

# Erica M Sparkenbaugh

## List of Publications by Year in descending order

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39  
papers

1,558  
citations

304743

22  
h-index

414414

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39  
docs citations

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times ranked

2157  
citing authors

#	ARTICLE	IF	CITATIONS
1	Targeting the AnxA1/Fpr2/ALX pathway regulates neutrophil function, promoting thrombin inflammation resolution in sickle cell disease. <i>Blood</i> , 2021, 137, 1538-1549.	1.4	35
2	Pathologically stiff erythrocytes impede contraction of blood clots: Comment. <i>Journal of Thrombosis and Haemostasis</i> , 2021, 19, 2893-2894.	3.8	2
3	Inhibition of Factor XII Attenuates Prothrombotic Complications in Sickle Cell Mice. <i>Blood</i> , 2021, 138, 189-189.	1.4	0
4	Synthetic Heparan Sulfate Compounds Attenuate Vascular Complications Associated with Sickle Cell Disease. <i>Blood</i> , 2021, 138, 857-857.	1.4	0
5	P-Selectin Deficiency Reduces Acute Vascular Complications but Not Chronic Organ Damage in Sickle Cell Mice. <i>Blood</i> , 2021, 138, 187-187.	1.4	0
6	Thrombin activation of PAR-1 contributes to microvascular stasis in mouse models of sickle cell disease. <i>Blood</i> , 2020, 135, 1783-1787.	1.4	32
7	Design of anti-inflammatory heparan sulfate to protect against acetaminophen-induced acute liver failure. <i>Science Translational Medicine</i> , 2020, 12, .	12.4	60
8	Enzymatic Synthesis of Chondroitin Sulfate E to Attenuate Bacteria Lipopolysaccharide-Induced Organ Damage. <i>ACS Central Science</i> , 2020, 6, 1199-1207.	11.3	23
9	High molecular weight kininogen contributes to early mortality and kidney dysfunction in a mouse model of sickle cell disease. <i>Journal of Thrombosis and Haemostasis</i> , 2020, 18, 2329-2340.	3.8	7
10	Novel Role for the AnxA1-Fpr2/ALX Signaling Axis as a Key Regulator of Platelet Function to Promote Resolution of Inflammation. <i>Circulation</i> , 2019, 140, 319-335.	1.6	98
11	Red blood cells modulate structure and dynamics of venous clot formation in sickle cell disease. <i>Blood</i> , 2019, 133, 2529-2541.	1.4	51
12	High Molecular Weight Kininogen but Not Factor XII Deficiency Attenuates Acetaminophen-Induced Liver Injury in Mice. <i>Blood</i> , 2019, 134, 3621-3621.	1.4	1
13	Hypercoagulable state in sickle cell disease. <i>Clinical Hemorheology and Microcirculation</i> , 2018, 68, 301-318.	1.7	26
14	High Molecular Weight Kininogen Contributes to End-Organ Damage and Mortality in a Mouse Model of Sickle Cell Disease. <i>Blood</i> , 2018, 132, 268-268.	1.4	1
15	Thrombin-Mediated Activation of PAR-1 Contributes to Microvascular Stasis in Mouse Models of Sickle Cell Disease Via Increased Endothelial Expression of P-Selectin and VWF. <i>Blood</i> , 2018, 132, 266-266.	1.4	0
16	In vitro activation of coagulation by human neutrophil DNA and histone proteins but not neutrophil extracellular traps. <i>Blood</i> , 2017, 129, 1021-1029.	1.4	183
17	Synthetic oligosaccharides can replace animal-sourced low molecular weight heparins. <i>Science Translational Medicine</i> , 2017, 9, .	12.4	82
18	Prothrombotic aspects of sickle cell disease. <i>Journal of Thrombosis and Haemostasis</i> , 2017, 15, 1307-1316.	3.8	36

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19	Thrombin-independent contribution of tissue factor to inflammation and cardiac hypertrophy in a mouse model of sickle cell disease. <i>Blood</i> , 2016, 127, 1371-1373.	1.4	17
20	Hepatocyte tissue factor contributes to the hypercoagulable state in a mouse model of chronic liver injury. <i>Journal of Hepatology</i> , 2016, 64, 53-59.	3.7	36
21	Protective and detrimental effects of neuroectodermal cell-derived tissue factor in mouse models of stroke. <i>JCI Insight</i> , 2016, 1, .	5.0	6
22	FXIIa Differentially Regulates Thrombin Generation during Steady State and Vaso-Occlusive Crisis in Sickle Cell Mice. <i>Blood</i> , 2016, 128, 162-162.	1.4	5
23	Alteration of the Structure and Dynamics of Venous Clot Formation in Human and Murine Sickle Cell Disease. <i>Blood</i> , 2016, 128, 2478-2478.	1.4	2
24	Excess of heme induces tissue factor-dependent activation of coagulation in mice. <i>Haematologica</i> , 2015, 100, 308-314.	3.5	77
25	Abstract 594: Characterization of Anti-thrombotic and Anti-inflammatory Properties of New Synthetic, Protamine Reversible Low Molecular Weight Heparin. <i>Arteriosclerosis, Thrombosis, and Vascular Biology</i> , 2015, 35, .	2.4	0
26	Tissue factor, protease activated receptors and pathologic heart remodelling. <i>Thrombosis and Haemostasis</i> , 2014, 112, 893-900.	3.4	24
27	Homogeneous low-molecular-weight heparins with reversible anticoagulant activity. <i>Nature Chemical Biology</i> , 2014, 10, 248-250.	8.0	173
28	De novo synthesis of a narrow size distribution low-molecular-weight heparin. <i>Glycobiology</i> , 2014, 24, 476-486.	2.5	16
29	Differential contribution of FXa and thrombin to vascular inflammation in a mouse model of sickle cell disease. <i>Blood</i> , 2014, 123, 1747-1756.	1.4	98
30	Interplay between coagulation and vascular inflammation in sickle cell disease. <i>British Journal of Haematology</i> , 2013, 162, 3-14.	2.5	127
31	Protease Activated Receptor-2 Contributes to Heart Failure. <i>PLoS ONE</i> , 2013, 8, e81733.	2.5	41
32	Tissue factor promotes activation of coagulation and inflammation in a mouse model of sickle cell disease. <i>Blood</i> , 2012, 120, 636-646.	1.4	94
33	Heme Induces Systemic Activation of Coagulation in Vivo in a Tissue Factor-Dependent Manner. <i>Blood</i> , 2012, 120, 820-820.	1.4	0
34	Protease Activated Receptor 2 (PAR-2) Promotes Vascular Inflammation in a Mouse Model of Sickle Cell Disease. <i>Blood</i> , 2012, 120, 375-375.	1.4	0
35	The Role of Hypoxia-Inducible Factor-1 $\alpha$ in Acetaminophen Hepatotoxicity. <i>Journal of Pharmacology and Experimental Therapeutics</i> , 2011, 338, 492-502.	2.5	39
36	Trovafloxacin Enhances TNF-Induced Inflammatory Stress and Cell Death Signaling and Reduces TNF Clearance in a Murine Model of Idiosyncratic Hepatotoxicity. <i>Toxicological Sciences</i> , 2009, 111, 288-301.	3.1	38

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37	Hepatotoxic Interaction of Sulindac with Lipopolysaccharide: Role of the Hemostatic System. <i>Toxicological Sciences</i> , 2009, 108, 184-193.	3.1	55
38	Sulindac Metabolism and Synergy with Tumor Necrosis Factor- $\alpha$ in a Drug-Inflammation Interaction Model of Idiosyncratic Liver Injury. <i>Journal of Pharmacology and Experimental Therapeutics</i> , 2009, 331, 114-121.	2.5	46
39	Gene Expression Profiles in Livers from Diclofenac-Treated Rats Reveal Intestinal Bacteria-Dependent and -Independent Pathways Associated with Liver Injury. <i>Journal of Pharmacology and Experimental Therapeutics</i> , 2008, 327, 634-644.	2.5	27