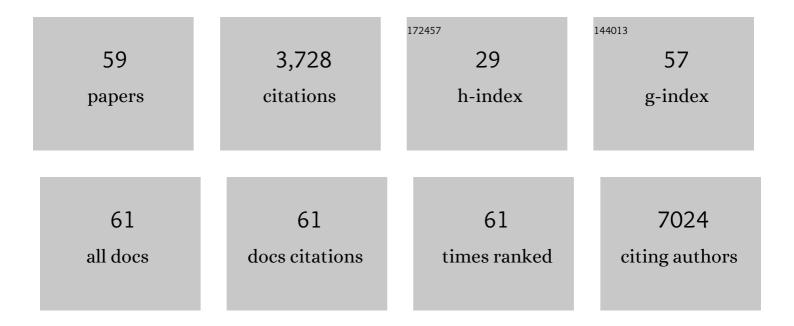
## **Catherine Meyer-Schwesinger**

List of Publications by Year in descending order

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#	Article	lF	CITATIONS
1	Ectodomain shedding by ADAM proteases as a central regulator in kidney physiology and disease. Biochimica Et Biophysica Acta - Molecular Cell Research, 2022, 1869, 119165.	4.1	6
2	The calcium-sensing receptor stabilizes podocyte function in proteinuric humans and mice. Kidney International, 2022, 101, 1186-1199.	5.2	6
3	Antigen Cross-Presentation by Murine Proximal Tubular Epithelial Cells Induces Cytotoxic and Inflammatory CD8+ T Cells. Cells, 2022, 11, 1510.	4.1	6
4	Loss of Renal Mass Exacerbates Renal Damage in a Mouse Model of Highâ€Renin Hypertension. FASEB Journal, 2022, 36, .	0.5	0
5	Lysosome function in glomerular health and disease. Cell and Tissue Research, 2021, 385, 371-392.	2.9	21
6	ADAM10-Mediated Ectodomain Shedding Is an Essential Driver of Podocyte Damage. Journal of the American Society of Nephrology: JASN, 2021, 32, 1389-1408.	6.1	7
7	ADP-Ribosylation Regulates the Signaling Function of IFN-Î <sup>3</sup> . Frontiers in Immunology, 2021, 12, 642545.	4.8	7
8	Tripartite Separation of Glomerular Cell Types and Proteomes from Reporter-Free Mice. Journal of the American Society of Nephrology: JASN, 2021, 32, 2175-2193.	6.1	16
9	The Intertwining of Autophagy and the Ubiquitin Proteasome System in Podocyte (Patho)Physiology. Cellular Physiology and Biochemistry, 2021, 55, 68-95.	1.6	10
10	CD73-mediated adenosine production by CD8 T cell-derived extracellular vesicles constitutes an intrinsic mechanism of immune suppression. Nature Communications, 2021, 12, 5911.	12.8	66
11	A novel mouse model of phospholipase A2 receptor 1-associated membranous nephropathyÂmimics podocyte injury in patients. Kidney International, 2020, 97, 913-919.	5.2	65
12	Pathogen-induced tissue-resident memory T <sub>H</sub> 17 (T <sub>RM</sub> 17) cells amplify autoimmune kidney disease. Science Immunology, 2020, 5, .	11.9	58
13	The ins-and-outs of podocyte lipid metabolism. Kidney International, 2020, 98, 1087-1090.	5.2	15
14	Podocytes Produce and Secrete Functional Complement C3 and Complement Factor H. Frontiers in Immunology, 2020, 11, 1833.	4.8	19
15	Inhibition of mTOR delayed but could not prevent experimental collapsing focal segmental glomerulosclerosis. Scientific Reports, 2020, 10, 8580.	3.3	3
16	Distinct Modes of Balancing Glomerular Cell Proteostasis in Mucolipidosis Type II and III Prevent Proteinuria. Journal of the American Society of Nephrology: JASN, 2020, 31, 1796-1814.	6.1	7
17	Enzyme replacement therapy in mice lacking arylsulfatase B targets bone-remodeling cells, but not chondrocytes. Human Molecular Genetics, 2020, 29, 803-816.	2.9	15
18	Interleukin-9 protects from early podocyte injury and progressive glomerulosclerosis in Adriamycin-induced nephropathy. Kidney International, 2020, 98, 615-629.	5.2	18

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19	Deubiquitinating Enzyme UCH-L1 Promotes Dendritic Cell Antigen Cross-Presentation by Favoring Recycling of MHC Class I Molecules. Journal of Immunology, 2019, 203, 1730-1742.	0.8	10
20	Thrombospondin Type 1 Domain–Containing 7A Localizes to the Slit Diaphragm and Stabilizes Membrane Dynamics of Fully Differentiated Podocytes. Journal of the American Society of Nephrology: JASN, 2019, 30, 824-839.	6.1	42
21	An unexpected role of steroid on podocytes: from zebrafish to human nephrotic syndrome?. Kidney International, 2019, 95, 1015-1017.	5.2	0
22	The ubiquitin–proteasome system inÂkidney physiology and disease. Nature Reviews Nephrology, 2019, 15, 393-411.	9.6	86
23	Ubiquitin C-terminal hydrolase L1 (UCH-L1) loss causes neurodegeneration by altering protein turnover in the first postnatal weeks. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 7963-7972.	7.1	36
24	Isolation of Glomeruli and <em>In Vivo</em> Labeling of Glomerular Cell Surface Proteins. Journal of Visualized Experiments, 2019, , .	0.3	3
25	Protecting the kidney against autoimmunity and inflammation. Nature Reviews Nephrology, 2019, 15, 66-68.	9.6	10
26	Glomerular endothelial cell maturation depends on ADAM10, a key regulator of Notch signaling. Angiogenesis, 2018, 21, 335-347.	7.2	31
27	Ubiquitin C-Terminal Hydrolase L1 is required forÂregulated protein degradation through theÂubiquitin proteasome system in kidney. Kidney International, 2018, 93, 110-127.	5.2	25
28	Disease-Linked Glutarylation Impairs Function and Interactions of Mitochondrial Proteins and Contributes to Mitochondrial Heterogeneity. Cell Reports, 2018, 24, 2946-2956.	6.4	42
29	Role of Apolipoprotein L1 in Human Parietal Epithelial Cell Transition. American Journal of Pathology, 2018, 188, 2508-2528.	3.8	25
30	IL-33–Mediated Expansion of Type 2 Innate Lymphoid Cells Protects from Progressive Glomerulosclerosis. Journal of the American Society of Nephrology: JASN, 2017, 28, 2068-2080.	6.1	93
31	A Heterologous Model of Thrombospondin Type 1 Domain-Containing 7A-Associated Membranous Nephropathy. Journal of the American Society of Nephrology: JASN, 2017, 28, 3262-3277.	6.1	64
32	A Mechanism for Cancer-Associated Membranous Nephropathy. New England Journal of Medicine, 2016, 374, 1995-1996.	27.0	158
33	Nanobodies that block gating of the P2X7 ion channel ameliorate inflammation. Science Translational Medicine, 2016, 8, 366ra162.	12.4	139
34	The Role of Renal Progenitors in Renal Regeneration. Nephron, 2016, 132, 101-109.	1.8	14
35	Autoantibodies against thrombospondin type 1 domain–containing 7A induce membranous nephropathy. Journal of Clinical Investigation, 2016, 126, 2519-2532.	8.2	181
36	T <sub>H1</sub> and T <sub>H17</sub> cells promote crescent formation in experimental autoimmune glomerulonephritis. Journal of Pathology, 2015, 237, 62-71.	4.5	27

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37	CD38 Is Expressed on Inflammatory Cells of the Intestine and Promotes Intestinal Inflammation. PLoS ONE, 2015, 10, e0126007.	2.5	48
38	Thrombospondin Type-1 Domain-Containing 7A in Idiopathic Membranous Nephropathy. New England Journal of Medicine, 2015, 372, 1073-1075.	27.0	58
39	Glomerulopathy Induced by Immunization with a Peptide Derived from the Goodpasture Antigen $\hat{I}\pm 3$ IV-NC1. Journal of Immunology, 2015, 194, 3646-3655.	0.8	12
40	MicroRNA-193a Regulates the Transdifferentiation of Human Parietal Epithelial Cells toward a Podocyte Phenotype. Journal of the American Society of Nephrology: JASN, 2015, 26, 1389-1401.	6.1	64
41	Alterations in the Ubiquitin Proteasome System in Persistent but Not Reversible Proteinuric Diseases. Journal of the American Society of Nephrology: JASN, 2014, 25, 2511-2525.	6.1	31
42	Thrombospondin Type-1 Domain-Containing 7A in Idiopathic Membranous Nephropathy. New England Journal of Medicine, 2014, 371, 2277-2287.	27.0	729
43	UCH-L1 induces podocyte hypertrophy in membranous nephropathy by protein accumulation. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2014, 1842, 945-958.	3.8	13
44	The proteases HtrA2/Omi and UCH-L1 regulate TNF-induced necroptosis. Cell Communication and Signaling, 2013, 11, 76.	6.5	55
45	Prognostic relevance of ubiquitin C-terminal hydrolase L1 (UCH-L1) mRNA and protein expression in breast cancer patients. Journal of Cancer Research and Clinical Oncology, 2013, 139, 1745-1755.	2.5	23
46	The expression of podocyte-specific proteins in parietal epithelial cells is regulated by protein degradation. Kidney International, 2013, 84, 532-544.	5.2	34
47	MicroRNA-155 Drives TH17 Immune Response and Tissue Injury in Experimental Crescentic GN. Journal of the American Society of Nephrology: JASN, 2013, 24, 1955-1965.	6.1	41
48	Mouse models of membranous nephropathy: the road less travelled by. American Journal of Clinical and Experimental Immunology, 2013, 2, 135-45.	0.2	13
49	Rho-kinase inhibition prevents proteinuria in immune-complex-mediated antipodocyte nephritis. American Journal of Physiology - Renal Physiology, 2012, 303, F1015-F1025.	2.7	13
50	IL-17A Production by Renal γδT Cells Promotes Kidney Injury in Crescentic GN. Journal of the American Society of Nephrology: JASN, 2012, 23, 1486-1495.	6.1	78
51	Bone Marrow–Derived Progenitor Cells Do Not Contribute to Podocyte Turnover in the Puromycin Aminoglycoside and Renal Ablation Models in Rats. American Journal of Pathology, 2011, 178, 494-499.	3.8	25
52	Ubiquitin C-Terminal Hydrolase-L1 Activity Induces Polyubiquitin Accumulation in Podocytes and Increases Proteinuria in Rat Membranous Nephropathy. American Journal of Pathology, 2011, 178, 2044-2057.	3.8	50
53	Ubiquitin carboxyl-terminal hydrolase 1 (UCHL1) is a potential tumour suppressor in prostate cancer and is frequently silenced by promoter methylation. Molecular Cancer, 2011, 10, 129.	19.2	88
54	Nephrotic Syndrome and Subepithelial Deposits in a Mouse Model of Immune-Mediated Anti-Podocyte Glomerulonephritis. Journal of Immunology, 2011, 187, 3218-3229.	0.8	46

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55	CIN85/RukL Is a Novel Binding Partner of Nephrin and Podocin and Mediates Slit Diaphragm Turnover in Podocytes. Journal of Biological Chemistry, 2010, 285, 25285-25295.	3.4	51
56	Autophagy influences glomerular disease susceptibility and maintains podocyte homeostasis in aging mice. Journal of Clinical Investigation, 2010, 120, 1084-1096.	8.2	604
57	Rho kinase inhibition attenuates LPS-induced renal failure in mice in part by attenuation of NF-κB p65 signaling. American Journal of Physiology - Renal Physiology, 2009, 296, F1088-F1099.	2.7	50
58	Kidney dendritic cell activation is required for progression of renal disease in a mouse model of glomerular injury. Journal of Clinical Investigation, 2009, 119, 1286-1297.	8.2	180
59	Chemokine Receptor CXCR3 Mediates T Cell Recruitment and Tissue Injury in Nephrotoxic Nephritis in Mice. Journal of the American Society of Nephrology: JASN, 2007, 18, 2071-2084.	6.1	89