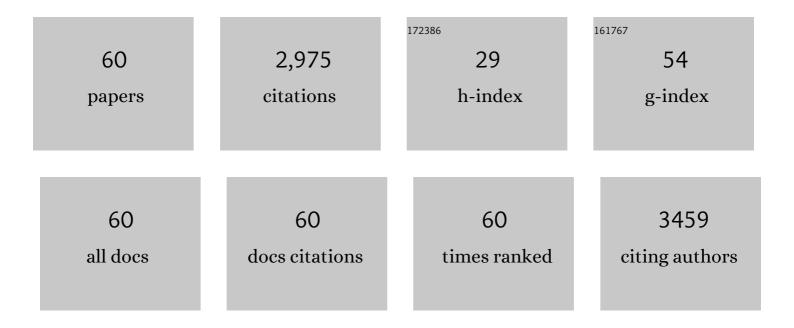
Robert C Smart

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	TIN2 is an architectural protein that facilitates TRF2-mediated <i>trans</i> - and <i>cis-</i> interactions on telomeric DNA. Nucleic Acids Research, 2021, 49, 13000-13018.	6.5	6
2	Measurement of Novel, Drinking Water-Associated PFAS in Blood from Adults and Children in Wilmington, North Carolina. Environmental Health Perspectives, 2020, 128, 77005.	2.8	118
3	C/EBPβ suppresses keratinocyte autonomous type 1 IFN response and p53 to increase cell survival and susceptibility to UVB-induced skin cancer. Carcinogenesis, 2019, 40, 1099-1109.	1.3	2
4	C/EBPβ deletion in oncogenic Ras skin tumors is a synthetic lethal event. Cell Death and Disease, 2018, 9, 1054.	2.7	17
5	Long noncoding RNA lincRNA-p21 is the major mediator of UVB-induced and p53-dependent apoptosis in keratinocytes. Cell Death and Disease, 2015, 6, e1700-e1700.	2.7	100
6	C/EBPα regulates CRL4 ^{Cdt2} -mediated degradation of p21 in response to UVB-induced DNA damage to control the G ₁ /S checkpoint. Cell Cycle, 2014, 13, 3602-3610.	1.3	19
7	Control of skin cancer by the circadian rhythm. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 18790-18795.	3.3	191
8	CD34 antigen: Determination of specific sites of phosphorylation in vitro and in vivo. International Journal of Mass Spectrometry, 2011, 301, 12-21.	0.7	6
9	PTEN Positively Regulates UVB-Induced DNA Damage Repair. Cancer Research, 2011, 71, 5287-5295.	0.4	81
10	C/EBPα Expression Is Downregulated in Human Nonmelanoma Skin Cancers and Inactivation of C/EBPα Confers Susceptibility to UVB-Induced Skin Squamous Cell Carcinomas. Journal of Investigative Dermatology, 2011, 131, 1339-1346.	0.3	19
11	C/EBPα and C/EBPβ Are Required for Sebocyte Differentiation and Stratified Squamous Differentiation in Adult Mouse Skin. PLoS ONE, 2010, 5, e9837.	1.1	38
12	RSK-Mediated Phosphorylation in the C/EBPβ Leucine Zipper Regulates DNA Binding, Dimerization, and Growth Arrest Activity. Molecular and Cellular Biology, 2010, 30, 2621-2635.	1.1	63
13	Overexpression of Transcription Factor Sp2 Inhibits Epidermal Differentiation and Increases Susceptibility to Wound- and Carcinogen-Induced Tumorigenesis. Cancer Research, 2010, 70, 8507-8516.	0.4	17
14	Ablation of TAK1 Upregulates Reactive Oxygen Species and Selectively Kills Tumor Cells. Cancer Research, 2010, 70, 8417-8425.	0.4	37
15	C/EBPα expression is partially regulated by C/EBPβ in response to DNA damage and C/EBPα-deficient fibroblasts display an impaired G1 checkpoint. Oncogene, 2009, 28, 3235-3245.	2.6	11
16	C/EBPβ represses p53 to promote cell survival downstream of DNA damage independent of oncogenic Ras and p19Arf. Cell Death and Differentiation, 2008, 15, 1734-1744.	5.0	30
17	Genetic Ablation of CCAAT/Enhancer Binding Protein α in Epidermis Reveals Its Role in Suppression of Epithelial Tumorigenesis. Cancer Research, 2007, 67, 6768-6776.	0.4	35
18	Decreased survival of C/EBPβ-deficient keratinocytes is due to aberrant regulation of p53 levels and function. Oncogene, 2007, 26, 360-367.	2.6	22

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19	Conditional ablation of C/EBPÎ ² demonstrates its keratinocyte-specific requirement for cell survival and mouse skin tumorigenesis. Oncogene, 2006, 25, 1272-1276.	2.6	68
20	TAK1 Is a Master Regulator of Epidermal Homeostasis Involving Skin Inflammation and Apoptosis. Journal of Biological Chemistry, 2006, 281, 19610-19617.	1.6	136
21	Diminished expression of C/EBPalpha in skin carcinomas is linked to oncogenic Ras and reexpression of C/EBPalpha in carcinoma cells inhibits proliferation. Cancer Research, 2005, 65, 861-7.	0.4	45
22	Cell Cycle-Dependent Phosphorylation of C/EBPβ Mediates Oncogenic Cooperativity between C/EBPβ and H-Ras V12. Molecular and Cellular Biology, 2004, 24, 7380-7391.	1.1	72
23	C/EBPα Is a DNA Damage-Inducible p53-Regulated Mediator of the G 1 Checkpoint in Keratinocytes. Molecular and Cellular Biology, 2004, 24, 10650-10660.	1.1	60
24	Lithium Stabilizes the CCAAT/Enhancer-binding Protein α (C/EBPα) through a Glycogen Synthase Kinase 3 (GSK3)-independent Pathway Involving Direct Inhibition of Proteasomal Activity. Journal of Biological Chemistry, 2003, 278, 19674-19681.	1.6	45
25	CCAAT/enhancer binding protein-Â is a mediator of keratinocyte survival and skin tumorigenesis involving oncogenic Ras signaling. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 207-212.	3.3	179
26	17beta-Estradiol Is a Hormonal Regulator of Mirex Tumor Promotion Sensitivity in Mice. Toxicological Sciences, 2002, 69, 42-48.	1.4	12
27	Deficiency of either cyclooxygenase (COX)-1 or COX-2 alters epidermal differentiation and reduces mouse skin tumorigenesis. Cancer Research, 2002, 62, 3395-401.	0.4	284
28	Protein Kinase C-α Coordinately Regulates Cytosolic Phospholipase A ₂ Activity and the Expression of Cyclooxygenase-2 through Different Mechanisms in Mouse Keratinocytes. Molecular Pharmacology, 2001, 59, 860-866.	1.0	62
29	17β-Estradiol and ICI-182780 regulate the hair follicle cycle in mice through an estrogen receptor-α pathway. American Journal of Physiology - Endocrinology and Metabolism, 2000, 278, E202-E210.	1.8	71
30	Effects of 17-β-Estradiol and ICI 182 780 on Hair Growth in Various Strains of Mice. Journal of Investigative Dermatology Symposium Proceedings, 1999, 4, 285-289.	0.8	30
31	A multihit, multistage model of chemical carcinogenesis. Carcinogenesis, 1999, 20, 1837-1844.	1.3	49
32	Correlation between expression of peroxisome proliferator-activated receptor β and squamous differentiation in epidermal and tracheobronchial epithelial cells. Molecular and Cellular Endocrinology, 1999, 147, 85-92.	1.6	84
33	C/EBPβ Modulates the Early Events of Keratinocyte Differentiation Involving Growth Arrest and Keratin 1 and Keratin 10 Expression. Molecular and Cellular Biology, 1999, 19, 7181-7190.	1.1	138
34	Expression of CCAAT/Enhancer Binding Proteins (C/EBP) is Associated with Squamous Differentiation in Epidermis and Isolated Primary Keratinocytes and is Altered in Skin Neoplasms. Journal of Investigative Dermatology, 1998, 110, 939-945.	0.3	82
35	On the effect of estrogen receptor agonists and antagonists on the mouse hair follicle cycle. Journal of Investigative Dermatology, 1998, 111, 175.	0.3	10
36	Evidence that mirex promotes a unique population of epidermal cells that cannot be distinguished by their mutant Ha-ras genotype. Molecular Carcinogenesis, 1997, 20, 115-124.	1.3	9

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37	An estrogen receptor pathway regulates the telogen-anagen hair follicle transition and influences epidermal cell proliferation Proceedings of the National Academy of Sciences of the United States of America, 1996, 93, 12525-12530.	3.3	155
38	Localization and Expression of Cornifin-α/SPRR1 in Mouse Epidermis, Anagen Follicles, and Skin Neoplasms. Journal of Investigative Dermatology, 1996, 106, 647-654.	0.3	23
39	Epidermal Protein Kinase C-β2 Is Highly Sensitive to Downregulation and Is Exclusively Expressed in Langerhans Cells: Downregulation Is Associated with Attenuated Contact Hypersensitivity. Journal of Investigative Dermatology, 1996, 107, 354-359.	0.3	18
40	Lack of effect of retinoic acid and fluocinolone acetonide on mirex tumor promotion indicates a novel mirex mechanism. Carcinogenesis, 1995, 16, 2199-2204.	1.3	4
41	Synergistic interaction between the non-phorbol ester-type promoter mixer and 12-0-tetradecanoylphorbol-13-acetate in mouse skin tumor promotion. Carcinogenesis, 1994, 15, 47-52.	1.3	11
42	Minimal Role of Enhanced Cell Proliferation in Skin Tumor Promotion by Mirex: A Nonphorbol Ester-Type Promoter. Environmental Health Perspectives, 1993, 101, 265.	2.8	1
43	Characterization of skin tumor promotion by mirex: structure-activity relationships, sexual dimorphism and presence of Ha-ras mutation. Carcinogenesis, 1993, 14, 1155-1160.	1.3	18
44	Characterization of benzo[a]pyrene-initiated mouse skin papillomas for Ha-ras mutations and protein kinase C levels. Carcinogenesis, 1993, 14, 2289-2295.	1.3	55
45	Diacylglycerol is an effector of the clonal expansion of cells containing activated Ha-ras genes. Carcinogenesis, 1993, 14, 2645-2648.	1.3	15
46	Alterations in protein kinase C isozymes α and β2 in activated Ha-ras containing papillomas in the absence of an increase in diacyiglycerol. Carcinogenesis, 1992, 13, 1113-1120.	1.3	32
47	Effect of ascorbic acid and its synthetic lipophilic derivative ascorbyl palmitate on phorbol ester-induced skin-tumor promotion in mice. American Journal of Clinical Nutrition, 1991, 54, 1266S-1273S.	2.2	25
48	Hepatic tumor-promoting chlorinated hydrocarbons stimulate protein kinase C activity. Carcinogenesis, 1989, 10, 851-856.	1.3	47
49	Comparison of epidermal protein kinase C activity, ornithine decarboxylase induction and DNA synthesis stimulated by TPA or dioctanoylglycerol in mouse strains with differing susceptibility to TPA-induced tumor promotion. Carcinogenesis, 1989, 10, 833-838.	1.3	23
50	Comparison of the effect of sn-1,2-didecanoylglycerol and 12-O-tetradecanoylphorbol-13-acetate on cutaneous morphology, inflammation and tumor promotion in CD-1 mice. Carcinogenesis, 1988, 9, 2221-2226.	1.3	23
51	Effect of dietary ascorbate on covalent binding of benzene to bone marrow and hepatic tissue in vivo. Biochemical Pharmacology, 1986, 35, 3180-3182.	2.0	4
52	sn-l, 2-Diacylglycerols mimic the effects of 12-0-tetradecanoylphorbol-13-acetate in vivo by inducing biochemical changes associated with tumor promotion in mouse epidermis. Carcinogenesis, 1986, 7, 1865-1870.	1.3	138
53	Disposition of the naturally occurring antimutagenic plant phenol, ellagic acid, and its synthetic derivatives, 3-O-decylellagic acid and 3, 3'-di-O-methylellagic acid in mice. Carcinogenesis, 1986, 7, 1663-1667.	1.3	88
54	Effect of ellagic acid and 3-O-decylellagic acid on the formation of benzo[a]pyrene-derived DNA adducts in vivo and on the tumorigenicity of 3-methylcholanthrene in mice. Carcinogenesis, 1986, 7, 1669-1675.	1.3	28

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55	Effect of ascorbate on covalent binding of benzene and phenol metabolites to isolated tissue preparations. Toxicology and Applied Pharmacology, 1985, 77, 334-343.	1.3	14
56	Chemical Carcinogenesis. , 0, , 225-250.		2
57	Introduction to Biochemical and Molecular Methods in Toxicology. , 0, , 13-22.		1
58	Carcinogenesis. , 0, , 537-586.		1
59	Molecular and Biochemical Toxicology: Definition and Scope. , 0, , 1-4.		1
60	Overview of Molecular Techniques in Toxicology: Genes and Transgenes. , 0, , 5-24.		0