Zhiyong Zhao

List of Publications by Year
in descending order

47 papers	1,591 citations	279487 23 h-index	301761 39 g-index
47 all docs	47 docs citations	47 times ranked	1663 citing authors

#	Article	IF	Citations
1	Rho kinases play an obligatory role in vertebrate embryonic organogenesis. Development (Cambridge), 2001, 128, 2953-2962.	1.2	198
2	Nicotine-induced embryonic malformations mediated by apoptosis from increasing intracellular calcium and oxidative stress. Birth Defects Research Part B: Developmental and Reproductive Toxicology, 2005, 74, 383-391.	1.4	97
3	Influences of Adenosine on the Fetus and Newborn. Molecular Genetics and Metabolism, 2001, 74, 160-171.	0.5	90
4	Activation of oxidative stress signaling that is implicated in apoptosis with a mouse model of diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2008, 198, 130.e1-130.e7.	0.7	85
5	Genomic Analysis of a New Mammalian Distal-less Gene: Dlx7. Genomics, 1996, 38, 314-324.	1.3	74
6	Programmed cell death in the developing heart: Regulation by BMP4 and FGF2. , 2000, 217, 388-400.		71
7	Dietary vitamin and lipid therapy rescues aberrant signaling and apoptosis and prevents hyperglycemia-induced diabetic embryopathy in rats. American Journal of Obstetrics and Gynecology, 2006, 194, 580-585.	0.7	69
8	MyoD-Dependent Induction during Myoblast Differentiation of p204, a Protein Also Inducible by Interferon. Molecular and Cellular Biology, 2000, 20, 7024-7036.	1.1	65
9	Rho-associated kinases play an essential role in cardiac morphogenesis and cardiomyocyte proliferation. Developmental Dynamics, 2003, 226, 24-32.	0.8	63
10	New Concepts in Diabetic Embryopathy. Clinics in Laboratory Medicine, 2013, 33, 207-233.	0.7	63
11	Patterning of the mammalian dentition in development and evolution. BioEssays, 1997, 19, 481-490.	1.2	58
12	Experimental Mechanisms of Diabetic Embryopathy and Strategies for Developing Therapeutic Interventions. Journal of the Society for Gynecologic Investigation, 2005, 12, 549-557.	1.9	52
13	Involvement of c-Jun N-terminal kinases activation in diabetic embryopathy. Biochemical and Biophysical Research Communications, 2007, 357, 749-754.	1.0	50
14	Characterization of the Murine A1 Adenosine Receptor Promoter, Potent Regulation by GATA-4 and Nkx2.5. Journal of Biological Chemistry, 1999, 274, 14204-14209.	1.6	45
15	Aberrant patterns of cellular communication in diabetes-induced embryopathy in rats: II, Apoptotic pathways. American Journal of Obstetrics and Gynecology, 2005, 192, 967-972.	0.7	43
16	Characterization of differential gene expression profiles in diabetic embryopathy using DNA microarray analysis. American Journal of Obstetrics and Gynecology, 2006, 195, 1075-1080.	0.7	43
17	Caspaseâ€8: a key role in the pathogenesis of diabetic embryopathy. Birth Defects Research Part B: Developmental and Reproductive Toxicology, 2009, 86, 72-77.	1.4	42
18	Rho-associated kinases play a role in endocardial cell differentiation and migration. Developmental Biology, 2004, 275, 183-191.	0.9	32

#	Article	IF	Citations
19	Amelioration of intracellular stress and reduction of neural tube defects in embryos of diabetic mice by phytochemical quercetin. Scientific Reports, 2016, 6, 21491.	1.6	30
20	Blockade of c-Jun N-terminal kinase activation abrogates hyperglycemia-induced yolk sac vasculopathy in vitro. American Journal of Obstetrics and Gynecology, 2008, 198, 321.e1-321.e7.	0.7	29
21	Reduction in Embryonic Malformations and Alleviation of Endoplasmic Reticulum Stress by Nitric Oxide Synthase Inhibition in Diabetic Embryopathy. Reproductive Sciences, 2012, 19, 823-831.	1.1	25
22	Impact of protein O-GlcNAcylation on neural tube malformation in diabetic embryopathy. Scientific Reports, 2017, 7, 11107.	1.6	25
23	Inhibition of cell proliferation in the embryonic myocardium by A1 adenosine receptor activation. Developmental Dynamics, 2001, 221, 194-200.	0.8	24
24	Cardiac malformations and alteration of TGFÎ² signaling system in diabetic embryopathy. Birth Defects Research Part B: Developmental and Reproductive Toxicology, 2010, 89, 97-105.	1.4	24
25	Modulation of nuclear factor-ÎºB signaling and reduction of neural tube defects by quercetin-3-glucoside in embryos of diabetic mice. American Journal of Obstetrics and Gynecology, 2018, 219, 197.e1-197.e8.	0.7	22
26	Elevated extracellular glucose and uncontrolled type 1 diabetes enhance NFAT5 signaling and disrupt the transverse tubular network in mouse skeletal muscle. Experimental Biology and Medicine, 2012, 237, 1068-1083.	1.1	19
27	Tissue-specific expression of GTPas RalA and RalB during embryogenesis and regulation by epithelial-mesenchymal interaction. Mechanisms of Development, 2000, 97, 201-204.	1.7	18
28	Numerous Members of the Sox Family of HMG Box-Containing Genes Are Expressed in Developing Mouse Teeth. Genomics, 1996, 37, 234-237.	1.3	17
29	Demonstration of the Essential Role of Protein Kinase C Isoforms in Hyperglycemia-Induced Embryonic Malformations. Reproductive Sciences, 2008, 15, 349-356.	1.1	16
30	Endoplasmic Reticulum Stress in Maternal Diabetesâ€Induced Cardiac Malformations During Critical Cardiogenesis Period. Birth Defects Research Part B: Developmental and Reproductive Toxicology, 2012, 95, 1-6.	1.4	16
31	Formation of neurodegenerative aggresome and death-inducing signaling complex in maternal diabetes-induced neural tube defects. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 4489-4494.	3.3	16
32	Putative tumor suppressor protein 4.1B is differentially expressed in kidney and brain via alternative promoters and 5â€² alternative splicing. Biochimica Et Biophysica Acta Gene Regulatory Mechanisms, 2004, 1680, 71-82.	2.4	15
33	Protein kinase CÎ²2 inhibition reduces hyperglycemia-induced neural tube defects through suppression of a caspase 8-triggered apoptotic pathway. American Journal of Obstetrics and Gynecology, 2011, 204, 226.e1-226.e5.	0.7	14
34	TGFÎ² and Wnt in Cardiac Outflow Tract Defects in Offspring of Diabetic Pregnancies. Birth Defects Research Part B: Developmental and Reproductive Toxicology, 2014, 101, 364-370.	1.4	11
35	The essential role of protein kinase CΊ in diabetes-induced neural tube defects. Journal of Maternal-Fetal and Neonatal Medicine, 2012, 25, 2020-2024.	0.7	10
36	Reevaluation of Antioxidative Strategies for Birth Defect Prevention in Diabetic Pregnancies. Journal of Biomolecular Research & Therapeutics, 2016, 5, .	0.2	6

#	Article	IF	Citations
37	MicroRNA Biomarkers for Early Detection of Embryonic Malformations in Pregnancy. Journal of Biomolecular Research & Therapeutics, 2014, 03, .	0.2	4
38	Identification of novel cell survival regulation in diabetic embryopathy via phospholipidomic profiling. Biochemical and Biophysical Research Communications, 2016, 470, 599-605.	1.0	4
39	Disturbed intracellular calcium homeostasis in neural tube defects in diabetic embryopathy. Biochemical and Biophysical Research Communications, 2019, 514, 960-966.	1.0	4
40	Activinâ€A in Diabetesâ€Induced Cardiac Malformations in Embryos. Birth Defects Research Part B: Developmental and Reproductive Toxicology, 2013, 98, 260-267.	1.4	2
41	Key membrane signaling intermediates (PKC and cPLA2) in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2005, 193, S24.	0.7	0
42	Characterization of differential gene expression profiles in diabetic embryopathy using cDNA microarray analysis. American Journal of Obstetrics and Gynecology, 2005, 193, S95.	0.7	0
43	44: PKCÎ²2 inhibition reduces neural tube malformations and suppresses caspase activation. American Journal of Obstetrics and Gynecology, 2011, 204, S27.	0.7	0
44	286: Protein kinase CÎ´ mediated-oxidative stress in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2011, 204, S119.	0.7	0
45	72: Oral treatment with anti-oxidant N-acetylcysteine reduces maternal diabetes-induced embryonic neural tube defects. American Journal of Obstetrics and Gynecology, 2012, 206, S46.	0.7	0
46	275: Diabetic embryopathy and excess apoptosis: the role of protein kinase C Î´. American Journal of Obstetrics and Gynecology, 2012, 206, S133.	0.7	0
47	Characterization of the Murine A1 Adenosine Receptor Promoter: Potent Regulation by GATA-4 and Nkx2.5. Pediatric Research, 1999, 45, 60A-60A.	1.1	0