

Dean S Rosenthal

List of Publications by Year in descending order

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docs citations

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times ranked

2979
citing authors

#	ARTICLE	IF	CITATIONS
1	Employing CRISPR-Cas9 to Generate CD133 Synthetic Lethal Melanoma Stem Cells. International Journal of Molecular Sciences, 2022, 23, 2333.	4.1	4
2	A novel chemo-phenotypic method identifies mixtures of salpn, vitamin D3, and pesticides involved in the development of colorectal and pancreatic cancer. Ecotoxicology and Environmental Safety, 2022, 233, 113330.	6.0	2
3	Hypopigmented burn hypertrophic scar contains melanocytes that can be signaled to re-pigment by synthetic alpha-melanocyte stimulating hormone in vitro. PLoS ONE, 2021, 16, e0248985.	2.5	12
4	Promoter Methylation Status in Pro-opiomelanocortin Does Not Contribute to Dyspigmentation in Hypertrophic Scar. Journal of Burn Care and Research, 2020, 41, 339-346.	0.4	7
5	Inorganic polyphosphate in platelet rich plasma accelerates re-epithelialization in vitro and in vivo. Regenerative Therapy, 2020, 15, 138-148.	3.0	11
6	55 Melanocytes in Hypopigmented Burn Scar Can Be Stimulated to Produce Melanin. Journal of Burn Care and Research, 2020, 41, S36-S37.	0.4	1
7	CD133 Is Associated with Increased Melanoma Cell Survival after Multikinase Inhibition. Journal of Oncology, 2019, 2019, 1-19.	1.3	15
8	Reactive Oxygen Species Scavenging Potential Contributes to Hypertrophic Scar Formation. Journal of Surgical Research, 2019, 244, 312-323.	1.6	19
9	CRISPR-Cas9 Knockdown and Induced Expression of CD133 Reveal Essential Roles in Melanoma Invasion and Metastasis. Cancers, 2019, 11, 1490.	3.7	23
10	Pigmentation Diathesis of Hypertrophic Scar: An Examination of Known Signaling Pathways to Elucidate the Molecular Pathophysiology of Injury-Related Dyschromia. Journal of Burn Care and Research, 2019, 40, 58-71.	0.4	24
11	Treatment Strategies for Hypopigmentation in the Context of Burn Hypertrophic Scars. Plastic and Reconstructive Surgery - Global Open, 2018, 6, e1642.	0.6	10
12	The repurposed anthelmintic mebendazole in combination with trametinib suppresses refractory NRASQ61K melanoma. Oncotarget, 2017, 8, 12576-12595.	1.8	43
13	ROCK inhibitor reduces Myc-induced apoptosis and mediates immortalization of human keratinocytes. Oncotarget, 2016, 7, 66740-66753.	1.8	26
14	Abstract 2501: Combination therapy with mebendazole, trametinib and metformin eliminates recalcitrant NRASQ61K melanoma cells. , 2016, , .		0
15	Abstract 3860: The repurposed anthelmintic mebendazole in combination with trametinib suppresses refractory NRASQ61K melanoma. , 2016, , .		0
16	Id3 induces an Elk-1/caspase-8 dependent apoptotic pathway in squamous carcinoma cells. Cancer Medicine, 2015, 4, 914-924.	2.8	12
17	Inorganic polyphosphates are important for cell survival and motility of human skin keratinocytes. Experimental Dermatology, 2015, 24, 636-639.	2.9	16
18	Inhibitor of differentiation 4 (Id4) stimulates pigmentation in melanoma leading to histiocyte infiltration. Experimental Dermatology, 2015, 24, 101-107.	2.9	9

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19	Abstract 4224: CD133 knockdown sensitizes melanoma to kinase inhibitors. , 2015, , .		1
20	Abstract 5145: Y-27632 inhibits Myc-induced apoptosis and cooperates with Myc to immortalize human keratinocytes. , 2015, , .		0
21	Id2, Id3 and Id4 overcome a Smad7-mediated block in tumorigenesis, generating TGF- β -independent melanoma. Carcinogenesis, 2014, 35, 951-958.	2.8	25
22	Abstract 3891: CD133 is associated with resistance of melanoma to multikinase inhibition. , 2014, , .		1
23	Abstract 825: Id3: Tumor suppressor of squamous cell carcinoma.. , 2013, , .		0
24	Clonal dominance of CD133+ subset population as risk factor in tumor progression and disease recurrence of human cutaneous melanoma. International Journal of Oncology, 2012, 41, 1570-1576.	3.3	23
25	High throughput transcriptomic analysis of the effects of radiation exposure in a mouse model. FASEB Journal, 2012, 26, 774.1.	0.5	0
26	Purification and Characterization of Poly(ADP-Ribosyl)ated DNA Replication/Repair Complexes. Methods in Molecular Biology, 2011, 780, 165-190.	0.9	5
27	Proteomic Analysis of Pathways Involved in Estrogen-Induced Growth and Apoptosis of Breast Cancer Cells. PLoS ONE, 2011, 6, e20410.	2.5	28
28	A Review of the Local Pathophysiologic Bases of Burn Wound Progression. Journal of Burn Care and Research, 2010, 31, 849-873.	0.4	181
29	Sulfur mustard induces apoptosis in lung epithelial cells via a caspase amplification loop. Toxicology, 2010, 271, 94-99.	4.2	25
30	VMY-1-103, a dansylated analog of purvalanol B, induces caspase-3-dependent apoptosis in LNCaP prostate cancer cells. Cancer Biology and Therapy, 2010, 10, 320-325.	3.4	18
31	Smad7 restricts melanoma invasion by restoring N-cadherin expression and establishing heterotypic cell-cell interactions in vivo. Pigment Cell and Melanoma Research, 2010, 23, 795-808.	3.3	24
32	Abstract LB-240: Smad7 blocks melanoma invasion by suppressing n-cadherin cleavage and preserving heterotypic cell-cell interactions in vivo.. , 2010, , .		0
33	Sequestration of E12/E47 and suppression of p27KIP1 play a role in Id2-induced proliferation and tumorigenesis. Carcinogenesis, 2009, 30, 1252-1259.	2.8	18
34	UVB upregulates the <i>bax</i> promoter in immortalized human keratinocytes via ROS induction of <i>Id3</i> . Experimental Dermatology, 2009, 18, 387-395.	2.9	14
35	Sulfur Mustard Induces Apoptosis in Cultured Normal Human Airway Epithelial Cells: Evidence of a Dominant Caspase-8-mediated Pathway and Differential Cellular Responses. Drug and Chemical Toxicology, 2008, 31, 137-148.	2.3	46
36	Peripheral-type benzodiazepine receptor overexpression and knockdown in human breast cancer cells indicate its prominent role in tumor cell proliferation. Biochemical Pharmacology, 2007, 73, 491-503.	4.4	106

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37	Sulfur mustard-induced apoptosis in human airway epithelial cells appears to be via the death receptor (Fas) pathway. <i>FASEB Journal</i> , 2007, 21, A258.	0.5	0
38	Apoptosis induced by ultraviolet B in HPV-immortalized human keratinocytes requires caspase-9 and is death receptor independent. <i>Experimental Dermatology</i> , 2006, 15, 23-34.	2.9	16
39	Id3 induces a caspase-3- and -9-dependent apoptosis and mediates UVB sensitization of HPV16 E6/7 immortalized human keratinocytes. <i>Oncogene</i> , 2006, 25, 3649-3660.	5.9	22
40	Calmodulin mediates sulfur mustard toxicity in human keratinocytes. <i>Toxicology</i> , 2006, 227, 21-35.	4.2	45
41	Id2 protein is selectively upregulated by UVB in primary, but not in immortalized human keratinocytes and inhibits differentiation. <i>Oncogene</i> , 2005, 24, 5443-5458.	5.9	18
42	PARP-1 binds E2F-1 independently of its DNA binding and catalytic domains, and acts as a novel coactivator of E2F-1-mediated transcription during re-entry of quiescent cells into S-phase. <i>Oncogene</i> , 2003, 22, 8460-8471.	5.9	98
43	Expression of Dominant-negative Fas-associated Death Domain Blocks Human Keratinocyte Apoptosis and Vesication Induced by Sulfur Mustard. <i>Journal of Biological Chemistry</i> , 2003, 278, 8531-8540.	3.4	54
44	HPV-16 E6/7 Immortalization Sensitizes Human Keratinocytes to Ultraviolet B by Altering the Pathway from Caspase-8 to Caspase-9-dependent Apoptosis. <i>Journal of Biological Chemistry</i> , 2002, 277, 24709-24716.	3.4	21
45	Gene therapy for prostate cancer by targeting poly(ADP-ribose) polymerase. <i>Cancer Research</i> , 2002, 62, 6879-83.	0.9	16
46	Poly(ADP-ribosyl)ation of p53 In Vitro and In Vivo Modulates Binding to its DNA Consensus Sequence. <i>Neoplasia</i> , 2001, 3, 179-188.	5.3	45
47	Poly(ADP-ribose) polymerase and aging. <i>Advances in Cell Aging and Gerontology</i> , 2001, 4, 113-133.	0.1	0
48	PARP Determines the Mode of Cell Death in Skin Fibroblasts, but not Keratinocytes, Exposed to Sulfur Mustard. <i>Journal of Investigative Dermatology</i> , 2001, 117, 1566-1573.	0.7	50
49	Calmodulin, poly(ADP-ribose)polymerase and p53 are targets for modulating the effects of sulfur mustard. <i>Journal of Applied Toxicology</i> , 2001, 20, S43-S49.	2.8	24
50	Mechanisms of JP-8 Jet Fuel Toxicity. I. Induction of Apoptosis in Rat Lung Epithelial Cells. <i>Toxicology and Applied Pharmacology</i> , 2001, 171, 94-106.	2.8	33
51	Mechanisms of JP-8 Jet Fuel Cell Toxicity. II. Induction of Necrosis in Skin Fibroblasts and Keratinocytes and Modulation of Levels of Bcl-2 Family Members. <i>Toxicology and Applied Pharmacology</i> , 2001, 171, 107-116.	2.8	27
52	Inhibition of poly(ADP-ribose) polymerase activity is insufficient to induce tetraploidy. <i>Nucleic Acids Research</i> , 2001, 29, 841-849.	14.5	28
53	Misregulation of gene expression in primary fibroblasts lacking poly(ADP-ribose) polymerase. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2000, 97, 11274-11279.	7.1	130
54	Roles of poly(ADP-ribosyl)ation and PARP in apoptosis, DNA repair, genomic stability and functions of p53 and E2F-1. <i>Advances in Enzyme Regulation</i> , 2000, 40, 183-215.	2.6	115

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55	Chromosomal aberrations in PARP-/- mice: Genome stabilization in immortalized cells by reintroduction of poly(ADP-ribose) polymerase cDNA. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 13191-13196.	7.1	108
56	Poly(ADP-ribose) polymerase upregulates E2F-1 promoter activity and DNA pol β expression during early S phase. Oncogene, 1999, 18, 5015-5023.	5.9	40
57	Involvement of PARP and poly(ADP-ribosylation) in the early stages of apoptosis and DNA replication. Molecular and Cellular Biochemistry, 1999, 193, 137-148.	3.1	84
58	Involvement of PARP and poly(ADP-ribosylation) in the early stages of apoptosis and DNA replication. , 1999, , 137-148.		28
59	The E7 protein of human papillomavirus type 16 sensitizes primary human keratinocytes to apoptosis. Oncogene, 1998, 17, 1207-1214.	5.9	101
60	Sulfur Mustard Induces Markers of Terminal Differentiation and Apoptosis in Keratinocytes Via a Ca ²⁺ -Calmodulin and Caspase-Dependent Pathway. Journal of Investigative Dermatology, 1998, 111, 64-71.	0.7	109
61	Regulation of the Expression or Recruitment of Components of the DNA Synthesome by Poly(ADP-Ribose) Polymerase. Biochemistry, 1998, 37, 9363-9370.	2.5	92
62	Prolongation of the p53 Response to DNA Strand Breaks in Cells Depleted of PARP by Antisense RNA Expression. Biochemical and Biophysical Research Communications, 1998, 253, 864-868.	2.1	20
63	Transient Poly(ADP-ribosylation) of Nuclear Proteins and Role of Poly(ADP-ribose) Polymerase in the Early Stages of Apoptosis. Journal of Biological Chemistry, 1998, 273, 13703-13712.	3.4	249
64	Sphingosine 1-Phosphate Inhibits Activation of Caspases that Cleave Poly(ADP-ribose) Polymerase and Lamins during Fas- and Ceramide-mediated Apoptosis in Jurkat T Lymphocytes. Journal of Biological Chemistry, 1998, 273, 2910-2916.	3.4	243
65	Detection of DNA breaks in apoptotic cells utilizing the DNA binding domain of poly(ADP-ribose) polymerase with fluorescence microscopy. Nucleic Acids Research, 1997, 25, 1437-1441.	14.5	21
66	Intact Cell Evidence for the Early Synthesis, and Subsequent Late Apoptin-Mediated Suppression, of Poly(ADP-ribose) during Apoptosis. Experimental Cell Research, 1997, 232, 313-321.	2.6	80
67	The Expression of Poly(ADP-ribose) Polymerase during Differentiation-Linked DNA Replication Reveals That It is a Component of the Multiprotein DNA Replication Complex. Biochemistry, 1996, 35, 11622-11633.	2.5	133
68	Depletion of Nuclear Poly(ADP-ribose) Polymerase by Antisense RNA Expression: Influence on Genomic Stability, Chromatin Organization, DNA Repair, and DNA Replication. Progress in Molecular Biology and Translational Science, 1996, 55, 135-156.	1.9	17
69	Sphingosylphosphocholine, a signaling molecule which accumulates in Niemann-Pick disease type A, stimulates DNA-binding activity of the transcription activator protein AP-1.. Proceedings of the National Academy of Sciences of the United States of America, 1995, 92, 5885-5889.	7.1	45
70	Requirement for the Expression of Poly(ADP-ribose) Polymerase during the Early Stages of Differentiation of 3T3-L1 Preadipocytes, as Studied by Antisense RNA Induction. Journal of Biological Chemistry, 1995, 270, 119-127.	3.4	60
71	Engineered Human Skin Model Using Poly(ADP-Ribose) Polymerase Antisense Expression Shows a Reduced Response to DNA Damage. Journal of Investigative Dermatology, 1995, 105, 38-43.	0.7	29
72	Model systems for the study of the role of PADPRP in essential biological processes. Biochimie, 1995, 77, 439-443.	2.6	3

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73	Chromosomal locations of major tRNA gene clusters of <i>Xenopus laevis</i> . <i>Chromosoma</i> , 1995, 104, 68-74.	2.2	3
74	Chromosomal locations of major tRNA gene clusters of <i>Xenopus laevis</i> . <i>Chromosoma</i> , 1995, 104, 68-74.	2.2	0
75	Expression and Characterization of a Fusion Protein Between the Catalytic Domain of Poly(ADP-Ribose) polymerase and the DNA Binding Domain of the Glucocorticoid Receptor. <i>Biochemical and Biophysical Research Communications</i> , 1994, 202, 880-887.	2.1	5
76	Acute or Chronic Topical Retinoic Acid Treatment of Human Skin In Vivo Alters the Expression of Epidermal Transglutaminase, Loricrin, Involucrin, Filaggrin, and Keratins 6 and 13 but not Keratins 1, 10, and 14. <i>Journal of Investigative Dermatology</i> , 1992, 98, 343-350.	0.7	110
77	Short-Term Retinoic Acid Treatment Increases In Vivo, but Decreases In Vitro, Epidermal Transglutaminase-K Enzyme Activity and Immunoreactivity. <i>Journal of Investigative Dermatology</i> , 1992, 99, 283-288.	0.7	29
78	Changes in Photo-Aged Human Skin Following Topical Application of All-Trans Retinoic Acid. <i>Journal of Investigative Dermatology</i> , 1990, 95, 510-515.	0.7	53
79	Inorganic Polyphosphates Are Important for Cell Survival and Motility of Human Skin Keratinocytes and Play a Role in Wound Healing. , 0, , .		5