Annick MÃ¼hlethaler-Mottet

List of Publications by Year
in descending order

28 papers	1,486 citations	361413 20 h-index	552781 26 g-index
28 all docs	28 docs citations	28 times ranked	2132 citing authors

#	Article	IF	Citations
1	TWIST1 expression is associated with high-risk neuroblastoma and promotes primary and metastatic tumor growth. Communications Biology, 2022, 5, 42.	4.4	1
2	The noradrenergic profile of plasma metanephrine in neuroblastoma patients is reproduced in xenograft mice models and arise from PNMT downregulation. Oncotarget, 2021, 12, 49-60.	1.8	2
3	Frequency and Prognostic Impact of ALK Amplifications and Mutations in the European Neuroblastoma Study Group (SIOPEN) High-Risk Neuroblastoma Trial (HR-NBL1). Journal of Clinical Oncology, 2021, 39, 3377-3390.	1.6	30
4	Expression of the Neuroblastoma-Associated ALK-F1174L Activating Mutation During Embryogenesis Impairs the Differentiation of Neural Crest Progenitors in Sympathetic Ganglia. Frontiers in Oncology, 2019, 9, 275.	2.8	10
5	Aldehyde dehydrogenase activity plays a Key role in the aggressive phenotype of neuroblastoma. BMC Cancer, 2016, 16, 781.	2.6	44
6	The CXCR4/CXCR7/CXCL12 Axis Is Involved in a Secondary but Complex Control of Neuroblastoma Metastatic Cell Homing. PLoS ONE, 2015, 10, e0125616.	2.5	26
7	Wild-type ALK and activating ALK-R1275Q and ALK-F1174L mutations upregulate Myc and initiate tumor formation in murine neural crest progenitor cells. Oncotarget, 2014, 5, 4452-4466.	1.8	32
8	Abstract B59: Wild-type ALK and both ALK-R1275Q and ALK-F1174L activating mutations display a strong oncogenic activity in vivo in murine neural crest progenitor cells via cooperation with c-myc. , 2014, , .		0
9	Involvement of the CXCR7/CXCR4/CXCL12 Axis in the Malignant Progression of Human Neuroblastoma. PLoS ONE, 2012, 7, e43665.	2.5	58
10	Functional Sphere Profiling Reveals the Complexity of Neuroblastoma Tumor-Initiating Cell Model. Neoplasia, 2011, 13, 991-IN30.	5.3	61
11	Individual caspase-10 isoforms play distinct and opposing roles in the initiation of death receptor-mediated tumour cell apoptosis. Cell Death and Disease, 2011, 2, e125-e125.	6.3	26
12	Fenretinide-induced caspase-8 activation and apoptosis in an established model of metastatic neuroblastoma. BMC Cancer, 2009, 9, 97.	2.6	13
13	Complex molecular mechanisms cooperate to mediate histone deacetylase inhibitors anti-tumour activity in neuroblastoma cells. Molecular Cancer, 2008, 7, 55.	19.2	54
14	The Chemokine Receptor CXCR4 Strongly Promotes Neuroblastoma Primary Tumour and Metastatic Growth, but not Invasion. PLoS ONE, 2007, 2, e1016.	2.5	52
15	Histone deacetylase inhibitors strongly sensitise neuroblastoma cells to TRAIL-induced apoptosis by a caspases-dependent increase of the pro- to anti-apoptotic proteins ratio. BMC Cancer, 2006, 6, 214.	2.6	40
16	Molecular cytogenetic characterization of doxorubicin-resistant neuroblastoma cell lines: Evidence that acquired multidrug resistance results from a unique large amplification of the 7q21 region. Genes Chromosomes and Cancer, 2006, 45, 495-508.	2.8	18
17	In vivoechographic evidence of tumoral vascularization and microenvironment interactions in metastatic orthotopic human neuroblastoma xenografts. International Journal of Cancer, 2005, 113, 881-890.	5.1	21
18	The S Box of Major Histocompatibility Complex Class II Promoters Is a Key Determinant for Recruitment of the Transcriptional Co-activator CIITA. Journal of Biological Chemistry, 2004, 279, 40529-40535.	3.4	25

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19	Drug-mediated sensitization to TRAIL-induced apoptosis in caspase-8-complemented neuroblastoma cells proceeds via activation of intrinsic and extrinsic pathways and caspase-dependent cleavage of XIAP, Bcl-xL and RIP. Oncogene, 2004, 23, 5415-5425.	5.9	66
20	Restoration of TRAIL-Induced Apoptosis in a Caspase-8-Deficient Neuroblastoma Cell Line by Stable Re-expression of Caspase-8. Annals of the New York Academy of Sciences, 2003, 1010, 195-199.	3.8	25
21	CIITA and the MHCII Enhanceosome in the Regulation of MHCII Expression. Current Genomics, 2003, 4, 343-363.	1.6	1
22	Analysis of the Sequence Polymorphism within Class II Transactivator Gene Promoters. Experimental and Clinical Immunogenetics, 2001, 18, 199-205.	1.2	10
23	Maturation of Dendritic Cells Is Accompanied by Rapid Transcriptional Silencing of Class II Transactivator (Ciita) Expression. Journal of Experimental Medicine, 2001, 194, 379-392.	8.5	142
24	Lessons from the bare lymphocyte syndrome: molecular mechanisms regulating MHC class II expression. Immunological Reviews, 2000, 178, 148-165.	6.0	75
25	CIITA is a transcriptional coactivator that is recruited to MHC class II promoters by multiple synergistic interactions with an enhanceosome complex. Genes and Development, 2000, 14, 1156-1166.	5.9	275
26	CIITA-induced occupation of MHC class II promoters is independent of the cooperative stabilization of the promoter-bound multi-protein complexes. International Immunology, 1999, 11, 461-469.	4.0	26
27	The molecular basis of MHC class II deficiency and transcriptional control of MHC class II gene expression. Microbes and Infection, 1999, 1, 839-846.	1.9	23
28	Activation of the MHC Class II Transactivator CIITA by Interferon-Î³ Requires Cooperative Interaction between Stat1 and USF-1. Immunity, 1998, 8, 157-166.	14.3	330