Shaoping Zhang

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

36
papers1,258
citations23
h-index35
g-index36
ext. papers1,359
ext. citations5.5
avg, IF3.64
L-index

#	Paper	IF	Citations
36	Restoration of myocellular copper-trafficking proteins and mitochondrial copper enzymes repairs cardiac function in rats with diabetes-evoked heart failure. <i>Metallomics</i> , 2020 , 12, 259-272	4.5	5
35	Altered metabolic gene expression in the brain of a triprolyl-human amylin transgenic mouse model of type 2 diabetes. <i>Scientific Reports</i> , 2019 , 9, 14588	4.9	1
34	Elevation of brain glucose and polyol-pathway intermediates with accompanying brain-copper deficiency in patients with Alzheimer's disease: metabolic basis for dementia. <i>Scientific Reports</i> , 2016 , 6, 27524	4.9	46
33	Diabetes-induced alterations in tissue collagen and carboxymethyllysine in rat kidneys: Association with increased collagen-degrading proteinases and amelioration by Cu(II)-selective chelation. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2015 , 1852, 1610-8	6.9	14
32	Low-dose copper infusion into the coronary circulation induces acute heart failure in diabetic rats: New mechanism of heart disease. <i>Biochemical Pharmacology</i> , 2015 , 97, 62-76	6	3
31	Essential roles of insulin, AMPK signaling and lysyl and prolyl hydroxylases in the biosynthesis and multimerization of adiponectin. <i>Molecular and Cellular Endocrinology</i> , 2015 , 399, 164-77	4.4	11
30	11EHSD1 modulates LPS-induced innate immune responses in adipocytes by altering expression of PTEN. <i>Molecular Endocrinology</i> , 2015 , 29, 558-70		5
29	The pathogenic mechanism of diabetes varies with the degree of overexpression and oligomerization of human amylin in the pancreatic islet Itells. <i>FASEB Journal</i> , 2014 , 28, 5083-96	0.9	35
28	Diabetic cardiomyopathy is associated with defective myocellular copper regulation and both defects are rectified by divalent copper chelation. <i>Cardiovascular Diabetology</i> , 2014 , 13, 100	8.7	42
27	Protection of the heart by treatment with a divalent-copper-selective chelator reveals a novel mechanism underlying cardiomyopathy in diabetic rats. <i>Cardiovascular Diabetology</i> , 2013 , 12, 123	8.7	28
26	Mice lacking the neuropeptide alpha-calcitonin gene-related peptide are protected against diet-induced obesity. <i>Endocrinology</i> , 2010 , 151, 4257-69	4.8	56
25	Tetracycline treatment retards the onset and slows the progression of diabetes in human amylin/islet amyloid polypeptide transgenic mice. <i>Diabetes</i> , 2010 , 59, 161-71	0.9	44
24	The chaperone proteins HSP70, HSP40/DnaJ and GRP78/BiP suppress misfolding and formation of Bheet-containing aggregates by human amylin: a potential role for defective chaperone biology in Type 2 diabetes. <i>Biochemical Journal</i> , 2010 , 432, 113-21	3.8	37
23	Copper(II)-selective chelation improves function and antioxidant defences in cardiovascular tissues of rats as a model of diabetes: comparisons between triethylenetetramine and three less copper-selective transition-metal-targeted treatments. <i>Diabetologia</i> , 2010 , 53, 1217-26	10.3	30
22	Is type 2 diabetes an amyloidosis and does it really matter (to patients)?. <i>Diabetologia</i> , 2010 , 53, 1011-6	10.3	23
21	Illuminating the molecular basis of diabetic arteriopathy: a proteomic comparison of aortic tissue from diabetic and healthy rats. <i>Proteomics</i> , 2010 , 10, 3367-78	4.8	8
20	Quantitative proteomic profiling identifies new renal targets of copper(II)-selective chelation in the reversal of diabetic nephropathy in rats. <i>Proteomics</i> , 2009 , 9, 4309-20	4.8	30

(1996-2008)

Spontaneous diabetes in hemizygous human amylin transgenic mice that developed neither islet amyloid nor peripheral insulin resistance. <i>Diabetes</i> , 2008 , 57, 2737-44 A copper(II)-selective chelator ameliorates diabetes-evoked renal fibrosis and albuminuria, and suppresses pathogenic TGF-beta activation in the kidneys of rats used as a model of diabetes. <i>Diabetologia</i> , 2008 , 51, 1741-51 Molecular changes evoked by triethylenetetramine treatment in the extracellular matrix of the	0.9	24
suppresses pathogenic TGF-beta activation in the kidneys of rats used as a model of diabetes. Diabetologia, 2008 , 51, 1741-51	10.3	
Molecular changes evoked by triethylenetetramine treatment in the extracellular matrix of the		44
heart and aorta in diabetic rats. <i>Molecular Pharmacology</i> , 2006 , 70, 2045-51	4.3	35
The aggregation potential of human amylin determines its cytotoxicity towards islet beta-cells. <i>FEBS Journal</i> , 2006 , 273, 3614-24	5.7	185
Activation of activating transcription factor 2 by p38 MAP kinase during apoptosis induced by human amylin in cultured pancreatic beta-cells. <i>FEBS Journal</i> , 2006 , 273, 3779-91	5.7	31
Thiol reducing compounds prevent human amylin-evoked cytotoxicity. FEBS Journal, 2005, 272, 4949-59	9 5.7	43
Fibrillogenic amylin evokes islet beta-cell apoptosis through linked activation of a caspase cascade and JNK1. <i>Journal of Biological Chemistry</i> , 2003 , 278, 52810-9	5.4	84
A cytosolic tRNA with an unmodified adenosine in the wobble position reads a codon ending with the non-complementary nucleoside cytidine. <i>Journal of Molecular Biology</i> , 2002 , 317, 481-92	6.5	22
Increased expression and activation of c-Jun contributes to human amylin-induced apoptosis in pancreatic islet beta-cells. <i>Journal of Molecular Biology</i> , 2002 , 324, 271-85	6.5	56
Ultrastructural evidence that apoptosis is the mechanism by which human amylin evokes death in RINm5F pancreatic islet beta-cells. <i>Cell Biology International</i> , 2001 , 25, 339-50	4.5	62
Induction of apoptosis by human amylin in RINm5F islet beta-cells is associated with enhanced expression of p53 and p21WAF1/CIP1. <i>FEBS Letters</i> , 1999 , 455, 315-20	3.8	51
Interaction between a mutant release factor one and P-site peptidyl-tRNA is influenced by the identity of the two bases downstream of the stop codon UAG. <i>FEBS Letters</i> , 1999 , 455, 355-8	3.8	9
Role of Ca2+ in apoptosis evoked by human amylin in pancreatic islet £ells. <i>Biochemical Journal</i> , 1999 , 343, 53-61	3.8	42
Role of Ca2+ in apoptosis evoked by human amylin in pancreatic islet Etells. <i>Biochemical Journal</i> , 1999 , 343, 53	3.8	18
Functional interaction between tRNA2Gly2 at the ribosomal P-site and RF1 during termination at UAG. <i>Journal of Molecular Biology</i> , 1998 , 284, 1243-6	6.5	10
The efficiency of a cis-cleaving ribozyme in an mRNA coding region is influenced by the translating ribosome in vivo. <i>Nucleic Acids Research</i> , 1997 , 25, 4301-6	20.1	4
Functional interaction between release factor one and P-site peptidyl-tRNA on the ribosome. Journal of Molecular Biology, 1996, 261, 98-107	6.5	38
	Molecular changes evoked by triethylenetetramine treatment in the extracellular matrix of the heart and aorta in diabetic rats. <i>Molecular Pharmacology</i> , 2006, 70, 2045-51 The aggregation potential of human amylin determines its cytotoxicity towards islet beta-cells. <i>FEBS Journal</i> , 2006, 273, 3614-24 Activation of activating transcription factor 2 by p38 MAP kinase during apoptosis induced by human amylin in cultured pancreatic beta-cells. <i>FEBS Journal</i> , 2006, 273, 3779-91 Thiol reducing compounds prevent human amylin-evoked cytotoxicity. <i>FEBS Journal</i> , 2005, 272, 4949-59 Fibrillogenic amylin evokes islet beta-cell apoptosis through linked activation of a caspase cascade and JNK1. <i>Journal of Biological Chemistry</i> , 2003, 278, 52810-9 A cytosolic tRNA with an unmodified adenosine in the wobble position reads a codon ending with the non-complementary nucleoside cytidine. <i>Journal of Molecular Biology</i> , 2002, 317, 481-92 Increased expression and activation of c-Jun contributes to human amylin-induced apoptosis in pancreatic islet beta-cells. <i>Journal of Molecular Biology</i> , 2002, 324, 271-85 Ultrastructural evidence that apoptosis is the mechanism by which human amylin evokes death in RINmSF pancreatic islet beta-cells. <i>Cell Biology International</i> , 2001, 25, 339-50 Induction of apoptosis by human amylin in RINmSF islet beta-cells is associated with enhanced expression of p53 and p21WAF1/CIP1. <i>FEBS Letters</i> , 1999, 455, 315-20 Interaction between a mutant release factor one and P-site peptidyl-tRNA is influenced by the identity of the two bases downstream of the stop codon UAG. <i>FEBS Letters</i> , 1999, 455, 355-8 Role of Ca2+ in apoptosis evoked by human amylin in pancreatic islet Eells. <i>Biochemical Journal</i> , 1999, 343, 53-61 Role of Ca2+ in apoptosis evoked by human amylin in pancreatic islet Eells. <i>Biochemical Journal</i> , 1999, 343, 53-61 Functional interaction between tRNA2Gly2 at the ribosomal P-site and RF1 during termination at UAG. <i>Journal of Molecular Biology</i> , 1998, 284, 1243-6 The efficiency	Molecular changes evoked by triethylenetetramine treatment in the extracellular matrix of the heart and aorta in diabetic rats. <i>Molecular Pharmacology</i> , 2006, 70, 2045-51 The aggregation potential of human amylin determines its cytotoxicity towards islet beta-cells. <i>FEBS Journal</i> , 2006, 273, 3614-24 Activation of activating transcription factor 2 by p38 MAP kinase during apoptosis induced by human amylin in cultured pancreatic beta-cells. <i>FEBS Journal</i> , 2006, 273, 3779-91 57 Thiol reducing compounds prevent human amylin-evoked cytotoxicity. <i>FEBS Journal</i> , 2005, 272, 4949-595,7 Fibrillogenic amylin evokes islet beta-cell apoptosis through linked activation of a caspase cascade and JNK1. <i>Journal of Biological Chemistry</i> , 2003, 278, 52810-9 A cytosolic tRNA with an unmodified adenosine in the wobble position reads a codon ending with the non-complementary nucleoside cytidine. <i>Journal of Molecular Biology</i> , 2002, 317, 481-92 Increased expression and activation of c-Jun contributes to human amylin-induced apoptosis in pancreatic islet beta-cells. <i>Journal of Molecular Biology</i> , 2002, 324, 271-85 Outrastructural evidence that apoptosis is the mechanism by which human amylin evokes death in RINmSF pancreatic islet beta-cells. <i>Cell Biology International</i> , 2001, 25, 339-50 4.5 Induction of apoptosis by human amylin in RINmSF islet beta-cells is associated with enhanced expression of p53 and p21WAF1/CIP1. <i>FEBS Letters</i> , 1999, 455, 315-20 3.8 Interaction between a mutant release factor one and P-site peptidyl-tRNA is influenced by the identity of the two bases downstream of the stop codon UAG. <i>FEBS Letters</i> , 1999, 455, 355-8 Role of Ca2+ in apoptosis evoked by human amylin in pancreatic islet itels. <i>Biochemical Journal</i> , 1999, 343, 53-61 Role of Ca2+ in apoptosis evoked by human amylin in pancreatic islet itels. <i>Biochemical Journal</i> , 1999, 343, 53-61 Functional interaction between tRNA2Gly2 at the ribosomal P-site and RF1 during termination at UAG. <i>Journal of Molecular Biology</i> , 1998, 284, 1243-

Genetic implication for an interaction between release factor one and ribosomal protein L7/L12 in vivo. *Journal of Molecular Biology*, **1994**, 242, 614-8

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