Laurence Legeai-Mallet

List of Publications by Year
in descending order

58 papers	4,110 citations	136950 32 h-index	155660 55 g-index
61 all docs	61 docs citations	61 times ranked	4095 citing authors

#	Article	IF	Citations
1	Theobroma cacao improves bone growth by modulating defective ciliogenesis in a mouse model of achondroplasia. Bone Research, 2022, 10, 8.	11.4	0
2	Growth charts in FGFR2- and FGFR3-related faciocraniosynostoses. Bone Reports, 2022, 16, 101524.	0.4	2
3	FGFR3 overactivation in the brain is responsible for memory impairments in Crouzon syndrome mouse model.. Journal of Experimental Medicine, 2022, 219, .	8.5	2
4	Prevention of guanylyl cyclaseâ€“B dephosphorylation rescues achondroplastic dwarfism. JCI Insight, 2021, 6, .	5.0	12
5	AnFgfr3-activating mutation in immature murine osteoblasts affects the appendicular and craniofacial skeleton. DMM Disease Models and Mechanisms, 2021, 14, .	2.4	6
6	Phosphatase inhibition by LB-100 enhances BMN-111 stimulation of bone growth. JCI Insight, 2021, 6, .	5.0	9
7	FGFR3 in Periosteal Cells Drives Cartilage-to-Bone Transformation in Bone Repair. Stem Cell Reports, 2020, 15, 955-967.	4.8	27
8	Novel therapeutic approaches for the treatment of achondroplasia. Bone, 2020, 141, 115579.	2.9	31
9	Fgfr3 Is a Positive Regulator of Osteoblast Expansion and Differentiation During Zebrafish Skull Vault Development. Journal of Bone and Mineral Research, 2020, 35, 1782-1797.	2.8	18
10	Constitutively-active FGFR3 disrupts primary cilium length and IFT20 trafficking in various chondrocyte models of achondroplasia. Human Molecular Genetics, 2018, 27, 1-13.	2.9	37
11	Achondroplasia: Development, pathogenesis, and therapy. Developmental Dynamics, 2017, 246, 291-309.	1.8	160
12	C-Type Natriuretic Peptide Analog as Therapy for Achondroplasia. Endocrine Development, 2016, 30, 98-105.	1.3	31
13	Meckelâ€™s and condylar cartilages anomalies in achondroplasia result in defective development and growth of the mandible. Human Molecular Genetics, 2016, 25, ddw153.	2.9	21
14	Tyrosine kinase inhibitor NVP-BGJ398 functionally improves FGFR3-related dwarfism in mouse model. Journal of Clinical Investigation, 2016, 126, 1871-1884.	8.2	84
15	The impact of polyphenols on chondrocyte growth and survival: a preliminary report. Food and Nutrition Research, 2015, 59, 29311.	2.6	1
16	Molecular modeling study of the induced-fit effect on kinase inhibition: the case of fibroblast growth factor receptor 3 (FGFR3). Journal of Computer-Aided Molecular Design, 2015, 29, 619-641.	2.9	3
17	Key challenges in the treatment of rare pediatric skeletal genetic disorders: from bench to bedside. Drug Discovery Today, 2015, 20, 781-783.	6.4	1
18	FGFR3 mutation causes abnormal membranous ossification in achondroplasia. Human Molecular Genetics, 2014, 23, 2914-2925.	2.9	57

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19	The Impairment of MAGMAS Function in Human Is Responsible for a Severe Skeletal Dysplasia. PLoS Genetics, 2014, 10, e1004311.	3.5	34
20	Chondrocytes Play a Major Role in the Stimulation of Bone Growth by Thyroid Hormone. Endocrinology, 2014, 155, 3123-3135.	2.8	34
21	Polymerase Îµ1 mutation in a human syndrome with facial dysmorphism, immunodeficiency, livedo, and short stature (â€œFILS syndromeâ€). Journal of Experimental Medicine, 2012, 209, 2323-2330.	8.5	83
22	An activating Fgfr3 mutation affects trabecular bone formation via a paracrine mechanism during growth. Human Molecular Genetics, 2012, 21, 2503-2513.	2.9	40
23	A novel tyrosine kinase inhibitor restores chondrocyte differentiation and promotes bone growth in a gain-of-function Fgfr3 mouse model. Human Molecular Genetics, 2012, 21, 841-851.	2.9	40
24	Central nervous system malformations and deformations in FGFR2â€related craniosynostosis. American Journal of Medical Genetics, Part A, 2012, 158A, 2797-2806.	1.2	23
25	Evaluation of the Therapeutic Potential of a CNP Analog in a Fgfr3 Mouse Model Recapitulating Achondroplasia. American Journal of Human Genetics, 2012, 91, 1108-1114.	6.2	155
26	Crouzon syndrome with acanthosis nigricans: a case-based update. Child's Nervous System, 2011, 27, 349-354.	1.1	22
27	Corrigendum to â€œHyperphosphatasia With Seizures, Neurologic Deficit, and Characteristic Facial Features: Five New Patients With Mabry Syndromeâ€•Am J Med Genet 152A: 1661â€1669. American Journal of Medical Genetics, Part A, 2011, 155, 1215-1215.	1.2	2
28	Distinct Effects of Allelic NFIX Mutations on Nonsense-Mediated mRNA Decay Engender Either a Sotos-like or a Marshall-Smith Syndrome. American Journal of Human Genetics, 2010, 87, 189-198.	6.2	131
29	Hyperphosphatasia with seizures, neurologic deficit, and characteristic facial features: Five new patients with Mabry syndrome. American Journal of Medical Genetics, Part A, 2010, 152A, 1661-1669.	1.2	42
30	Synthesis and biological evaluation of a triazole-based library of pyrido[2,3-d]pyrimidines as FGFR3 tyrosine kinase inhibitors. Organic and Biomolecular Chemistry, 2010, 8, 2164.	2.8	53
31	Delayed bone age due to a dual effect of FGFR3 mutation in Achondroplasia. Bone, 2010, 47, 905-915.	2.9	23
32	Thanatophoric dysplasia caused by double missense FGFR3 mutations. American Journal of Medical Genetics, Part A, 2009, 149A, 1296-1301.	1.2	19
33	Activating Fgfr3 Y367C mutation causes hearing loss and inner ear defect in a mouse model of chondrodysplasia. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2009, 1792, 140-147.	3.8	49
34	New Insight on FGFR3-Related Chondrodysplasias Molecular Physiopathology Revealed by Human Chondrocyte Gene Expression Profiling. PLoS ONE, 2009, 4, e7633.	2.5	19
35	Thromboxane synthase mutations in an increased bone density disorder (Ghosal syndrome). Nature Genetics, 2008, 40, 284-286.	21.4	61
36	Hereditary multiple exostoses and enchondromatosis. Best Practice and Research in Clinical Rheumatology, 2008, 22, 45-54.	3.3	104

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37	Achondroplasia. Best Practice and Research in Clinical Rheumatology, 2008, 22, 3-18.	3.3	117
38	Human immortalized chondrocytes carrying heterozygous FGFR3 mutations: An in vitro model to study chondrodysplasias. FEBS Letters, 2007, 581, 2593-2598.	2.8	10
39	A cluster of translocation breakpoints in 2q37 is associated with overexpression of NPPC in patients with a similar overgrowth phenotype. Human Mutation, 2007, 28, 1183-1188.	2.5	79
40	FGFR3 intracellular mutations induce tyrosine phosphorylation in the Golgi and defective glycosylation. Biochimica Et Biophysica Acta - Molecular Cell Research, 2007, 1773, 502-512.	4.1	30
41	Defective chondrocyte proliferation and differentiation in osteochondromas of MHE patients. Bone, 2006, 39, 17-26.	2.9	44
42	Novel FGFR3 mutations creating cysteine residues in the extracellular domain of the receptor cause achondroplasia or severe forms of hypochondroplasia. European Journal of Human Genetics, 2006, 14, 1240-1247.	2.8	75
43	Null Leukemia Inhibitory Factor Receptor (LIFR) Mutations in StÃ¼ve-Wiedemann/Schwartz-Jampel Type 2 Syndrome. American Journal of Human Genetics, 2004, 74, 298-305.	6.2	162
44	ADAMTS10 Mutations in Autosomal Recessive Weill-Marchesani Syndrome. American Journal of Human Genetics, 2004, 75, 801-806.	6.2	255
45	Parathyroid Hormone Receptor Type 1/Indian Hedgehog Expression Is Preserved in the Growth Plate of Human Fetuses Affected with Fibroblast Growth Factor Receptor Type 3 Activating Mutations. American Journal of Pathology, 2002, 161, 1325-1335.	3.8	20
46	Les exostosines : des protÃ©ines impliquÃ©es dans la biosynthÃ¨se des hÃ©paranes sulfates. Medecine/Sciences, 2002, 18, 23-25.	0.2	0
47	Homozygosity mapping of a Weill-Marchesani syndrome locus to chromosome 19p13.3-p13.2. Human Genetics, 2002, 110, 366-370.	3.8	41
48	Mutations in the basic domain and the loop-helix II junction of TWIST abolish DNA binding in Saethre-Chotzen syndrome. FEBS Letters, 2001, 492, 112-118.	2.8	55
49	EXT 1 Gene Mutation Induces Chondrocyte Cytoskeletal Abnormalities and Defective Collagen Expression in the Exostoses. Journal of Bone and Mineral Research, 2000, 15, 1489-1500.	2.8	28
50	Spatio-temporal expression of FGFR 1, 2 and 3 genes during human embryo-fetal ossification. Mechanisms of Development, 1998, 77, 19-30.	1.7	161
51	Fibroblast Growth Factor Receptor 3 Mutations Promote Apoptosis but Do Not Alter Chondrocyte Proliferation in Thanatophoric Dysplasia. Journal of Biological Chemistry, 1998, 273, 13007-13014.	3.4	127
52	An extension of the admixture test for the study of genetic heterogeneity in hereditary multiple exostoses. Human Genetics, 1997, 99, 298-302.	3.8	36
53	Incomplete penetrance and expressivity skewing in hereditary multiple exostoses. Clinical Genetics, 1997, 52, 12-16.	2.0	132
54	Mutations of the Fibroblast Growth Factor Receptor-3 Gene in Achondroplasia. Hormone Research, 1996, 45, 108-110.	1.8	47

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55	A gene for achondroplasiaâ€“hypochondroplasia maps to chromosome 4p. Nature Genetics, 1994, 6, 318-321.	21.4	128
56	A gene for Holtâ€“Oram syndrome maps to the distal long arm of chromosome 12. Nature Genetics, 1994, 6, 405-408.	21.4	51
57	Mutations in the gene encoding fibroblast growth factor receptor-3 in achondroplasia. Nature, 1994, 371, 252-254.	27.8	873
58	A gene for hereditary multiple exostoses maps to chromosome 19p. Human Molecular Genetics, 1994, 3, 717-722.	2.9	199