Lucia Pirisi

List of Publications by Year in descending order

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Ι μαι Ριριςι

#	Article	IF	CITATIONS
1	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
2	Identification and characterization of HPV-independent cervical cancers. Oncotarget, 2017, 8, 13375-13386.	1.8	82
3	Gene expression changes during HPVâ€mediated carcinogenesis: A comparison between an <i>in vitro</i> cell model and cervical cancer. International Journal of Cancer, 2008, 123, 32-40.	5.1	74
4	Intimate Partner Violence and Cervical Neoplasia. Journal of Women's Health and Gender-Based Medicine, 2000, 9, 1015-1023.	1.5	67
5	Six1 promotes colorectal cancer growth and metastasis by stimulating angiogenesis and recruiting tumor-associated macrophages. Carcinogenesis, 2017, 38, 281-292.	2.8	60
6	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
7	NFI-Ski Interactions Mediate Transforming Growth Factor β Modulation of Human Papillomavirus Type 16 Early Gene Expression. Journal of Virology, 2004, 78, 3953-3964.	3.4	39
8	Six1 promotes epithelial-mesenchymal transition and malignant conversion in human papillomavirus type 16-immortalized human keratinocytes. Carcinogenesis, 2014, 35, 1379-1388.	2.8	36
9	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
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	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-β. Virology, 2000, 270, 397-407.	2.4	32
12	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
13	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
14	Loss of Transforming Growth Factor-β (TGF-β) Receptor Type I Mediates TGF-β Resistance in Human Papillomavirus Type 16-Transformed Human Keratinocytes at Late Stages of in Vitro Progression. Virology, 2000, 270, 408-416.	2.4	23
15	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
16	TGF-Î ² 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	The translational significance of epithelialâ€mesenchymal transition in head and neck cancer. Clinical and Translational Medicine, 2014, 3, 60.	4.0	18
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

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#	ARTICLE	IF	CITATIONS
19	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
20	Human keratinocytes and tumor-derived cell lines express alternatively spliced forms of transforming growth factor-α mRNA, encoding precursors lacking carboxyl-terminal valine residues. Oncogene, 1999, 18, 5554-5562.	5.9	15
21	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
22	Self-assembling 3D spheroid cultures of human neonatal keratinocytes have enhanced regenerative properties. Stem Cell Research, 2020, 49, 102048.	0.7	13
23	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
24	Unique carboxyl-terminal sequences of wild type and alternatively spliced variant forms of transforming growth factor-1± precursors mediate specific interactions with ErbB4 and ErbB2. Oncogene, 2000, 19, 3172-3181.	5.9	9
25	Partial loss of Smad signaling during in vitroprogression of HPV16-immortalized human keratinocytes. BMC Cancer, 2013, 13, 424.	2.6	9
26	HPV-inactive cell populations arise from HPV16-transformed human keratinocytes after p53 knockout. Virology, 2021, 554, 9-16.	2.4	8
27	Stem Cell Properties of Normal Human Keratinocytes Determine Transformation Responses to Human Papillomavirus 16 DNA. Journal of Virology, 2018, 92, .	3.4	7
28	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
29	HPV16-transformed human keratinocytes depend on SIX1 expression for proliferation and HPV E6/E7 gene expression. Virology, 2019, 537, 20-30.	2.4	4
30	Establishing a High Throughput Epidermal Spheroid Culture System to Model Keratinocyte Stem Cell Plasticity. Journal of Visualized Experiments, 2021, , .	0.3	0
31	Human papillomavirus-mediated carcinogenesis and tumor progression. Genome Instability & Disease, 2021, 2, 71-91.	1.1	0