Katarzyna A PirÃ³g

List of Publications by Year
in descending order

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Version: 2024-02-01

18	421	840776 11	888059 17
papers	citations	h-index	g-index

18	18	18	640
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	microRNA-seq of cartilage reveals an overabundance of miR-140-3p which contains functional isomiRs. Rna, 2020, 26, 1575-1588.	3.5	17
2	<scp>CRELD2</scp> Is a Novel <scp>LRP1</scp> Chaperone That Regulates Noncanonical <scp>WNT</scp> Signaling in Skeletal Development. Journal of Bone and Mineral Research, 2020, 35, 1452-1469.	2.8	12
3	New developments in chondrocyte ER-stress andÂrelated diseases. F1000Research, 2020, 9, 290.	1.6	17
4	XBP1 signalling is essential for alleviating mutant protein aggregation in ER-stress related skeletal disease. PLoS Genetics, 2019, 15, e1008215.	3.5	16
5	Calcium activated nucleotidase 1 (CANT1) is critical for glycosaminoglycan biosynthesis in cartilage and endochondral ossification. Matrix Biology, 2019, 81, 70-90.	3.6	27
6	Mesencephalic astrocyte-derived neurotropic factor is an important factor in chondrocyte ER homeostasis. Cell Stress and Chaperones, 2019, 24, 159-173.	2.9	19
7	Pseudoachondroplasia and Multiple Epiphyseal Dysplasia: Molecular Genetics, Disease Mechanisms and Therapeutic Targets. , 2017, , 135-153.		0
8	The utility of mouse models to provide information regarding the pathomolecular mechanisms in human genetic skeletal diseases: The emerging role of endoplasmic reticulum stress (Review). International Journal of Molecular Medicine, 2015, 35, 1483-1492.	4.0	23
9	Cartilage-specific ablation of XBP1 signaling in mouse results in a chondrodysplasia characterized by reduced chondrocyte proliferation and delayed cartilage maturation and mineralization. Osteoarthritis and Cartilage, 2015, 23, 661-670.	1.3	38
10	New therapeutic targets in rare genetic skeletal diseases. Expert Opinion on Orphan Drugs, 2015, 3, 1137-1154.	0.8	34
11	Abnormal Chondrocyte Apoptosis in the Cartilage Growth Plate is Influenced by Genetic Background and Deletion of CHOP in a Targeted Mouse Model of Pseudoachondroplasia. PLoS ONE, 2014, 9, e85145.	2.5	27
12	Mild Myopathy Is Associated with COMP but Not MATN3 Mutations in Mouse Models of Genetic Skeletal Diseases. PLoS ONE, 2013, 8, e82412.	2.5	6
13	Loss of matrilin 1 does not exacerbate the skeletal phenotype in a mouse model of multiple epiphyseal dysplasia caused by a Matn3 V194D mutation. Arthritis and Rheumatism, 2012, 64, 1529-1539.	6.7	9
14	A novel form of chondrocyte stress is triggered by a COMP mutation causing pseudoachondroplasia. Human Mutation, 2012, 33, 218-231.	2.5	42
15	A mouse model offers novel insights into the myopathy and tendinopathy often associated with pseudoachondroplasia and multiple epiphyseal dysplasia. Human Molecular Genetics, 2010, 19, 52-64.	2.9	39
16	Skeletal Dysplasias Associated with Mild Myopathyâ€"A Clinical and Molecular Review. Journal of Biomedicine and Biotechnology, 2010, 2010, 1-13.	3.0	9
17	Reduced cell proliferation and increased apoptosis are significant pathological mechanisms in a murine model of mild pseudoachondroplasia resulting from a mutation in the C-terminal domain of COMP. Human Molecular Genetics, 2007, 16, 2072-2088.	2.9	84
18	Changes inBcl-2Expression in Vaccinia Virus-Infected Human Peripheral Blood Monocytes. Viral Immunology, 2005, 18, 224-231.	1.3	2