

Florian L Muller

List of Publications by Year in descending order

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Version: 2024-02-01

70
papers

12,120
citations

76326

40
h-index

106344

65
g-index

89
all docs

89
docs citations

89
times ranked

21036
citing authors

#	ARTICLE	IF	CITATIONS
1	Quantification of Phosphonate Drugs by ¹ H- ³¹ P HSQC Shows That Rats Are Better Models of Primate Drug Exposure than Mice. <i>Analytical Chemistry</i> , 2022, 94, 10045-10053.	6.5	5
2	Remdesivir for COVID-19: Why Not Dose Higher?. <i>Antimicrobial Agents and Chemotherapy</i> , 2021, 65, .	3.2	6
3	Impaired anaplerosis is a major contributor to glycolysis inhibitor toxicity in glioma. <i>Cancer & Metabolism</i> , 2021, 9, 27.	5.0	11
4	Homozygous MTAP deletion in primary human glioblastoma is not associated with elevation of methylthioadenosine. <i>Nature Communications</i> , 2021, 12, 4228.	12.8	21
5	Structure-guided microbial targeting of antistaphylococcal prodrugs. <i>ELife</i> , 2021, 10, .	6.0	7
6	Why Remdesivir Failed: Preclinical Assumptions Overestimate the Clinical Efficacy of Remdesivir for COVID-19 and Ebola. <i>Antimicrobial Agents and Chemotherapy</i> , 2021, 65, e0111721.	3.2	22
7	Targeting Host Glycolysis as a Strategy for Antimalarial Development. <i>Frontiers in Cellular and Infection Microbiology</i> , 2021, 11, 730413.	3.9	6
8	Single-Cell RNA Sequencing Supports Preferential Bioactivation of Remdesivir in the Liver. <i>Antimicrobial Agents and Chemotherapy</i> , 2021, 65, e0133321.	3.2	1
9	NEAT1 is essential for metabolic changes that promote breast cancer growth and metastasis. <i>Cell Metabolism</i> , 2021, 33, 2380-2397.e9.	16.2	73
10	Enhancer Reprogramming Confers Dependence on Glycolysis and IGF Signaling in KMT2D Mutant Melanoma. <i>Cell Reports</i> , 2020, 33, 108293.	6.4	39
11	Captisol and GS-704277, but Not GS-441524, Are Credible Mediators of Remdesivir's Nephrotoxicity. <i>Antimicrobial Agents and Chemotherapy</i> , 2020, 64, .	3.2	10
12	Aliphatic amines are viable pro-drug moieties in phosphonoamidate drugs. <i>Bioorganic and Medicinal Chemistry Letters</i> , 2020, 30, 127656.	2.2	3
13	Antimicrobial Prodrug Activation by the Staphylococcal Glyoxalase GloB. <i>ACS Infectious Diseases</i> , 2020, 6, 3064-3075.	3.8	9
14	An enolase inhibitor for the targeted treatment of ENO1-deleted cancers. <i>Nature Metabolism</i> , 2020, 2, 1413-1426.	11.9	49
15	Superoxide-mediated oxidative stress accelerates skeletal muscle atrophy by synchronous activation of proteolytic systems. <i>GeroScience</i> , 2020, 42, 1579-1591.	4.6	24
16	Advantages of the Parent Nucleoside GS-441524 over Remdesivir for Covid-19 Treatment. <i>ACS Medicinal Chemistry Letters</i> , 2020, 11, 1361-1366.	2.8	137
17	Why Great Mitotic Inhibitors Make Poor Cancer Drugs. <i>Trends in Cancer</i> , 2020, 6, 924-941.	7.4	33
18	Bioreducible Phosphonoamidate Pro-drug Inhibitor of Enolase: Proof of Concept Study. <i>ACS Medicinal Chemistry Letters</i> , 2020, 11, 1484-1489.	2.8	2

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19	Robust detection of oncometabolic aberrations by ^1H - ^{13}C heteronuclear single quantum correlation in intact biological specimens. <i>Communications Biology</i> , 2020, 3, 328.	4.4	3
20	KMT2D Deficiency Impairs Super-Enhancers to Confer a Glycolytic Vulnerability in Lung Cancer. <i>Cancer Cell</i> , 2020, 37, 599-617.e7.	16.8	137
21	The 3S Enantiomer Drives Enolase Inhibitory Activity in SF2312 and Its Analogues. <i>Molecules</i> , 2019, 24, 2510.	3.8	10
22	Mechanism-Specific Pharmacodynamics of a Novel Complex-I Inhibitor Quantified by Imaging Reversal of Consumptive Hypoxia with [^{18}F]FAZA PET In Vivo. <i>Cells</i> , 2019, 8, 1487.	4.1	20
23	Functional Genomics Reveals Synthetic Lethality between Phosphogluconate Dehydrogenase and Oxidative Phosphorylation. <i>Cell Reports</i> , 2019, 26, 469-482.e5.	6.4	47
24	<scp>FOXO</scp> protects against ageâ€progressive axonal degeneration. <i>Aging Cell</i> , 2018, 17, e12701.	6.7	52
25	Mutations in the SWI/SNF complex induce a targetable dependence on oxidative phosphorylation in lung cancer. <i>Nature Medicine</i> , 2018, 24, 1047-1057.	30.7	175
26	An inhibitor of oxidative phosphorylation exploits cancer vulnerability. <i>Nature Medicine</i> , 2018, 24, 1036-1046.	30.7	622
27	Genomic deletion of malic enzyme 2 confers collateral lethality in pancreatic cancer. <i>Nature</i> , 2017, 542, 119-123.	27.8	209
28	Synthetic vulnerabilities of mesenchymal subpopulations in pancreatic cancer. <i>Nature</i> , 2017, 542, 362-366.	27.8	105
29	Tumor Evolution of Glioma-Intrinsic Gene Expression Subtypes Associates with Immunological Changes in the Microenvironment. <i>Cancer Cell</i> , 2017, 32, 42-56.e6.	16.8	1,282
30	ENOblock Does Not Inhibit the Activity of the Glycolytic Enzyme Enolase. <i>PLoS ONE</i> , 2016, 11, e0168739.	2.5	34
31	SF2312 is a natural phosphonate inhibitor of enolase. <i>Nature Chemical Biology</i> , 2016, 12, 1053-1058.	8.0	90
32	Collateral Lethality: A New Therapeutic Strategy in Oncology. <i>Trends in Cancer</i> , 2015, 1, 161-173.	7.4	106
33	Whole-genome and multisector exome sequencing of primary and post-treatment glioblastoma reveals patterns of tumor evolution. <i>Genome Research</i> , 2015, 25, 316-327.	5.5	343
34	The â€mitoflashâ€™ probe cpYFP does not respond to superoxide. <i>Nature</i> , 2014, 514, E12-E14.	27.8	109
35	Oncogene ablation-resistant pancreatic cancer cells depend on mitochondrial function. <i>Nature</i> , 2014, 514, 628-632.	27.8	998
36	Oncogenic NRAS signaling differentially regulates survival and proliferation in melanoma. <i>Nature Medicine</i> , 2012, 18, 1503-1510.	30.7	333

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37	Dietary restriction attenuates age-associated muscle atrophy by lowering oxidative stress in mice even in complete absence of CuZnSOD. <i>Aging Cell</i> , 2012, 11, 770-782.	6.7	82
38	Dietary restriction but not rapamycin extends disease onset and survival of the H46R/H48Q mouse model of ALS. <i>Neurobiology of Aging</i> , 2012, 33, 1829-1832.	3.1	48
39	Passenger deletions generate therapeutic vulnerabilities in cancer. <i>Nature</i> , 2012, 488, 337-342.	27.8	294
40	Complex I generated, mitochondrial matrix-directed superoxide is released from the mitochondria through voltage dependent anion channels. <i>Biochemical and Biophysical Research Communications</i> , 2012, 422, 515-521.	2.1	75
41	Mitochondrial "flashes": a radical concept rephined. <i>Trends in Cell Biology</i> , 2012, 22, 503-508.	7.9	74
42	MnSOD deficiency results in elevated oxidative stress and decreased mitochondrial function but does not lead to muscle atrophy during aging. <i>Aging Cell</i> , 2011, 10, 493-505.	6.7	76
43	Telomerase reactivation reverses tissue degeneration in aged telomerase-deficient mice. <i>Nature</i> , 2011, 469, 102-106.	27.8	674
44	Telomere dysfunction induces metabolic and mitochondrial compromise. <i>Nature</i> , 2011, 470, 359-365.	27.8	1,093
45	An Objective Appraisal of the Free Radical Theory of Aging. , 2011, , 177-202.		7
46	Increased superoxide <i>in vivo</i> accelerates age-associated muscle atrophy through mitochondrial dysfunction and neuromuscular junction degeneration. <i>FASEB Journal</i> , 2010, 24, 1376-1390.	0.5	250
47	Denervation Induces Cytosolic Phospholipase A2-mediated Fatty Acid Hydroperoxide Generation by Muscle Mitochondria. <i>Journal of Biological Chemistry</i> , 2009, 284, 46-55.	3.4	82
48	A critical evaluation of cpYFP as a probe for superoxide. <i>Free Radical Biology and Medicine</i> , 2009, 47, 1779-1780.	2.9	46
49	Conditional knockout of Mn-SOD targeted to type IIB skeletal muscle fibers increases oxidative stress and is sufficient to alter aerobic exercise capacity. <i>American Journal of Physiology - Cell Physiology</i> , 2009, 297, C1520-C1532.	4.6	67
50	FoxOs Cooperatively Regulate Diverse Pathways Governing Neural Stem Cell Homeostasis. <i>Cell Stem Cell</i> , 2009, 5, 540-553.	11.1	418
51	MnSOD deficiency has a differential effect on disease progression in two different ALS mutant mouse models. <i>Muscle and Nerve</i> , 2008, 38, 1173-1183.	2.2	27
52	The Basics of Oxidative Biochemistry. , 2008, , 11-35.		9
53	GAPDH Is Conformationally and Functionally Altered in Association with Oxidative Stress in Mouse Models of Amyotrophic Lateral Sclerosis. <i>Journal of Molecular Biology</i> , 2008, 382, 1195-1210.	4.2	70
54	Deleterious action of FA metabolites on ATP synthesis: possible link between lipotoxicity, mitochondrial dysfunction, and insulin resistance. <i>American Journal of Physiology - Endocrinology and Metabolism</i> , 2008, 295, E678-E685.	3.5	117

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55	The in vivo gene expression signature of oxidative stress. <i>Physiological Genomics</i> , 2008, 34, 112-126.	2.3	204
56	High rates of superoxide production in skeletal-muscle mitochondria respiring on both complex I- and complex II-linked substrates. <i>Biochemical Journal</i> , 2008, 409, 491-499.	3.7	138
57	Does Oxidative Stress Limit Mouse Life Span?. , 2008, , 129-146.		0
58	Oxidative stress induced mitochondrial dysfunction leads to age related loss of muscle mass via myonuclear apoptosis and proteolysis. <i>FASEB Journal</i> , 2008, 22, 758.33.	0.5	0
59	Denervation-induced skeletal muscle atrophy is associated with increased mitochondrial ROS production. <i>American Journal of Physiology - Regulatory Integrative and Comparative Physiology</i> , 2007, 293, R1159-R1168.	1.8	285
60	Trends in oxidative aging theories. <i>Free Radical Biology and Medicine</i> , 2007, 43, 477-503.	2.9	897
61	Absence of CuZn superoxide dismutase leads to elevated oxidative stress and acceleration of age-dependent skeletal muscle atrophy. <i>Free Radical Biology and Medicine</i> , 2006, 40, 1993-2004.	2.9	378
62	Alterations in mitochondrial function, hydrogen peroxide release and oxidative damage in mouse hind-limb skeletal muscle during aging. <i>Mechanisms of Ageing and Development</i> , 2006, 127, 298-306.	4.6	203
63	Acceleration of age-related skeletal muscle atrophy and oxidative stress in the mice lacking of CuZnSOD. <i>FASEB Journal</i> , 2006, 20, A818.	0.5	0
64	Absence of CuZn superoxide dismutase (SOD1) leads to increased proteolysis of skeletal muscle. <i>FASEB Journal</i> , 2006, 20, LB32.	0.5	0
65	H2S-Induced Ectothermy: Relevance to Aging. <i>Rejuvenation Research</i> , 2005, 8, 135-137.	1.8	2
66	Complex III Releases Superoxide to Both Sides of the Inner Mitochondrial Membrane. <i>Journal of Biological Chemistry</i> , 2004, 279, 49064-49073.	3.4	859
67	Q-Cycle Bypass Reactions at the Qo Site of the Cytochrome bc1 (and Related) Complexes. <i>Methods in Enzymology</i> , 2004, 382, 21-45.	1.0	43
68	Architecture of the QoSite of the Cytochromebc1Complex Probed by Superoxide Production. <i>Biochemistry</i> , 2003, 42, 6493-6499.	2.5	132
69	Multiple Q-Cycle Bypass Reactions at the QoSite of the Cytochromebc1Complex. <i>Biochemistry</i> , 2002, 41, 7866-7874.	2.5	160
70	The nature and mechanism of superoxide production by the electron transport chain: Its relevance to aging. <i>Age</i> , 2000, 23, 227-253.	3.0	110