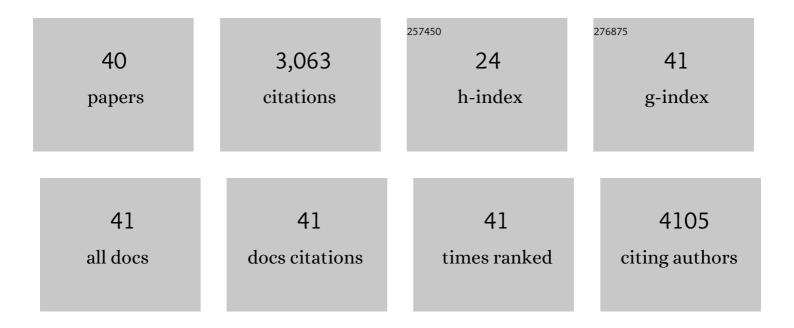
Markus Christmann

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	The Mitochondrial Disruptor Devimistat (CPI-613) Synergizes with Genotoxic Anticancer Drugs in Colorectal Cancer Therapy in a Bim-Dependent Manner. Molecular Cancer Therapeutics, 2022, 21, 100-112.	4.1	9
2	Senescence Is the Main Trait Induced by Temozolomide in Glioblastoma Cells. Cancers, 2022, 14, 2233.	3.7	19
3	Alterations in Molecular Profiles Affecting Glioblastoma Resistance to Radiochemotherapy: Where Does the Good Go?. Cancers, 2022, 14, 2416.	3.7	13
4	Oxaliplatin-Induced Senescence in Colorectal Cancer Cells Depends on p14ARF-Mediated Sustained p53 Activation. Cancers, 2021, 13, 2019.	3.7	14
5	Repair of O6-carboxymethylguanine adducts by O6-methylguanine-DNA methyltransferase in human colon epithelial cells. Carcinogenesis, 2021, 42, 1110-1118.	2.8	5
6	Localization matters: nuclear-trapped Survivin sensitizes glioblastoma cells to temozolomide by elevating cellular senescence and impairing homologous recombination. Cellular and Molecular Life Sciences, 2021, 78, 5587-5604.	5.4	9
7	Natural Merosesquiterpenes Activate the DNA Damage Response via DNA Strand Break Formation and Trigger Apoptotic Cell Death in p53-Wild-Type and Mutant Colorectal Cancer. Cancers, 2021, 13, 3282.	3.7	7
8	Targeting c-IAP1, c-IAP2, and Bcl-2 Eliminates Senescent Glioblastoma Cells Following Temozolomide Treatment. Cancers, 2021, 13, 3585.	3.7	19
9	Benzo[a]pyrene represses DNA repair through altered E2F1/E2F4 function marking an early event in DNA damage-induced cellular senescence. Nucleic Acids Research, 2020, 48, 12085-12101.	14.5	23
10	Lipoic Acid Synergizes with Antineoplastic Drugs in Colorectal Cancer by Targeting p53 for Proteasomal Degradation. Cells, 2019, 8, 794.	4.1	17
11	Functional mismatch repair and inactive p53 drive sensitization of colorectal cancer cells to irinotecan via the IAP antagonist BV6. Archives of Toxicology, 2019, 93, 2265-2277.	4.2	13
12	DNA repair in personalized brain cancer therapy with temozolomide and nitrosoureas. DNA Repair, 2019, 78, 128-141.	2.8	89
13	Temozolomide Induces Senescence and Repression of DNA Repair Pathways in Glioblastoma Cells via Activation of ATR–CHK1, p21, and NF-κB. Cancer Research, 2019, 79, 99-113.	0.9	126
14	Epigenetic regulation of DNA repair genes and implications for tumor therapy. Mutation Research - Reviews in Mutation Research, 2019, 780, 15-28.	5.5	59
15	Repair gene O ⁶ â€methylguanineâ€DNA methyltransferase is controlled by SP1 and upâ€regulated by glucocorticoids, but not by temozolomide and radiation. Journal of Neurochemistry, 2018, 144, 139-151.	3.9	41
16	Targeting anticancer drug-induced senescence in glioblastoma therapy. Oncotarget, 2018, 9, 37466-37467.	1.8	4
17	Inherent and toxicant-provoked reduction in DNA repair capacity: A key mechanism for personalized risk assessment, cancer prevention and intervention, and response to therapy. International Journal of Hygiene and Environmental Health, 2018, 221, 993-1006.	4.3	13
18	Sensitization of colorectal cancer cells to irinotecan by the Survivin inhibitor LLP3 depends on XAF1 proficiency in the context of mutated p53. Archives of Toxicology, 2018, 92, 2645-2648.	4.2	13

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19	Epigenetic silencing of XAF1 in high-grade gliomas is associated with IDH1 status and improved clinical outcome. Oncotarget, 2017, 8, 15071-15084.	1.8	13
20	Integrin αVβ3 silencing sensitizes malignant glioma cells to temozolomide by suppression of homologous recombination repair. Oncotarget, 2017, 8, 27754-27771.	1.8	28
21	MGMT promoter methylation determined by HRM in comparison to MSP and pyrosequencing for predicting high-grade glioma response. Clinical Epigenetics, 2016, 8, 49.	4.1	59
22	Adaptive upregulation of DNA repair genes following benzo(a)pyrene diol epoxide protects against cell death at the expense of mutations. Nucleic Acids Research, 2016, 44, 10727-10743.	14.5	37
23	Apoptosis induced by temozolomide and nimustine in glioblastoma cells is supported by JNK/c-Jun-mediated induction of the BH3-only protein BIM. Oncotarget, 2015, 6, 33755-33768.	1.8	42
24	Translesion Polymerase η Is Upregulated by Cancer Therapeutics and Confers Anticancer Drug Resistance. Cancer Research, 2014, 74, 5585-5596.	0.9	48
25	Human three prime exonuclease TREX1 is induced by genotoxic stress and involved in protection of glioma and melanoma cells to anticancer drugs. Biochimica Et Biophysica Acta - Molecular Cell Research, 2013, 1833, 1832-1843.	4.1	23
26	Transcriptional regulation of human DNA repair genes following genotoxic stress: trigger mechanisms, inducible responses and genotoxic adaptation. Nucleic Acids Research, 2013, 41, 8403-8420.	14.5	201
27	Survival and Death Strategies in Glioma Cells: Autophagy, Senescence and Apoptosis Triggered by a Single Type of Temozolomide-Induced DNA Damage. PLoS ONE, 2013, 8, e55665.	2.5	218
28	O6-methylguanine-DNA methyltransferase (MGMT): impact on cancer risk in response to tobacco smoke. Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis, 2012, 736, 64-74.	1.0	40
29	O6-Methylguanine-DNA methyltransferase (MGMT) in normal tissues and tumors: Enzyme activity, promoter methylation and immunohistochemistry. Biochimica Et Biophysica Acta: Reviews on Cancer, 2011, 1816, 179-190.	7.4	142
30	Delayed c-Fos activation in human cells triggers XPF induction and an adaptive response to UVC-induced DNA damage and cytotoxicity. Cellular and Molecular Life Sciences, 2011, 68, 1785-1798.	5.4	29
31	Targeting O 6-methylguanine-DNA methyltransferase with specific inhibitors as a strategy in cancer therapy. Cellular and Molecular Life Sciences, 2010, 67, 3663-3681.	5.4	124
32	MGMT activity, promoter methylation and immunohistochemistry of pretreatment and recurrent malignant gliomas: a comparative study on astrocytoma and glioblastoma. International Journal of Cancer, 2010, 127, 2106-2118.	5.1	97
33	Three prime exonuclease I (TREX1) is Fos/AP-1 regulated by genotoxic stress and protects against ultraviolet light and benzo(a)pyrene-induced DNA damage. Nucleic Acids Research, 2010, 38, 6418-6432.	14.5	52
34	Differential Sensitivity of Malignant Glioma Cells to Methylating and Chloroethylating Anticancer Drugs: p53 Determines the Switch by Regulating <i>xpc, ddb2</i> , and DNA Double-Strand Breaks. Cancer Research, 2007, 67, 11886-11895.	0.9	96
35	MGMT: Key node in the battle against genotoxicity, carcinogenicity and apoptosis induced by alkylating agents. DNA Repair, 2007, 6, 1079-1099.	2.8	549
36	Inhibition of O6-Methylguanine-DNA Methyltransferase by Glucose-Conjugated Inhibitors: Comparison with Nonconjugated Inhibitors and Effect on Fotemustine and Temozolomide-Induced Cell Death. Journal of Pharmacology and Experimental Therapeutics, 2004, 311, 585-593.	2.5	54

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37	Mechanisms of human DNA repair: an update. Toxicology, 2003, 193, 3-34.	4.2	486
38	Phosphorylation of mismatch repair proteins MSH2 and MSH6 affecting MutSalpha mismatch-binding activity. Nucleic Acids Research, 2002, 30, 1959-1966.	14.5	60
39	Acquired resistance of melanoma cells to the antineoplastic agent fotemustine is caused by reactivation of the DNA repair gene mgmt. International Journal of Cancer, 2001, 92, 123-129.	5.1	82
40	Nuclear Translocation of Mismatch Repair Proteins MSH2 and MSH6 as a Response of Cells to Alkylating Agents. Journal of Biological Chemistry, 2000, 275, 36256-36262.	3.4	85