

Zi-Gang Zhao

List of Publications by Year in descending order

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Version: 2024-02-01

22
papers

125
citations

1478505

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docs citations

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64
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#	ARTICLE	IF	CITATIONS
1	Controlled Hemorrhage Sensitizes Angiotensin II-Elicited Hypertension through Activation of the Brain Renin-Angiotensin System Independently of Endoplasmic Reticulum Stress. <i>Oxidative Medicine and Cellular Longevity</i> , 2022, 2022, 1-13.	4.0	1
2	Postconditioning of stellate ganglion block improves intestinal barrier function by inhibiting autophagy in conscious rats following hemorrhagic shock and resuscitation. <i>Chinese Medical Journal</i> , 2022, Publish Ahead of Print, .	2.3	0
3	TLR2/TLR4-Enhanced TIPE2 Expression Is Involved in Post-Hemorrhagic Shock Mesenteric Lymph-Induced Activation of CD4+T Cells. <i>Frontiers in Immunology</i> , 2022, 13, 838618.	4.8	1
4	Estradiol-induced inhibition of endoplasmic reticulum stress normalizes splenic CD4+T lymphocytes following hemorrhagic shock. <i>Scientific Reports</i> , 2021, 11, 7508.	3.3	4
5	Therapeutic mechanisms of mesenchymal stem cells in acute respiratory distress syndrome reveal potentials for Covid-19 treatment. <i>Journal of Translational Medicine</i> , 2021, 19, 198.	4.4	15
6	Stellate Ganglion Block Improves the Proliferation and Function of Splenic CD4+T Cells Through Inhibition of Posthemorrhagic Shock Mesenteric Lymph-Mediated Autophagy. <i>Inflammation</i> , 2021, 44, 2543-2553.	3.8	6
7	Autophagy Is Involved in Stellate Ganglion Block Reversing Posthemorrhagic Shock Mesenteric Lymph-Mediated Vascular Hyporeactivity. <i>Frontiers in Physiology</i> , 2021, 12, 728191.	2.8	2
8	Mesenteric Lymph Drainage Improves Cardiac Papillary Contractility and Calcium Sensitivity in Rats with Hemorrhagic Shock. <i>Journal of Surgical Research</i> , 2021, 266, 245-253.	1.6	2
9	Engagement of Posthemorrhagic Shock Mesenteric Lymph on CD4+ T Lymphocytes In Vivo and In Vitro. <i>Journal of Surgical Research</i> , 2020, 256, 220-230.	1.6	6
10	Stellate Ganglion Blockade repairs Intestinal Mucosal Barrier through suppression of Endoplasmic Reticulum Stress following Hemorrhagic Shock. <i>International Journal of Medical Sciences</i> , 2020, 17, 2147-2154.	2.5	7
11	Blockade of Stellate Ganglion Remediate Hemorrhagic Shock-Induced Intestinal Barrier Dysfunction. <i>Journal of Surgical Research</i> , 2019, 244, 69-76.	1.6	9
12	Mesenteric lymph drainage alleviates hemorrhagic shock-induced spleen injury and inflammation. <i>Acta Cirurgica Brasileira</i> , 2019, 34, e201900903.	0.7	5
13	Nitric Oxide Regulates The Lymphatic Reactivity Following Hemorrhagic Shock Through Atp-Sensitive Potassium Channel. <i>Shock</i> , 2016, 45, 668-676.	2.1	7
14	Posthemorrhagic shock mesenteric lymph enhances monolayer permeability via F-actin and VE-cadherin. <i>Journal of Surgical Research</i> , 2016, 203, 47-55.	1.6	8
15	Inhibitory effect of post-hemorrhagic shock mesenteric lymph drainage on the HMGB1 and RAGE in mouse kidney. <i>Renal Failure</i> , 2016, 38, 131-136.	2.1	5
16	Post-hemorrhagic shock mesenteric lymph is an important contributor to cardiac dysfunction following hemorrhagic shock. <i>Acta Cirurgica Brasileira</i> , 2015, 30, 439-444.	0.7	5
17	Post-shock Mesenteric Lymph Drainage Ameliorates Cellular Immune Function in Rats Following Hemorrhagic Shock. <i>Inflammation</i> , 2015, 38, 584-594.	3.8	9
18	Exogenous normal lymph alleviates lipopolysaccharide-induced acute lung injury through lessening the adhesion molecules. <i>Acta Cirurgica Brasileira</i> , 2014, 29, 287-291.	0.7	2

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19	Effect of mesenteric lymph duct ligation on gene expression profiles of renal tissue in hemorrhagic shock rats with fluid resuscitation. <i>Renal Failure</i> , 2014, 36, 271-277.	2.1	2
20	Changes in Renal Tissue Proteome Induced by Mesenteric Lymph Drainage in Rats After Hemorrhagic Shock With Resuscitation. <i>Shock</i> , 2014, 42, 350-355.	2.1	7
21	Postshock mesenteric lymph drainage ameliorates vascular reactivity and calcium sensitivity through RhoA. <i>Journal of Surgical Research</i> , 2014, 186, 304-309.	1.6	8
22	Mesenteric Lymph Return Is an Important Contributor to Vascular Hyporeactivity and Calcium Desensitization After Hemorrhagic Shock. <i>Shock</i> , 2012, 38, 186-195.	2.1	14