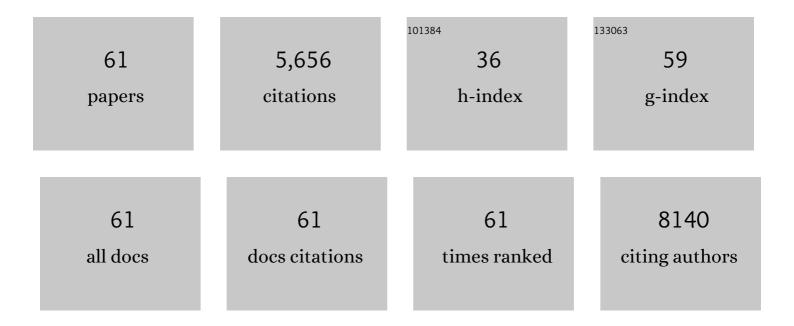
## David G Johnson

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/5374105/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	Expression of transcription factor E2F1 induces quiescent cells to enter S phase. Nature, 1993, 365, 349-352.	13.7	935
2	Distinct and Overlapping Roles for E2F Family Members in Transcription, Proliferation and Apoptosis. Current Molecular Medicine, 2006, 6, 739-748.	0.6	423
3	Inactivating E2f1 reverts apoptosis resistance and cancer sensitivity in Trp53-deficient mice. Nature Cell Biology, 2003, 5, 655-660.	4.6	391
4	Regulation of epidermal apoptosis and DNA repair by E2F1 in response to ultraviolet B radiation. Oncogene, 2005, 24, 2449-2460.	2.6	374
5	Oncogenic capacity of the E2F1 gene Proceedings of the National Academy of Sciences of the United States of America, 1994, 91, 12823-12827.	3.3	234
6	Role of E2F in cell cycle control and cancer. Frontiers in Bioscience - Landmark, 1998, 3, d447-458.	3.0	166
7	Increased E2F1 activity induces skin tumors in mice heterozygous and nullizygous for p53. Proceedings of the National Academy of Sciences of the United States of America, 1998, 95, 8858-8863.	3.3	153
8	Putting the Oncogenic and Tumor Suppressive Activities of E2F into Context. Current Molecular Medicine, 2006, 6, 731-738.	0.6	151
9	Transcriptional and Nontranscriptional Functions of E2F1 in Response to DNA Damage. Cancer Research, 2012, 72, 13-17.	0.4	145
10	ATM promotes apoptosis and suppresses tumorigenesis in response to Myc. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 1446-1451.	3.3	142
11	E2F1 Has Both Oncogenic and Tumor-Suppressive Properties in a Transgenic Model. Molecular and Cellular Biology, 1999, 19, 6408-6414.	1.1	136
12	GCN5 and E2F1 stimulate nucleotide excision repair by promoting H3K9 acetylation at sites of damage. Nucleic Acids Research, 2011, 39, 1390-1397.	6.5	135
13	Deregulated expression of E2F1 induces hyperplasia and cooperates with ras in skin tumor development. Oncogene, 1998, 16, 1267-1276.	2.6	125
14	E2F1 Uses the ATM Signaling Pathway to Induce p53 and Chk2 Phosphorylation and Apoptosis. Molecular Cancer Research, 2004, 2, 203-214.	1.5	125
15	Regulation of BRCA1 Expression by the Rb-E2F Pathway. Journal of Biological Chemistry, 2000, 275, 4532-4536.	1.6	108
16	A High-Frequency Regulatory Polymorphism in the p53 Pathway Accelerates Tumor Development. Cancer Cell, 2010, 18, 220-230.	7.7	108
17	The RB-E2F1 Pathway Regulates Autophagy. Cancer Research, 2010, 70, 7882-7893.	0.4	107
18	Lack of Cyclin-Dependent Kinase 4 Inhibits c- myc Tumorigenic Activities in Epithelial Tissues. Molecular and Cellular Biology, 2004, 24, 7538-7547.	1.1	96

DAVID G JOHNSON

#	Article	IF	CITATIONS
19	E2F4 and E2F1 Have Similar Proliferative Properties but Different Apoptotic and Oncogenic Properties In Vivo. Molecular and Cellular Biology, 2000, 20, 3417-3424.	1.1	87
20	INO80 chromatin remodeling complex promotes the removal of UV lesions by the nucleotide excision repair pathway. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 17274-17279.	3.3	87
21	The paradox ofE2F1: Oncogene and tumor suppressor gene. , 2000, 27, 151-157.		85
22	RB localizes to DNA double-strand breaks and promotes DNA end resection and homologous recombination through the recruitment of BRG1. Genes and Development, 2016, 30, 2500-2512.	2.7	83
23	ARF Differentially Modulates Apoptosis Induced by E2F1 and Myc. Molecular and Cellular Biology, 2002, 22, 1360-1368.	1.1	75
24	The Retinoblastoma (RB) Tumor Suppressor: Pushing Back against Genome Instability on Multiple Fronts. International Journal of Molecular Sciences, 2017, 18, 1776.	1.8	72
25	E2F1 promotes the recruitment of DNA repair factors to sites of DNA double-strand breaks. Cell Cycle, 2011, 10, 1287-1294.	1.3	66
26	Overexpression of the Low Molecular Weight Cyclin E in Transgenic Mice Induces Metastatic Mammary Carcinomas through the Disruption of the ARF-p53 Pathway. Cancer Research, 2007, 67, 7212-7222.	0.4	64
27	Chromatin: Receiver and Quarterback for Cellular Signals. Cell, 2013, 152, 685-689.	13.5	62
28	Expression of Transcription Factor E2F1 and Telomerase in Glioblastomas: Mechanistic Linkage and Prognostic Significance. Journal of the National Cancer Institute, 2005, 97, 1589-1600.	3.0	57
29	E2F1 Localizes to Sites of UV-induced DNA Damage to Enhance Nucleotide Excision Repair. Journal of Biological Chemistry, 2010, 285, 19308-19315.	1.6	55
30	E2F1 uses the ATM signaling pathway to induce p53 and Chk2 phosphorylation and apoptosis. Molecular Cancer Research, 2004, 2, 203-14.	1.5	54
31	Mouse Models for the p53 R72P Polymorphism Mimic Human Phenotypes. Cancer Research, 2010, 70, 5851-5859.	0.4	49
32	E2F1 acetylation directs p300/CBP-mediated histone acetylation at DNA double-strand breaks to facilitate repair. Nature Communications, 2019, 10, 4951.	5.8	45
33	E2F3a Stimulates Proliferation, p53-Independent Apoptosis and Carcinogenesis in a Transgenic Mouse Model. Cell Cycle, 2006, 5, 184-190.	1.3	43
34	SAGE profiling of UV-induced mouse skin squamous cell carcinomas, comparison with acute UV irradiation effects. Molecular Carcinogenesis, 2005, 42, 40-52.	1.3	40
35	Oncogenes and the DNA Damage Response: Myc and E2F1 Engage the ATM Signaling Pathway to Activate p53 and Induce Apoptosis. Cell Cycle, 2006, 5, 801-803.	1.3	40
36	Tumor formation in mice with conditional inactivation of Brca1 in epithelial tissues. Oncogene, 2003, 22, 5415-5426.	2.6	38

DAVID G JOHNSON

#	Article	IF	CITATIONS
37	Distinct and Overlapping Roles for E2F Family Members in Transcription, Proliferation and Apoptosis. Current Molecular Medicine, 2006, 6, 739-748.	0.6	38
38	E2F2 suppresses Mycâ€induced proliferation and tumorigenesis. Molecular Carcinogenesis, 2010, 49, 152-156.	1.3	37
39	Effects of <i>MDM2</i> promoter polymorphisms and <i>p53</i> codon 72 polymorphism on risk and age at onset of squamous cell carcinoma of the head and neck. Molecular Carcinogenesis, 2011, 50, 697-706.	1.3	37
40	Myc lacks E2F1's ability to suppress skin carcinogenesis. Oncogene, 2001, 20, 5341-5349.	2.6	36
41	Differential activities of E2F family members: Unique functions in regulating transcription. Molecular Carcinogenesis, 1998, 22, 190-198.	1.3	35
42	Deregulated expression of cell-cycle proteins during premalignant progression in SENCAR mouse skin. Oncogene, 1998, 17, 2251-2258.	2.6	34
43	Inactivation of E2f1 enhances tumorigenesis in a Myc transgenic model. Cancer Research, 2002, 62, 3276-81.	0.4	34
44	Transgenic E2F1 Expression in the Mouse Brain Induces a Human-Like Bimodal Pattern of Tumors. Cancer Research, 2007, 67, 4005-4009.	0.4	29
45	Combined effects of <i>E2F1</i> and <i>E2F2</i> polymorphisms on risk and early onset of squamous cell carcinoma of the head and neck. Molecular Carcinogenesis, 2012, 51, E132-41.	1.3	28
46	Deregulated expression of DP1 induces epidermal proliferation and enhances skin carcinogenesis. Molecular Carcinogenesis, 2001, 31, 90-100.	1.3	27
47	Repression of Androgen Receptor Transcription through the E2F1/DNMT1 Axis. PLoS ONE, 2011, 6, e25187.	1.1	25
48	E2F1 Responds to Ultraviolet Radiation by Directly Stimulating DNA Repair and Suppressing Carcinogenesis. Cancer Research, 2014, 74, 3369-3377.	0.4	24
49	Identification of prohibitin and prohibiton as novel factors binding to the p53 induced gene 3 (PIG3) promoter (TGYCC)15 motif. Biochemical and Biophysical Research Communications, 2014, 443, 1239-1244.	1.0	20
50	E2F1 and E2F3 activate ATM through distinct mechanisms to promote E1A-induced apoptosis. Cell Cycle, 2008, 7, 391-400.	1.3	17
51	The E2F1 transcription factor and RB tumor suppressor moonlight as DNA repair factors. Cell Cycle, 2020, 19, 2260-2269.	1.3	17
52	Molecular Cloning and Characterization of a Novel Mouse Epidermal Differentiation Gene and Its Promoter. Genomics, 2001, 73, 284-290.	1.3	13
53	E2F1 and p53 Transcription Factors as Accessory Factors for Nucleotide Excision Repair. International Journal of Molecular Sciences, 2012, 13, 13554-13568.	1.8	13
54	E2F4 and E2F1 Have Similar Proliferative Properties but Different Apoptotic and Oncogenic Properties In Vivo. Molecular and Cellular Biology, 2000, 20, 3417-3424.	1.1	11

DAVID G JOHNSON

#	Article	IF	CITATIONS
55	Modeling gene-environment interactions in oral cavity and esophageal cancers demonstrates a role for the p53 R72P polymorphism in modulating susceptibility. Molecular Carcinogenesis, 2014, 53, 648-658.	1.3	10
56	Enhanced skin carcinogenesis and lack of thymus hyperplasia in transgenic mice expressing human cyclin D1b ( <i>CCND1b</i> ). Molecular Carcinogenesis, 2009, 48, 508-516.	1.3	8
57	Direct Regulation of DNA Repair by E2F and RB in Mammals and Plants: Core Function or Convergent Evolution?. Cancers, 2021, 13, 934.	1.7	3
58	Variability of Alkaloid Production in Submerged Culture. Quarterly Journal of Crude Drug Research = Vierteljahrliche Zietschrift Fur Drogen-Forschung = Revue Trimestrielle Des Recherches Sur Les Matieres Premieres, 1964, 4, 577-581.	0.2	2
59	Putting the Oncogenic and Tumor Suppressive Activities of E2F into Context. Current Molecular Medicine, 2006, 6, 731-738.	0.6	1
60	The <i>p53</i> R72P polymorphism does not affect the physiological response to ionizing radiation in a mouse model. Cell Cycle, 2017, 16, 1153-1163.	1.3	1
61	Slug Expression in Mouse Skin and Skin Tumors Is Not Regulated by p53. Journal of Investigative Dermatology, 2014, 134, 566-568.	0.3	0