

Francoise Coustry

List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/2244313/publications.pdf>

Version: 2024-02-01

10
papers

353
citations

1040056

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h-index

1372567

10
g-index

11
all docs

11
docs citations

11
times ranked

381
citing authors

#	ARTICLE	IF	CITATIONS
1	Cartilage oligomeric matrix protein: COMPopathies and beyond. Matrix Biology, 2018, 71-72, 161-173.	3.6	131
2	D469del-COMP Retention in Chondrocytes Stimulates Caspase-Independent Necroptosis. American Journal of Pathology, 2012, 180, 738-748.	3.8	40
3	Chop (Ddit3) Is Essential for D469del-COMP Retention and Cell Death in Chondrocytes in an Inducible Transgenic Mouse Model of Pseudoachondroplasia. American Journal of Pathology, 2012, 180, 727-737.	3.8	35
4	Chondrocyte-Specific Pathology During Skeletal Growth and Therapeutics in a Murine Model of Pseudoachondroplasia. Journal of Bone and Mineral Research, 2014, 29, 1258-1268.	2.8	34
5	Antioxidant and anti-inflammatory agents mitigate pathology in a mouse model of pseudoachondroplasia. Human Molecular Genetics, 2015, 24, 3918-3928.	2.9	34
6	Mutant cartilage oligomeric matrix protein (COMP) compromises bone integrity, joint function and the balance between adipogenesis and osteogenesis. Matrix Biology, 2018, 67, 75-89.	3.6	26
7	Novel mTORC1 Mechanism Suggests Therapeutic Targets for COMPopathies. American Journal of Pathology, 2019, 189, 132-146.	3.8	15
8	Primary Osteoarthritis Early Joint Degeneration Induced by Endoplasmic Reticulum Stress Is Mitigated by Resveratrol. American Journal of Pathology, 2021, 191, 1624-1637.	3.8	14
9	Resveratrol Reduces <scp>COMPopathy</scp> in Mice Through Activation of Autophagy. JBMR Plus, 2021, 5, e10456.	2.7	14
10	Joint Degeneration in a Mouse Model of Pseudoachondroplasia: ER Stress, Inflammation, and Block of Autophagy. International Journal of Molecular Sciences, 2021, 22, 9239.	4.1	7