

# Angela J Glading

## List of Publications by Year in descending order

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34  
papers

2,396  
citations

471371

17  
h-index

642610

23  
g-index

34  
all docs

34  
docs citations

34  
times ranked

2373  
citing authors

#	ARTICLE	IF	CITATIONS
1	Contribution of protein-protein interactions to the endothelial-barrier-stabilizing function of KRIT1. <i>Journal of Cell Science</i> , 2022, 135, .	1.2	4
2	KRIT1 stabilizes endothelial adherens junctions independent of Rap1 via regulation of $\beta$ 1-integrin. <i>FASEB Journal</i> , 2021, 35, .	0.2	0
3	Protein Kinase C $\delta$ (PKC $\delta$ ) Regulates the Nucleocytoplasmic Shuttling of KRIT1. <i>FASEB Journal</i> , 2021, 35, .	0.2	0
4	Protein kinase C $\delta$ (PKC $\delta$ ) regulates the nucleocytoplasmic shuttling of KRIT1. <i>Journal of Cell Science</i> , 2021, 134, .	1.2	8
5	Disease models in cerebral cavernous malformations. <i>Drug Discovery Today: Disease Models</i> , 2020, 31, 21-29.	1.2	0
6	VEGF signalling enhances lesion burden in KRIT1 deficient mice. <i>Journal of Cellular and Molecular Medicine</i> , 2020, 24, 632-639.	1.6	22
7	Microvascular Mimetics for the Study of Leukocyte-Endothelial Interactions. <i>Cellular and Molecular Bioengineering</i> , 2020, 13, 125-139.	1.0	16
8	Isolation of Cerebral Endothelial Cells from CCM1/KRIT1 Null Mouse Brain. <i>Methods in Molecular Biology</i> , 2020, 2152, 259-265.	0.4	0
9	Measurement of Endothelial Barrier Function in Mouse Models of Cerebral Cavernous Malformations Using Intravital Microscopy. <i>Methods in Molecular Biology</i> , 2020, 2152, 387-400.	0.4	0
10	VEGF is required for the initiation of Cerebral Cavernous Malformations. <i>FASEB Journal</i> , 2018, 32, 35.7.	0.2	1
11	Up-regulation of NADPH oxidase-mediated redox signaling contributes to the loss of barrier function in KRIT1 deficient endothelium. <i>Scientific Reports</i> , 2017, 7, 8296.	1.6	51
12	Phospholipase C $\mu$ Modulates Rap1 Activity and the Endothelial Barrier. <i>PLoS ONE</i> , 2016, 11, e0162338.	1.1	4
13	Oxidative stress and inflammation in cerebral cavernous malformation disease pathogenesis: Two sides of the same coin. <i>International Journal of Biochemistry and Cell Biology</i> , 2016, 81, 254-270.	1.2	80
14	Control of vascular permeability by adhesion molecules. <i>Tissue Barriers</i> , 2015, 3, e985954.	1.6	57
15	Destabilization of endothelial cell-cell contacts modifies inflammatory responses. <i>FASEB Journal</i> , 2015, 29, 418.6.	0.2	0
16	KRIT1 Depletion Modifies Endothelial Cell Behavior Through Increased VEGF Signaling. <i>FASEB Journal</i> , 2015, 29, 418.4.	0.2	0
17	Measurement of blood flow velocity for <i>in vivo</i> video sequences with motion estimation methods. <i>Proceedings of SPIE</i> , 2014, , .	0.8	0
18	KRIT1 Protein Depletion Modifies Endothelial Cell Behavior via Increased Vascular Endothelial Growth Factor (VEGF) Signaling. <i>Journal of Biological Chemistry</i> , 2014, 289, 33054-33065.	1.6	54

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19	Decreased Krev Interactionâ€“Trapped 1 Expression Leads to Increased Vascular Permeability and Modifies Inflammatory Responses In Vivo. <i>Arteriosclerosis, Thrombosis, and Vascular Biology</i> , 2012, 32, 2702-2710.	1.1	36
20	Rap1 and its effector KRIT1/CCM1 regulate $\beta$ -catenin signaling. <i>DMM Disease Models and Mechanisms</i> , 2010, 3, 73-83.	1.2	104
21	Rap1 and its effector KRIT1/CCM1 regulate $\beta$ -catenin signaling. <i>Journal of Cell Science</i> , 2010, 123, e1-e1.	1.2	0
22	KRIT-1/CCM1 is a Rap1 effector that regulates endothelial cellâ€“cell junctions. <i>Journal of Cell Biology</i> , 2007, 179, 247-254.	2.3	280
23	PEA-15 Inhibits Tumor Cell Invasion by Binding to Extracellular Signal-Regulated Kinase 1/2. <i>Cancer Research</i> , 2007, 67, 1536-1544.	0.4	73
24	Phosphorylation of Phosphoprotein Enriched in Astrocytes (PEA-15) Regulates Extracellular Signal-regulated Kinase-dependent Transcription and Cell Proliferation. <i>Molecular Biology of the Cell</i> , 2005, 16, 3552-3561.	0.9	75
25	Interferon-Inducible Protein 9 (CXCL11)-Induced Cell Motility in Keratinocytes Requires Calcium Flux-Dependent Activation of $\beta$ -Calpain. <i>Molecular and Cellular Biology</i> , 2005, 25, 1922-1941.	1.1	75
26	Epidermal Growth Factor Activates m-Calpain (Calpain II), at Least in Part, by Extracellular Signal-Regulated Kinase-Mediated Phosphorylation. <i>Molecular and Cellular Biology</i> , 2004, 24, 2499-2512.	1.1	250
27	PEA-15 Binding to ERK1/2 MAPKs Is Required for Its Modulation of Integrin Activation. <i>Journal of Biological Chemistry</i> , 2003, 278, 52587-52597.	1.6	52
28	Activation of m-Calpain (Calpain II) by Epidermal Growth Factor Is Limited by Protein Kinase A Phosphorylation of m-Calpain. <i>Molecular and Cellular Biology</i> , 2002, 22, 2716-2727.	1.1	162
29	Cutting to the chase: calpain proteases in cell motility. <i>Trends in Cell Biology</i> , 2002, 12, 46-54.	3.6	350
30	Membrane Proximal ERK Signaling Is Required for M-calpain Activation Downstream of Epidermal Growth Factor Receptor Signaling. <i>Journal of Biological Chemistry</i> , 2001, 276, 23341-23348.	1.6	186
31	Epidermal Growth Factor Receptor Activation of Calpain Is Required for Fibroblast Motility and Occurs via an ERK/MAP Kinase Signaling Pathway. <i>Journal of Biological Chemistry</i> , 2000, 275, 2390-2398.	1.6	240
32	Ip-10 Inhibits Epidermal Growth Factorâ€“Induced Motility by Decreasing Epidermal Growth Factor Receptorâ€“Mediated Calpain Activity. <i>Journal of Cell Biology</i> , 1999, 146, 243-254.	2.3	127
33	Epidermal growth factor receptor-mediated motility in fibroblasts. , 1998, 43, 395-411.		87
34	Is Location Everything? Regulation of the Endothelial CCM Signaling Complex. <i>Frontiers in Cardiovascular Medicine</i> , 0, 9, .	1.1	2