

Janice Y Chou

List of Publications by Year in descending order

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Version: 2024-02-01

56
papers

2,284
citations

257450

24
h-index

223800

46
g-index

56
all docs

56
docs citations

56
times ranked

2081
citing authors

#	ARTICLE	IF	CITATIONS
1	Correction of metabolic abnormalities in a mouse model of glycogen storage disease type Ia by CRISPR/Cas9-based gene editing. <i>Molecular Therapy</i> , 2021, 29, 1602-1610.	8.2	15
2	Activation of tumor-promoting pathways implicated in hepatocellular adenoma/carcinoma, a long-term complication of glycogen storage disease type Ia. <i>Biochemical and Biophysical Research Communications</i> , 2020, 522, 1-7.	2.1	11
3	Gene therapy using a novel G6PC-S298C variant enhances the long-term efficacy for treating glycogen storage disease type Ia. <i>Biochemical and Biophysical Research Communications</i> , 2020, 527, 824-830.	2.1	8
4	The signaling pathways implicated in impairment of hepatic autophagy in glycogen storage disease type Ia. <i>Human Molecular Genetics</i> , 2020, 29, 834-844.	2.9	14
5	An evolutionary approach to optimizing glucose-6-phosphatase enzymatic activity for gene therapy of glycogen storage disease type Ia. <i>Journal of Inherited Metabolic Disease</i> , 2019, 42, 470-479.	3.6	9
6	Gene therapy prevents hepatic tumor initiation in murine glycogen storage disease type Ia at the tumor-developing stage. <i>Journal of Inherited Metabolic Disease</i> , 2019, 42, 459-469.	3.6	9
7	Molecular biology and gene therapy for glycogen storage disease type Ib. <i>Journal of Inherited Metabolic Disease</i> , 2018, 41, 1007-1014.	3.6	23
8	Hepatic glucose-6-phosphatase deficiency leads to metabolic reprogramming in glycogen storage disease type Ia. <i>Biochemical and Biophysical Research Communications</i> , 2018, 498, 925-931.	2.1	21
9	Response letter. <i>Journal of Inherited Metabolic Disease</i> , 2018, 41, 915-915.	3.6	1
10	Sirtuin signaling controls mitochondrial function in glycogen storage disease type Ia. <i>Journal of Inherited Metabolic Disease</i> , 2018, 41, 997-1006.	3.6	21
11	Glycogen storage disease type Ia mice with less than 2% of normal hepatic glucose-6-phosphatase activity restored are at risk of developing hepatic tumors. <i>Molecular Genetics and Metabolism</i> , 2017, 120, 229-234.	1.1	26
12	Downregulation of pathways implicated in liver inflammation and tumorigenesis of glycogen storage disease type Ia mice receiving gene therapy. <i>Human Molecular Genetics</i> , 2017, 26, 1890-1899.	2.9	21
13	Liver-directed gene therapy for murine glycogen storage disease type Ib. <i>Human Molecular Genetics</i> , 2017, 26, 4395-4405.	2.9	15
14	Glycogen storage disease type Ib neutrophils exhibit impaired cell adhesion and migration. <i>Biochemical and Biophysical Research Communications</i> , 2017, 482, 569-574.	2.1	20
15	Recent development and gene therapy for glycogen storage disease type Ia. <i>Liver Research</i> , 2017, 1, 174-180.	1.4	12
16	Downregulation of SIRT1 signaling underlies hepatic autophagy impairment in glycogen storage disease type Ia. <i>PLoS Genetics</i> , 2017, 13, e1006819.	3.5	53
17	Minimal hepatic glucose-6-phosphatase activity required to sustain survival and prevent hepatocellular adenoma formation in murine glycogen storage disease type Ia. <i>Molecular Genetics and Metabolism Reports</i> , 2015, 3, 28-32.	1.1	14
18	Mice expressing reduced levels of hepatic glucose-6-phosphatase activity do not develop age-related insulin resistance or obesity. <i>Human Molecular Genetics</i> , 2015, 24, 5115-5125.	2.9	16

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19	Type I glycogen storage diseases: disorders of the glucose-6-phosphatase/glucose-6-phosphate transporter complexes. <i>Journal of Inherited Metabolic Disease</i> , 2015, 38, 511-519.	3.6	90
20	Molecular mechanisms of neutrophil dysfunction in glycogen storage disease type Ib. <i>Blood</i> , 2014, 123, 2843-2853.	1.4	99
21	The SLC37 Family of Sugar-Phosphate/Phosphate Exchangers. <i>Current Topics in Membranes</i> , 2014, 73, 357-382.	0.9	47
22	Development of hepatocellular adenomas and carcinomas in mice with liver-specific G6Pase-1± deficiency. <i>DMM Disease Models and Mechanisms</i> , 2014, 7, 1083-1091.	2.4	20
23	The SLC37 family of phosphate-linked sugar phosphate antiporters. <i>Molecular Aspects of Medicine</i> , 2013, 34, 601-611.	6.4	26
24	The upstream enhancer elements of the G6PC promoter are critical for optimal G6PC expression in murine glycogen storage disease type Ia. <i>Molecular Genetics and Metabolism</i> , 2013, 110, 275-280.	1.1	21
25	Glucose-6-phosphatase-1±, implicated in a congenital neutropenia syndrome, is essential for macrophage energy homeostasis and functionality. <i>Blood</i> , 2012, 119, 4047-4055.	1.4	52
26	Prevention of hepatocellular adenoma and correction of metabolic abnormalities in murine glycogen storage disease type Ia by gene therapy. <i>Hepatology</i> , 2012, 56, 1719-1729.	7.3	62
27	Treatment of newborn G6pc mice with bone marrow-derived myelomonocytes induces liver repair. <i>Journal of Hepatology</i> , 2011, 55, 1263-1271.	3.7	8
28	G-CSF improves murine G6PC3-deficient neutrophil function by modulating apoptosis and energy homeostasis. <i>Blood</i> , 2011, 117, 3881-3892.	1.4	42
29	Recombinant AAV-directed gene therapy for type I glycogen storage diseases. <i>Expert Opinion on Biological Therapy</i> , 2011, 11, 1011-1024.	3.1	21
30	Glycogen Storage Disease Type Ia in Canines: A Model for Human Metabolic and Genetic Liver Disease. <i>Journal of Biomedicine and Biotechnology</i> , 2011, 2011, 1-9.	3.0	10
31	SLC37A1 and SLC37A2 Are Phosphate-Linked, Glucose-6-Phosphate Antiporters. <i>PLoS ONE</i> , 2011, 6, e23157.	2.5	59
32	Neutropenia in type Ib glycogen storage disease. <i>Current Opinion in Hematology</i> , 2010, 17, 36-42.	2.5	75
33	Lack of glucose recycling between endoplasmic reticulum and cytoplasm underlies cellular dysfunction in glucose-6-phosphatase-1± deficient neutrophils in a congenital neutropenia syndrome. <i>Blood</i> , 2010, 116, 2783-2792.	1.4	81
34	Severe congenital neutropenia resulting from G6PC3 deficiency with increased neutrophil CXCR4 expression and myelokathexis. <i>Blood</i> , 2010, 116, 2793-2802.	1.4	78
35	Oxidative stress mediates nephropathy in type Ia glycogen storage disease. <i>Laboratory Investigation</i> , 2010, 90, 620-629.	3.7	28
36	Adeno-Associated Virus-Mediated Correction of a Canine Model of Glycogen Storage Disease Type Ia. <i>Human Gene Therapy</i> , 2010, 21, 903-910.	2.7	52

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37	Neonatal Gene Therapy of Glycogen Storage Disease Type Ia Using a Feline Immunodeficiency Virus-based Vector. <i>Molecular Therapy</i> , 2010, 18, 1592-1598.	8.2	23
38	Complete Normalization of Hepatic G6PC Deficiency in Murine Glycogen Storage Disease Type Ia Using Gene Therapy. <i>Molecular Therapy</i> , 2010, 18, 1076-1084.	8.2	75
39	Glycogen storage disease type I and G6Pase-1 ² deficiency: etiology and therapy. <i>Nature Reviews Endocrinology</i> , 2010, 6, 676-688.	9.6	176
40	Renal Disease in Type I Glycogen Storage Disease. , 2009, , 693-708.		2
41	Generation of mice with a conditional allele for G6pc. <i>Genesis</i> , 2009, 47, 590-594.	1.6	15
42	Structure-function study of the glucose-6-phosphate transporter, an eukaryotic antiporter deficient in glycogen storage disease type Ib. <i>Molecular Genetics and Metabolism</i> , 2009, 96, 32-37.	1.1	21
43	Normoglycemia alone is insufficient to prevent long-term complications of hepatocellular adenoma in glycogen storage disease type Ib mice. <i>Journal of Hepatology</i> , 2009, 51, 909-917.	3.7	25
44	Mutations in the glucose-6-phosphatase-1 ² (G6PC) gene that cause type Ia glycogen storage disease. <i>Human Mutation</i> , 2008, 29, 921-930.	2.5	124
45	Necrotic foci, elevated chemokines and infiltrating neutrophils in the liver of glycogen storage disease type Ia. <i>Journal of Hepatology</i> , 2008, 48, 479-485.	3.7	26
46	Functional analysis of mutations in the glucose-6-phosphate transporter that cause glycogen storage disease type Ib. <i>Molecular Genetics and Metabolism</i> , 2008, 95, 220-223.	1.1	13
47	The glucose-6-phosphate transporter is a phosphate-linked antiporter deficient in glycogen storage disease type Ib and Ic. <i>FASEB Journal</i> , 2008, 22, 2206-2213.	0.5	52
48	Neutrophil stress and apoptosis underlie myeloid dysfunction in glycogen storage disease type Ib. <i>Blood</i> , 2008, 111, 5704-5711.	1.4	80
49	Gene Therapy for Type I Glycogen Storage Diseases. <i>Current Gene Therapy</i> , 2007, 7, 79-88.	2.0	27
50	Neutrophilia and elevated serum cytokines are implicated in glycogen storage disease type Ia. <i>FEBS Letters</i> , 2007, 581, 3833-3838.	2.8	19
51	Impaired neutrophil activity and increased susceptibility to bacterial infection in mice lacking glucose-6-phosphatase-1 ² . <i>Journal of Clinical Investigation</i> , 2007, 117, 784-793.	8.2	105
52	Increased scavenger receptor class B type I-mediated cellular cholesterol efflux and antioxidant capacity in the sera of glycogen storage disease type Ia patients. <i>Molecular Genetics and Metabolism</i> , 2006, 89, 233-238.	1.1	11
53	Glycogen storage disease type Ia in Argentina: two novel glucose-6-phosphatase mutations affecting protein stability. <i>Molecular Genetics and Metabolism</i> , 2004, 83, 276-279.	1.1	11
54	Type I Glycogen Storage Diseases: Disorders of the Glucose-6- Phosphatase Complex. <i>Current Molecular Medicine</i> , 2002, 2, 121-143.	1.3	254

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55	Human variant glucose-6-phosphate transporter is active in microsomal transport. Human Genetics, 2000, 107, 526-529.	3.8	31
56	Two New Mutations in the Glucose-6-Phosphatase Gene Cause Glycogen Storage Disease in Hungarian Patients. European Journal of Human Genetics, 1997, 5, 191-195.	2.8	14