Raffaella Gozzelino

List of Publications by Year
in descending order

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28 papers	3,539 citations	331670 21 h-index	526287 27 g-index
29 all docs	29 docs citations	29 times ranked	5893 citing authors

#	Article	IF	Citations
1	Cell Death-Osis of Dopaminergic Neurons and the Role of Iron in Parkinson's Disease. Antioxidants and Redox Signaling, 2021, 35, 453-473.	5.4	5
2	TNFÎ± Controls the Delicate Balance between Erythropoiesis and Stem Cell Exhaustion during Inflammatory Stress. Blood, 2021, 138, 2184-2184.	1.4	0
3	Multilevel Impacts of Iron in the Brain: The Cross Talk between Neurophysiological Mechanisms, Cognition, and Social Behavior. Pharmaceuticals, 2019, 12, 126.	3.8	65
4	Renal control of disease tolerance to malaria. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 5681-5686.	7.1	58
5	Iron as Therapeutic Target in Human Diseases. Pharmaceuticals, 2019, 12, 178.	3.8	3
6	Iron Metabolism and the Inflammatory Response. IUBMB Life, 2017, 69, 442-450.	3.4	33
7	An Iron-Rich Diet Decreases the Mycobacterial Burden and Correlates With Hepcidin Upregulation, Lower Levels of Proinflammatory Mediators, and Increased T-Cell Recruitment in a Model of Mycobacterium bovis Bacille Calmette-Guerin Infection. Journal of Infectious Diseases, 2017, 216, 907-918.	4.0	18
8	Iron Homeostasis in Health and Disease. International Journal of Molecular Sciences, 2016, 17, 130.	4.1	274
9	The Pathophysiology of Heme in the Brain. Current Alzheimer Research, 2016, 13, 174-184.	1.4	58
10	The importance of eukaryotic ferritins in iron handling and cytoprotection. Biochemical Journal, 2015, 472, 1-15.	3.7	79
11	The importance of iron in pathophysiologic conditions. Frontiers in Pharmacology, 2015, 6, 26.	3.5	24
12	Iron overload in Plasmodium berghei-infected placenta as a pathogenesis mechanism of fetal death. Frontiers in Pharmacology, 2014, 5, 155.	3.5	14
13	Gut Microbiota Elicits a Protective Immune Response against Malaria Transmission. Cell, 2014, 159, 1277-1289.	28.9	279
14	Coupling Heme and Iron MetabolismviaFerritin H Chain. Antioxidants and Redox Signaling, 2014, 20, 1754-1769.	5.4	126
15	Tissue damage control in disease tolerance. Trends in Immunology, 2014, 35, 483-494.	6.8	147
16	Anthracyclines Induce DNA Damage Response-Mediated Protection against Severe Sepsis. Immunity, 2013, 39, 874-884.	14.3	131
17	NF-ÎºB activation fails to protect cells to TNFÎ±-induced apoptosis in the absence of Bcl-xL, but not Mcl-1, Bcl-2 or Bcl-w. Biochimica Et Biophysica Acta - Molecular Cell Research, 2013, 1833, 1085-1095.	4.1	10
18	TNFÎ± induces survival through the FLIP-L-dependent activation of the MAPK/ERK pathway. Cell Death and Disease, 2013, 4, e493-e493.	6.3	71

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19	Metabolic Adaptation to Tissue Iron Overload Confers Tolerance to Malaria. Cell Host and Microbe, 2012, 12, 693-704.	11.0	123
20	Heme Cytotoxicity and the Pathogenesis of Immune-Mediated Inflammatory Diseases. Frontiers in Pharmacology, 2012, 3, 77.	3.5	86
21	Heme Sensitization to TNF-Mediated Programmed Cell Death. Advances in Experimental Medicine and Biology, 2011, 691, 211-219.	1.6	21
22	A Central Role for Free Heme in the Pathogenesis of Severe Sepsis. Science Translational Medicine, 2010, 2, 51ra71.	12.4	412
23	Mechanisms of Cell Protection by Heme Oxygenase-1. Annual Review of Pharmacology and Toxicology, 2010, 50, 323-354.	9.4	1,057
24	The Death Receptor Antagonist FLIP-L Interacts with Trk and Is Necessary for Neurite Outgrowth Induced by Neurotrophins. Journal of Neuroscience, 2010, 30, 6094-6105.	3.6	13
25	Heme oxygenase-1 affords protection against noncerebral forms of severe malaria. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 15837-15842.	7.1	246
26	BCL-XL regulates TNF-Î±-mediated cell death independently of NF-ÎºB, FLIP and IAPs. Cell Research, 2008, 18, 1020-1036.	12.0	37
27	The Long Form of Fas Apoptotic Inhibitory Molecule Is Expressed Specifically in Neurons and Protects Them against Death Receptor-Triggered Apoptosis. Journal of Neuroscience, 2007, 27, 11228-11241.	3.6	73
28	The death receptor antagonist FAIM promotes neurite outgrowth by a mechanism that depends on ERK and NF-ÎºB signaling. Journal of Cell Biology, 2004, 167, 479-492.	5.2	75