Peixin Yang

List of Publications by Year
in descending order

87 papers	7,846 citations	101543 36 h-index	60623 81 g-index
91 all docs	91 docs citations	91 times ranked	16727 citing authors

#	Article	IF	Citations
1	Maternal obesity increases DNA methylation and decreases RNA methylation in the human placenta. Reproductive Toxicology, 2022, 107, 90-96.	2.9	16
2	Microtentacle Formation in Ovarian Carcinoma. Cancers, 2022, 14, 800.	3.7	3
3	Epigenetics in Congenital Heart Disease. Journal of the American Heart Association, 2022, 11, e025163.	3.7	13
4	AMPK Signaling Regulates Mitophagy and Mitochondrial ATP Production in Human Trophoblast Cell Line BeWo. Frontiers in Bioscience, 2022, 27, 118.	2.1	5
5	Functional cargos of exosomes derived from Flk-1+ vascular progenitors enable neurulation and ameliorate embryonic anomalies in diabetic pregnancy. Communications Biology, 2022, 5, .	4.4	6
6	Maternal diabetes induces senescence and neural tube defects sensitive to the senomorphic rapamycin. Science Advances, 2021, 7, .	10.3	29
7	mTOR deletion in neural crest cells disrupts cardiac outflow tract remodeling and causes a spectrum of cardiac defects through the mTORC1 pathway. Developmental Biology, 2021, 477, 241-250.	2.0	2
8	Restoring BMP4 expression in vascular endothelial progenitors ameliorates maternal diabetes-induced apoptosis and neural tube defects. Cell Death and Disease, 2020, 11, 859.	6.3	7
9	Transamniotic mesenchymal stem cell therapy for neural tube defects preserves neural function through lesion-specific engraftment and regeneration. Cell Death and Disease, 2020, 11, 523.	6.3	14
10	mTOR plays a pivotal role in multiple processes of enamel organ development principally through the mTORC1 pathway and in part via regulating cytoskeleton dynamics. Developmental Biology, 2020, 467, 77-87.	2.0	4
11	Deficiency of the oxidative stressâ€"responsive kinase p70S6K1 restores autophagy and ameliorates neural tube defects in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2020, 223, 753.e1-753.e14.	1.3	13
12	Chronic-plus-binge alcohol intake induces production of proinflammatory mtDNA-enriched extracellular vesicles and steatohepatitis via ASK1/p38MAPKÎ±-dependent mechanisms. JCI Insight, 2020, 5, .	5.0	34
13	Tip60- and sirtuin 2-regulated MARCKS acetylation and phosphorylation are required for diabetic embryopathy. Nature Communications, 2019, 10, 282.	12.8	26
14	Circulating exosomes derived from transplanted progenitor cells aid the functional recovery of ischemic myocardium. Science Translational Medicine, 2019, 11, .	12.4	69
15	Preventing and Diagnosing Diabetic Complications: Epigenetics, miRNA, DNA Methylation, and Histone Modifications. , 2019, , 1347-1359.		0
16	Embryopathy as a Model for the Epigenetics Regulation of Complications in Diabetes. , 2019, , 1361-1379.		0
17	Trehalose restores functional autophagy suppressed by high glucose. Reproductive Toxicology, 2019, 85, 51-58.	2.9	21
18	Loss-of-function mutations with circadian rhythm regulator Per1/Per2 lead to premature ovarian insufficiencyâ€. Biology of Reproduction, 2019, 100, 1066-1072.	2.7	23

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19	The increased activity of a transcription factor inhibits autophagy in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2019, 220, 108.e1-108.e12.	1.3	7
20	Effect of Postmortem Interval and Years in Storage on RNA Quality of Tissue at a Repository of the NIH NeuroBioBank. Biopreservation and Biobanking, 2018, 16, 148-157.	1.0	51
21	High Glucose Inhibits Neural Stem Cell Differentiation Through Oxidative Stress and Endoplasmic Reticulum Stress. Stem Cells and Development, 2018, 27, 745-755.	2.1	38
22	Oxidative stress-induced miR-27a targets the redox gene nuclear factor erythroid 2-related factor 2 in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2018, 218, 136.e1-136.e10.	1.3	35
23	The current status and future of cardiac stem/progenitor cell therapy for congenital heart defects from diabetic pregnancy. Pediatric Research, 2018, 83, 275-282.	2.3	9
24	A novel human IL-2 mutein with minimal systemic toxicity exerts greater antitumor efficacy than wild-type IL-2. Cell Death and Disease, 2018, 9, 989.	6.3	26
25	<i>Î±</i>4 Coordinates Small Intestinal Epithelium Homeostasis by Regulating Stability of HuR. Molecular and Cellular Biology, 2018, 38, .	2.3	20
26	Cellular stress and apoptosis contribute to the pathogenesis of autism spectrum disorder. Autism Research, 2018, 11, 1076-1090.	3.8	71
27	Yolk Sac. , 2018, , 551-558.		3
28	Regenerative medicine therapy for single ventricle congenital heart disease. Translational Pediatrics, 2018, 7, 176-187.	1.2	12
29	Pregestational type 2 diabetes mellitus induces cardiacÂhypertrophy in the murine embryo through cardiacÂremodeling and fibrosis. American Journal of Obstetrics and Gynecology, 2017, 217, 216.e1-216.e13.	1.3	23
30	Protein kinase C-alpha suppresses autophagy and induces neural tube defects via miR-129-2 in diabetic pregnancy. Nature Communications, 2017, 8, 15182.	12.8	67
31	Endoplasmic Reticulum Stress-Induced CHOP Inhibits PGC-1Î± and Causes Mitochondrial Dysfunction in Diabetic Embryopathy. Toxicological Sciences, 2017, 158, 275-285.	3.1	34
32	A step-wise approach for analysis of the mouse embryonic heart using 17.6 Tesla MRI. Magnetic Resonance Imaging, 2017, 35, 46-53.	1.8	6
33	Nuclear export of misfolded SOD1 mediated by a normally buried NES-like sequence reduces proteotoxicity in the nucleus. ELife, 2017, 6, .	6.0	32
34	Preventing and Diagnosing Diabetic Complications: Epigenetics, miRNA, DNA Methylation, and Histone Modifications. , 2017, , 1-12.		0
35	Embryopathy as a Model for the Epigenetics Regulation of Complications in Diabetes. , 2017, , 1-19.		0
36	High Glucoseâ€“Repressed CITED2 Expression Through miR-200b Triggers the Unfolded Protein Response and Endoplasmic Reticulum Stress. Diabetes, 2016, 65, 149-163.	0.6	37

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37	High glucoseâ€induced oxidative stress represses sirtuin deacetylase expression and increases histone acetylation leading to neural tube defects. Journal of Neurochemistry, 2016, 137, 371-383.	3.9	73
38	High glucose suppresses embryonic stem cell differentiation into cardiomyocytes. Stem Cell Research and Therapy, 2016, 7, 187.	5.5	22
39	Effect of Two Lipoprotein (a)-Associated Genetic Variants on Plasminogen Levels and Fibrinolysis. G3: Genes, Genomes, Genetics, 2016, 6, 3525-3532.	1.8	7
40	Type 2 diabetes mellitus induces congenital heart defects in murine embryos by increasing oxidative stress, endoplasmic reticulum stress, and apoptosis. American Journal of Obstetrics and Gynecology, 2016, 215, 366.e1-366.e10.	1.3	73
41	Superoxide dismutase 2 overexpression alleviates maternal diabetes-induced neural tube defects, restores mitochondrial function and suppresses cellular stress in diabetic embryopathy. Free Radical Biology and Medicine, 2016, 96, 234-244.	2.9	34
42	microRNA expression profiling and functional annotation analysis of their targets modulated by oxidative stress during embryonic heart development in diabetic mice. Reproductive Toxicology, 2016, 65, 365-374.	2.9	29
43	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	9.1	4,701
44	The green tea polyphenol EGCG alleviates maternal diabetesâ€“induced neural tube defects by inhibiting DNA hypermethylation. American Journal of Obstetrics and Gynecology, 2016, 215, 368.e1-368.e10.	1.3	48
45	The Nrf2 Activator Vinylsulfone Reduces High Glucose-Induced Neural Tube Defects by Suppressing Cellular Stress and Apoptosis. Reproductive Sciences, 2016, 23, 993-1000.	2.5	21
46	High glucose suppresses embryonic stem cell differentiation into neural lineage cells. Biochemical and Biophysical Research Communications, 2016, 472, 306-312.	2.1	19
47	MiR-17 Downregulation by High Glucose Stabilizes Thioredoxin-Interacting Protein and Removes Thioredoxin Inhibition on ASK1 Leading to Apoptosis. Toxicological Sciences, 2016, 150, 84-96.	3.1	52
48	New development of the yolk sac theory in diabetic embryopathy: molecular mechanism and link to structural birth defects. American Journal of Obstetrics and Gynecology, 2016, 214, 192-202.	1.3	42
49	The Hippo/ <scp>YAP</scp> pathway interacts with <scp>EGFR</scp> signaling and <scp>HPV</scp> oncoproteins to regulate cervical cancer progression. EMBO Molecular Medicine, 2015, 7, 1426-1449.	6.9	221
50	Birth defects in pregestational diabetes: Defect range, glycemic threshold and pathogenesis. World Journal of Diabetes, 2015, 6, 481.	3.5	117
51	Transgenic Expression of miR-222 Disrupts Intestinal Epithelial Regeneration by Targeting Multiple Genes Including Frizzled-7. Molecular Medicine, 2015, 21, 676-687.	4.4	22
52	Identification of ERAD components essential for dislocation of the null Hong Kong variant of Î±-1-antitrypsin (NHK). Biochemical and Biophysical Research Communications, 2015, 458, 424-428.	2.1	18
53	Oxidative stress is responsible for maternal diabetes-impaired transforming growth factor beta signaling in the developing mouse heart. American Journal of Obstetrics and Gynecology, 2015, 212, 650.e1-650.e11.	1.3	39
54	Curcumin ameliorates high glucose-induced neural tube defects by suppressing cellular stress and apoptosis. American Journal of Obstetrics and Gynecology, 2015, 212, 802.e1-802.e8.	1.3	59

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55	Cellular Stress, Excessive Apoptosis, and the Effect of Metformin in a Mouse Model of Type 2 Diabetic Embryopathy. Diabetes, 2015, 64, 2526-2536.	0.6	64
56	Superoxide Dismutase 1 In Vivo Ameliorates Maternal Diabetes Mellitusâ€“Induced Apoptosis and Heart Defects Through Restoration of Impaired Wnt Signaling. Circulation: Cardiovascular Genetics, 2015, 8, 665-676.	5.1	54
57	Advances in revealing the molecular targets downstream of oxidative stressâ€“induced proapoptotic kinase signaling in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2015, 213, 125-134.	1.3	51
58	ASK1 mediates the teratogenicity of diabetes in the developing heart by inducing ER stress and inhibiting critical factors essential for cardiac development. American Journal of Physiology - Endocrinology and Metabolism, 2015, 309, E487-E499.	3.5	41
59	Punicalagin exerts protective effect against high glucose-induced cellular stress and neural tube defects. Biochemical and Biophysical Research Communications, 2015, 467, 179-184.	2.1	39
60	Maternal diabetes triggers DNA damage and DNA damage response in neurulation stage embryos through oxidative stress. Biochemical and Biophysical Research Communications, 2015, 467, 407-412.	2.1	25
61	Dominant negative FADD dissipates the proapoptotic signalosome of the unfolded protein response in diabetic embryopathy. American Journal of Physiology - Endocrinology and Metabolism, 2015, 309, E861-E873.	3.5	17
62	The miR-322-TRAF3 Circuit Mediates the Pro-apoptotic Effect of High Glucose on Neural Stem Cells. Toxicological Sciences, 2015, 144, 186-196.	3.1	61
63	<i>Ask1</i> Gene Deletion Blocks Maternal Diabetesâ€“Induced Endoplasmic Reticulum Stress in the Developing Embryo by Disrupting the Unfolded Protein Response Signalosome. Diabetes, 2015, 64, 973-988.	0.6	60
64	Decoding the oxidative stress hypothesis in diabetic embryopathy through proapoptotic kinase signaling. American Journal of Obstetrics and Gynecology, 2015, 212, 569-579.	1.3	72
65	<i>Jnk2</i> deletion disrupts intestinal mucosal homeostasis and maturation by differentially modulating RNA-binding proteins HuR and CUGBP1. American Journal of Physiology - Cell Physiology, 2014, 306, C1167-C1175.	4.6	9
66	Cardiac myocyte proliferation: Not as simple as counting sheep. Journal of Molecular and Cellular Cardiology, 2014, 74, 125-126.	1.9	2
67	Trehalose prevents neural tube defects by correcting maternal diabetes-suppressed autophagy and neurogenesis. American Journal of Physiology - Endocrinology and Metabolism, 2013, 305, E667-E678.	3.5	71
68	Superoxide dismutase 1 overexpression in mice abolishes maternal diabetesâ€“induced endoplasmic reticulum stress inÂdiabetic embryopathy. American Journal of Obstetrics and Gynecology, 2013, 209, 345.e1-345.e7.	1.3	48
69	Maternal Hyperglycemia Activates an ASK1â€“FoxO3aâ€“Caspase 8 Pathway That Leads to Embryonic Neural Tube Defects. Science Signaling, 2013, 6, ra74.	3.6	81
70	c-Jun NH2-Terminal Kinase 1/2 and Endoplasmic Reticulum Stress as Interdependent and Reciprocal Causation in Diabetic Embryopathy. Diabetes, 2013, 62, 599-608.	0.6	72
71	Oxidative Stressâ€“Induced JNK1/2 Activation Triggers Proapoptotic Signaling and Apoptosis That Leads to Diabetic Embryopathy. Diabetes, 2012, 61, 2084-2092.	0.6	70
72	SOD1 suppresses maternal hyperglycemia-increased iNOS expression and consequent nitrosative stress in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2012, 206, 448.e1-448.e7.	1.3	36

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73	Role of HIF-1Î± in maternal hyperglycemia-induced embryonic vasculopathy. American Journal of Obstetrics and Gynecology, 2011, 204, 332.e1-332.e7.	1.3	26
74	SOD1 overexpression in vivo blocks hyperglycemia-induced specific PKC isoforms: substrate activation and consequent lipid peroxidation in diabetic embryopathy. American Journal of Obstetrics and Gynecology, 2011, 205, 84.e1-84.e6.	1.3	47
75	Epigallocatechin-3-gallate ameliorates hyperglycemia-induced embryonic vasculopathy and malformation by inhibition of Foxo3a activation. American Journal of Obstetrics and Gynecology, 2010, 203, 75.e1-75.e6.	1.3	44
76	Hyperglycemia induces inducible nitric oxide synthase gene expression and consequent nitrosative stress via c-Jun N-terminal kinase activation. American Journal of Obstetrics and Gynecology, 2010, 203, 185.e5-185.e11.	1.3	54
77	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
78	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.	1.3	29
79	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.	1.3	85
80	Involvement of c-Jun N-terminal kinases activation in diabetic embryopathy. Biochemical and Biophysical Research Communications, 2007, 357, 749-754.	2.1	50
81	A Novel Mechanism of FSH Regulation of DNA Synthesis in the Granulosa Cells of Hamster Preantral Follicles: Involvement of a Protein Kinase C-Mediated MAP Kinase 3/1 Self-Activation Loop1. Biology of Reproduction, 2006, 75, 149-157.	2.7	25
82	Transforming Growth Factor B1 Stimulated DNA Synthesis in the Granulosa Cells of Preantral Follicles: Negative Interaction with Epidermal Growth Factor1. Biology of Reproduction, 2006, 75, 140-148.	2.7	11
83	Developmental Expression of Estrogen Receptor (ER) Î± and ERÎ² in the Hamster Ovary: Regulation by Follicle-Stimulating Hormone. Endocrinology, 2004, 145, 5757-5766.	2.8	30
84	Follicle Stimulating Hormone-Induced DNA Synthesis in the Granulosa Cells of Hamster Preantral Follicles Involves Activation of Cyclin-Dependent Kinase-4 Rather Than Cyclin D2 Synthesis1. Biology of Reproduction, 2004, 70, 509-517.	2.7	19
85	Expression of ER-Î± and ER-Î² in the Hamster Ovary: Differential Regulation by Gonadotropins and Ovarian Steroid Hormones. Endocrinology, 2002, 143, 2385-2398.	2.8	59
86	Mining estrogen microarray data: an approach using contrast data analysis. , 0, , .		0
87	Endoplasmic reticulum stress and IRE-1 signaling cause apoptosis in colon cancer cells in response to andrographolide treatment. Oncotarget, 0, 7, 41432-41444.	1.8	63