## Imanol Arozarena

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Usefulness of an immunohistochemical score in advanced pancreatic neuroendocrine tumors treated with CAPTEM or everolimus. Pancreatology, 2021, 21, 215-223.	1.1	2
2	Understanding the Molecular Mechanism of miR-877-3p Could Provide Potential Biomarkers and Therapeutic Targets in Squamous Cell Carcinoma of the Cervix. Cancers, 2021, 13, 1739.	3.7	4
3	Identification of a Dexamethasone Mediated Radioprotection Mechanism Reveals New Therapeutic Vulnerabilities in Glioblastoma. Cancers, 2021, 13, 361.	3.7	8
4	Novel Insights into the Role of the Mineralocorticoid Receptor in Human Glioblastoma. International Journal of Molecular Sciences, 2021, 22, 11656.	4.1	3
5	Tyrosine Kinase Inhibitors in Adult Glioblastoma: An (Un)Closed Chapter?. Cancers, 2021, 13, 5799.	3.7	18
6	Cooperative behaviour and phenotype plasticity evolve during melanoma progression. Pigment Cell and Melanoma Research, 2020, 33, 695-708.	3.3	18
7	Phenotype plasticity as enabler ofÂmelanoma progression and therapyÂresistance. Nature Reviews Cancer, 2019, 19, 377-391.	28.4	262
8	RAS at the Golgi antagonizes malignant transformation through PTPRÎ <sup>®</sup> -mediated inhibition of ERK activation. Nature Communications, 2018, 9, 3595.	12.8	18
9	Targeting invasive properties of melanoma cells. FEBS Journal, 2017, 284, 2148-2162.	4.7	36
10	An adaptive signaling network in melanoma inflammatory niches confers tolerance to MAPK signaling inhibition. Journal of Experimental Medicine, 2017, 214, 1691-1710.	8.5	71
11	PDL1 Signals through Conserved Sequence Motifs to Overcome Interferon-Mediated Cytotoxicity. Cell Reports, 2017, 20, 1818-1829.	6.4	220
12	Targeting endothelin receptor signalling overcomes heterogeneity driven therapy failure. EMBO Molecular Medicine, 2017, 9, 1011-1029.	6.9	63
13	Overcoming resistance to BRAF inhibitors. Annals of Translational Medicine, 2017, 5, 387-387.	1.7	109
14	Report from the II Melanoma Translational Meeting of the Spanish Melanoma Group (GEM). Annals of Translational Medicine, 2017, 5, 390-390.	1.7	0
15	Glucose availability controls ATF4-mediated MITF suppression to drive melanoma cell growth. Oncotarget, 2017, 8, 32946-32959.	1.8	46
16	The Complexity of the ERK/MAP-Kinase Pathway and the Treatment of Melanoma Skin Cancer. Frontiers in Cell and Developmental Biology, 2016, 4, 33.	3.7	84
17	Inhibiting Drivers of Non-mutational Drug Tolerance Is a Salvage Strategy for Targeted Melanoma Therapy. Cancer Cell, 2016, 29, 270-284.	16.8	198
18	Targeting MITF in the tolerance-phase. Oncotarget, 2016, 7, 54094-54095.	1.8	4

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19	MGMT Expression Predicts PARP-Mediated Resistance to Temozolomide. Molecular Cancer Therapeutics, 2015, 14, 1236-1246.	4.1	36
20	Microphthalmiaâ€associated transcription factor in melanoma development and <scp>MAP</scp> â€kinase pathway targeted therapy. Pigment Cell and Melanoma Research, 2015, 28, 390-406.	3.3	168
21	Differential chemosensitivity to antifolate drugs between RAS and BRAF melanoma cells. Molecular Cancer, 2014, 13, 154.	19.2	2
22	Effect of SMURF2 Targeting on Susceptibility to MEK Inhibitors in Melanoma. Journal of the National Cancer Institute, 2013, 105, 33-46.	6.3	85
23	Oncogenic BRAF Induces Melanoma Cell Invasion by Downregulating the cGMP-Specific Phosphodiesterase PDE5A. Cancer Cell, 2011, 19, 45-57.	16.8	190
24	Ras, an Actor on Many Stages: Posttranslational Modifications, Localization, and Site-Specified Events. Genes and Cancer, 2011, 2, 182-194.	1.9	49
25	Ras Subcellular Localization Defines Extracellular Signal-Regulated Kinase 1 and 2 Substrate Specificity through Distinct Utilization of Scaffold Proteins. Molecular and Cellular Biology, 2009, 29, 1338-1353.	2.3	100
26	FGF-2 protects small cell lung cancer cells from apoptosis through a complex involving PKCÉ›, B-Raf and S6K2. EMBO Journal, 2006, 25, 3078-3088.	7.8	173
27	Distinct Utilization of Effectors and Biological Outcomes Resulting from Site-Specific Ras Activation: Ras Functions in Lipid Rafts and Golgi Complex Are Dispensable for Proliferation and Transformation. Molecular and Cellular Biology, 2006, 26, 100-116.	2.3	110
28	Activation of H-Ras in the Endoplasmic Reticulum by the RasGRF Family Guanine Nucleotide Exchange Factors. Molecular and Cellular Biology, 2004, 24, 1516-1530.	2.3	87
29	Differences on the Inhibitory Specificities of H-Ras, K-Ras, and N-Ras (N17) Dominant Negative Mutants Are Related to Their Membrane Microlocalization. Journal of Biological Chemistry, 2003, 278, 4572-4581.	3.4	102
30	Maintenance of Cdc42 GDP-bound State by Rho-GDI Inhibits MAP Kinase Activation by the Exchange Factor Ras-GRF. Journal of Biological Chemistry, 2001, 276, 21878-21884.	3.4	32
31	H-, K- and N-Ras inhibit myeloid leukemia cell proliferation by a p21WAF1-dependent mechanism. Oncogene, 2000, 19, 783-790.	5.9	53
32	The Rho Family GTPase Cdc42 Regulates the Activation of Ras/MAP Kinase by the Exchange Factor Ras-GRF. Journal of Biological Chemistry, 2000, 275, 26441-26448.	3.4	40