

# Jack Uetrecht

## List of Publications by Year in descending order

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84  
papers

4,024  
citations

101543

36  
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123424

61  
g-index

85  
all docs

85  
docs citations

85  
times ranked

3153  
citing authors

#	ARTICLE	IF	CITATIONS
1	Biochemical mechanisms of drug toxicity. , 2022, , 267-302.		0
2	Clozapine Induces an Acute Proinflammatory Response That Is Attenuated by Inhibition of Inflammasome Signaling: Implications for Idiosyncratic Drug-Induced Agranulocytosis. Toxicological Sciences, 2022, 186, 70-82.	3.1	17
3	Idiosyncratic Drug Reactions: A 35-Year <i>Chemical Research in Toxicology</i> Perspective. Chemical Research in Toxicology, 2022, 35, 1649-1654.	3.3	6
4	Investigating the Mechanism of Trimethoprim-Induced Skin Rash and Liver Injury. Toxicological Sciences, 2021, 180, 17-25.	3.1	5
5	Liver Injury Caused by Green Tea Extract in PD-1 <sup>−/−</sup> Mice: An Impaired Immune Tolerance Model for Idiosyncratic Drug-Induced Liver Injury. Chemical Research in Toxicology, 2021, 34, 849-856.	3.3	12
6	Testing Possible Risk Factors for Idiosyncratic Drug-Induced Liver Injury Using an Amodiaquine Mouse Model and Co-treatment with 1-Methyl- <sup>d</sup> -Tryptophan or Acetaminophen. ACS Omega, 2021, 6, 4656-4662.	3.5	5
7	Idiosyncratic Drug-Induced Liver Injury: Mechanistic and Clinical Challenges. International Journal of Molecular Sciences, 2021, 22, 2954.	4.1	48
8	Severe cutaneous adverse reaction associated with antiseizure medications: Diagnosis, management, and prevention. Epilepsy and Behavior, 2021, 117, 107844.	1.7	4
9	Mechanism of idiosyncratic drug induced liver injury (DILI): unresolved basic issues. Annals of Translational Medicine, 2021, 9, 730-730.	1.7	27
10	Response to the Letter to the Editor Concerning the Article “Rotenone Increases Isoniazid Toxicity but Does Not Cause Liver Injury: Implications for the Hypothesis That Inhibition of the Mitochondrial Electron Transport Chain Is a Common Mechanism of Idiosyncratic Drug-Induced Liver Injury” by Bernard Fromenty. Chemical Research in Toxicology, 2020, 33, 5-6.	3.3	1
11	Reactive metabolite of gefitinib activates inflammasomes: implications for gefitinib-induced idiosyncratic reaction. Journal of Toxicological Sciences, 2020, 45, 673-680.	1.5	18
12	Mechanistic Studies of Idiosyncratic DILI: Clinical Implications. Frontiers in Pharmacology, 2019, 10, 837.	3.5	61
13	Rotenone Increases Isoniazid Toxicity but Does Not Cause Significant Liver Injury: Implications for the Hypothesis that Inhibition of the Mitochondrial Electron Transport Chain Is a Common Mechanism of Idiosyncratic Drug-Induced Liver Injury. Chemical Research in Toxicology, 2019, 32, 1423-1431.	3.3	10
14	Involvement of CCL2/CCR2 macrophage recruitment in amodiaquine-induced liver injury. Journal of Immunotoxicology, 2019, 16, 28-33.	1.7	12
15	Mechanisms of idiosyncratic drug-induced liver injury. Advances in Pharmacology, 2019, 85, 133-163.	2.0	26
16	The 2-Hydroxyiminostilbene Metabolite of Carbamazepine or the Supernatant from Incubation of Hepatocytes with Carbamazepine Activates Inflammasomes: Implications for Carbamazepine-Induced Hypersensitivity Reactions. Drug Metabolism and Disposition, 2019, 47, 1093-1096.	3.3	15
17	The skin as a metabolic and immune-competent organ: Implications for drug-induced skin rash. Journal of Immunotoxicology, 2019, 16, 1-12.	1.7	20
18	Editor’s Highlight: An Impaired Immune Tolerance Animal Model Distinguishes the Potential of Troglitazone/Pioglitazone and Tolcapone/Entacapone to Cause IDILI. Toxicological Sciences, 2018, 161, 412-420.	3.1	32

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19	Nevirapine-induced liver lipid-SER inclusions and other ultrastructural aberrations. <i>Ultrastructural Pathology</i> , 2018, 42, 108-115.	0.9	5
20	Dose-dependent acute liver injury with hypersensitivity features in humans due to a novel microsomal prostaglandin E synthase 1 inhibitor. <i>British Journal of Clinical Pharmacology</i> , 2018, 84, 179-188.	2.4	19
21	The Effects of Immune Modulators on Amodiaquine-Induced Liver Injury. <i>Chemical Research in Toxicology</i> , 2018, 31, 739-744.	3.3	2
22	Do In Vitro Assays Predict Drug Candidate Idiosyncratic Drug-Induced Liver Injury Risk?. <i>Drug Metabolism and Disposition</i> , 2018, 46, 1658-1669.	3.3	23
23	Use of an animal model to test whether non-alcoholic fatty liver disease increases the risk of idiosyncratic drug-induced liver injury. <i>Journal of Immunotoxicology</i> , 2018, 15, 90-95.	1.7	7
24	Supernatant from Hepatocyte Cultures with Drugs That Cause Idiosyncratic Liver Injury Activates Macrophage Inflammasomes. <i>Chemical Research in Toxicology</i> , 2017, 30, 1327-1332.	3.3	48
25	Effects of immunization and checkpoint inhibition on amodiaquine-induced liver injury. <i>Journal of Immunotoxicology</i> , 2017, 14, 89-94.	1.7	10
26	How Reactive Metabolites Induce an Immune Response That Sometimes Leads to an Idiosyncratic Drug Reaction. <i>Chemical Research in Toxicology</i> , 2017, 30, 295-314.	3.3	109
27	Immune mechanisms of idiosyncratic drug-induced liver injury. <i>Journal of Clinical and Translational Research</i> , 2017, 3, 145-156.	0.3	16
28	Protein Targets of Isoniazid-Reactive Metabolites in Mouse Liver <i>in Vivo</i> . <i>Chemical Research in Toxicology</i> , 2016, 29, 1064-1072.	3.3	11
29	Mechanism of isoniazid-induced hepatotoxicity: then and now. <i>British Journal of Clinical Pharmacology</i> , 2016, 81, 1030-1036.	2.4	140
30	Deferiprone-induced agranulocytosis: 20 years of clinical observations. <i>American Journal of Hematology</i> , 2016, 91, 1026-1031.	4.1	43
31	Exploring an animal model of amodiaquine-induced liver injury in rats and mice. <i>Journal of Immunotoxicology</i> , 2016, 13, 694-712.	1.7	7
32	Treatment of PD-1 <sup>-/-</sup> mice with amodiaquine and anti-CTLA4 leads to liver injury similar to idiosyncratic liver injury in patients. <i>Hepatology</i> , 2015, 61, 1332-1342.	7.3	123
33	Inhibition of immune tolerance unmasks drug-induced allergic hepatitis. <i>Hepatology</i> , 2015, 62, 346-348.	7.3	16
34	Immunization with amodiaquine-modified hepatic proteins prevents amodiaquine-induced liver injury. <i>Journal of Immunotoxicology</i> , 2015, 12, 361-367.	1.7	13
35	Hepatic effects of aminoglutethimide: A model aromatic amine. <i>Journal of Immunotoxicology</i> , 2015, 12, 24-32.	1.7	8
36	Development of a novel mouse model of amodiaquine-induced liver injury with a delayed onset. <i>Journal of Immunotoxicology</i> , 2015, 12, 247-260.	1.7	36

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37	The Role of CD8 T Cells in Amodiaquine-Induced Liver Injury in PD1 <sup>-/-</sup> Mice Cotreated with Anti-CTLA-4. <i>Chemical Research in Toxicology</i> , 2015, 28, 1567-1573.	3.3	47
38	The Combination of Anti-CTLA-4 and PD1 <sup>-/-</sup> Mice Unmasks the Potential of Isoniazid and Nevirapine To Cause Liver Injury. <i>Chemical Research in Toxicology</i> , 2015, 28, 2287-2291.	3.3	64
39	Current understanding of the mechanisms of idiosyncratic drug-induced agranulocytosis. <i>Expert Opinion on Drug Metabolism and Toxicology</i> , 2015, 11, 243-257.	3.3	69
40	Isoniazid-induced liver injury and immune response in mice. <i>Journal of Immunotoxicology</i> , 2014, 11, 383-392.	1.7	27
41	Bioactivation of drugs in the skin: relationship to cutaneous adverse drug reactions. <i>Drug Metabolism Reviews</i> , 2014, 46, 1-18.	3.6	29
42	Paradoxical Attenuation of Autoimmune Hepatitis by Oral Isoniazid in Wild-Type and N-Acetyltransferase <sup>-/-</sup> Deficient Mice. <i>Drug Metabolism and Disposition</i> , 2014, 42, 963-973.	3.3	13
43	IgG <sub>3</sub> Is the Dominant Subtype of Anti-isoniazid Antibodies in Patients with Isoniazid-Induced Liver Failure. <i>Chemical Research in Toxicology</i> , 2014, 27, 738-740.	3.3	8
44	Clozapine Promotes the Proliferation of Granulocyte Progenitors in the Bone Marrow Leading to Increased Granulopoiesis and Neutrophilia in Rats. <i>Chemical Research in Toxicology</i> , 2014, 27, 1109-1119.	3.3	15
45	Involvement of Myeloperoxidase and NADPH Oxidase in the Covalent Binding of Amodiaquine and Clozapine to Neutrophils: Implications for Drug-Induced Agranulocytosis. <i>Chemical Research in Toxicology</i> , 2014, 27, 699-709.	3.3	34
46	Activation of Inflammasomes by Agents Causing Idiosyncratic Skin Reactions: A Possible Biomarker. <i>Chemical Research in Toxicology</i> , 2014, 27, 949-951.	3.3	27
47	Lack of liver injury in Wistar rats treated with the combination of isoniazid and rifampicin. <i>Molecular and Cellular Biochemistry</i> , 2014, 387, 9-17.	3.1	13
48	D-penicillamine-induced granulomatous hepatitis in brown Norway rats. <i>Molecular and Cellular Biochemistry</i> , 2014, 393, 229-235.	3.1	6
49	Effect of Clozapine and Olanzapine on Neutrophil Kinetics: Implications for Drug-Induced Agranulocytosis. <i>Chemical Research in Toxicology</i> , 2014, 27, 1104-1108.	3.3	46
50	Mild Isoniazid-Induced Liver Injury in Humans Is Associated with an Increase in Th17 Cells and T Cells Producing IL-10. <i>Chemical Research in Toxicology</i> , 2014, 27, 683-689.	3.3	33
51	Detection of anti-isoniazid and anti-cytochrome P450 antibodies in patients with isoniazid-induced liver failure. <i>Hepatology</i> , 2014, 59, 1084-1093.	7.3	107
52	Identification of Danger Signals in Nevirapine-Induced Skin Rash. <i>Chemical Research in Toxicology</i> , 2013, 26, 1378-1383.	3.3	19
53	A novel T <sub>H</sub> 17-type cell is rapidly increased in the liver in response to acetaminophen-induced liver injury: T <sub>H</sub> 17 cells and the innate immune response. <i>Journal of Immunotoxicology</i> , 2013, 10, 287-291.	1.7	19
54	Role of the Adaptive Immune System in Idiosyncratic Drug-Induced Liver Injury. , 2013, , 175-193.		5

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55	Effect of Aminoglutethimide on Neutrophils in Rats: Implications for Idiosyncratic Drug-Induced Blood Dyscrasias. <i>Chemical Research in Toxicology</i> , 2013, 26, 1272-1281.	3.3	4
56	Nevirapine Bioactivation and Covalent Binding in the Skin. <i>Chemical Research in Toxicology</i> , 2013, 26, 410-421.	3.3	36
57	Changes in gene expression induced by aromatic amine drugs: Testing the danger hypothesis. <i>Journal of Immunotoxicology</i> , 2013, 10, 178-191.	1.7	8
58	Idiosyncratic Adverse Drug Reactions: Current Concepts. <i>Pharmacological Reviews</i> , 2013, 65, 779-808.	16.0	253
59	Direct Oxidation and Covalent Binding of Isoniazid to Rodent Liver and Human Hepatic Microsomes: Humans Are More Like Mice than Rats. <i>Chemical Research in Toxicology</i> , 2012, 25, 2567-2576.	3.3	89
60	Bioactivation of Nevirapine to a Reactive Quinone Methide: Implications for Liver Injury. <i>Chemical Research in Toxicology</i> , 2012, 25, 1708-1719.	3.3	52
61	Animal Models of Idiosyncratic Drug Reactions. <i>Advances in Pharmacology</i> , 2012, 63, 81-135.	2.0	52
62	Involvement of the Immune System in Idiosyncratic Drug Reactions. <i>Drug Metabolism and Pharmacokinetics</i> , 2011, 26, 47-59.	2.2	41
63	A Fresh Look at the Mechanism of Isoniazid-Induced Hepatotoxicity. <i>Clinical Pharmacology and Therapeutics</i> , 2011, 89, 911-914.	4.7	148
64	Cytokine and autoantibody patterns in acute liver failure. <i>Journal of Immunotoxicology</i> , 2010, 7, 157-164.	1.7	44
65	Mechanisms of Immune-Mediated Liver Injury. <i>Toxicological Sciences</i> , 2010, 115, 307-321.	3.1	254
66	Bioactivation of Minocycline to Reactive Intermediates by Myeloperoxidase, Horseradish Peroxidase, and Hepatic Microsomes: Implications for Minocycline-Induced Lupus and Hepatitis. <i>Drug Metabolism and Disposition</i> , 2009, 37, 1806-1818.	3.3	21
67	Immunoallergic Drug-Induced Liver Injury in Humans. <i>Seminars in Liver Disease</i> , 2009, 29, 383-392.	3.6	79
68	Immune-Mediated Adverse Drug Reactions. <i>Chemical Research in Toxicology</i> , 2009, 22, 24-34.	3.3	92
69	Idiosyncratic Drug Reactions: Past, Present, and Future. <i>Chemical Research in Toxicology</i> , 2008, 21, 84-92.	3.3	244
70	Demonstration of the Metabolic Pathway Responsible for Nevirapine-Induced Skin Rash. <i>Chemical Research in Toxicology</i> , 2008, 21, 1862-1870.	3.3	81
71	Idiosyncratic Drug Reactions: Current Understanding. <i>Annual Review of Pharmacology and Toxicology</i> , 2007, 47, 513-539.	9.4	276
72	Evaluation of Which Reactive Metabolite, If Any, Is Responsible for a Specific Idiosyncratic Reaction. <i>Drug Metabolism Reviews</i> , 2006, 38, 745-753.	3.6	48

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73	Current trends in drug-induced autoimmunity. <i>Autoimmunity Reviews</i> , 2005, 4, 309-314.	5.8	65
74	Role of drug metabolism for breaking tolerance and the localization of drug hypersensitivity. <i>Toxicology</i> , 2005, 209, 113-118.	4.2	26
75	Role of animal models in the study of drug-induced hypersensitivity reactions. <i>AAPS Journal</i> , 2005, 7, E914-E921.	4.4	50
76	d-Penicillamine-Induced Autoimmunity in the Brown Norway Rat: A Role for Both T and Non-T Splenocytes in Adoptive Transfer of Tolerance. <i>Chemical Research in Toxicology</i> , 2004, 17, 1299-1302.	3.3	13
77	Screening for the potential of a drug candidate to cause idiosyncratic drug reactions. <i>Drug Discovery Today</i> , 2003, 8, 832-837.	6.4	127
78	Bioactivation of Clozapine by Murine Cardiac Tissue in Vivo and in Vitro. <i>Chemical Research in Toxicology</i> , 2003, 16, 1359-1364.	3.3	46
79	The danger hypothesis applied to idiosyncratic drug reactions. <i>Current Opinion in Allergy and Clinical Immunology</i> , 2003, 3, 235-242.	2.3	73
80	N-OXIDATION OF DRUGS ASSOCIATED WITH IDIOSYNCRATIC DRUG REACTIONS. <i>Drug Metabolism Reviews</i> , 2002, 34, 651-665.	3.6	65
81	Hypochlorous Acid, a Major Oxidant Produced by Activated Neutrophils, Has Low Effect on Two Pyridobenzazepine Derivatives, JL 3 and JL 13. <i>Archiv Der Pharmazie</i> , 2000, 333, 63-67.	4.1	5
82	Structural features associated with reactive metabolite formation in clozapine analogues. <i>Chemico-Biological Interactions</i> , 1997, 104, 117-129.	4.0	58
83	Antibodies to myeloperoxidase in propylthiouracil-induced autoimmune disease in the cat. <i>Toxicology</i> , 1996, 114, 155-162.	4.2	47
84	Drug Metabolism by Leukocytes and Its Role in Drug-Induced Lupus and Other Idiosyncratic Drug Reactions. <i>Critical Reviews in Toxicology</i> , 1990, 20, 213-235.	3.9	91