Michael D Briggs

List of Publications by Year
in descending order

35 papers	2,148 citations	218677 26 h-index	377865 34 g-index
35 all docs	35 docs citations	35 times ranked	1805 citing authors

#	Article	IF	Citations
1	Multiple epiphyseal dysplasia and related disorders: Molecular genetics, disease mechanisms, and therapeutic avenues. Developmental Dynamics, 2021, 250, 345-359.	1.8	15
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	Cartilage endoplasmic reticulum stress may influence the onset but not the progression of experimental osteoarthritis. Arthritis Research and Therapy, 2019, 21, 206.	3.5	14
6	Increased intracellular proteolysis reduces disease severity in an ER stressâ€“associated dwarfism. Journal of Clinical Investigation, 2017, 127, 3861-3865.	8.2	50
7	The aggrecanopathies; an evolving phenotypic spectrum of human genetic skeletal diseases. Orphanet Journal of Rare Diseases, 2016, 11, 86.	2.7	63
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	Increased Classical Endoplasmic Reticulum Stress Is Sufficient to Reduce Chondrocyte Proliferation Rate in the Growth Plate and Decrease Bone Growth. PLoS ONE, 2015, 10, e0117016.	2.5	32
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	Genotype to phenotype correlations in cartilage oligomeric matrix protein associated chondrodysplasias. European Journal of Human Genetics, 2014, 22, 1278-1282.	2.8	40
13	Armet/Manf and Creld2 are components of a specialized ER stress response provoked by inappropriate formation of disulphide bonds: implications for genetic skeletal diseases. Human Molecular Genetics, 2013, 22, 5262-5275.	2.9	62
14	Pseudoachondroplasia and multiple epiphyseal dysplasia: A 7â€year comprehensive analysis of the known disease genes identify novel and recurrent mutations and provides an accurate assessment of their relative contribution. Human Mutation, 2012, 33, 144-157.	2.5	104
15	A novel form of chondrocyte stress is triggered by a COMP mutation causing pseudoachondroplasia. Human Mutation, 2012, 33, 218-231.	2.5	42
16	The unfolded protein response and its relevance to connective tissue diseases. Cell and Tissue Research, 2010, 339, 197-211.	2.9	124
17	An unfolded protein response is the initial cellular response to the expression of mutant matrilin-3 in a mouse model of multiple epiphyseal dysplasia. Cell Stress and Chaperones, 2010, 15, 835-849.	2.9	59
18	Type IX collagen gene mutations can result in multiple epiphyseal dysplasia that is associated with osteochondritis dissecans and a mild myopathy. American Journal of Medical Genetics, Part A, 2010, 152A, 863-869.	1.2	44

#	Article	IF	Citations
19	Targeted Induction of Endoplasmic Reticulum Stress Induces Cartilage Pathology. PLoS Genetics, 2009, 5, e1000691.	3.5	127
20	Novel mutations in exon 2 ofMATN3affect residues within the Î±-helices of the A-domain and can result in the intracellular retention of mutant matrilin-3. Human Mutation, 2008, 29, 330-330.	2.5	18
21	Structural and Functional Characterization of Recombinant Matrilin-3 A-domain and Implications for Human Genetic Bone Diseases. Journal of Biological Chemistry, 2007, 282, 34634-34643.	3.4	39
22	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
23	Decreased chondrocyte proliferation and dysregulated apoptosis in the cartilage growth plate are key features of a murine model of epiphyseal dysplasia caused by a matn3 mutation. Human Molecular Genetics, 2007, 16, 1728-1741.	2.9	67
24	Preselection of cases through expert clinical and radiological review significantly increases mutation detection rate in multiple epiphyseal dysplasia. European Journal of Human Genetics, 2007, 15, 150-154.	2.8	28
25	Mutations in the known genes are not the major cause of MED; distinctive phenotypic entities among patients with no identified mutations. European Journal of Human Genetics, 2005, 13, 292-301.	2.8	32
26	Multiple epiphyseal dysplasia mutations inMATN3 cause misfolding of the A-domain and prevent secretion of mutant matrilin-3. Human Mutation, 2005, 26, 557-565.	2.5	63
27	Novel and recurrent mutations in the C-terminal domain ofCOMP cluster in two distinct regions and result in a spectrum of phenotypes within the pseudoachondroplasia - multiple epiphyseal dysplasia disease group. Human Mutation, 2005, 25, 593-594.	2.5	44
28	Clinical and radiographic findings in multiple epiphyseal dysplasia caused by MATN3 mutations: Description of 12 patients. , 2004, 125A, 278-284.		55
29	Pseudoachondroplasia and multiple epiphyseal dysplasia: Mutation review, molecular interactions, and genotype to phenotype correlations. Human Mutation, 2002, 19, 465-478.	2.5	261
30	Multiple epiphyseal dysplasia: radiographic abnormalities correlated with genotype. Pediatric Radiology, 2001, 31, 10-18.	2.0	61
31	Mutations in the region encoding the von Willebrand factor A domain of matrilin-3 are associated with multiple epiphyseal dysplasia. Nature Genetics, 2001, 28, 393-396.	21.4	183
32	Clinical and radiographic features of multiple epiphyseal dysplasia not linked to the COMP or type IX collagen genes. European Journal of Human Genetics, 2001, 9, 606-612.	2.8	26
33	Cartilage Oligomeric Matrix Protein Interacts with Type IX Collagen, and Disruptions to These Interactions Identify a Pathogenetic Mechanism in a Bone Dysplasia Family. Journal of Biological Chemistry, 2001, 276, 6046-6055.	3.4	188
34	Exon skipping mutation in the COL9A2 gene in a family with multiple epiphyseal dysplasia. Matrix Biology, 2000, 19, 121-128.	3.6	30
35	Identification of Novel pro-Î±2(IX) Collagen Gene Mutations in Two Families with Distinctive Oligo-Epiphyseal Forms of Multiple Epiphyseal Dysplasia. American Journal of Human Genetics, 1999, 65, 31-38.	6.2	64