Lucia Pirisi

List of Publications by Year
in descending order

31	908	471509 17	477307 29
papers	citations	h-index	g-index

31	31	31	1141
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	HPV-inactive cell populations arise from HPV16-transformed human keratinocytes after p53 knockout. Virology, 2021, 554, 9-16.	2.4	8
2	Establishing a High Throughput Epidermal Spheroid Culture System to Model Keratinocyte Stem Cell Plasticity. Journal of Visualized Experiments, 2021, , .	0.3	0
3	Human papillomavirus-mediated carcinogenesis and tumor progression. Genome Instability & Disease, 2021, 2, 71-91.	1.1	0
4	Self-assembling 3D spheroid cultures of human neonatal keratinocytes have enhanced regenerative properties. Stem Cell Research, 2020, 49, 102048.	0.7	13
5	The Marine Natural Product Manzamine A Inhibits Cervical Cancer by Targeting the SIX1 Protein. Journal of Natural Products, 2020, 83, 286-295.	3.0	31
6	HPV16-transformed human keratinocytes depend on SIX1 expression for proliferation and HPV E6/E7 gene expression. Virology, 2019, 537, 20-30.	2.4	4
7	Stem Cell Properties of Normal Human Keratinocytes Determine Transformation Responses to Human Papillomavirus 16 DNA. Journal of Virology, 2018, 92, .	3.4	7
8	Six1 promotes colorectal cancer growth and metastasis by stimulating angiogenesis and recruiting tumor-associated macrophages. Carcinogenesis, 2017, 38, 281-292.	2.8	60
9	Identification and characterization of HPV-independent cervical cancers. Oncotarget, 2017, 8, 13375-13386.	1.8	82
10	Human papillomavirus status and gene expression profiles of oropharyngeal and oral cancers from European American and African American patients. Head and Neck, 2016, 38, E694-704.	2.0	33
11	Disparity in the Persistence of High-Risk Human Papillomavirus Genotypes Between African American and European American Women of College Age. Journal of Infectious Diseases, 2015, 211, 100-108.	4.0	34
12	Six1 overexpression at early stages of HPV16-mediated transformation of human keratinocytes promotes differentiation resistance and EMT. Virology, 2015, 474, 144-153.	2.4	15
13	The translational significance of epithelialâ€mesenchymal transition in head and neck cancer. Clinical and Translational Medicine, 2014, 3, 60.	4.0	18
14	Six1 promotes epithelial-mesenchymal transition and malignant conversion in human papillomavirus type 16-immortalized human keratinocytes. Carcinogenesis, 2014, 35, 1379-1388.	2.8	36
15	Ski protein levels increase during in vitro progression of HPV16-immortalized human keratinocytes and in cervical cancer. Virology, 2013, 444, 100-108.	2.4	10
16	TGF-Î² regulation of gene expression at early and late stages of HPV16-mediated transformation of human keratinocytes. Virology, 2013, 447, 63-73.	2.4	21
17	Partial loss of Smad signaling during in vitroprogression of HPV16-immortalized human keratinocytes. BMC Cancer, 2013, 13, 424.	2.6	9
18	Mechanisms of decreased expression of transforming growth factor-beta receptor type I at late stages of HPV16-mediated transformation. Cancer Letters, 2009, 282, 177-186.	7.2	16

#	Article	IF	Citations
19	Gene expression changes during HPVâ€mediated carcinogenesis: A comparison between an in vitro cell model and cervical cancer. International Journal of Cancer, 2008, 123, 32-40.	5.1	74
20	NFI is an Essential Positive Transcription Factor for Human Papillomavirus Type 16 Early Gene Expression. The Open Virology Journal, 2007, 1, 33-38.	1.8	6
21	NFI-Ski Interactions Mediate Transforming Growth Factor Î² Modulation of Human Papillomavirus Type 16 Early Gene Expression. Journal of Virology, 2004, 78, 3953-3964.	3.4	39
22	Hormonal and Barrier Methods of Contraception, Oncogenic Human Papillomaviruses, and Cervical Squamous Intraepithelial Lesion Development. Journal of Women's Health and Gender-Based Medicine, 2001, 10, 441-449.	1.5	23
23	Genotyping single nucleotide polymorphisms using intact polymerase chain reaction products by electrospray quadrupole mass spectrometry. Rapid Communications in Mass Spectrometry, 2001, 15, 1752-1759.	1.5	24
24	Unique carboxyl-terminal sequences of wild type and alternatively spliced variant forms of transforming growth factor-Î± precursors mediate specific interactions with ErbB4 and ErbB2. Oncogene, 2000, 19, 3172-3181.	5.9	9
25	Retinoic Acid Resistance at Late Stages of Human Papillomavirus Type 16-Mediated Transformation of Human Keratinocytes Arises Despite Intact Retinoid Signaling and Is Due to a Loss of Sensitivity to Transforming Growth Factor-Î². Virology, 2000, 270, 397-407.	2.4	32
26	Loss of Transforming Growth Factor-Î² (TGF-Î²) Receptor Type I Mediates TGF-Î² Resistance in Human Papillomavirus Type 16-Transformed Human Keratinocytes at Late Stages of in Vitro Progression. Virology, 2000, 270, 408-416.	2.4	23
27	Intimate Partner Violence and Cervical Neoplasia. Journal of Women's Health and Gender-Based Medicine, 2000, 9, 1015-1023.	1.5	67
28	Human keratinocytes and tumor-derived cell lines express alternatively spliced forms of transforming growth factor-Î± mRNA, encoding precursors lacking carboxyl-terminal valine residues. Oncogene, 1999, 18, 5554-5562.	5.9	15
29	Progressive Loss of Sensitivity to Growth Control by Retinoic Acid and Transforming Growth Factor-Beta at Late Stages of Human Papillomavirus Type 16-Initiated Transformation of Human Keratinocytes. Advances in Experimental Medicine and Biology, 1995, 375, 117-135.	1.6	41
30	Retinoic Acid Suppresses Human Papillomavirus Type 16 (HPV16)-Mediated Transformation of Human Keratinocytes and Inhibits the Expression of the HPV16 Oncogenes. Advances in Experimental Medicine and Biology, 1994, 354, 19-35.	1.6	16
31	Continuous cell lines with altered growth and differentiation properties originate after transfection of human keratinocytes with human papillomavirus type 16 DNA. Carcinogenesis, 1988, 9, 1573-1579.	2.8	142