

# Orestes Foresto-Neto

## List of Publications by Year in descending order

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Version: 2024-02-01

22  
papers

940  
citations

759233

12  
h-index

713466

21  
g-index

23  
all docs

23  
docs citations

23  
times ranked

1463  
citing authors

#	ARTICLE	IF	CITATIONS
1	Inflammation in Renal Diseases: New and Old Players. <i>Frontiers in Pharmacology</i> , 2019, 10, 1192.	3.5	203
2	The macrophage phenotype and inflammasome component NLRP3 contributes to nephrocalcinosis-related chronic kidney disease independent from IL-1 $\beta$ -mediated tissue injury. <i>Kidney International</i> , 2018, 93, 656-669.	5.2	159
3	Particles of different sizes and shapes induce neutrophil necroptosis followed by the release of neutrophil extracellular trap-like chromatin. <i>Scientific Reports</i> , 2017, 7, 15003.	3.3	97
4	Mitochondria Permeability Transition versus Necroptosis in Oxalate-Induced AKI. <i>Journal of the American Society of Nephrology: JASN</i> , 2019, 30, 1857-1869.	6.1	81
5	Cellular and Molecular Mechanisms of Kidney Injury in 2,8-Dihydroxyadenine Nephropathy. <i>Journal of the American Society of Nephrology: JASN</i> , 2020, 31, 799-816.	6.1	54
6	TLR2 and TLR4 play opposite role in autophagy associated with cisplatin-induced acute kidney injury. <i>Clinical Science</i> , 2018, 132, 1725-1739.	4.3	50
7	The role of uric acid in inflammasome-mediated kidney injury. <i>Current Opinion in Nephrology and Hypertension</i> , 2020, 29, 423-431.	2.0	46
8	Phagocytosis of environmental or metabolic crystalline particles induces cytotoxicity by triggering necroptosis across a broad range of particle size and shape. <i>Scientific Reports</i> , 2017, 7, 15523.	3.3	45
9	NLRP3 inflammasome inhibition ameliorates tubulointerstitial injury in the remnant kidney model. <i>Laboratory Investigation</i> , 2018, 98, 773-782.	3.7	45
10	NF- $\kappa$ B System Is Chronically Activated and Promotes Glomerular Injury in Experimental Type 1 Diabetic Kidney Disease. <i>Frontiers in Physiology</i> , 2020, 11, 84.	2.8	27
11	Aristolochic acid I determine the phenotype and activation of macrophages in acute and chronic kidney disease. <i>Scientific Reports</i> , 2018, 8, 12169.	3.3	24
12	STAT1 regulates macrophage number and phenotype and prevents renal fibrosis after ischemia-reperfusion injury. <i>American Journal of Physiology - Renal Physiology</i> , 2019, 316, F277-F291.	2.7	24
13	Renal lipotoxicity: Insights from experimental models. <i>Clinical and Experimental Pharmacology and Physiology</i> , 2021, 48, 1579-1588.	1.9	15
14	Gut Microbiota and Intestinal Epithelial Myd88 Signaling Are Crucial for Renal Injury in UUO Mice. <i>Frontiers in Immunology</i> , 2020, 11, 578623.	4.8	13
15	Simultaneous activation of innate and adaptive immunity participates in the development of renal injury in a model of heavy proteinuria. <i>Bioscience Reports</i> , 2018, 38, .	2.4	12
16	Pathogenic role of innate immunity in a model of chronic NO inhibition associated with salt overload. <i>American Journal of Physiology - Renal Physiology</i> , 2019, 317, F1058-F1067.	2.7	12
17	Renal Sensing of Bacterial Metabolites in the Gut-kidney Axis. <i>Kidney360</i> , 2021, 2, 1501-1509.	2.1	12
18	Pathogenic role of angiotensin II and the NF- $\kappa$ B system in a model of malignant hypertensive nephrosclerosis. <i>Hypertension Research</i> , 2019, 42, 779-789.	2.7	9

#	ARTICLE	IF	CITATIONS
19	Chronic exposure to hypoxia attenuates renal injury and innate immunity activation in the remnant kidney model. <i>American Journal of Physiology - Renal Physiology</i> , 2019, 317, F1285-F1292.	2.7	6
20	Renal Inflammation and Innate Immune Activation Underlie the Transition From Gentamicin-Induced Acute Kidney Injury to Renal Fibrosis. <i>Frontiers in Physiology</i> , 2021, 12, 606392.	2.8	5
21	FP411INHIBITION OF THE TLR4/NF- $\kappa$ B AXIS ATTENUATED GLOMERULAR INFLAMMATION AND SCLEROSIS IN LONG TERM EXPERIMENTAL DIABETIC KIDNEY DISEASE. <i>Nephrology Dialysis Transplantation</i> , 2018, 33, i174-i174.	0.7	0
22	SUN-311 NF-KAPPA B ACTIVATION PROMOTES GLOMERULAR INJURY AND INFLAMMATION IN LONG-TERM EXPERIMENTAL DIABETIC KIDNEY DISEASE. <i>Kidney International Reports</i> , 2019, 4, S289.	0.8	0