	9.4	310
5	Complementation of dominant suppression implicates CD98 in integrin activation. Nature, 1997, 390, 81-85.	27.8	274
6	Targeted Therapy and Checkpoint Immunotherapy Combinations for the Treatment of Cancer. Trends in Immunology, 2016, 37, 462-476.	6.8	232
7	The inner world of cell adhesion: integrin cytoplasmic domains. Trends in Cell Biology, 1994, 4, 109-112.	7.9	182
8	The Conserved Membrane-proximal Region of an Integrin Cytoplasmic Domain Specifies Ligand Binding Affinity. Journal of Biological Chemistry, 1995, 270, 12411-12417.	3.4	177
9	Identification of a New Biological Function for the Integrin Î±_vÎ²₃: Initiation of Fibronectin Matrix Assembly. Cell Adhesion and Communication, 1996, 4, 149-158.	1.7	99
10	The Small GTP-binding Protein R-Ras Can Influence Integrin Activation by Antagonizing a Ras/Raf-initiated Integrin Suppression Pathway. Molecular Biology of the Cell, 1999, 10, 1799-1809.	2.1	89
11	Evolution of a Highly Selective and Potent 2-(Pyridin-2-yl)-1,3,5-triazine Tie-2 Kinase Inhibitor. Journal of Medicinal Chemistry, 2007, 50, 611-626.	6.4	88
12	The Death Effector Domain of PEA-15 Is Involved in Its Regulation of Integrin Activation. Journal of Biological Chemistry, 1998, 273, 33897-33900.	3.4	87
13	AMG 757, a Half-Life Extended, DLL3-Targeted Bispecific T-Cell Engager, Shows High Potency and Sensitivity in Preclinical Models of Small-Cell Lung Cancer. Clinical Cancer Research, 2021, 27, 1526-1537.	7.0	86
14	Exploiting MCL1 Dependency with Combination MEK + MCL1 Inhibitors Leads to Induction of Apoptosis and Tumor Regression in KRAS-Mutant Nonâ€“Small Cell Lung Cancer. Cancer Discovery, 2018, 8, 1598-1613.	9.4	71
15	Death Effector Domain Protein PEA-15 Potentiates Ras Activation of Extracellular Signal Receptor-activated Kinase by an Adhesion-independent Mechanism. Molecular Biology of the Cell, 2000, 11, 2863-2872.	2.1	66
16	Phospshoinositide 3-Kinase (PI3K)/Mammalian Target of Rapamycin (mTOR) Dual Inhibitors: Discovery and Structureâ€“Activity Relationships of a Series of Quinoline and Quinoxaline Derivatives. Journal of Medicinal Chemistry, 2011, 54, 4735-4751.	6.4	54
17	In Vitro and In Vivo Activity of AMG 337, a Potent and Selective MET Kinase Inhibitor, in MET-Dependent Cancer Models. Molecular Cancer Therapeutics, 2016, 15, 1568-1579.	4.1	50
18	The effector loop and prenylation site of R-Ras are involved in the regulation of integrin function. Oncogene, 2000, 19, 4961-4969.	5.9	45

#	Article	IF	Citations
19	MDM2 antagonists synergize broadly and robustly with compounds targeting fundamental oncogenic signaling pathways. Oncotarget, 2014, 5, 2030-2043.	1.8	45
20	Structure-Based Design of a Novel Series of Potent, Selective Inhibitors of the Class I Phosphatidylinositol 3-Kinases. Journal of Medicinal Chemistry, 2012, 55, 5188-5219.	6.4	43
21	Suppression of Integrin Activation by Activated Ras or Raf Does Not Correlate with Bulk Activation of ERK MAP Kinase. Molecular Biology of the Cell, 2002, 13, 2256-2265.	2.1	42
22	Selective Class I Phosphoinositide 3-Kinase Inhibitors: Optimization of a Series of Pyridyltriazines Leading to the Identification of a Clinical Candidate, AMG 511. Journal of Medicinal Chemistry, 2012, 55, 7796-7816.	6.4	42
23	Structureâ€“Activity Relationships of Phosphoinositide 3-Kinase (PI3K)/Mammalian Target of Rapamycin (mTOR) Dual Inhibitors: Investigations of Various 6,5-Heterocycles to Improve Metabolic Stability. Journal of Medicinal Chemistry, 2011, 54, 5174-5184.	6.4	40
24	Ligand binding and affinity modulation of integrins. Biochemistry and Cell Biology, 1996, 74, 785-798.	2.0	39
25	MAPK pathway inhibition induces MET and GAB1 levels, priming BRAF mutant melanoma for rescue by hepatocyte growth factor. Oncotarget, 2017, 8, 17795-17809.	1.8	35
26	Synthesis, structural analysis, and SAR studies of triazine derivatives as potent, selective Tie-2 inhibitors. Bioorganic and Medicinal Chemistry Letters, 2007, 17, 2886-2889.	2.2	29
27	C-terminal sequences in R-Ras are involved in integrin regulation and in plasma membrane microdomain distribution. Biochemical and Biophysical Research Communications, 2003, 311, 829-838.	2.1	24
28	Synthesis and structureâ€“activity relationships of dual PI3K/mTOR inhibitors based on a 4-amino-6-methyl-1,3,5-triazine sulfonamide scaffold. Bioorganic and Medicinal Chemistry Letters, 2012, 22, 5714-5720.	2.2	24
29	The imidazo[1,2-a]pyridine ring system as a scaffold for potent dual phosphoinositide-3-kinase (PI3K)/mammalian target of rapamycin (mTOR) inhibitors. Bioorganic and Medicinal Chemistry Letters, 2015, 25, 4136-4142.	2.2	19
30	R-Ras C-terminal sequences are sufficient to confer R-Ras specificity toH-Ras. Oncogene, 2002, 21, 4448-4461.	5.9	18
31	[16] R-Ras regulation of integrin function. Methods in Enzymology, 2001, 333, 163-171.	1.0	13
32	Discovery and in Vivo Evaluation of Macrocyclic Mcl-1 Inhibitors Featuring an Î±-Hydroxy Phenylacetic Acid Pharmacophore or Bioisostere. Journal of Medicinal Chemistry, 2019, 62, 10258-10271.	6.4	11
33	Phosphoinositide-3-kinase inhibitors: Evaluation of substituted alcohols as replacements for the piperazine sulfonamide portion of AMG 511. Bioorganic and Medicinal Chemistry Letters, 2014, 24, 5630-5634.	2.2	4