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List of Publications by Year
in descending order

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28 papers	3,454 citations	361413 20 h-index	526287 27 g-index
28 all docs	28 docs citations	28 times ranked	5451 citing authors

#	Article	IF	Citations
1	Genome-Wide Analysis of c-MYC-Regulated mRNAs and miRNAs and c-MYC DNA-Binding by Next-Generation Sequencing. Methods in Molecular Biology, 2021, 2318, 119-160.	0.9	0
2	Targeting c-MYC through Interference with NAMPT and SIRT1 and Their Association to Oncogenic Drivers in Murine Serrated Intestinal Tumorigenesis. Neoplasia, 2019, 21, 974-988.	5.3	9
3	The c-MYC/NAMPT/SIRT1 feedback loop is activated in early classical and serrated route colorectal cancer and represents a therapeutic target. Medical Oncology, 2019, 36, 5.	2.5	19
4	Expression of n-MYC, NAMPT and SIRT1 in Basal Cell Carcinomas and their Cells of Origin. Acta Dermato-Venereologica, 2018, 99, 63-71.	1.3	4
5	Genome-Wide Analysis of c-MYC-Regulated mRNAs and miRNAs, and c-MYC DNA Binding by Next-Generation Sequencing. Methods in Molecular Biology, 2013, 1012, 145-185.	0.9	6
6	AP4 is a mediator of epithelialâ€“mesenchymal transition and metastasis in colorectal cancer. Journal of Experimental Medicine, 2013, 210, 1331-1350.	8.5	136
7	AP4 is a mediator of epithelialâ€“mesenchymal transition and metastasis in colorectal cancer. Journal of Cell Biology, 2013, 201, 2017OIA33.	5.2	1
8	The c-MYC oncoprotein, the NAMPT enzyme, the SIRT1-inhibitor DBC1, and the SIRT1 deacetylase form a positive feedback loop. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, E187-96.	7.1	226
9	c-MYC and SIRT1 locked in a vicious cycle. Oncotarget, 2012, 3, 112-113.	1.8	18
10	Up-regulation of c-MYC and SIRT1 expression correlates with malignant transformation in the serrated route to colorectal cancer. Oncotarget, 2012, 3, 1182-1193.	1.8	72
11	miR-34 and SNAIL form a double-negative feedback loop to regulate epithelial-mesenchymal transitions. Cell Cycle, 2011, 10, 4256-4271.	2.6	539
12	OTT-MAL Is a Deregulated Activator of Serum Response Factor-Dependent Gene Expression. Molecular and Cellular Biology, 2008, 28, 6171-6181.	2.3	38
13	AP4 encodes a c-MYC-inducible repressor of p21. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 15046-15051.	7.1	140
14	Digital Karyotyping Reveals Frequent Inactivation of the dystrophin/DMD Gene in Malignant Melanoma. Cell Cycle, 2007, 6, 189-198.	2.6	23
15	c-MYC Delays Prometaphase by Direct Transactivation of MAD2 and BubR1: Identification of Mechanisms Underlying c-MYC-Induced DNA Damage and Chromosomal Instability. Cell Cycle, 2007, 6, 339-352.	2.6	85
16	Differential Regulation of microRNAs by p53 Revealed by Massively Parallel Sequencing: miR-34a is a p53 Target That Induces Apoptosis and G1-arrest. Cell Cycle, 2007, 6, 1586-1593.	2.6	859
17	Large-Scale Identification of c-MYC-Associated Proteins Using a Combined TAP/MudPIT Approach. Cell Cycle, 2007, 6, 205-217.	2.6	134
18	Induction of Cullin 7 by DNA damage attenuates p53 function. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 11388-11393.	7.1	28

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19	Inducible microRNA expression by an all-in-one episomal vector system. Nucleic Acids Research, 2006, 34, e119-e119.	14.5	21
20	Functional Epigenomics Identifies Genes Frequently Silenced in Prostate Cancer. Cancer Research, 2005, 65, 4218-4227.	0.9	272
21	Characterization of the c-MYC-regulated transcriptome by SAGE: Identification and analysis of c-MYC target genes. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 6274-6279.	7.1	356
22	Induction of the Cdk inhibitor p21 by LY83583 inhibits tumor cell proliferation in a p53-independent manner. Journal of Clinical Investigation, 2002, 110, 1717-1727.	8.2	59
23	Induction of the Cdk inhibitor p21 by LY83583 inhibits tumor cell proliferation in a p53-independent manner. Journal of Clinical Investigation, 2002, 110, 1717-1727.	8.2	37
24	Characterization of epithelial senescence by serial analysis of gene expression: identification of genes potentially involved in prostate cancer. Cancer Research, 2002, 62, 6255-62.	0.9	66
25	Dominant Lesional T Cell Receptor Rearrangements Persist in Relapsing Psoriasis but are Absent from Nonlesional Skin: Evidence for a Stable Antigen-Specific Pathogenic T Cell Response in Psoriasis Vulgaris. Journal of Investigative Dermatology, 2001, 117, 1296-1301.	0.7	73
26	Analysis of the TCRBV Repertoire of T Cells in Normal, Human Skin: Evidence for a Restricted Diversity. Journal of Investigative Dermatology, 2000, 115, 66-73.	0.7	10
27	Selection of conserved TCR VDJ rearrangements in chronic psoriatic plaques indicates a common antigen in psoriasis vulgaris. European Journal of Immunology, 1999, 29, 3360-3368.	2.9	90
28	T lymphocytes derived from skin lesions of patients with psoriasis vulgaris express a novel cytokine pattern that is distinct from that of T helper type 1 and T helper type 2 cells. European Journal of Immunology, 1994, 24, 2377-2382.	2.9	133