Zhiyong Zhao

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Disturbed intracellular calcium homeostasis in neural tube defects in diabetic embryopathy. Biochemical and Biophysical Research Communications, 2019, 514, 960-966.	1.0	4
2	Modulation of nuclear factor-l [°] B 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
3	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
4	Impact of protein O-GlcNAcylation on neural tube malformation in diabetic embryopathy. Scientific Reports, 2017, 7, 11107.	1.6	25
5	Reevaluation of Antioxidative Strategies for Birth Defect Prevention in Diabetic Pregnancies. Journal of Biomolecular Research & Therapeutics, 2016, 5, .	0.2	6
6	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
7	Identification of novel cell survival regulation in diabetic embryopathy via phospholipidomic profiling. Biochemical and Biophysical Research Communications, 2016, 470, 599-605.	1.0	4
8	MicroRNA Biomarkers for Early Detection of Embryonic Malformations in Pregnancy. Journal of Biomolecular Research & Therapeutics, 2014, 03, .	0.2	4
9	TGFβ 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
10	New Concepts in Diabetic Embryopathy. Clinics in Laboratory Medicine, 2013, 33, 207-233.	0.7	63
11	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
12	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
13	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
14	The essential role of protein kinase Cδ in diabetes-induced neural tube defects. Journal of Maternal-Fetal and Neonatal Medicine, 2012, 25, 2020-2024.	0.7	10
15	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
16	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
17	275: Diabetic embryopathy and excess apoptosis: the role of protein kinase C δ. American Journal of Obstetrics and Gynecology, 2012, 206, S133.	0.7	0
18	44: PKCβ2 inhibition reduces neural tube malformations and suppresses caspase activation. American Journal of Obstetrics and Gynecology, 2011, 204, S27.	0.7	0

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19	286: Protein kinase Cδ mediated-oxidative stress in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2011, 204, S119.	0.7	0
20	Protein kinase Cβ2 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
21	Cardiac malformations and alteration of TCFÎ ² signaling system in diabetic embryopathy. Birth Defects Research Part B: Developmental and Reproductive Toxicology, 2010, 89, 97-105.	1.4	24
22	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
23	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
24	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
25	Demonstration of the Essential Role of Protein Kinase C Isoforms in Hyperglycemia-Induced Embryonic Malformations. Reproductive Sciences, 2008, 15, 349-356.	1.1	16
26	Involvement of c-Jun N-terminal kinases activation in diabetic embryopathy. Biochemical and Biophysical Research Communications, 2007, 357, 749-754.	1.0	50
27	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
28	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
29	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
30	Key membrane signaling intermediates (PKC and cPLA2) in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2005, 193, S24.	0.7	0
31	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
32	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
33	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
34	Putative tumor suppressor protein 4.1B is differentially expressed in kidney and brain via alternative promoters and 5′ alternative splicing. Biochimica Et Biophysica Acta Gene Regulatory Mechanisms, 2004, 1680, 71-82.	2.4	15
35	Rho-associated kinases play a role in endocardial cell differentiation and migration. Developmental Biology, 2004, 275, 183-191.	0.9	32
36	Rho-associated kinases play an essential role in cardiac morphogenesis and cardiomyocyte proliferation. Developmental Dynamics, 2003, 226, 24-32.	0.8	63

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37	Influences of Adenosine on the Fetus and Newborn. Molecular Genetics and Metabolism, 2001, 74, 160-171.	0.5	90
38	Inhibition of cell proliferation in the embryonic myocardium by A1 adenosine receptor activation. Developmental Dynamics, 2001, 221, 194-200.	0.8	24
39	Rho kinases play an obligatory role in vertebrate embryonic organogenesis. Development (Cambridge), 2001, 128, 2953-2962.	1.2	198
40	Programmed cell death in the developing heart: Regulation by BMP4 and FGF2. , 2000, 217, 388-400.		71
41	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
42	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
43	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
44	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
45	Patterning of the mammalian dentition in development and evolution. BioEssays, 1997, 19, 481-490.	1.2	58
46	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
47	Genomic Analysis of a New Mammalian Distal-less Gene: Dlx7. Genomics, 1996, 38, 314-324.	1.3	74