Simona Buelli

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Shiga Toxin 2 Triggers C3a-Dependent Glomerular and Tubular Injury through Mitochondrial Dysfunction in Hemolytic Uremic Syndrome. Cells, 2022, 11, 1755.	4.1	3
2	Endothelin-targeted new treatments for proteinuric and inflammatory glomerular diseases: focus on the added value to anti-renin-angiotensin system inhibition. Pediatric Nephrology, 2021, 36, 763-775.	1.7	17
3	C3a receptor blockade protects podocytes from injury in diabetic nephropathy. JCI Insight, 2020, 5, .	5.0	46
4	Complement Activation Contributes to the Pathophysiology of Shiga Toxin-Associated Hemolytic Uremic Syndrome. Microorganisms, 2019, 7, 15.	3.6	23
5	Shiga toxin triggers endothelial and podocyte injury: the role of complement activation. Pediatric Nephrology, 2019, 34, 379-388.	1.7	34
6	SGLT2 inhibitor dapagliflozin limits podocyte damage in proteinuric nondiabetic nephropathy. JCI Insight, 2018, 3, .	5.0	114
7	A previously unrecognized role of C3a in proteinuric progressive nephropathy. Scientific Reports, 2016, 6, 28445.	3.3	22
8	Untangling the Knot in Diabetic Nephropathy: The Unanticipated Role of Glycocalyx in the Antiproteinuric Effect of Endothelin Receptor Antagonists. Diabetes, 2016, 65, 2115-2117.	0.6	5
9	Sirtuin3 Dysfunction Is the Key Determinant of Skeletal Muscle Insulin Resistance by Angiotensin II. PLoS ONE, 2015, 10, e0127172.	2.5	16
10	Effects of MCP-1 Inhibition by Bindarit Therapy in a Rat Model of Polycystic Kidney Disease. Nephron, 2015, 129, 52-61.	1.8	43
11	Mitochondrial-dependent Autoimmunity in Membranous Nephropathy of IgG4-related Disease. EBioMedicine, 2015, 2, 456-466.	6.1	24
12	Shiga Toxin Promotes Podocyte Injury in Experimental Hemolytic Uremic Syndrome via Activation of the Alternative Pathway of Complement. Journal of the American Society of Nephrology: JASN, 2014, 25, 1786-1798.	6.1	52
13	β-Arrestin-1 Drives Endothelin-1–Mediated Podocyte Activation and Sustains Renal Injury. Journal of the American Society of Nephrology: JASN, 2014, 25, 523-533.	6.1	63
14	Angiotensin II Contributes to Diabetic Renal Dysfunction in Rodents and Humans via Notch1/Snail Pathway. American Journal of Pathology, 2013, 183, 119-130.	3.8	39
15	Membranous Nephropathy Associated With IgG4-Related Disease. American Journal of Kidney Diseases, 2011, 58, 272-275.	1.9	64
16	<i>MYO1E</i> Mutations and Childhood Familial Focal Segmental Glomerulosclerosis. New England Journal of Medicine, 2011, 365, 295-306.	27.0	221
17	Alternative Pathway Activation of Complement by Shiga Toxin Promotes Exuberant C3a Formation That Triggers Microvascular Thrombosis. Journal of Immunology, 2011, 187, 172-180.	0.8	220
18	Endothelin in Chronic Proteinuric Kidney Disease. Contributions To Nephrology, 2011, 172, 171-184.	1.1	15

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19	Shiga toxin-associated hemolytic uremic syndrome: pathophysiology of endothelial dysfunction. Pediatric Nephrology, 2010, 25, 2231-2240.	1.7	156
20	Protein load impairs factor H binding promoting complement-dependent dysfunction of proximal tubular cells. Kidney International, 2009, 75, 1050-1059.	5.2	28
21	Proteasomal Processing of Albumin by Renal Dendritic Cells Generates Antigenic Peptides. Journal of the American Society of Nephrology: JASN, 2009, 20, 123-130.	6.1	88
22	Fractalkine and CX3CR1 Mediate Leukocyte Capture by Endothelium in Response to Shiga Toxin. Journal of Immunology, 2008, 181, 1460-1469.	0.8	37
23	Permselective Dysfunction of Podocyte-Podocyte Contact upon Angiotensin II Unravels the Molecular Target for Renoprotective Intervention. American Journal of Pathology, 2006, 168, 1073-1085.	3.8	82
24	Shigatoxin-Induced Endothelin-1 Expression in Cultured Podocytes Autocrinally Mediates Actin Remodeling. American Journal of Pathology, 2006, 169, 1965-1975.	3.8	92
25	Activation of porcine endothelium in response to xenogeneic serum causes thrombosis independently of platelet activation. Xenotransplantation, 2005, 12, 110-120.	2.8	14
26	In Response to Protein Load Podocytes Reorganize Cytoskeleton and Modulate Endothelin-1 Gene. American Journal of Pathology, 2005, 166, 1309-1320.	3.8	151
27	Protein Overload Induces Fractalkine Upregulation in Proximal Tubular Cells through Nuclear Factor κB– and p38 Mitogen-Activated Protein Kinase–Dependent Pathways. Journal of the American Society of Nephrology: JASN, 2003, 14, 2436-2446.	6.1	118
28	Protein overload-induced NF-kappaB activation in proximal tubular cells requires H(2)O(2) through a PKC-dependent pathway. Journal of the American Society of Nephrology: JASN, 2002, 13, 1179-89.	6.1	135