Anatoly Zhitkovich

List of Publications by Year
in descending order

70	6,365	76326	91884
papers	citations	40 h-index	69 g-index

71	71	71	5888
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Heat Shock Proteins and HSF1 in Cancer. Frontiers in Oncology, 2022, 12, 860320.	2.8	30
2	Vulnerability of HIF1Î± and HIF2Î± to damage by proteotoxic stressors. Toxicology and Applied Pharmacology, 2022, 445, 116041.	2.8	2
3	NAD⁺ metabolism controls growth inhibition by HIF1 in normoxia and determines differential sensitivity of normal and cancer cells. Cell Cycle, 2021, 20, 1812-1827.	2.6	4
4	Ascorbate: antioxidant and biochemical activities and their importance for in vitro models. Archives of Toxicology, 2021, 95, 3623-3631.	4.2	12
5	Nuclear and Cytoplasmic Functions of Vitamin C. Chemical Research in Toxicology, 2020, 33, 2515-2526.	3.3	42
6	p53 Activation by Cr(VI): A Transcriptionally Limited Response Induced by ATR Kinase in S-Phase. Toxicological Sciences, 2019, 172, 11-22.	3.1	6
7	Vitamin C as a Modulator of the Response to Cancer Therapy. Molecules, 2019, 24, 453.	3.8	58
8	N-Acetylcysteine: Antioxidant, Aldehyde Scavenger, and More. Chemical Research in Toxicology, 2019, 32, 1318-1319.	3.3	106
9	Vitamin C increases DNA breaks and suppresses DNA damage-independent activation of ATM by bleomycin. Free Radical Biology and Medicine, 2019, 136, 12-21.	2.9	15
10	Toxicological Antagonism among Welding Fume Metals: Inactivation of Soluble Cr(VI) by Iron. Chemical Research in Toxicology, 2018, 31, 1172-1184.	3.3	6
11	Monoubiquitinated Î³-H2AX: Abundant product and specific biomarker for non-apoptotic DNA double-strand breaks. Toxicology and Applied Pharmacology, 2018, 355, 238-246.	2.8	38
12	Nickel-induced HIF-1Î± promotes growth arrest and senescence in normal human cells but lacks toxic effects in transformed cells. Toxicology and Applied Pharmacology, 2017, 331, 94-100.	2.8	15
13	20S immunoproteasomes remove formaldehyde-damaged cytoplasmic proteins suppressing caspase-independent cell death. Scientific Reports, 2017, 7, 654.	3.3	4
14	Variation in Extracellular Detoxification Is a Link to Different Carcinogenicity among Chromates in Rodent and Human Lungs. Chemical Research in Toxicology, 2017, 30, 1720-1729.	3.3	10
15	Different ATM Signaling in Response to Chromium(VI) Metabolism via Ascorbate and Nonascorbate Reduction: Implications for in Vitro Models and Toxicogenomics. Environmental Health Perspectives, 2016, 124, 61-66.	6.0	22
16	ATM and KAT5 safeguard replicating chromatin against formaldehyde damage. Nucleic Acids Research, 2016, 44, 198-209.	14.5	24
17	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
18	Proteasome activity is important for replication recovery, CHK1 phosphorylation and prevention of G2 arrest after low-dose formaldehyde. Toxicology and Applied Pharmacology, 2015, 286, 135-141.	2.8	18

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19	DNA Double-Strand Breaks by Cr(VI) Are Targeted to Euchromatin and Cause ATR-Dependent Phosphorylation of Histone H2AX and Its Ubiquitination. Toxicological Sciences, 2015, 143, 54-63.	3.1	47
20	Role of mismatch repair proteins in the processing of cisplatin interstrand cross-links. DNA Repair, 2015, 35, 126-136.	2.8	65
21	Monitoring Cr Intermediates and Reactive Oxygen Species with Fluorescent Probes during Chromate Reduction. Chemical Research in Toxicology, 2014, 27, 843-851.	3.3	39
22	Role of direct reactivity with metals in chemoprotection by N-acetylcysteine against chromium(VI), cadmium(II), and cobalt(II). Free Radical Biology and Medicine, 2013, 65, 262-269.	2.9	58
23	p53 activation by Ni(II) is a HIF-1Î± independent response causing caspases 9/3-mediated apoptosis in human lung cells. Toxicology and Applied Pharmacology, 2013, 269, 233-239.	2.8	31
24	Chromium(VI) Causes Interstrand DNA Cross-Linking in Vitro but Shows No Hypersensitivity in Cross-Link Repair-Deficient Human Cells. Chemical Research in Toxicology, 2013, 26, 1591-1598.	3.3	17
25	Uptake, p53 Pathway Activation, and Cytotoxic Responses for Co(II) and Ni(II) in Human Lung Cells: Implications for Carcinogenicity. Toxicological Sciences, 2013, 136, 467-477.	3.1	18
26	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
27	Metabolism of Cr(VI) by ascorbate but not glutathione is a low oxidant-generating process. Journal of Trace Elements in Medicine and Biology, 2012, 26, 192-196.	3.0	15
28	S-phase sensing of DNA-protein crosslinks triggers TopBP1-independent ATR activation and p53-mediated cell death by formaldehyde. Cell Cycle, 2012, 11, 2526-2537.	2.6	48
29	Chromium in Drinking Water: Sources, Metabolism, and Cancer Risks. Chemical Research in Toxicology, 2011, 24, 1617-1629.	3.3	823
30	Bioavailability, Intracellular Mobilization of Nickel, and HIF-1Î± Activation in Human Lung Epithelial Cells Exposed to Metallic Nickel and Nickel Oxide Nanoparticles. Toxicological Sciences, 2011, 124, 138-148.	3.1	142
31	XPA impacts formation but not proteasome-sensitive repair of DNA-protein cross-links induced by chromate. Mutagenesis, 2010, 25, 381-388.	2.6	24
32	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
33	Mechanism of DNAâ^'Protein Cross-Linking by Chromium. Chemical Research in Toxicology, 2010, 23, 341-347.	3.3	79
34	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
35	Rapid DNA Double-Strand Breaks Resulting from Processing of Cr-DNA Cross-Links by Both MutS Dimers. Cancer Research, 2009, 69, 1071-1079.	0.9	85
36	Genetic and Epigenetic Mechanisms in Metal Carcinogenesis and Cocarcinogenesis: Nickel, Arsenic, and Chromium. Chemical Research in Toxicology, 2008, 21, 28-44.	3.3	774

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37	Reduction with Glutathione Is a Weakly Mutagenic Pathway in Chromium(VI) Metabolism. Chemical Research in Toxicology, 2008, 21, 2188-2194.	3.3	29
38	The Chromate-Inducible chrBACF Operon from the Transposable Element Tn OtChr Confers Resistance to Chromium(VI) and Superoxide. Journal of Bacteriology, 2008, 190, 6996-7003.	2.2	118
39	Cellular vitamin C increases chromate toxicity via a death program requiring mismatch repair but not p53. Carcinogenesis, 2007, 28, 1613-1620.	2.8	58
40	Causes of DNA single-strand breaks during reduction of chromate by glutathione in vitro and in cells. Free Radical Biology and Medicine, 2006, 40, 1981-1992.	2.9	71
41	Ascorbate depletion mediates up-regulation of hypoxia-associated proteins by cell density and nickel. Journal of Cellular Biochemistry, 2006, 97, 1025-1035.	2.6	64
42	Ascorbate acts as a highly potent inducer of chromate mutagenesis and clastogenesis: linkage to DNA breaks in G2 phase by mismatch repair. Nucleic Acids Research, 2006, 35, 465-476.	14.5	118
43	Lower mutagenicity but higher stability of Cr-DNA adducts formed during gradual chromate activation with ascorbate. Carcinogenesis, 2006, 27, 2316-2321.	2.8	21
44	Killing of Chromium-Damaged Cells by Mismatch Repair and its Relevance to Carcinogenesis. Cell Cycle, 2005, 4, 4050-4052.	2.6	25
45	Mismatch Repair Proteins Are Activators of Toxic Responses to Chromium-DNA Damage. Molecular and Cellular Biology, 2005, 25, 3596-3607.	2.3	116
46	Importance of Chromiumâ”DNA Adducts in Mutagenicity and Toxicity of Chromium(VI). Chemical Research in Toxicology, 2005, 18, 3-11.	3.3	494
47	Killing of chromium-damaged cells by mismatch repair and its relevance to carcinogenesis. Cell Cycle, 2005, 4, 1050-2.	2.6	13
48	Depletion of Intracellular Ascorbate by the Carcinogenic Metals Nickel and Cobalt Results in the Induction of Hypoxic Stress. Journal of Biological Chemistry, 2004, 279, 40337-40344.	3.4	268
49	Differentiation of DNA reactive and non-reactive genotoxic mechanisms using gene expression profile analysis. Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis, 2004, 549, 29-41.	1.0	72
50	Human Nucleotide Excision Repair Efficiently Removes Chromium-DNA Phosphate Adducts and Protects Cells against Chromate Toxicity. Journal of Biological Chemistry, 2004, 279, 30419-30424.	3.4	89
51	Genotoxicity and Mutagenicity of Chromium(VI)/Ascorbate-Generated DNA Adducts in Human and Bacterial Cellsâ€. Biochemistry, 2003, 42, 1062-1070.	2.5	158
52	Carcinogenic Chromium(VI) Induces Cross-Linking of Vitamin C to DNA in Vitro and in Human Lung A549 Cells. Biochemistry, 2002, 41, 3156-3167.	2.5	96
53	Reductive activation with cysteine represents a chromium(III)-dependent pathway in the induction of genotoxicity by carcinogenic chromium(VI).. Environmental Health Perspectives, 2002, 110, 729-731.	6.0	110
54	Title is missing!. Molecular and Cellular Biochemistry, 2001, 222, 107-118.	3.1	44

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55	Non-Oxidative Mechanisms Are Responsible for the Induction of Mutagenesis by Reduction of Cr(VI) with Cysteine:Â Role of Ternary DNA Adducts in Cr(III)-Dependent Mutagenesisâ€. Biochemistry, 2001, 40, 549-560.	2.5	113
56	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
57	Loss of DNAâ€“protein crosslinks from formaldehyde-exposed cells occurs through spontaneous hydrolysis and an active repair process linked to proteosome function. Carcinogenesis, 2000, 21, 1573-1580.	2.8	165
58	Loss of DNAâ€“protein crosslinks from formaldehyde-exposed cells occurs through spontaneous hydrolysis and an active repair process linked to proteosome function. Carcinogenesis, 2000, 21, 1573-1580.	2.8	154
59	Reductive Metabolism of Cr(VI) by Cysteine Leads to the Formation of Binary and Ternary Crâ”DNA Adducts in the Absence of Oxidative DNA Damage. Chemical Research in Toxicology, 2000, 13, 1114-1124.	3.3	58
60	Analysis of DNAâ€“protein crosslinking activity of malondialdehyde in vitro. Mutation Research - Fundamental and Molecular Mechanisms of Mutagenesis, 1999, 424, 97-106.	1.0	89
61	Cr(III)-mediated crosslinks of glutathione or amino acids to the DNA phosphate backbone are mutagenic in human cells. Nucleic Acids Research, 1998, 26, 2024-2030.	14.5	136
62	DNA-PROTEIN CROSS-LINKS PRODUCED BY VARIOUS CHEMICALS IN CULTURED HUMAN LYMPHOMA CELLS. Journal of Toxicology and Environmental Health - Part A: Current Issues, 1997, 50, 433-449.	2.3	83
63	Formation of the Amino Acidâ”DNA Complexes by Hexavalent and Trivalent Chromium in Vitro: Importance of Trivalent Chromium and the Phosphate Group. Biochemistry, 1996, 35, 7275-7282.	2.5	157
64	Interlaboratory validation of a new assay for DNA-protein crosslinks. Mutation Research - Genetic Toxicology Testing and Biomonitoring of Environmental Or Occupational Exposure, 1996, 369, 13-21.	1.2	66
65	DNA-protein crosslinks in peripheral lymphocytes of individuals exposed to hexavalent chromium compounds. Biomarkers, 1996, 1, 86-93.	1.9	41
66	Increased DNA-protein crosslinks in lymphocytes of residents living in chromium-contaminated areas. Biological Trace Element Research, 1995, 50, 175-180.	3.5	41
67	Glutathione and free amino acids form stable complexes with DNA following exposure of intact mammalian cells to chromate. Carcinogenesis, 1995, 16, 907-913.	2.8	137
68	Development and utilization of a new simple assay for DNA-protein crosslinks as a biomarker of exposure to welding fumes. International Archives of Occupational and Environmental Health, 1993, 65, S87-S89.	2.3	12
69	Analysis of the binding sites of chromium to DNA and protein in vitro and in intact cells. Carcinogenesis, 1992, 13, 2341-2346.	2.8	94
70	A simple, sensitive assay to detect DNAâ€“protein cromlinks in intact cells and in vivo. Carcinogenesis, 1992, 13, 1485-1489.	2.8	237