

Nicolas Dumaz

List of Publications by Year in descending order

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Version: 2024-02-01

63
papers

3,193
citations

218677

26
h-index

149698

56
g-index

65
all docs

65
docs citations

65
times ranked

4089
citing authors

#	ARTICLE	IF	CITATIONS
1	Novel treatment strategy for NRAS-mutated melanoma through a selective inhibitor of CD147/VEGFR-2 interaction. <i>Oncogene</i> , 2022, 41, 2254-2264.	5.9	5
2	New perspectives on targeting RAF, MEK and ERK in melanoma. <i>Current Opinion in Oncology</i> , 2021, 33, 120-126.	2.4	9
3	RICTOR Affects Melanoma Tumorigenesis and Its Resistance to Targeted Therapy. <i>Biomedicines</i> , 2021, 9, 1498.	3.2	10
4	Mitogen-activated protein kinase blockade in melanoma: intermittent versus continuous therapy, from preclinical to clinical data. <i>Current Opinion in Oncology</i> , 2021, 33, 127-132.	2.4	4
5	Targeted therapies in melanoma beyond BRAF: targeting NRAS-mutated and KIT-mutated melanoma. <i>Current Opinion in Oncology</i> , 2020, 32, 79-84.	2.4	25
6	Mechanisms of resistance and predictive biomarkers of response to targeted therapies and immunotherapies in metastatic melanoma. <i>Current Opinion in Oncology</i> , 2020, 32, 91-97.	2.4	7
7	A Melanoma-Tailored Next-Generation Sequencing Panel Coupled with a Comprehensive Analysis to Improve Routine Melanoma Genotyping. <i>Targeted Oncology</i> , 2020, 15, 759-771.	3.6	2
8	FGF2 Induces Resistance to Nilotinib through MAPK Pathway Activation in KIT Mutated Melanoma. <i>Cancers</i> , 2020, 12, 1062.	3.7	7
9	Atypical BRAF and NRAS Mutations in Mucosal Melanoma. <i>Cancers</i> , 2019, 11, 1133.	3.7	47
10	Vismodegib resistant mutations are not selected in multifocal relapses of locally advanced basal cell carcinoma after vismodegib discontinuation. <i>Journal of the European Academy of Dermatology and Venereology</i> , 2019, 33, e422-e424.	2.4	1
11	Baseline Genomic Features in BRAFV600-Mutated Metastatic Melanoma Patients Treated with BRAF Inhibitor + MEK Inhibitor in Routine Care. <i>Cancers</i> , 2019, 11, 1203.	3.7	10
12	490 The role of PDE4D in resistance to targeted therapy in melanoma. <i>Journal of Investigative Dermatology</i> , 2019, 139, S299.	0.7	0
13	RASA1 loss in a BRAF-mutated Langerhans cell sarcoma: a mechanism of resistance to BRAF inhibitor. <i>Annals of Oncology</i> , 2019, 30, 1170-1172.	1.2	12
14	Large expert-curated database for benchmarking document similarity detection in biomedical literature search. <i>Database: the Journal of Biological Databases and Curation</i> , 2019, 2019, .	3.0	15
15	A targeted genomic alteration analysis predicts survival of melanoma patients under BRAF inhibitors. <i>Oncotarget</i> , 2019, 10, 1669-1687.	1.8	12
16	The role of RICTOR downstream of receptor tyrosine kinase in cancers. <i>Molecular Cancer</i> , 2018, 17, 39.	19.2	42
17	STAT3 Mediates Nilotinib Response in KIT-Altered Melanoma: A Phase II Multicenter Trial of the French Skin Cancer Network. <i>Journal of Investigative Dermatology</i> , 2018, 138, 58-67.	0.7	47
18	A targeted genomic analysis uncovered a large spectrum of acquired resistance mechanisms to BRAF inhibitor therapy in metastatic melanoma patients. <i>Annals of Oncology</i> , 2018, 29, iii25-iii26.	1.2	0

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19	SMO mutations do not seem to drive multifocal relapse of locally advanced basal cell carcinoma after vismodegib discontinuation.. Journal of Clinical Oncology, 2018, 36, e21559-e21559.	1.6	0
20	Modulation of signaling through GPCR-cAMP-PKA pathways by PDE4 depends on stimulus intensity: Possible implications for the pathogenesis of acrodysostosis without hormone resistance. Molecular and Cellular Endocrinology, 2017, 442, 1-11.	3.2	13
21	PDE4D promotes FAK-mediated cell invasion in BRAF-mutated melanoma. Oncogene, 2017, 36, 3252-3262.	5.9	25
22	533 PDE4D is a therapeutic target in melanoma. Journal of Investigative Dermatology, 2017, 137, S283.	0.7	1
23	Mutations causing acrodysostosis-2 facilitate activation of phosphodiesterase 4D3. Human Molecular Genetics, 2017, 26, 3883-3894.	2.9	17
24	Phospho-proteomic analyses of B-Raf protein complexes reveal new regulatory principles. Oncotarget, 2016, 7, 26628-26652.	1.8	25
25	TERT promoter mutations in melanoma render TERT expression dependent on MAPK pathway activation. Oncotarget, 2016, 7, 53127-53136.	1.8	54
26	Association of Vemurafenib and Pipobroman Enhances BRAF-CRAF Dimerization in Squamous Cell Carcinoma. Journal of Investigative Dermatology, 2016, 136, 1302-1305.	0.7	1
27	Hypoxia and MITF regulate KIT oncogenic properties in melanocytes. Oncogene, 2016, 35, 5070-5077.	5.9	5
28	<i>PARKIN</i> Inactivation Links Parkinson's Disease to Melanoma. Journal of the National Cancer Institute, 2016, 108, djv340.	6.3	56
29	Validation of a preclinical model for assessment of drug efficacy in melanoma. Oncotarget, 2016, 7, 13069-13081.	1.8	12
30	RICTOR involvement in the PI3K/AKT pathway regulation in melanocytes and melanoma. Oncotarget, 2015, 6, 28120-28131.	1.8	26
31	Driver KIT mutations in melanoma cluster in four hotspots. Melanoma Research, 2015, 25, 88-90.	1.2	11
32	Phase II multicentric uncontrolled national trial assessing the efficacy of nilotinib in the treatment of advanced melanomas with c-KIT mutation or amplification: Results of the pharmacodynamic study.. Journal of Clinical Oncology, 2015, 33, e20062-e20062.	1.6	1
33	A New KIT Mutation (N505I) in Acral Melanoma Confers Constitutive Signaling, Favors Tumorigenic Properties, and Is Sensitive to Imatinib. Journal of Investigative Dermatology, 2014, 134, 1473-1476.	0.7	4
34	A Large French Case-Control Study Emphasizes the Role of Rare <i>Mc1R</i> Variants in Melanoma Risk. BioMed Research International, 2014, 2014, 1-10.	1.9	19
35	Phase II multicentric uncontrolled national trial assessing the efficacy of nilotinib in the treatment of advanced melanomas with c-KIT mutation or amplification.. Journal of Clinical Oncology, 2014, 32, 9032-9032.	1.6	4
36	Oncogene abnormalities in a series of primary melanomas of the sinonasal tract: NRAS mutations and cyclin D1 amplification are more frequent than KIT or BRAF mutations. Human Pathology, 2013, 44, 1902-1911.	2.0	54

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37	Genetic variation at <i>KIT</i> locus may predispose to melanoma. <i>Pigment Cell and Melanoma Research</i> , 2013, 26, 88-96.	3.3	5
38	Skin Tumors Induced by Sorafenib; Paradoxical RAS-RAF Pathway Activation and Oncogenic Mutations of <i>HRAS</i> , <i>TP53</i> , and <i>TGFBR1</i> . <i>Clinical Cancer Research</i> , 2012, 18, 263-272.	7.0	119
39	130 INVTED TKI's, BRAF Inhibitors and the Problem of New Toxicities Such as Keratoacanthoma and Induction of Invasive SCC. <i>European Journal of Cancer</i> , 2011, 47, S32.	2.8	0
40	ERK and PDE4 cooperate to induce RAF isoform switching in melanoma. <i>Nature Structural and Molecular Biology</i> , 2011, 18, 584-591.	8.2	81
41	Mechanism of RAF isoform switching induced by oncogenic RAS in melanoma. <i>Small GTPases</i> , 2011, 2, 289-292.	1.6	30
42	c-Kit mutants require hypoxia-inducible factor 1 α to transform melanocytes. <i>Oncogene</i> , 2010, 29, 227-236.	5.9	70
43	Inhibition of the Proprotein Convertases Represses the Invasiveness of Human Primary Melanoma Cells with Altered p53, CDKN2A and N-Ras Genes. <i>PLoS ONE</i> , 2010, 5, e9992.	2.5	16
44	Abstract 2341: The Subtilisin-like proprotein convertases blockade inhibits the invasiveness of human primary melanoma with altered P53, CDKN2A and N-Ras genes. , 2010, , .		0
45	Recent discoveries in the genetics of melanoma and their therapeutic implications. <i>Archivum Immunologiae Et Therapiae Experimentalis</i> , 2007, 55, 363-372.	2.3	19
46	In Melanoma, RAS Mutations Are Accompanied by Switching Signaling from BRAF to CRAF and Disrupted Cyclic AMP Signaling. <i>Cancer Research</i> , 2006, 66, 9483-9491.	0.9	271
47	Integrating signals between cAMP and the RAS/RAF/MEK/ERK signalling pathways. Based on The Anniversary Prize of the Gesellschaft fur Biochemie und Molekularbiologie Lecture delivered on 5 July 2003 at the Special FEBS Meeting in Brussels. <i>FEBS Journal</i> , 2005, 272, 3491-3504.	4.7	274
48	Raf Phosphorylation. <i>Molecular Cell</i> , 2005, 17, 164-166.	9.7	17
49	Protein Kinase A Blocks Raf-1 Activity by Stimulating 14-3-3 Binding and Blocking Raf-1 Interaction with Ras. <i>Journal of Biological Chemistry</i> , 2003, 278, 29819-29823.	3.4	224
50	Cyclic AMP Blocks Cell Growth through Raf-1-Dependent and Raf-1-Independent Mechanisms. <i>Molecular and Cellular Biology</i> , 2002, 22, 3717-3728.	2.3	71
51	Phosphorylation of murine double minute clone 2 (MDM2) protein at serine-267 by protein kinase CK2 in vitro and in cultured cells. <i>Biochemical Journal</i> , 2001, 355, 347-356.	3.7	37
52	Critical roles for the serine 20, but not the serine 15, phosphorylation site and for the polyproline domain in regulating p53 turnover. <i>Biochemical Journal</i> , 2001, 359, 459-464.	3.7	60
53	Critical roles for the serine 20, but not the serine 15, phosphorylation site and for the polyproline domain in regulating p53 turnover. <i>Biochemical Journal</i> , 2001, 359, 459.	3.7	53
54	Phosphorylation of murine double minute clone 2 (MDM2) protein at serine-267 by protein kinase CK2 in vitro and in cultured cells. <i>Biochemical Journal</i> , 2001, 355, 347.	3.7	27

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55	Phosphorylation of murine p53, but not human p53, by MAP kinase in vitro and in cultured cells highlights species-dependent variation in post-translational modification. <i>Oncogene</i> , 1999, 18, 7602-7607.	5.9	11
56	Protein kinase CK1 is a p53-threonine 18 kinase which requires prior phosphorylation of serine 15. <i>FEBS Letters</i> , 1999, 463, 312-316.	2.8	119
57	Serine15 phosphorylation stimulates p53 transactivation but does not directly influence interaction with HDM2. <i>EMBO Journal</i> , 1999, 18, 7002-7010.	7.8	390
58	Recovery of the normal p53 response after UV treatment in DNA repair- deficient fibroblasts by retroviral-mediated correction with the XPD gene. <i>Carcinogenesis</i> , 1998, 19, 1701-1704.	2.8	23
59	The role of UV-B light in skin carcinogenesis through the analysis of p53 mutations in squamous cell carcinomas of hairless mice. <i>Carcinogenesis</i> , 1997, 18, 897-904.	2.8	139
60	Prolonged p53 protein accumulation in trichothiodystrophy fibroblasts dependent on unrepaired pyrimidine dimers on the transcribed strands of cellular genes. <i>Molecular Carcinogenesis</i> , 1997, 20, 340-347.	2.7	59
61	The specificity of p53 mutation spectra in sunlight induced human cancers. <i>Journal of Photochemistry and Photobiology B: Biology</i> , 1995, 28, 115-124.	3.8	137
62	Can we predict solar ultraviolet radiation as the causal event in human tumours by analysing the mutation spectra of the p53 gene?. <i>Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis</i> , 1994, 307, 375-386.	1.0	92
63	Specific UV-induced mutation spectrum in the p53 gene of skin tumors from DNA-repair-deficient xeroderma pigmentosum patients.. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 1993, 90, 10529-10533.	7.1	263