

Danish Sayed

List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/12192368/publications.pdf>

Version: 2024-02-01

23
papers

3,575
citations

516710

16
h-index

642732

23
g-index

24
all docs

24
docs citations

24
times ranked

5945
citing authors

#	ARTICLE	IF	CITATIONS
1	G3bp1 $\hat{\alpha}$ microRNA-1 axis regulates cardiomyocyte hypertrophy. Cellular Signalling, 2022, 91, 110245.	3.6	2
2	A protocol for transdifferentiation of human cardiac fibroblasts into endothelial cells via activation of innate immunity. STAR Protocols, 2021, 2, 100556.	1.2	2
3	HIF1 $\hat{\alpha}$ Regulates Early Metabolic Changes due to Activation of Innate Immunity in Nuclear Reprogramming. Stem Cell Reports, 2020, 14, 192-200.	4.8	22
4	Acute NelfA knockdown restricts compensatory gene expression and precipitates ventricular dysfunction during cardiac hypertrophy. Journal of Molecular and Cellular Cardiology, 2020, 142, 93-104.	1.9	3
5	Oxoglutarate dehydrogenase and acetyl-CoA acyltransferase 2 selectively associate with H2A.Z-occupied promoters and are required for histone modifications. Biochimica Et Biophysica Acta - Gene Regulatory Mechanisms, 2019, 1862, 194436.	1.9	15
6	Glucocorticoid Receptor $\hat{\alpha}$ Binding and Transcriptome Signature in Cardiomyocytes. Journal of the American Heart Association, 2019, 8, e011484.	3.7	42
7	Recruitment of RNA Polymerase II to Metabolic Gene Promoters Is Inhibited in the Failing Heart Possibly Through PGC-1 $\hat{\alpha}$ (Peroxisome Proliferator-Activated Receptor-1 $\hat{\alpha}$ Coactivator-1 $\hat{\alpha}$) Dysregulation. Circulation: Heart Failure, 2019, 12, e005529.	3.9	19
8	Transcriptional regulation mediated by H2A.Z via ANP32e-dependent inhibition of protein phosphatase 2A. Biochimica Et Biophysica Acta - Gene Regulatory Mechanisms, 2018, 1861, 481-496.	1.9	11
9	Acute Targeting of General Transcription Factor IIB Restricts Cardiac Hypertrophy via Selective Inhibition of Gene Transcription. Circulation: Heart Failure, 2015, 8, 138-148.	3.9	22
10	miR-206 Mediates YAP-Induced Cardiac Hypertrophy and Survival. Circulation Research, 2015, 117, 891-904.	4.5	133
11	GTPase Activating Protein (Sh3 Domain) Binding Protein 1 Regulates the Processing of MicroRNA-1 during Cardiac Hypertrophy. PLoS ONE, 2015, 10, e0145112.	2.5	25
12	Transcriptional Regulation Patterns Revealed by High Resolution Chromatin Immunoprecipitation during Cardiac Hypertrophy. Journal of Biological Chemistry, 2013, 288, 2546-2558.	3.4	54
13	GATA4 expression is primarily regulated via a miR-26b-dependent post-transcriptional mechanism during cardiac hypertrophy. Cardiovascular Research, 2012, 93, 645-654.	3.8	83
14	MicroRNAs in Development and Disease. Physiological Reviews, 2011, 91, 827-887.	28.8	959
15	An antagonism between the AKT and beta-adrenergic signaling pathways mediated through their reciprocal effects on miR-199a-5p. Cellular Signalling, 2010, 22, 1054-1062.	3.6	83
16	MicroRNA-21 Is a Downstream Effector of AKT That Mediates Its Antiapoptotic Effects via Suppression of Fas Ligand. Journal of Biological Chemistry, 2010, 285, 20281-20290.	3.4	282
17	AKT-ing via microRNA. Cell Cycle, 2010, 9, 3233-3237.	2.6	73
18	MicroRNAs Challenge the Status Quo of Therapeutic Targeting. Journal of Cardiovascular Translational Research, 2009, 2, 100-107.	2.4	7

#	ARTICLE	IF	CITATIONS
19	Downregulation of MiR-199a Derepresses Hypoxia-Inducible Factor-1 α and Sirtuin 1 and Recapitulates Hypoxia Preconditioning in Cardiac Myocytes. <i>Circulation Research</i> , 2009, 104, 879-886.	4.5	546
20	MicroRNA-21 Targets Sprouty2 and Promotes Cellular Outgrowths. <i>Molecular Biology of the Cell</i> , 2008, 19, 3272-3282.	2.1	354
21	MicroRNA with a MacroFunction. <i>Cell Cycle</i> , 2007, 6, 1850-1855.	2.6	33
22	MicroRNAs Play an Essential Role in the Development of Cardiac Hypertrophy. <i>Circulation Research</i> , 2007, 100, 416-424.	4.5	716
23	Histone H2A.z Is Essential for Cardiac Myocyte Hypertrophy but Opposed by Silent Information Regulator 2 β . <i>Journal of Biological Chemistry</i> , 2006, 281, 19369-19377.	3.4	89