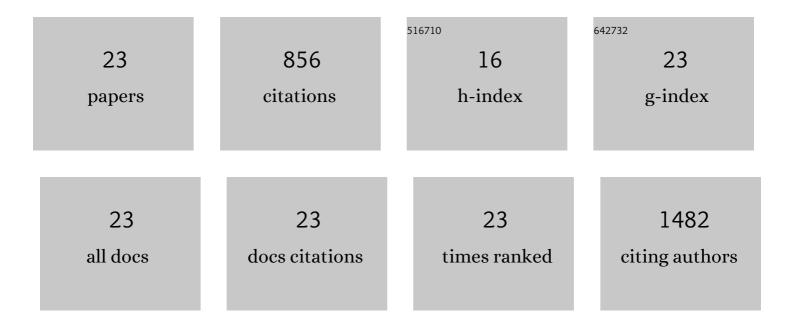
Mariusz L Hartman

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	MITF in melanoma: mechanisms behind its expression and activity. Cellular and Molecular Life Sciences, 2015, 72, 1249-1260.	5.4	229
2	Pro-Survival Role of MITF in Melanoma. Journal of Investigative Dermatology, 2015, 135, 352-358.	0.7	72
3	Anti-apoptotic proteins on guard of melanoma cell survival. Cancer Letters, 2013, 331, 24-34.	7.2	67
4	BCL-w: apoptotic and non-apoptotic role in health and disease. Cell Death and Disease, 2020, 11, 260.	6.3	53
5	Natural Compounds' Activity against Cancer Stem-Like or Fast-Cycling Melanoma Cells. PLoS ONE, 2014, 9, e90783.	2.5	44
6	Dissecting Mechanisms of Melanoma Resistance to BRAF and MEK Inhibitors Revealed Genetic and Non-Genetic Patient- and Drug-Specific Alterations and Remarkable Phenotypic Plasticity. Cells, 2020, 9, 142.	4.1	41
7	Non-Apoptotic Cell Death Signaling Pathways in Melanoma. International Journal of Molecular Sciences, 2020, 21, 2980.	4.1	39
8	Wholeâ€exome sequencing reveals novel genetic variants associated with diverse phenotypes of melanoma cells. Molecular Carcinogenesis, 2019, 58, 588-602.	2.7	37
9	Inhibitors of HSP90 in melanoma. Apoptosis: an International Journal on Programmed Cell Death, 2020, 25, 12-28.	4.9	35
10	Vemurafenib and trametinib reduce expression of CTGF and IL-8 in V600EBRAF melanoma cells. Laboratory Investigation, 2017, 97, 217-227.	3.7	28
11	Plasticity of Drug-NaÃ ⁻ ve and Vemurafenib- or Trametinib-Resistant Melanoma Cells in Execution of Differentiation/Pigmentation Program. Journal of Oncology, 2019, 2019, 1-15.	1.3	26
12	Gene Expression Profiling Identifies Microphthalmia-Associated Transcription Factor (MITF) and Dickkopf-1 (DKK1) as Regulators of Microenvironment-Driven Alterations in Melanoma Phenotype. PLoS ONE, 2014, 9, e95157.	2.5	26
13	Pro-apoptotic Activity of BH3-only Proteins and BH3 Mimetics: from Theory to Potential Cancer Therapy. Anti-Cancer Agents in Medicinal Chemistry, 2012, 12, 966-981.	1.7	24
14	Parthenolide enhances dacarbazine activity against melanoma cells. Anti-Cancer Drugs, 2013, 24, 835-845.	1.4	23
15	Phenotypic diversity of patient-derived melanoma populations in stem cell medium. Laboratory Investigation, 2015, 95, 672-683.	3.7	22
16	Physiologically Relevant Oxygen Concentration (6% O2) as an Important Component of the Microenvironment Impacting Melanoma Phenotype and Melanoma Response to Targeted Therapeutics In Vitro. International Journal of Molecular Sciences, 2019, 20, 4203.	4.1	17
17	Parthenolide induces MITF-M downregulation and senescence in patient-derived MITF-Mhigh melanoma cell populations. Oncotarget, 2016, 7, 9026-9040.	1.8	16
18	17-Aminogeldanamycin selectively diminishes IRE1α-XBP1s pathway activity and cooperatively induces apoptosis with MEK1/2 and BRAFV600E inhibitors in melanoma cells of different genetic subtypes. Apoptosis: an International Journal on Programmed Cell Death, 2019, 24, 596-611.	4.9	14

#	Article	IF	CITATIONS
19	BH3 mimetics potentiate pro-apoptotic activity of encorafenib in BRAFV600E melanoma cells. Cancer Letters, 2021, 499, 122-136.	7.2	13
20	MCL-1, BCL-XL and MITF Are Diversely Employed in Adaptive Response of Melanoma Cells to Changes in Microenvironment. PLoS ONE, 2015, 10, e0128796.	2.5	10
21	Exogenous growth factors bFGF, EGF and HGF do not influence viability and phenotype of V600EBRAF melanoma cells and their response to vemurafenib and trametinib in vitro. PLoS ONE, 2017, 12, e0183498.	2.5	10
22	17-Aminogeldanamycin Inhibits Constitutive Nuclear Factor-Kappa B (NF-κB) Activity in Patient-Derived Melanoma Cell Lines. International Journal of Molecular Sciences, 2020, 21, 3749.	4.1	7
23	TYRP1 mRNA level is stable and MITF-M-independent in drug-naÃ⁻ve, vemurafenib- and trametinib-resistant BRAFV600E melanoma cells. Archives of Dermatological Research, 2020, 312, 385-392.	1.9	3