

# Anne M Filppula

## List of Publications by Year in descending order

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Version: 2024-02-01

20  
papers

980  
citations

567281  
15  
h-index

752698  
20  
g-index

20  
all docs

20  
docs citations

20  
times ranked

1293  
citing authors

#	ARTICLE	IF	CITATIONS
1	Translational aspects of cytochrome P450-mediated drug-drug interactions: A case study with clopidogrel. <i>Basic and Clinical Pharmacology and Toxicology</i> , 2022, 130, 48-59.	2.5	3
2	Comparative Hepatic and Intestinal Metabolism and Pharmacodynamics of Statins. <i>Drug Metabolism and Disposition</i> , 2021, 49, 658-667.	3.3	19
3	Comparative Hepatic and Intestinal Efflux Transport of Statins. <i>Drug Metabolism and Disposition</i> , 2021, 49, 750-759.	3.3	31
4	An automated cocktail method for in vitro assessment of direct and time-dependent inhibition of nine major cytochrome P450 enzymes – application to establishing CYP2C8 inhibitor selectivity. <i>European Journal of Pharmaceutical Sciences</i> , 2021, 162, 105810.	4.0	7
5	Clinical Studies on Drug-Drug Interactions Involving Metabolism and Transport: Methodology, Pitfalls, and Interpretation. <i>Clinical Pharmacology and Therapeutics</i> , 2019, 105, 1345-1361.	4.7	107
6	Improved predictions of time-dependent drug-drug interactions by determination of cytosolic drug concentrations. <i>Scientific Reports</i> , 2019, 9, 5850.	3.3	15
7	Critical Differences between Enzyme Sources in Sensitivity to Detect Time-Dependent Inactivation of CYP2C8. <i>Drug Metabolism and Disposition</i> , 2019, 47, 436-443.	3.3	7
8	Clopidogrel but Not Prasugrel Significantly Inhibits the CYP2C8-Mediated Metabolism of Montelukast in Humans. <i>Clinical Pharmacology and Therapeutics</i> , 2018, 104, 495-504.	4.7	14
9	Clopidogrel Carboxylic Acid Glucuronidation is Mediated Mainly by UGT2B7, UGT2B4, and UGT2B17: Implications for Pharmacogenetics and Drug-Drug Interactions. <i>Drug Metabolism and Disposition</i> , 2018, 46, 141-150.	3.3	22
10	In Vitro Screening of Six Protein Kinase Inhibitors for Time-Dependent Inhibition of CYP2C8 and CYP3A4: Possible Implications with regard to Drug-Drug Interactions. <i>Basic and Clinical Pharmacology and Toxicology</i> , 2018, 123, 739-748.	2.5	14
11	Validation and development of MTH1 inhibitors for treatment of cancer. <i>Annals of Oncology</i> , 2016, 27, 2275-2283.	1.2	111
12	Neurotoxicity and low paclitaxel clearance associated with concomitant clopidogrel therapy in a 60-year-old Caucasian woman with ovarian carcinoma. <i>British Journal of Clinical Pharmacology</i> , 2016, 81, 313-315.	2.4	20
13	Role of Cytochrome P450 2C8 in Drug Metabolism and Interactions. <i>Pharmacological Reviews</i> , 2016, 68, 168-241.	16.0	175
14	Glucuronidation Converts Clopidogrel to a Strong Time-Dependent Inhibitor of CYP2C8: A Phase II Metabolite as a Perpetrator of Drug-Drug Interactions. <i>Clinical Pharmacology and Therapeutics</i> , 2014, 96, 498-507.	4.7	124
15	In Vitro Assessment of Time-Dependent Inhibitory Effects on CYP2C8 and CYP3A Activity by Fourteen Protein Kinase Inhibitors. <i>Drug Metabolism and Disposition</i> , 2014, 42, 1202-1209.	3.3	56
16	Autoinhibition of CYP3A4 Leads to Important Role of CYP2C8 in Imatinib Metabolism: Variability in CYP2C8 Activity May Alter Plasma Concentrations and Response. <i>Drug Metabolism and Disposition</i> , 2013, 41, 50-59.	3.3	57
17	Gemfibrozil Impairs Imatinib Absorption and Inhibits the CYP2C8-Mediated Formation of Its Main Metabolite. <i>Clinical Pharmacology and Therapeutics</i> , 2013, 94, 383-393.	4.7	28
18	Potent mechanism-based inhibition of CYP3A4 by imatinib explains its liability to interact with CYP3A4 substrates. <i>British Journal of Pharmacology</i> , 2012, 165, 2787-2798.	5.4	74

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19	Reevaluation of the Microsomal Metabolism of Montelukast: Major Contribution by CYP2C8 at Clinically Relevant Concentrations. Drug Metabolism and Disposition, 2011, 39, 904-911.	3.3	42
20	Gemfibrozil Markedly Increases the Plasma Concentrations of Montelukast: A Previously Unrecognized Role for CYP2C8 in the Metabolism of Montelukast. Clinical Pharmacology and Therapeutics, 2010, 88, 223-230.	4.7	54