## David P Basile

List of Publications by Year in descending order

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DAVID P RASHE

#	Article	IF	CITATIONS
1	Orai1: A New Therapeutic Target for the Acute Kidney Injury-to-Chronic Kidney Disease Transition. Nephron, 2022, 146, 264-267.	1.8	2
2	Regeneration and replacement of endothelial cells and renal vascular repair. , 2022, , 129-144.		1
3	Macrophage dynamics in kidney repair: elucidation of a COX-2–dependent MafB pathway to affect macrophage differentiation. Kidney International, 2022, 101, 15-18.	5.2	4
4	Serum IL-17 levels are higher in critically ill patients with AKI and associated with worse outcomes. Critical Care, 2022, 26, 107.	5.8	4
5	Oxidative Stress and Inflammation Contribute to Kidney Injury Risk During Prolonged Passive Extreme Heat Exposure. FASEB Journal, 2022, 36, .	0.5	1
6	Kidney injury risk during prolonged exposure to current and projected wet bulb temperatures occurring during extreme heat events in healthy young men. Journal of Applied Physiology, 2022, 133, 27-40.	2.5	6
7	Crystals or His(stones): Rethinking AKI in Tumor Lysis Syndrome. Journal of the American Society of Nephrology: JASN, 2022, 33, 1055-1057.	6.1	1
8	T helper 17 cells in the pathophysiology of acute and chronic kidney disease. Kidney Research and Clinical Practice, 2021, 40, 12-28.	2.2	12
9	Contribution of Th17 cells to tissue injury in hypertension. Current Opinion in Nephrology and Hypertension, 2021, 30, 151-158.	2.0	10
10	Pathogenesis of Acute Kidney Injury. , 2021, , 1-38.		0
11	Mutation of RORÎ <sup>3</sup> T reveals a role for Th17 cells in both injury and recovery from renal ischemia-reperfusion injury. American Journal of Physiology - Renal Physiology, 2020, 319, F796-F808.	2.7	12
12	The case for capillary rarefaction in the AKI to CKD progression: insights from multiple injury models. American Journal of Physiology - Renal Physiology, 2019, 317, F1253-F1254.	2.7	7
13	Role of Renal Hypoxia in the Progression From Acute Kidney Injury to Chronic Kidney Disease. Seminars in Nephrology, 2019, 39, 567-580.	1.6	47
14	Calcium channel Orai1 promotes lymphocyte IL-17 expression and progressive kidney injury. Journal of Clinical Investigation, 2019, 129, 4951-4961.	8.2	40
15	Exogenous Gene Transmission of Isocitrate Dehydrogenase 2 Mimics Ischemic Preconditioning Protection. Journal of the American Society of Nephrology: JASN, 2018, 29, 1154-1164.	6.1	29
16	Th17 cells contribute to pulmonary fibrosis and inflammation during chronic kidney disease progression after acute ischemia. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2018, 314, R265-R273.	1.8	22
17	Hydrodynamic Isotonic Fluid Delivery Ameliorates Moderate-to-Severe Ischemia-Reperfusion Injury in Rat Kidneys. Journal of the American Society of Nephrology: JASN, 2017, 28, 2081-2092.	6.1	31
18	IL-17 mediates neutrophil infiltration and renal fibrosis following recovery from ischemia reperfusion: compensatory role of natural killer cells in athymic rats. American Journal of Physiology - Renal Physiology, 2017, 312, F385-F397.	2.7	68

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19	Human adipose stromal cell therapy improves survival and reduces renal inflammation and capillary rarefaction in acute kidney injury. Journal of Cellular and Molecular Medicine, 2017, 21, 1420-1430.	3.6	19
20	Endothelial colony-forming cells ameliorate endothelial dysfunction via secreted factors following ischemia-reperfusion injury. American Journal of Physiology - Renal Physiology, 2017, 312, F897-F907.	2.7	42
21	Surprising Enhancement of Fibrosis by Tubule-Specific Deletion of the TGF-Î <sup>2</sup> Receptor: A New Twist on an Old Paradigm. Journal of the American Society of Nephrology: JASN, 2017, 28, 3427-3429.	6.1	4
22	Vitamin D deficiency contributes to vascular damage in sustained ischemic acute kidney injury. Physiological Reports, 2016, 4, e12829.	1.7	39
23	Progression after AKI. Journal of the American Society of Nephrology: JASN, 2016, 27, 687-697.	6.1	351
24	Pathogenesis of Acute Kidney Injury. , 2016, , 2101-2138.		2
25	Th-17 cell activation in response to high salt following acute kidney injury is associated with progressive fibrosis and attenuated by AT-1R antagonism. Kidney International, 2015, 88, 776-784.	5.2	84
26	Effect of Renal Shock Wave Lithotripsy on the Development of Metabolic Syndrome in a Juvenile Swine Model: A Pilot Study. Journal of Urology, 2015, 193, 1409-1416.	0.4	8
27	Renal Endothelial Dysfunction in Acute Kidney Ischemia Reperfusion Injury. Cardiovascular & Hematological Disorders Drug Targets, 2014, 14, 3-14.	0.7	112
28	Pathogenesis of Acute Kidney Injury. , 2014, , 1-45.		0
29	Circulating and tissue resident endothelial progenitor cells. Journal of Cellular Physiology, 2013, 229, n/a-n/a.	4.1	173
30	A method to facilitate and monitor expression of exogenous genes in the rat kidney using plasmid and viral vectors. American Journal of Physiology - Renal Physiology, 2013, 304, F1217-F1229.	2.7	46
31	Getting the "Inside―Scoop on EphrinB2 Signaling in Pericytes and the Effect on Peritubular Capillary Stability. Journal of the American Society of Nephrology: JASN, 2013, 24, 521-523.	6.1	4
32	Chromosome substitution modulates resistance to ischemia reperfusion injury in Brown Norway rats. Kidney International, 2013, 83, 242-250.	5.2	21
33	Distinct effects on long-term function of injured and contralateral kidneys following unilateral renal ischemia-reperfusion. American Journal of Physiology - Renal Physiology, 2012, 302, F625-F635.	2.7	41
34	Persistent oxidative stress following renal ischemia-reperfusion injury increases ANG II hemodynamic and fibrotic activity. American Journal of Physiology - Renal Physiology, 2012, 302, F1494-F1502.	2.7	67
35	Activated Pericytes and the Inhibition of Renal Vascular Stability: Obstacles for Kidney Repair. Journal of the American Society of Nephrology: JASN, 2012, 23, 767-769.	6.1	6
36	Low Proliferative Potential and Impaired Angiogenesis of Cultured Rat Kidney Endothelial Cells. Microcirculation, 2012, 19, 598-609.	1.8	18

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37	Pathophysiology of Acute Kidney Injury. , 2012, 2, 1303-1353.		801
38	A CAP in our knowledge of vascular signaling in acute kidney injury. Kidney International, 2011, 80, 233-235.	5.2	0
39	Impaired endothelial proliferation and mesenchymal transition contribute to vascular rarefaction following acute kidney injury. American Journal of Physiology - Renal Physiology, 2011, 300, F721-F733.	2.7	249
40	Increased ANG II sensitivity following recovery from acute kidney injury: role of oxidant stress in skeletal muscle resistance arteries. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2010, 298, R1682-R1691.	1.8	19
41	Expression of the RNA-stabilizing protein HuR in ischemia-reperfusion injury of rat kidney. American Journal of Physiology - Renal Physiology, 2009, 297, F95-F105.	2.7	32
42	Recovery from renal ischemia-reperfusion injury is associated with altered renal hemodynamics, blunted pressure natriuresis, and sodium-sensitive hypertension. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2009, 297, R1358-R1363.	1.8	73
43	VEGF-121 preserves renal microvessel structure and ameliorates secondary renal disease following acute kidney injury. American Journal of Physiology - Renal Physiology, 2008, 295, F1648-F1657.	2.7	143
44	Immune suppression blocks sodium-sensitive hypertension following recovery from ischemic acute renal failure. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2008, 294, R1234-R1239.	1.8	58
45	Renal ischemia reperfusion inhibits VEGF expression and induces ADAMTS-1, a novel VEGF inhibitor. American Journal of Physiology - Renal Physiology, 2008, 294, F928-F936.	2.7	154
46	Challenges of targeting vascular stability in acute kidney injury. Kidney International, 2008, 74, 257-258.	5.2	7
47	An expanding role of Toll-like receptors in sepsis-induced acute kidney injury. American Journal of Physiology - Renal Physiology, 2008, 294, F1048-F1049.	2.7	7
48	Recovery from acute renal failure predisposes hypertension and secondary renal disease in response to elevated sodium. American Journal of Physiology - Renal Physiology, 2007, 293, F269-F278.	2.7	100
49	Novel Approaches in the Investigation of Acute Kidney Injury. Journal of the American Society of Nephrology: JASN, 2007, 18, 7-9.	6.1	6
50	Immune suppression blocks sodium sensitive hypertension following recovery from ischemic acute renal failure. FASEB Journal, 2007, 21, A591.	0.5	0
51	Impaired sodium excretion following recovery from ischemic acute renal failure. FASEB Journal, 2006, 20, A341.	0.5	0
52	Transcriptome analysis and kidney research: Toward systems biology. Kidney International, 2005, 67, 2114-2122.	5.2	25
53	Enhanced skeletal muscle arteriolar reactivity to ANG II after recovery from ischemic acute renal failure. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2005, 289, R1770-R1776.	1.8	15
54	Identification of persistently altered gene expression in the kidney after functional recovery from ischemic acute renal failure. American Journal of Physiology - Renal Physiology, 2005, 288, F953-F963.	2.7	86

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55	Transforming growth factor-β in acute renal failure: receptor expression, effects on proliferation, cellularity, and vascularization after recovery from injury. American Journal of Physiology - Renal Physiology, 2005, 288, F568-F577.	2.7	90
56	Angiostatin and matrix metalloprotease expression following ischemic acute renal failure. American Journal of Physiology - Renal Physiology, 2004, 286, F893-F902.	2.7	110
57	Resistance to ischemic acute renal failure in the Brown Norway rat: A new model to study cytoprotection. Kidney International, 2004, 65, 2201-2211.	5.2	47
58	Rarefaction of peritubular capillaries following ischemic acute renal failure: a potential factor predisposing to progressive nephropathy. Current Opinion in Nephrology and Hypertension, 2004, 13, 1-7.	2.0	202
59	Chronic renal hypoxia after acute ischemic injury: effects of <scp>I</scp> -arginine on hypoxia and secondary damage. American Journal of Physiology - Renal Physiology, 2003, 284, F338-F348.	2.7	134
60	Renal ischemic injury results in permanent damage to peritubular capillaries and influences long-term function. American Journal of Physiology - Renal Fluid and Electrolyte Physiology, 2001, 281, F887-F899.	0.0	248
61	Renal ischemic injury results in permanent damage to peritubular capillaries and influences long-term function. American Journal of Physiology - Renal Physiology, 2001, 281, F887-F899.	2.7	426
62	Transforming growth factor-β as a target for treatment in diabetic nephropathy. American Journal of Kidney Diseases, 2001, 38, 887-890.	1.9	16
63	Renal ischemic injury results in permanent damage to peritubular capillaries and influences long-term function. American Journal of Physiology - Renal Physiology, 2001, 281, F887-F899.	2.7	340
64	Toward an effective gene therapy in renal disease. Kidney International, 1999, 55, 740-741.	5.2	1
65	The transforming growth factor beta system in kidney disease and repair: recent progress and future directions. Current Opinion in Nephrology and Hypertension, 1999, 8, 21-30.	2.0	106
66	Extracellular matrix-related genes in kidney after ischemic injury: potential role for TGF-β in repair. American Journal of Physiology - Renal Physiology, 1998, 275, F894-F903.	2.7	47