Suman Dalal

List of Publications by Year in descending order

16 papers	402 citations	759233 12 h-index	940533 16 g-index
16 all docs	16 docs citations	16 times ranked	552 citing authors

#	Article	IF	Citations
1	Î²-Adrenergic receptor stimulation induces endoplasmic reticulum stress in adult cardiac myocytes: role in apoptosis. Molecular and Cellular Biochemistry, 2012, 364, 59-70.	3.1	48
2	Extracellular ubiquitin inhibits Â-AR-stimulated apoptosis in cardiac myocytes: role of GSK-3Â and mitochondrial pathways. Cardiovascular Research, 2010, 86, 20-28.	3.8	44
3	Osteopontin: At the cross-roads of myocyte survival and myocardial function. Life Sciences, 2014, 118, 1-6.	4.3	42
4	Osteopontin stimulates apoptosis in adult cardiac myocytes via the involvement of CD44 receptors, mitochondrial death pathway, and endoplasmic reticulum stress. American Journal of Physiology - Heart and Circulatory Physiology, 2014, 306, H1182-H1191.	3.2	38
5	Deficiency of Ataxia Telangiectasia Mutated Kinase Modulates Cardiac Remodeling Following Myocardial Infarction: Involvement in Fibrosis and Apoptosis. PLoS ONE, 2013, 8, e83513.	2.5	35
6	Osteopontin-stimulated apoptosis in cardiac myocytes involves oxidative stress and mitochondrial death pathway: role of a pro-apoptotic protein BIK. Molecular and Cellular Biochemistry, 2016, 418, 1-11.	3.1	30
7	Extracellular Ubiquitin Increases Expression of Angiogenic Molecules and Stimulates Angiogenesis in Cardiac Microvascular Endothelial Cells. Microcirculation, 2014, 21, 324-332.	1.8	29
8	NF2 signaling pathway plays a pro-apoptotic role in Î²-adrenergic receptor stimulated cardiac myocyte apoptosis. PLoS ONE, 2018, 13, e0196626.	2.5	25
9	Exogenous ubiquitin modulates chronic Î²-adrenergic receptor-stimulated myocardial remodeling: role in Akt activity and matrix metalloproteinase expression. American Journal of Physiology - Heart and Circulatory Physiology, 2012, 303, H1459-H1468.	3.2	23
10	Ataxia telangiectasia-mutated kinase deficiency exacerbates left ventricular dysfunction and remodeling late after myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2016, 311, H445-H452.	3.2	22
11	Exogenous ubiquitin reduces inflammatory response and preserves myocardial function 3 days post-ischemia-reperfusion injury. American Journal of Physiology - Heart and Circulatory Physiology, 2019, 316, H617-H628.	3.2	21
12	Extracellular ubiquitin modulates cardiac fibroblast phenotype and function via its interaction with CXCR4. Life Sciences, 2018, 211, 8-16.	4.3	16
13	Exogenous ubiquitin attenuates hypoxia/reoxygenation-induced cardiac myocyte apoptosis via the involvement of CXCR4 and modulation of mitochondrial homeostasis. Biochemistry and Cell Biology, 2020, 98, 492-501.	2.0	11
14	Ataxia telangiectasia mutated kinase deficiency impairs the autophagic response early during myocardial infarction. American Journal of Physiology - Heart and Circulatory Physiology, 2018, 315, H48-H57.	3.2	10
15	Deficiency of ataxia-telangiectasia mutated kinase modulates functional and biochemical parameters of the heart in response to Western-type diet. American Journal of Physiology - Heart and Circulatory Physiology, 2021, 320, H2324-H2338.	3.2	5
16	Cardioprotective Potential of Exogenous Ubiquitin. Cardiovascular Drugs and Therapy, 2021, 35, 1227-1232.	2.6	3