Martha E O'donnell

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	High glucose-induced effects on Na ⁺ -K ⁺ -2Cl ^{â^'} cotransport and Na ⁺ /H ⁺ exchange of blood-brain barrier endothelial cells: involvement of SGK1, PKCl²II, and SPAK/OSR1. American Journal of Physiology - Cell Physiology, 2021, 320, C619-C634.	2.1	9
2	Diabetic ketoacidosis causes chronic elevation in renal C-C motif chemokine ligand 5. Endocrine, 2021, , 1.	1.1	1
3	Exacerbated brain edema in a rat streptozotocin model of hyperglycemic ischemic stroke: Evidence for involvement of blood–brain barrier Na–K–Cl cotransport and Na/H exchange. Journal of Cerebral Blood Flow and Metabolism, 2019, 39, 1678-1692.	2.4	20
4	Pathophysiology of stroke: what do cells of the neurovascular unit have to do with it?. American Journal of Physiology - Cell Physiology, 2019, 316, C1-C1.	2.1	4
5	Pathophysiology of stroke: the many and varied contributions of brain microvasculature. American Journal of Physiology - Cell Physiology, 2018, 315, C341-C342.	2.1	4
6	Hyperglycemiaâ€Induced Alteration of Brain Microvascular Endothelial Intracellular Ca Response to Ischemic Factors: Role of TRPV4 Channels. FASEB Journal, 2018, 32, lb445.	0.2	0
7	Treatment with the KCa3.1 inhibitor TRAM-34 during diabetic ketoacidosis reduces inflammatory changes in the brain. Pediatric Diabetes, 2017, 18, 356-366.	1.2	18
8	Histological and cognitive alterations in adult diabetic rats following an episode of juvenile diabetic ketoacidosis: Evidence of permanent cerebral injury. Neuroscience Letters, 2017, 650, 161-167.	1.0	8
9	Diabetic ketoacidosis in juvenile rats is associated with reactive gliosis and activation of microglia in the hippocampus. Pediatric Diabetes, 2016, 17, 127-139.	1.2	34
10	Blood–Brain Barrier KCa3.1 Channels. Stroke, 2015, 46, 237-244.	1.0	57
11	lschemic factor-induced increases in cerebral microvascular endothelial cell Na/H exchange activity and abundance: evidence for involvement of ERK1/2 MAP kinase. American Journal of Physiology - Cell Physiology, 2014, 306, C931-C942.	2.1	35
12	Blood–Brain Barrier Na Transporters in Ischemic Stroke. Advances in Pharmacology, 2014, 71, 113-146.	1.2	57
13	Intravenous HOE-642 Reduces Brain Edema and Na Uptake in the Rat Permanent Middle Cerebral Artery Occlusion Model of Stroke: Evidence for Participation of the Blood–Brain Barrier Na/H Exchanger. Journal of Cerebral Blood Flow and Metabolism, 2013, 33, 225-234.	2.4	62
14	lschemia-induced stimulation of cerebral microvascular endothelial cell Na-K-Cl cotransport involves p38 and JNK MAP kinases. American Journal of Physiology - Cell Physiology, 2012, 302, C505-C517.	2.1	31
15	Blood brain barrier KCa3.1 channels: evidence for a role in brain Na uptake and edema during ischemic stroke. FASEB Journal, 2012, 26, 695.13.	0.2	0
16	Engaging neuroscience to advance translational research in brain barrier biology. Nature Reviews Neuroscience, 2011, 12, 169-182.	4.9	508
17	Ischemia-induced stimulation of Na-K-Cl cotransport in cerebral microvascular endothelial cells involves AMP kinase. American Journal of Physiology - Cell Physiology, 2011, 301, C316-C326.	2.1	31
18	Cerebral microvascular endothelial cell Na/H exchange: evidence for the presence of NHE1 and NHE2 isoforms and regulation by arginine vasopressin. American Journal of Physiology - Cell Physiology, 2009, 297, C278-C289.	2.1	73

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19	Bumetanide and HOE642 administered after initiation of rat middle cerebral artery occlusion effectively reduce rat brain Na uptake and infarct. FASEB Journal, 2009, 23, 614.6.	0.2	0
20	Hypoxia effects on cell volume and ion uptake of cerebral microvascular endothelial cells. American Journal of Physiology - Cell Physiology, 2008, 294, C88-C96.	2.1	54
21	Estradiol Reduces Activity of the Blood–Brain Barrier Na–K–Cl Cotransporter and Decreases Edema Formation in Permanent Middle Cerebral Artery Occlusion. Journal of Cerebral Blood Flow and Metabolism, 2006, 26, 1234-1249.	2.4	93
22	Moderate-to-severe ischemic conditions increase activity and phosphorylation of the cerebral microvascular endothelial cell Na+-K+-Clâ^ cotransporter. American Journal of Physiology - Cell Physiology, 2005, 289, C1492-C1501.	2.1	75
23	Arginine vasopressin stimulation of cerebral microvascular endothelial cell Na-K-Cl cotransporter activity is V1 receptor and [Ca] dependent. American Journal of Physiology - Cell Physiology, 2005, 289, C283-C292.	2.1	65
24	Bumetanide Inhibition of the Blood-Brain Barrier Na-K-Cl Cotransporter Reduces Edema Formation in the Rat Middle Cerebral Artery Occlusion Model of Stroke. Journal of Cerebral Blood Flow and Metabolism, 2004, 24, 1046-1056.	2.4	213
25	The Role of the Blood-Brain Barrier Na-K-2Cl Cotransporter in Stroke. , 2004, 559, 67-75.		34
26	Flow-induced expression of endothelial Na-K-Cl cotransport: dependence on K ⁺ and Cl ^{â^'} channels. American Journal of Physiology - Cell Physiology, 2001, 280, C216-C227.	2.1	37