

# Anatoly Zhitkovich

## List of Publications by Year in descending order

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70  
papers

6,365  
citations

76326

40  
h-index

91884

69  
g-index

71  
all docs

71  
docs citations

71  
times ranked

5888  
citing authors

#	ARTICLE	IF	CITATIONS
1	Chromium in Drinking Water: Sources, Metabolism, and Cancer Risks. <i>Chemical Research in Toxicology</i> , 2011, 24, 1617-1629.	3.3	823
2	Genetic and Epigenetic Mechanisms in Metal Carcinogenesis and Cocarcinogenesis: Nickel, Arsenic, and Chromium. <i>Chemical Research in Toxicology</i> , 2008, 21, 28-44.	3.3	774
3	Importance of Chromium-DNA Adducts in Mutagenicity and Toxicity of Chromium(VI). <i>Chemical Research in Toxicology</i> , 2005, 18, 3-11.	3.3	494
4	Depletion of Intracellular Ascorbate by the Carcinogenic Metals Nickel and Cobalt Results in the Induction of Hypoxic Stress. <i>Journal of Biological Chemistry</i> , 2004, 279, 40337-40344.	3.4	268
5	A simple, sensitive assay to detect DNA-protein crosslinks in intact cells and in vivo. <i>Carcinogenesis</i> , 1992, 13, 1485-1489.	2.8	237
6	Loss of DNA-protein crosslinks from formaldehyde-exposed cells occurs through spontaneous hydrolysis and an active repair process linked to proteasome function. <i>Carcinogenesis</i> , 2000, 21, 1573-1580.	2.8	165
7	Genotoxicity and Mutagenicity of Chromium(VI)/Ascorbate-Generated DNA Adducts in Human and Bacterial Cells. <i>Biochemistry</i> , 2003, 42, 1062-1070.	2.5	158
8	Formation of the Amino Acid-DNA Complexes by Hexavalent and Trivalent Chromium in Vitro: Importance of Trivalent Chromium and the Phosphate Group. <i>Biochemistry</i> , 1996, 35, 7275-7282.	2.5	157
9	Loss of DNA-protein crosslinks from formaldehyde-exposed cells occurs through spontaneous hydrolysis and an active repair process linked to proteasome function. <i>Carcinogenesis</i> , 2000, 21, 1573-1580.	2.8	154
10	Bioavailability, Intracellular Mobilization of Nickel, and HIF-1 $\alpha$ Activation in Human Lung Epithelial Cells Exposed to Metallic Nickel and Nickel Oxide Nanoparticles. <i>Toxicological Sciences</i> , 2011, 124, 138-148.	3.1	142
11	Glutathione and free amino acids form stable complexes with DNA following exposure of intact mammalian cells to chromate. <i>Carcinogenesis</i> , 1995, 16, 907-913.	2.8	137
12	Cr(III)-mediated crosslinks of glutathione or amino acids to the DNA phosphate backbone are mutagenic in human cells. <i>Nucleic Acids Research</i> , 1998, 26, 2024-2030.	14.5	136
13	Ascorbate acts as a highly potent inducer of chromate mutagenesis and clastogenesis: linkage to DNA breaks in G2 phase by mismatch repair. <i>Nucleic Acids Research</i> , 2006, 35, 465-476.	14.5	118
14	The Chromate-Inducible <i>chrBACF</i> Operon from the Transposable Element Tn <i>OtChr</i> Confers Resistance to Chromium(VI) and Superoxide. <i>Journal of Bacteriology</i> , 2008, 190, 6996-7003.	2.2	118
15	Mismatch Repair Proteins Are Activators of Toxic Responses to Chromium-DNA Damage. <i>Molecular and Cellular Biology</i> , 2005, 25, 3596-3607.	2.3	116
16	Non-Oxidative Mechanisms Are Responsible for the Induction of Mutagenesis by Reduction of Cr(VI) with Cysteine: A Role of Ternary DNA Adducts in Cr(III)-Dependent Mutagenesis. <i>Biochemistry</i> , 2001, 40, 549-560.	2.5	113
17	Reductive activation with cysteine represents a chromium(III)-dependent pathway in the induction of genotoxicity by carcinogenic chromium(VI). <i>Environmental Health Perspectives</i> , 2002, 110, 729-731.	6.0	110
18	N-Acetylcysteine: Antioxidant, Aldehyde Scavenger, and More. <i>Chemical Research in Toxicology</i> , 2019, 32, 1318-1319.	3.3	106

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19	Carcinogenic Chromium(VI) Induces Cross-Linking of Vitamin C to DNA in Vitro and in Human Lung A549 Cells. <i>Biochemistry</i> , 2002, 41, 3156-3167.	2.5	96
20	Analysis of the binding sites of chromium to DNA and protein in vitro and in intact cells. <i>Carcinogenesis</i> , 1992, 13, 2341-2346.	2.8	94
21	Analysis of DNA-protein crosslinking activity of malondialdehyde in vitro. <i>Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis</i> , 1999, 424, 97-106.	1.0	89
22	Human Nucleotide Excision Repair Efficiently Removes Chromium-DNA Phosphate Adducts and Protects Cells against Chromate Toxicity. <i>Journal of Biological Chemistry</i> , 2004, 279, 30419-30424.	3.4	89
23	Rapid DNA Double-Strand Breaks Resulting from Processing of Cr-DNA Cross-Links by Both MutS Dimers. <i>Cancer Research</i> , 2009, 69, 1071-1079.	0.9	85
24	DNA-PROTEIN CROSS-LINKS PRODUCED BY VARIOUS CHEMICALS IN CULTURED HUMAN LYMPHOMA CELLS. <i>Journal of Toxicology and Environmental Health - Part A: Current Issues</i> , 1997, 50, 433-449.	2.3	83
25	Mechanism of DNA-Protein Cross-Linking by Chromium. <i>Chemical Research in Toxicology</i> , 2010, 23, 341-347.	3.3	79
26	Differentiation of DNA reactive and non-reactive genotoxic mechanisms using gene expression profile analysis. <i>Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis</i> , 2004, 549, 29-41.	1.0	72
27	Causes of DNA single-strand breaks during reduction of chromate by glutathione in vitro and in cells. <i>Free Radical Biology and Medicine</i> , 2006, 40, 1981-1992.	2.9	71
28	Interlaboratory validation of a new assay for DNA-protein crosslinks. <i>Mutation Research - Genetic Toxicology Testing and Biomonitoring of Environmental Or Occupational Exposure</i> , 1996, 369, 13-21.	1.2	66
29	Role of mismatch repair proteins in the processing of cisplatin interstrand cross-links. <i>DNA Repair</i> , 2015, 35, 126-136.	2.8	65
30	Ascorbate depletion mediates up-regulation of hypoxia-associated proteins by cell density and nickel. <i>Journal of Cellular Biochemistry</i> , 2006, 97, 1025-1035.	2.6	64
31	Reductive Metabolism of Cr(VI) by Cysteine Leads to the Formation of Binary and Ternary Cr-DNA Adducts in the Absence of Oxidative DNA Damage. <i>Chemical Research in Toxicology</i> , 2000, 13, 1114-1124.	3.3	58
32	Cellular vitamin C increases chromate toxicity via a death program requiring mismatch repair but not p53. <i>Carcinogenesis</i> , 2007, 28, 1613-1620.	2.8	58
33	Role of direct reactivity with metals in chemoprotection by N-acetylcysteine against chromium(VI), cadmium(II), and cobalt(II). <i>Free Radical Biology and Medicine</i> , 2013, 65, 262-269.	2.9	58
34	Vitamin C as a Modulator of the Response to Cancer Therapy. <i>Molecules</i> , 2019, 24, 453.	3.8	58
35	S-phase sensing of DNA-protein crosslinks triggers TopBP1-independent ATR activation and p53-mediated cell death by formaldehyde. <i>Cell Cycle</i> , 2012, 11, 2526-2537.	2.6	48
36	DNA Double-Strand Breaks by Cr(VI) Are Targeted to Euchromatin and Cause ATR-Dependent Phosphorylation of Histone H2AX and Its Ubiquitination. <i>Toxicological Sciences</i> , 2015, 143, 54-63.	3.1	47

#	ARTICLE	IF	CITATIONS
37	Title is missing!. Molecular and Cellular Biochemistry, 2001, 222, 107-118.	3.1	44
38	Nuclear and Cytoplasmic Functions of Vitamin C. Chemical Research in Toxicology, 2020, 33, 2515-2526.	3.3	42
39	Increased DNA-protein crosslinks in lymphocytes of residents living in chromium-contaminated areas. Biological Trace Element Research, 1995, 50, 175-180.	3.5	41
40	DNA-protein crosslinks in peripheral lymphocytes of individuals exposed to hexavalent chromium compounds. Biomarkers, 1996, 1, 86-93.	1.9	41
41	WRN helicase promotes repair of DNA double-strand breaks caused by aberrant mismatch repair of chromium-DNA adducts. Cell Cycle, 2009, 8, 2769-2778.	2.6	41
42	Monitoring Cr Intermediates and Reactive Oxygen Species with Fluorescent Probes during Chromate Reduction. Chemical Research in Toxicology, 2014, 27, 843-851.	3.3	39
43	Monoubiquitinated $\gamma$ -H2AX: Abundant product and specific biomarker for non-apoptotic DNA double-strand breaks. Toxicology and Applied Pharmacology, 2018, 355, 238-246.	2.8	38
44	Undetectable role of oxidative DNA damage in cell cycle, cytotoxic and clastogenic effects of Cr(VI) in human lung cells with restored ascorbate levels. Mutagenesis, 2012, 27, 437-443.	2.6	37
45	p53 activation by Ni(II) is a HIF-1 $\alpha$ independent response causing caspases 9/3-mediated apoptosis in human lung cells. Toxicology and Applied Pharmacology, 2013, 269, 233-239.	2.8	31
46	Heat Shock Proteins and HSF1 in Cancer. Frontiers in Oncology, 2022, 12, 860320.	2.8	30
47	Reduction with Glutathione Is a Weakly Mutagenic Pathway in Chromium(VI) Metabolism. Chemical Research in Toxicology, 2008, 21, 2188-2194.	3.3	29
48	Formaldehyde Is a Potent Proteotoxic Stressor Causing Rapid Heat Shock Transcription Factor 1 Activation and Lys48-Linked Polyubiquitination of Proteins. American Journal of Pathology, 2016, 186, 2857-2868.	3.8	28
49	Killing of Chromium-Damaged Cells by Mismatch Repair and its Relevance to Carcinogenesis. Cell Cycle, 2005, 4, 4050-4052.	2.6	25
50	XPA impacts formation but not proteasome-sensitive repair of DNA-protein cross-links induced by chromate. Mutagenesis, 2010, 25, 381-388.	2.6	24
51	ATM and KAT5 safeguard replicating chromatin against formaldehyde damage. Nucleic Acids Research, 2016, 44, 198-209.	14.5	24
52	Different ATM Signaling in Response to Chromium(VI) Metabolism via Ascorbate and Nonascorbate Reduction: Implications for <i>in Vitro</i> Models and Toxicogenomics. Environmental Health Perspectives, 2016, 124, 61-66.	6.0	22
53	Lower mutagenicity but higher stability of Cr-DNA adducts formed during gradual chromate activation with ascorbate. Carcinogenesis, 2006, 27, 2316-2321.	2.8	21
54	XRCC1 Deficiency Sensitizes Human Lung Epithelial Cells to Genotoxicity by Crocidolite Asbestos and Libby Amphibole. Environmental Health Perspectives, 2010, 118, 1707-1713.	6.0	19

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55	Uptake, p53 Pathway Activation, and Cytotoxic Responses for Co(II) and Ni(II) in Human Lung Cells: Implications for Carcinogenicity. <i>Toxicological Sciences</i> , 2013, 136, 467-477.	3.1	18
56	Proteasome activity is important for replication recovery, CHK1 phosphorylation and prevention of G2 arrest after low-dose formaldehyde. <i>Toxicology and Applied Pharmacology</i> , 2015, 286, 135-141.	2.8	18
57	Chromium(VI) Causes Interstrand DNA Cross-Linking <i>in Vitro</i> but Shows No Hypersensitivity in Cross-Link Repair-Deficient Human Cells. <i>Chemical Research in Toxicology</i> , 2013, 26, 1591-1598.	3.3	17
58	Metabolism of Cr(VI) by ascorbate but not glutathione is a low oxidant-generating process. <i>Journal of Trace Elements in Medicine and Biology</i> , 2012, 26, 192-196.	3.0	15
59	Nickel-induced HIF-1 $\alpha$ promotes growth arrest and senescence in normal human cells but lacks toxic effects in transformed cells. <i>Toxicology and Applied Pharmacology</i> , 2017, 331, 94-100.	2.8	15
60	Vitamin C increases DNA breaks and suppresses DNA damage-independent activation of ATM by bleomycin. <i>Free Radical Biology and Medicine</i> , 2019, 136, 12-21.	2.9	15
61	Killing of chromium-damaged cells by mismatch repair and its relevance to carcinogenesis. <i>Cell Cycle</i> , 2005, 4, 1050-2.	2.6	13
62	Development and utilization of a new simple assay for DNA-protein crosslinks as a biomarker of exposure to welding fumes. <i>International Archives of Occupational and Environmental Health</i> , 1993, 65, S87-S89.	2.3	12
63	Ascorbate: antioxidant and biochemical activities and their importance for in vitro models. <i>Archives of Toxicology</i> , 2021, 95, 3623-3631.	4.2	12
64	Reduction of Cr (VI) by cysteine: Significance in human lymphocytes and formation of DNA damage in reactions with variable reduction rates. , 2001, , 107-118.		11
65	Variation in Extracellular Detoxification Is a Link to Different Carcinogenicity among Chromates in Rodent and Human Lungs. <i>Chemical Research in Toxicology</i> , 2017, 30, 1720-1729.	3.3	10
66	Toxicological Antagonism among Welding Fume Metals: Inactivation of Soluble Cr(VI) by Iron. <i>Chemical Research in Toxicology</i> , 2018, 31, 1172-1184.	3.3	6
67	p53 Activation by Cr(VI): A Transcriptionally Limited Response Induced by ATR Kinase in S-Phase. <i>Toxicological Sciences</i> , 2019, 172, 11-22.	3.1	6
68	20S immunoproteasomes remove formaldehyde-damaged cytoplasmic proteins suppressing caspase-independent cell death. <i>Scientific Reports</i> , 2017, 7, 654.	3.3	4
69	NAD <sup>+</sup> metabolism controls growth inhibition by HIF1 in normoxia and determines differential sensitivity of normal and cancer cells. <i>Cell Cycle</i> , 2021, 20, 1812-1827.	2.6	4
70	Vulnerability of HIF1 $\alpha$ and HIF2 $\alpha$ to damage by proteotoxic stressors. <i>Toxicology and Applied Pharmacology</i> , 2022, 445, 116041.	2.8	2