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	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
2	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
3	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
4	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
5	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
6	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
7	Targeted Therapy and Checkpoint Immunotherapy Combinations for the Treatment of Cancer. Trends in Immunology, 2016, 37, 462-476.	6.8	232
8	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
9	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
10	MDM2 antagonists synergize broadly and robustly with compounds targeting fundamental oncogenic signaling pathways. Oncotarget, 2014, 5, 2030-2043.	1.8	45
11	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
12	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
13	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
14	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
15	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
16	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
17	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
18	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

#	Article	IF	Citations
19	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
20	R-Ras C-terminal sequences are sufficient to confer R-Ras specificity toH-Ras. Oncogene, 2002, 21, 4448-4461.	5.9	18
21	[16] R-Ras regulation of integrin function. Methods in Enzymology, 2001, 333, 163-171.	1.0	13
22	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
23	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
24	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
25	Integrin affinity modulation. Trends in Cell Biology, 1998, 8, 359-364.	7.9	416
26	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
27	Suppression of Integrin Activation: A Novel Function of a Ras/Raf-Initiated MAP Kinase Pathway. Cell, 1997, 88, 521-530.	28.9	480
28	Complementation of dominant suppression implicates CD98 in integrin activation. Nature, 1997, 390, 81-85.	27.8	274
29	Ligand binding and affinity modulation of integrins. Biochemistry and Cell Biology, 1996, 74, 785-798.	2.0	39
30	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
31	Breaking the Integrin Hinge. Journal of Biological Chemistry, 1996, 271, 6571-6574.	3.4	518
32	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
33	The inner world of cell adhesion: integrin cytoplasmic domains. Trends in Cell Biology, 1994, 4, 109-112.	7.9	182