List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	A novel imidazolinone metforminâ€methylglyoxal metabolite promotes endothelial cell angiogenesis via the eNOS/HIFâ€1α pathway. FASEB Journal, 2021, 35, e21645.	0.5	6
2	Cell-specific regulation of Nrf2 during ROS-Dependent cell death caused by 2,3,5-tris(glutathion-S-yl)hydroquinone (TGHQ). Chemico-Biological Interactions, 2019, 302, 1-10.	4.0	10
3	Toxicoproteomic Analysis of Poly(ADP-Ribose)-Associated Proteins Induced by Oxidative Stress in Human Proximal Tubule Cells. Toxicological Sciences, 2019, 171, 117-131.	3.1	0
4	Ameliorating Methylglyoxal-Induced Progenitor Cell Dysfunction for Tissue Repair in Diabetes. Diabetes, 2019, 68, 1287-1302.	0.6	25
5	Concurrent Inhibition of Vesicular Monoamine Transporter 2 Does Not Protect Against 3,4-Methylenedioxymethamphetamine (Ecstasy) Induced Neurotoxicity. Toxicological Sciences, 2019, 170, 157-166.	3.1	2
6	All- <i>trans</i> -retinoic acid-mediated cytoprotection in LLC-PK <sub>1</sub> renal epithelial cells is coupled to <i>p</i> -ERK activation in a ROS-independent manner. American Journal of Physiology - Renal Physiology, 2017, 313, F1200-F1208.	2.7	6
7	MiR-27b augments bone marrow progenitor cell survival via suppressing the mitochondrial apoptotic pathway in Type 2 diabetes. American Journal of Physiology - Endocrinology and Metabolism, 2017, 313, E391-E401.	3.5	25
8	From the Cover: ROS-Induced Store-Operated Ca2+ Entry Coupled to PARP-1 Hyperactivation Is Independent of PARG Activity in Necrotic Cell Death. Toxicological Sciences, 2017, 158, 444-453.	3.1	11
9	In situ, dual-mode monitoring of organ-on-a-chip with smartphone-based fluorescence microscope. Biosensors and Bioelectronics, 2016, 86, 697-705.	10.1	69
10	Exploration of earlyâ€life candidate biomarkers for childhood asthma using antibody arrays. Pediatric Allergy and Immunology, 2016, 27, 696-701.	2.6	9
11	From the Cover: Arsenic Induces Accumulation of α-Synuclein: Implications for Synucleinopathies and Neurodegeneration. Toxicological Sciences, 2016, 153, 271-281.	3.1	41
12	Transcriptional and postâ€translational modifications of Bâ€Raf in quinolâ€thioether induced tuberous sclerosis renal cell carcinoma. Molecular Carcinogenesis, 2016, 55, 1243-1250.	2.7	0
13	Metformin Scavenges Methylglyoxal To Form a Novel Imidazolinone Metabolite in Humans. Chemical Research in Toxicology, 2016, 29, 227-234.	3.3	72
14	Vesicular Monoamine Transporter 2 and the Acute and Long-Term Response to 3,4-(±)-Methylenedioxymethamphetamine. Toxicological Sciences, 2015, 143, 209-219.	3.1	13
15	Site specific modification of the human plasma proteome by methylglyoxal. Toxicology and Applied Pharmacology, 2015, 289, 155-162.	2.8	17
16	Glial Cell Response to 3,4-(±)-Methylenedioxymethamphetamine and Its Metabolites. Toxicological Sciences, 2014, 138, 130-138.	3.1	13
17	Catechol-O-Methyltransferase and 3,4-(±)-Methylenedioxymethamphetamine Toxicity. Toxicological Sciences, 2014, 139, 162-173.	3.1	4
18	PARP-1 Hyperactivation and Reciprocal Elevations in Intracellular Ca2+ During ROS-Induced Nonapoptotic Cell Death. Toxicological Sciences, 2014, 140, 118-134.	3.1	45

**TERRENCE J MONKS** 

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19	Reactive Intermediates. Toxicologic Pathology, 2013, 41, 315-321.	1.8	6
20	Pentoxifylline Initiates GSKâ€3βâ€Induced Proteasomal Degradation of Cyclin D1 and Arrests Renal Cancer Cells in the G1 Phase. FASEB Journal, 2013, 27, 1030.2.	0.5	0
21	Perturbations in Intracellular Ca2+ Concentrations and DNA Damage are Coupled to the Activation of PARPâ€1 During ROSâ€induced Necrotic Cell Death. FASEB Journal, 2013, 27, 890.5.	0.5	0
22	A Dual Role for Poly(ADP-Ribose) Polymerase-1 †During Caspase-Dependent Apoptosis. Toxicological Sciences, 2012, 128, 103-114.	3.1	36
23	Utilization of LC-MS/MS Analyses to Identify Site-Specific Chemical Protein Adducts In Vitro. Methods in Molecular Biology, 2011, 691, 317-326.	0.9	2
24	New site(s) of methylglyoxal-modified human serum albumin, identified by multiple reaction monitoring, alter warfarin binding and prostaglandin metabolism. Chemico-Biological Interactions, 2011, 192, 122-128.	4.0	22
25	ERK Crosstalks with 4EBP1 to Activate Cyclin D1 Translation during Quinol-Thioether–Induced Tuberous Sclerosis Renal Cell Carcinoma. Toxicological Sciences, 2011, 124, 75-87.	3.1	18
26	The Frequency of 1,4-Benzoquinone-Lysine Adducts in Cytochrome c Correlate with Defects in Apoptosome Activation. Toxicological Sciences, 2011, 122, 64-72.	3.1	9
27	The Cytoprotective Effect of N-acetyl-L-cysteine against ROS-Induced Cytotoxicity Is Independent of Its Ability to Enhance Clutathione Synthesis. Toxicological Sciences, 2011, 120, 87-97.	3.1	97
28	Utilization of MALDI-TOF to Determine Chemical-Protein Adduct Formation In Vitro. Methods in Molecular Biology, 2011, 691, 303-316.	0.9	2
29	One-Dimensional Western Blotting Coupled to LC-MS/MS Analysis to Identify Chemical-Adducted Proteins in Rat Urine. Methods in Molecular Biology, 2011, 691, 327-338.	0.9	2
30	Identification of Chemical-Adducted Proteins in Urine by Multi-dimensional Protein Identification Technology (LC/LC–MS/MS). Methods in Molecular Biology, 2011, 691, 339-347.	0.9	1
31	Role of hydroquinone–thiol conjugates in benzene-mediated toxicity. Chemico-Biological Interactions, 2010, 184, 212-217.	4.0	15
32	The fate of benzene-oxide. Chemico-Biological Interactions, 2010, 184, 201-206.	4.0	42
33	cAMPâ€dependent pathway(s) directs the Bâ€Raf MAPKâ€Mediated Cytosolic Mislocalization of p27kip•yclin D1 in Renal Cancer. FASEB Journal, 2010, 24, .	0.5	0
34	Neurotoxic Thioether Adducts of 3,4-Methylenedioxymethamphetamine Identified in Human Urine After Ecstasy Ingestion. Drug Metabolism and Disposition, 2009, 37, 1448-1455.	3.3	30
35	Protein Electrophile-Binding Motifs: Lysine-Rich Proteins Are Preferential Targets of Quinones. Drug Metabolism and Disposition, 2009, 37, 1211-1218.	3.3	25
36	Improved MALDI-TOF imaging yields increased protein signals at high molecular mass. Journal of the American Society for Mass Spectrometry, 2009, 20, 89-95.	2.8	50

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37	Serotonergic Neurotoxic Thioether Metabolites of 3,4-Methylenedioxymethamphetamine (MDMA,) Tj ETQq1 Toxicology, 2008, 21, 2272-2279.	1 0.784314 rg 3.3	gBT /Overloc 14
38	Modulation of Human Multidrug Resistance Protein (MRP) 1 (ABCC1) and MRP2 (ABCC2) Transport Activities by Endogenous and Exogenous Glutathione-Conjugated Catechol Metabolites. Drug Metabolism and Disposition, 2008, 36, 552-560.	3.3	33
39	Accumulation of Neurotoxic Thioether Metabolites of 3,4-(±)-Methylenedioxymethamphetamine in Rat Brain. Journal of Pharmacology and Experimental Therapeutics, 2008, 324, 284-291.	2.5	40
40	SITE SPECIFIC PHOSPHORYLATION OF HEAT SHOCK PROTEIN 27 (HSP27) REGULATES CELL SURVIVAL AND DEATH. FASEB Journal, 2008, 22, 1140.7.	0.5	0
41	1,4â€Benzoquinone forms an unstable thioether bond with cysteine residues that is eliminated in basic conditions. FASEB Journal, 2008, 22, 1131.6.	0.5	0
42	Quinone Electrophiles Selectively Adduct "Electrophile Binding Motifs―within Cytochrome <i>c</i> . Biochemistry, 2007, 46, 11090-11100.	2.5	51
43	Ros-Induced Histone Modifications and their Role in Cell Survival and Cell Death. Drug Metabolism Reviews, 2006, 38, 755-767.	3.6	48
44	Neurotoxic Metabolites of Ecstasy Accumulate in Rat Brain Following Multiple Injections. FASEB Journal, 2006, 20, A1134.	0.5	0
45	Identifying siteâ€specific chemical modifications of urinary excreted proteins using a proteomics approach. FASEB Journal, 2006, 20, A66.	0.5	0
46	Renal toxicantâ€specific heat shock protein 27 phosphorylation. FASEB Journal, 2006, 20, A66.	0.5	0
47	Arylation of cytochrome c by benzoquinone and benzoquinoneâ€ŧhioether causes a loss of protein function. FASEB Journal, 2006, 20, A66.	0.5	0
48	Immuohistochemical and MALDI Imaging Reveal Changes in Expression and Phosphorylation of Annexin I and II in Chemicalâ€Induced Renal Tumors. FASEB Journal, 2006, 20, A66.	0.5	0
49	Serotonergic Neurotoxic Metabolites of Ecstasy Identified in Rat Brain. Journal of Pharmacology and Experimental Therapeutics, 2005, 313, 422-431.	2.5	108
50	2,3,5-tris(Glutathion-S-yl)hydroquinone (TGHQ)-Mediated Apoptosis of Human Promyelocytic Leukemia Cells Is Preceded by Mitochondrial Cytochrome c Release in the Absence of a Decrease in the Mitochondrial Membrane Potential. Toxicological Sciences, 2005, 86, 92-100.	3.1	11
51	Alkylation of Cytochromecby (Glutathion-S-yl)-1,4-benzoquinone and Iodoacetamide Demonstrates Compound-Dependent Site Specificity. Chemical Research in Toxicology, 2005, 18, 41-50.	3.3	28
52	Age-dependent (+)MDMA-mediated Neurotoxicity in Mice. NeuroToxicology, 2005, 26, 1031-1040.	3.0	20
53	Response to Sprague and Nichols: Contribution of metabolic activation to MDMA neurotoxicity. Trends in Pharmacological Sciences, 2005, 26, 60-61.	8.7	5
54	Tuberous sclerosis-2 tumor suppressor modulates ERK and B-Raf activity in transformed renal epithelial cells. American Journal of Physiology - Renal Physiology, 2004, 286, F417-F424.	2.7	14

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55	Induction of ERK1/2 and Histone H3 Phosphorylation within the Outer Stripe of the Outer Medulla of the Eker Rat by 2,3,5-Tris-(Glutathion-S-yl)hydroquinone. Toxicological Sciences, 2004, 80, 350-357.	3.1	9
56	Thioether Metabolites of 3,4-Methylenedioxyamphetamine and 3,4-Methylenedioxymethamphetamine Inhibit Human Serotonin Transporter (hSERT) Function and Simultaneously Stimulate Dopamine Uptake into hSERT-Expressing SK-N-MC Cells. Journal of Pharmacology and Experimental Therapeutics, 2004, 311, 298-306.	2.5	46
57	Menadione metabolism to thiodione in hepatoblastoma by scanning electrochemical microscopy. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 17582-17587.	7.1	91
58	EGFR-independent activation of p38 MAPK and EGFR-dependent activation of ERK1/2 are required for ROS-induced renal cell death. American Journal of Physiology - Renal Physiology, 2004, 287, F1049-F1058.	2.7	83
59	Grp78 is essential for 11-deoxy-16,16-dimethyl PGE2-mediated cytoprotection in renal epithelial cells. American Journal of Physiology - Renal Physiology, 2004, 287, F1113-F1122.	2.7	29
60	Estradiol metabolites as isoform-specific inhibitors of human glutathione S-transferases. Chemico-Biological Interactions, 2004, 151, 21-32.	4.0	17
61	Hepatotoxicity of 3,4-methylenedioxyamphetamine and ?-methyldopamine in isolated rat hepatocytes: formation of glutathione conjugates. Archives of Toxicology, 2004, 78, 16-24.	4.2	82
62	The Role of Metabolism in 3,4-(±)-Methylenedioxyamphetamine and 3,4-(±)-Methylenedioxymethamphetamine (Ecstasy) toxicity. Therapeutic Drug Monitoring, 2004, 26, 132-136.	2.0	111
63	Changes in gene expression during chemical-induced nephrocarcinogenicity in the Eker rat. Molecular Carcinogenesis, 2003, 38, 141-154.	2.7	12
64	An Integrated Approach To Identifying Chemically Induced Posttranslational Modifications Using Comparative MALDI-MS and Targeted HPLC-ESI-MS/MS. Chemical Research in Toxicology, 2003, 16, 598-608.	3.3	42
65	11-Deoxy,16,16-Dimethyl Prostaglandin E2 Induces Specific Proteins in Association with Its Ability to Protect Against Oxidative Stress. Chemical Research in Toxicology, 2003, 16, 312-319.	3.3	11
66	Comparative Identification of Prostanoid Inducible Proteins by LC-ESI-MS/MS and MALDI-TOF Mass Spectrometry. Chemical Research in Toxicology, 2003, 16, 757-767.	3.3	28
67	Reduced constitutive 8-oxoguanine-DNA glycosylase expression and impaired induction following oxidative DNA damage in the tuberin deficient Eker rat. Carcinogenesis, 2003, 24, 573-582.	2.8	32
68	The Metabolism and Toxicity of Quinones, Quinonimines, Quinone Methides, and Quinone-Thioethers. Current Drug Metabolism, 2002, 3, 425-438.	1.2	271
69	Mitogen-Activated Protein Kinases Contribute to Reactive Oxygen Species-Induced Cell Death in Renal Proximal Tubule Epithelial Cells. Chemical Research in Toxicology, 2002, 15, 1635-1642.	3.3	87
70	Cell proliferation is insufficient, but loss of tuberin is necessary, for chemically induced nephrocarcinogenicity. American Journal of Physiology - Renal Physiology, 2002, 283, F262-F270.	2.7	17
71	Role of metabolites in MDMA (ecstasy)-induced nephrotoxicity: an in vitro study using rat and human renal proximal tubular cells. Archives of Toxicology, 2002, 76, 581-588.	4.2	72
72	Differential Regulation of Redox Responsive Transcription Factors by the Nephrocarcinogen 2,3,5-Tris(glutathion-S-yl)hydroquinone. Chemical Research in Toxicology, 2001, 14, 814-821.	3.3	14

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73	Carcinogenicity of a Nephrotoxic Metabolite of the "Nongenotoxic―Carcinogen Hydroquinone. Chemical Research in Toxicology, 2001, 14, 25-33.	3.3	64
74	Serotonergic Neurotoxicity of 3,4-(±)-Methylenedioxyamphetamine and 3,4-(±)-Methylendioxymethamphetamine (Ecstasy) Is Potentiated by Inhibition of γ-Glutamyl Transpeptidase. Chemical Research in Toxicology, 2001, 14, 863-870.	3.3	58
75	Histone H3 Phosphorylation Is Coupled to Poly-(ADP-Ribosylation) during Reactive Oxygen Species-Induced Cell Death in Renal Proximal Tubular Epithelial Cells. Molecular Pharmacology, 2001, 60, 394-402.	2.3	93
76	Transformation of kidney epithelial cells by a quinol thioether via inactivation of the tuberous sclerosis-2 tumor suppressor gene. Molecular Carcinogenesis, 2001, 31, 37-45.	2.7	16
77	Serotonergic Neurotoxicity of Methylenedioxyamphetamine and Methylenedioxymetamphetamine. Advances in Experimental Medicine and Biology, 2001, 500, 397-406.	1.6	6
78	Mutagenicity and Carcinogenicity of Biological Reactive Intermediate's Derived from a "Non-Genotoxic―Carcinogen. Advances in Experimental Medicine and Biology, 2001, , 83-92.	1.6	0
79	Role of Quinones in Toxicology. Chemical Research in Toxicology, 2000, 13, 135-160.	3.3	1,456
80	Stress- and Growth-Related Gene Expression Are Independent of Chemical-Induced Prostaglandin E2 Synthesis in Renal Epithelial Cells. Chemical Research in Toxicology, 2000, 13, 111-117.	3.3	26
81	The Putative Benzene Metabolite 2,3,5-Tris(glutathion-S-yl)hydroquinone Depletes Clutathione, Stimulates Sphingomyelin Turnover, and Induces Apoptosis in HL-60 Cells. Chemical Research in Toxicology, 2000, 13, 550-556.	3.3	33
82	Glutathione and <i>N</i> -Acetylcysteine Conjugates of α-Methyldopamine Produce Serotonergic Neurotoxicity:  Possible Role in Methylenedioxyamphetamine-Mediated Neurotoxicity. Chemical Research in Toxicology, 1999, 12, 1150-1157.	3.3	104
83	Immunochemical Detection of Quinolâ~'Thioether-Derived Protein Adducts. Chemical Research in Toxicology, 1998, 11, 1283-1290.	3.3	39
84	Immunochemical Analysis of Quinolâ^'Thioether-Derived Covalent Protein Adducts in Rodent Species Sensitive and Resistant to Quinolâ^'Thioether-Mediated Nephrotoxicity. Chemical Research in Toxicology, 1998, 11, 1291-1300.	3.3	28
85	THE PHARMACOLOGY AND TOXICOLOGY OF POLYPHENOLIC-GLUTATHIONE CONJUGATES. Annual Review of Pharmacology and Toxicology, 1998, 38, 229-255.	9.4	86
86	The Response of Renal Tubular Epithelial Cells to Physiologically and Chemically Induced Growth Arrest. Journal of Biological Chemistry, 1997, 272, 7511-7518.	3.4	19
87	Identification of Quinol Thioethers in Bone Marrow of Hydroquinone/Phenol-Treated Rats and Mice and Their Potential Role in Benzene-Mediated Hematotoxicity. Chemical Research in Toxicology, 1997, 10, 859-865.	3.3	33
88	2,5-bis-(Glutathion-S-yl)-α-methyldopamine, a putative metabolite of (±)-3,4-methylenedioxyamphetamine, decreases brain serotonin concentrations. European Journal of Pharmacology, 1997, 323, 173-180.	3.5	81
89	Biological Reactivity of Polyphenolicâ^Glutathione Conjugates. Chemical Research in Toxicology, 1997, 10, 1296-1313.	3.3	96
90	DNA Damage,gadd153Expression, and Cytotoxicity in Plateau-Phase Renal Proximal Tubular Epithelial Cells Treated with a Quinol Thioether. Archives of Biochemistry and Biophysics, 1997, 341, 300-308.	3.0	15

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91	PGE <sub>2</sub> -mediated cytoprotection in renal epithelial cells: evidence for a pharmacologically distinct receptor. American Journal of Physiology - Renal Physiology, 1997, 273, F507-F515.	2.7	16
92	Effects of Intracerebroventricular Administration of 5-(Glutathion- <i>S</i> -yl)-α-methyldopamine on Brain Dopamine, Serotonin, and Norepinephrine Concentrations in Male Sprague-Dawley Rats. Chemical Research in Toxicology, 1996, 9, 457-465.	3.3	56
93	Metabolism of tert-Butylhydroquinone to S-Substituted Conjugates in the Male Fischer 344 Rat. Chemical Research in Toxicology, 1996, 9, 133-139.	3.3	31
94	17β-Estradiol Metabolism by Hamster Hepatic Microsomes:  Comparison of Catechol Estrogen O-Methylation with Catechol Estrogen Oxidation and Glutathione Conjugation. Chemical Research in Toxicology, 1996, 9, 793-799.	3.3	50
95	Linking the Metabolism of Hydroquinone to Its Nephrotoxicity and Nephrocarcinogenicity. Advances in Experimental Medicine and Biology, 1996, 387, 267-273.	1.6	18
96	The Kidney as a Target for Biological Reactive Metabolites. Advances in Experimental Medicine and Biology, 1996, 387, 203-212.	1.6	1
97	Modulation of Quinol/Quinone-Thioether Toxicity by Intramolecular Detoxication. Drug Metabolism Reviews, 1995, 27, 93-106.	3.6	11
98	Metabolism of 5-(Glutathion-S-yl)alphamethyldopamine following Intracerebroventricular Administration to Male Sprague-Dawley Rats. Chemical Research in Toxicology, 1995, 8, 634-641.	3.3	50
99	Glutathione Conjugation as a Mechanism for the Transport of Reactive Metabolites. Advances in Pharmacology, 1994, 27, 183-210.	2.0	24
100	Oxidation and Acetylation as Determinants of 2-Bromocystein-S-ylhydroquinone-Mediated Nephrotoxicity. Chemical Research in Toxicology, 1994, 7, 495-502.	3.3	15
101	Identification of multi-S-substituted conjugates of hydroquinone by HPLC-coulometric electrode array analysis and mass spectroscopy. Chemical Research in Toxicology, 1993, 6, 459-469.	3.3	57
102	Toxicology of Quinone-Thioethers. Critical Reviews in Toxicology, 1992, 22, 243-270.	3.9	120
103	The effects of 2,3,5-(triglutathion-S-yl)hydroquinone on renal mitochondrial respiratory function in vitro: Possible role in cytotoxicity. Toxicology and Applied Pharmacology, 1992, 117, 165-171.	2.8	19
104	Quinone chemistry and toxicity. Toxicology and Applied Pharmacology, 1992, 112, 2-16.	2.8	697
105	Nephrotoxicity of 2-bromo-(cystein-s-yl) hydroquinone and 2-bromo-(N-acetyl-l-cystein-S-yl) hydroquinone thioethers. Toxicology and Applied Pharmacology, 1991, 111, 279-298.	2.8	18
106	Inhibition of γ-glutamyl transpeptidase potentiates the nephrotoxicity of glutathione-conjugated chlorohydroquinones. Toxicology and Applied Pharmacology, 1991, 110, 45-60.	2.8	28
107	Glutathione Conjugation as a Mechanism of Targeting Latent Quinones to the Kidney. Advances in Experimental Medicine and Biology, 1991, 283, 457-464.	1.6	4
108	The Role of Î <sup>3</sup> -Glutamyl Transpeptidase in Hydroquinone-Glutathione Conjugate Mediated Nephrotoxicity. Advances in Experimental Medicine and Biology, 1991, 283, 749-751.	1.6	6

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109	The in vivo disposition of 2-bromo-[14C]hydroquinone and the effect of γ-glutamyl transpeptidase inhibition. Toxicology and Applied Pharmacology, 1990, 103, 121-132.	2.8	40
110	Differences in the localization and extent of the renal proximal tubular necrosis caused by mercapturic acid and glutathione conjugates of 1,4-naphthoquinone and menadione. Toxicology and Applied Pharmacology, 1990, 104, 334-350.	2.8	34
111	Glutathione, Î <sup>3</sup> -glutamyl transpeptidase, and the mercapturic acid pathway as modulators of 2-bromohydroquinone oxidation. Toxicology and Applied Pharmacology, 1990, 103, 557-563.	2.8	35
112	Species differences in renal γ-glutamyl transpeptidase activity do not correlate with susceptibility to 2-bromo-(diglutathion-S-yl)-hydroquinone nephrotoxicity. Toxicology, 1990, 64, 291-311.	4.2	21
113	Epidermal ornithine decarboxylase induction and mouse skin tumor promotion by quinones. Carcinogenesis, 1990, 11, 1795-1801.	2.8	27
114	Nephrotoxicity of quinol/quinone-linked S-conjugates. Toxicology Letters, 1990, 53, 59-67.	0.8	8
115	2-Bromohydroquinone-induced toxicity to rabbit renal proximal tubules: The role of biotransformation, glutathione, and covalent binding. Toxicology and Applied Pharmacology, 1989, 99, 19-27.	2.8	25
116	Reactive intermediates and their toxicological significance. Toxicology, 1988, 52, 1-53.	4.2	89
117	Differential uptake of isomeric 2-bromohydroquinone-glutathione conjugates into kidney slices. Biochemical and Biophysical Research Communications, 1988, 152, 223-230.	2.1	24
118	The contribution of bromobenzene to our current understanding of chemically-induced toxicities. Life Sciences, 1988, 42, 1259-1269.	4.3	55
119	The role of ortho-bromophenol in the nephrotoxicity of bromobenzene in rats. Toxicology and Applied Pharmacology, 1984, 72, 539-549.	2.8	46
120	Activation and detoxification of bromobenzene in extrahepatic tissues. Life Sciences, 1984, 35, 561-568.	4.3	16
121	Intra- and extra-cellular formation of metabolites from chemically reactive species. Biochemical Society Transactions, 1984, 12, 4-7.	3.4	19
122	Bromobenzene and p-bromophenol toxicity and covalent binding. Life Sciences, 1982, 30, 841-848.	4.3	57
123	Acetaminophen-induced hepatotoxicity. Life Sciences, 1981, 29, 107-116.	4.3	152
124	Influence of methylxanthine-containing foods on theophylline metabolism and kinetics. Clinical Pharmacology and Therapeutics, 1979, 26, 513-524.	4.7	97