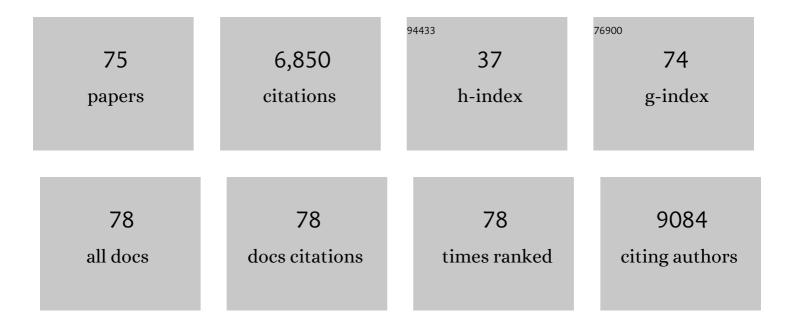
William R Wilson

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Radiosensitisation of SCCVII tumours and normal tissues in mice by the DNA-dependent protein kinase inhibitor AZD7648. Radiotherapy and Oncology, 2022, 166, 162-170.	0.6	7
2	Bioreductive prodrug PR-104 improves the tumour distribution and titre of the nitroreductase-armed oncolytic adenovirus ONYX-411NTR leading to therapeutic benefit. Cancer Gene Therapy, 2022, 29, 1021-1032.	4.6	4
3	Therapeutic targeting of the hypoxic tumour microenvironment. Nature Reviews Clinical Oncology, 2021, 18, 751-772.	27.6	185
4	Spatially-resolved pharmacokinetic/pharmacodynamic modelling of bystander effects of a nitrochloromethylbenzindoline hypoxia-activated prodrug. Cancer Chemotherapy and Pharmacology, 2021, 88, 673-687.	2.3	2
5	Subcellular Location of Tirapazamine Reduction Dramatically Affects Aerobic but Not Anoxic Cytotoxicity. Molecules, 2020, 25, 4888.	3.8	4
6	Benzotriazine Di-Oxide Prodrugs for Exploiting Hypoxia and Low Extracellular pH in Tumors. Molecules, 2019, 24, 2524.	3.8	3
7	Dual pH-sensitive liposomes with low pH-triggered sheddable PEG for enhanced tumor-targeted drug delivery. Nanomedicine, 2019, 14, 1971-1989.	3.3	58
8	Hypoxia-selective radiosensitisation by SN38023, a bioreductive prodrug of DNA-dependent protein kinase inhibitor IC87361. Biochemical Pharmacology, 2019, 169, 113641.	4.4	19
9	Radiosensitization of head and neck squamous cell carcinoma lines by DNA-PK inhibitors is more effective than PARP-1 inhibition and is enhanced by SLFN11 and hypoxia. International Journal of Radiation Biology, 2019, 95, 1597-1612.	1.8	26
10	Studies Towards Hypoxia-Activated Prodrugs of PARP Inhibitors. Molecules, 2019, 24, 1559.	3.8	11
11	Functional CRISPR and shRNA Screens Identify Involvement of Mitochondrial Electron Transport in the Activation of Evofosfamide. Molecular Pharmacology, 2019, 95, 638-651.	2.3	11
12	An Intratumor Pharmacokinetic/Pharmacodynamic Model for the Hypoxia-Activated Prodrug Evofosfamide (TH-302): Monotherapy Activity is Not Dependent on a Bystander Effect. Neoplasia, 2019, 21, 159-171.	5.3	22
13	Development of capability for genome-scale CRISPR-Cas9 knockout screens in New Zealand. Journal of the Royal Society of New Zealand, 2018, 48, 245-261.	1.9	1
14	Next-Generation Hypoxic Cell Radiosensitizers: Nitroimidazole Alkylsulfonamides. Journal of Medicinal Chemistry, 2018, 61, 1241-1254.	6.4	52
15	Bystander Effects of Hypoxia-Activated Prodrugs: Agent-Based Modeling Using Three Dimensional Cell Cultures. Frontiers in Pharmacology, 2018, 9, 1013.	3.5	21
16	An agent-based model for drug-radiation interactions in the tumour microenvironment: Hypoxia-activated prodrug SN30000 in multicellular tumour spheroids. PLoS Computational Biology, 2018, 14, e1006469.	3.2	33
17	Drug-DNA adducts as biomarkers for metabolic activation of the nitro-aromatic nitrogen mustard prodrug PR-104A. Biochemical Pharmacology, 2018, 154, 64-74.	4.4	6
18	Characterization of a smart pH-cleavable PEG polymer towards the development of dual pH-sensitive liposomes. International Journal of Pharmaceutics, 2018, 548, 288-296.	5.2	28

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19	Cellular pharmacology of evofosfamide (TH-302): A critical re-evaluation of its bystander effects. Biochemical Pharmacology, 2018, 156, 265-280.	4.4	22
20	PEG-Benzaldehyde-Hydrazone-Lipid Based PEG-Sheddable pH-Sensitive Liposomes: Abilities for Endosomal Escape and Long Circulation. Pharmaceutical Research, 2018, 35, 154.	3.5	45
21	Evofosfamide for the treatment of human papillomavirus-negative head and neck squamous cell carcinoma. JCI Insight, 2018, 3, .	5.0	44
22	DNA Adduct Profiles Predict in Vitro Cell Viability after Treatment with the Experimental Anticancer Prodrug PR104A. Chemical Research in Toxicology, 2017, 30, 830-839.	3.3	13
23	Reductive Metabolism Influences the Toxicity and Pharmacokinetics of the Hypoxia-Targeted Benzotriazine Di-Oxide Anticancer Agent SN30000 in Mice. Frontiers in Pharmacology, 2017, 8, 531.	3.5	16
24	Hypoxia-activated prodrugs: paths forward in the era of personalised medicine. British Journal of Cancer, 2016, 114, 1071-1077.	6.4	155
25	Mechanisms and biomaterials in pH-responsive tumour targeted drug delivery: A review. Biomaterials, 2016, 85, 152-167.	11.4	768
26	Identification of P450 Oxidoreductase as a Major Determinant of Sensitivity to Hypoxia-Activated Prodrugs. Cancer Research, 2015, 75, 4211-4223.	0.9	65
27	Dual Targeting of Hypoxia and Homologous Recombination Repair Dysfunction in Triple-Negative Breast Cancer. Molecular Cancer Therapeutics, 2014, 13, 2501-2514.	4.1	40
28	The flavoprotein FOXRED2 reductively activates nitro-chloromethylbenzindolines and other hypoxia-targeting prodrugs. Biochemical Pharmacology, 2014, 89, 224-235.	4.4	21
29	Identification of one-electron reductases that activate both the hypoxia prodrug SN30000 and diagnostic probe EF5. Biochemical Pharmacology, 2014, 91, 436-446.	4.4	33
30	Photodegradation of the Benzotriazine 1,4-Di-N-Oxide Hypoxia-Activated Prodrug SN30000 in Aqueous Solution. Journal of Pharmaceutical Sciences, 2014, 103, 3464-3472.	3.3	7
31	Zinc Finger Nuclease Knock-out of NADPH:Cytochrome P450 Oxidoreductase (POR) in Human Tumor Cell Lines Demonstrates That Hypoxia-activated Prodrugs Differ in POR Dependence. Journal of Biological Chemistry, 2013, 288, 37138-37153.	3.4	22
32	The Role of Bystander Effects in the Antitumor Activity of the Hypoxia-Activated Prodrug PR-104. Frontiers in Oncology, 2013, 3, 263.	2.8	46
33	The 2-Nitroimidazole EF5 Is a Biomarker for Oxidoreductases That Activate the Bioreductive Prodrug CEN-209 under Hypoxia. Clinical Cancer Research, 2012, 18, 1684-1695.	7.0	67
34	Diflavin Oxidoreductases Activate the Bioreductive Prodrug PR-104A under Hypoxia. Molecular Pharmacology, 2012, 81, 31-40.	2.3	61
35	PR-104 a bioreductive pre-prodrug combined with gemcitabine or docetaxel in a phase Ib study of patients with advanced solid tumours. BMC Cancer, 2012, 12, 496.	2.6	49
36	A phase I trial of PR-104, a pre-prodrug of the bioreductive prodrug PR-104A, given weekly to solid tumour patients. BMC Cancer, 2011, 11, 432.	2.6	56

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37	The Design of Selectively-activated Anti-cancer Prodrugs for use in Antibody-directed and Gene-directed Enzyme-Prodrug Therapies*. Journal of Pharmacy and Pharmacology, 2011, 50, 387-394.	2.4	66
38	Targeting hypoxia in cancer therapy. Nature Reviews Cancer, 2011, 11, 393-410.	28.4	2,607
39	Reductive metabolism of the dinitrobenzamide mustard anticancer prodrug PR-104 in mice. Cancer Chemotherapy and Pharmacology, 2011, 67, 543-555.	2.3	23
40	PR-104 plus sorafenib in patients with advanced hepatocellular carcinoma. Cancer Chemotherapy and Pharmacology, 2011, 68, 539-545.	2.3	29
41	Selective Treatment of Hypoxic Tumor Cells In Vivo: Phosphate Preâ€Prodrugs of Nitro Analogues of the Duocarmycins. Angewandte Chemie - International Edition, 2011, 50, 2606-2609.	13.8	43
42	Glucuronidation of Anticancer Prodrug PR-104A: Species Differences, Identification of Human UDP-Glucuronosyltransferases, and Implications for Therapy. Journal of Pharmacology and Experimental Therapeutics, 2011, 337, 692-702.	2.5	16
43	A phase I trial of PR-104, a nitrogen mustard prodrug activated by both hypoxia and aldo-keto reductase 1C3, in patients with solid tumors. Cancer Chemotherapy and Pharmacology, 2010, 65, 791-801.	2.3	86
44	Pharmacokinetic/Pharmacodynamic Modeling Identifies SN30000 and SN29751 as Tirapazamine Analogues with Improved Tissue Penetration and Hypoxic Cell Killing in Tumors. Clinical Cancer Research, 2010, 16, 4946-4957.	7.0	120
45	The Bioreductive Prodrug PR-104A Is Activated under Aerobic Conditions by Human Aldo-Keto Reductase 1C3. Cancer Research, 2010, 70, 1573-1584.	0.9	153
46	Nitro-chloromethylbenzindolines: hypoxia-activated prodrugs of potent adenine <i>N</i> 3 DNA minor groove alkylators. Molecular Cancer Therapeutics, 2009, 8, 2903-2913.	4.1	36
47	DNA Cross-Links in Human Tumor Cells Exposed to the Prodrug PR-104A: Relationships to Hypoxia, Bioreductive Metabolism, and Cytotoxicity. Cancer Research, 2009, 69, 3884-3891.	0.9	76
48	Roles of DNA repair and reductase activity in the cytotoxicity of the hypoxia-activated dinitrobenzamide mustard PR-104A. Molecular Cancer Therapeutics, 2009, 8, 1714-1723.	4.1	60
49	Hypoxia-Activated Prodrugs: Substituent Effects on the Properties of Nitro <i>seco</i> -1,2,9,9a-Tetrahydrocyclopropa[<i>c</i>]benz[<i>e</i>]indol-4-one (nitroCBI) Prodrugs of DNA Minor Groove Alkylating Agents. Journal of Medicinal Chemistry, 2009, 52, 7258-7272.	6.4	47
50	Mechanism of Action and Preclinical Antitumor Activity of the Novel Hypoxia-Activated DNA Cross-Linking Agent PR-104. Clinical Cancer Research, 2007, 13, 3922-3932.	7.0	208
51	Bystander Effects of Bioreductive Drugs: Potential for Exploiting Pathological Tumor Hypoxia with Dinitrobenzamide Mustards. Radiation Research, 2007, 167, 625-636.	1.5	61
52	Synthesis and Structureâ^'Activity Relationships for 2,4-Dinitrobenzamide-5-mustards as Prodrugs for the Escherichia coli nfsB Nitroreductase in Gene Therapy. Journal of Medicinal Chemistry, 2007, 50, 1197-1212.	6.4	38
53	Oxygen Dependence and Extravascular Transport of Hypoxia-Activated Prodrugs: Comparison of the Dinitrobenzamide Mustard PR-104A and Tirapazamine. International Journal of Radiation Oncology Biology Physics, 2007, 69, 560-571.	0.8	66
54	Use of Three-Dimensional Tissue Cultures to Model Extravascular Transport and Predict In Vivo Activity of Hypoxia-Targeted Anticancer Drugs. Journal of the National Cancer Institute, 2006, 98, 1118-1128.	6.3	139

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55	In Vitro and In Vivo Models for Evaluation of GDEPT: Quantifying Bystander Killing in Cell Cultures and Tumors. , 2004, 90, 403-432.		13
56	Oxygen Dependence of the Metabolic Activation and Cytotoxicity of Tirapazamine: Implications for Extravascular Transport andActivity in Tumors. Radiation Research, 2004, 161, 656-666.	1.5	46
57	Multicellular resistance to tirapazamine is due to restricted extravascular transport: a pharmacokinetic/pharmacodynamic study in HT29 multicellular layer cultures. Cancer Research, 2003, 63, 5970-7.	0.9	77
58	Quantitation of bystander effects in nitroreductase suicide gene therapy using three-dimensional cell cultures. Cancer Research, 2002, 62, 1425-32.	0.9	56
59	Synthesis, structures and hypoxia-selective cytotoxicity of cobalt(III) complexes containing tridentate amine and nitrogen mustard ligands. Dalton Transactions RSC, 2000, , 925-932.	2.3	71
60	Tirapazamine: a bioreductive anticancer drug that exploits tumour hypoxia. Expert Opinion on Investigational Drugs, 2000, 9, 2889-2901.	4.1	93
61	Cytotoxicity and DNA Interaction of the Enantiomers of 6-Amino-3-(chloromethyl)-1-[(5,6,7-trimethoxyindol-2-yl)carbonyl]indoline (Amino-seco-CI-TMI). Chemical Research in Toxicology, 1999, 12, 700-706.	3.3	13
62	Synthesis of 1-Substituted 3-(Chloromethyl)-6-aminoindoline (6-Amino-seco-CI) DNA Minor Groove Alkylating Agents and Structureâ^'Activity Relationships for Their Cytotoxicity. Journal of Medicinal Chemistry, 1999, 42, 649-658.	6.4	38
63	Synthesis and Cytotoxicity of Amino-seco-DSA:Â An Amino Analogue of the DNA Alkylating Agent Duocarmycin SA. Journal of Organic Chemistry, 1999, 64, 5946-5953.	3.2	33
64	Extravascular diffusion of tirapazamine: effect of metabolic consumption assessed using the multicellular layer model. International Journal of Radiation Oncology Biology Physics, 1998, 42, 641-649.	0.8	93
65	Synthesis and Cytotoxicity of 5-Amino-1-(chloromethyl)-3-[(5,6,7-trimethoxyindol-2-yl)carbonyl]-1,2- dihydro-3H-benz[e]indole (Amino-seco-CBI-TMI) and Related 5-Alkylamino Analogues:  New DNA Minor Groove Alkylating Agents. Journal of Organic Chemistry, 1998, 63, 9414-9420.	3.2	50
66	Hypoxia-Selective Antitumor Agents. 14. Synthesis and Hypoxic Cell Cytotoxicity of Regioisomers of the Hypoxia-Selective Cytotoxin 5-[N,N-Bis(2-chloroethyl)amino]-2,4-dinitrobenzamide. Journal of Medicinal Chemistry, 1996, 39, 2518-2528.	6.4	40
67	Hypoxia-Selective Antitumor Agents. 12. Nitrobenzyl Quaternary Salts as Bioreductive Prodrugs of the Alkylating Agent Mechlorethamine. Journal of Medicinal Chemistry, 1996, 39, 1084-1094.	6.4	41
68	Australasian Society of Clinical and Experimental Pharmacologists and Toxicologists, 1994: HYPOXIA-ACTIVATED PRODRUGS AS ANTITUMOUR AGENTS: STRATEGIES FOR MAXIMIZING TUMOUR CELL KILLING. Clinical and Experimental Pharmacology and Physiology, 1995, 22, 881-885.	1.9	8
69	Reductive Chemistry of the Novel Hypoxia-Selective Cytotoxin 5-[N,N-Bis(2-chloroethyl)amino]-2,4-dinitrobenzamide. Journal of Medicinal Chemistry, 1995, 38, 1229-1241.	6.4	58
70	Hypoxia-Selective Antitumor Agents. 9. Structure-Activity Relationships for Hypoxia-Selective Cytotoxicity among Analogs of 5-[N,N-Bis(2-chloroethyl)amino]-2,4-dinitrobenzamide. Journal of Medicinal Chemistry, 1994, 37, 2175-2184.	6.4	32
71	Proliferative assays for the assessment of radiosensitivity of tumor cell lines using 96-well microcultures. Radiation Oncology Investigations, 1993, 1, 261-269.	0.9	13
72	Quantitative Structure—Activity Relationships for the Cytotoxicity of Substituted Aniline Mustards in Tissue Culture. ACS Symposium Series, 1989, , 291-300.	0.5	1

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73	Hypoxia-selective Radiosensitization of Mammalian Cells by Nitracrine, an Electron-affinic DNA Intercalator. International Journal of Radiation Biology and Related Studies in Physics, Chemistry, and Medicine, 1987, 51, 641-654.	1.0	29
74	Considerations for the design of nitrophenyl mustards as agents with selective toxicity for hypoxic tumor cells. Journal of Medicinal Chemistry, 1986, 29, 879-887.	6.4	129
75	Nitroacridines with selective toxicity towards hypoxic mammalian cells: Synthesis and stability of tritiated derivatives. Journal of Labelled Compounds and Radiopharmaceuticals, 1985, 22, 995-1005.	1.0	9