Jay C Groppe

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/7687783/publications.pdf

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10	1,182	9	10
papers	citations	h-index	g-index
10	10	10	1319
all docs	docs citations	times ranked	citing authors

#	Article	IF	CITATIONS
1	An ACVR1 R375P pathogenic variant in two families with mild fibrodysplasia ossificans progressiva. American Journal of Medical Genetics, Part A, 2021, , .	1.2	3
2	Induced degradation of protein kinases by bifunctional small molecules: a next-generation strategy. Expert Opinion on Drug Discovery, 2019, 14, 1237-1253.	5.0	16
3	Hypoxia-selective allosteric destabilization of activin receptor-like kinases: A potential therapeutic avenue for prophylaxis of heterotopic ossification. Bone, 2018, 112, 71-89.	2.9	10
4	Cellular Hypoxia Promotes Heterotopic Ossification by Amplifying BMP Signaling. Journal of Bone and Mineral Research, 2016, 31, 1652-1665.	2.8	110
5	Multiâ€system involvement in a severe variant of fibrodysplasia ossificans progressiva (<i>ACVR1</i>) Tj ETQq1 1 2265-2271.	0.784314 1.2	rgBT /Ov <mark>erb</mark> 33
6	In vitro Analyses of the Dysregulated R206H ALK2 Kinase-FKBP12 Interaction Associated with Heterotopic Ossification in FOP. Cells Tissues Organs, 2011, 194, 291-295.	2.3	65
7	Classic and atypical fibrodysplasia ossificans progressiva (FOP) phenotypes are caused by mutations in the bone morphogenetic protein (BMP) type I receptor ACVR1. Human Mutation, 2009, 30, 379-390.	2.5	364
8	Cooperative Assembly of TGF- \hat{l}^2 Superfamily Signaling Complexes Is Mediated by Two Disparate Mechanisms and Distinct Modes of Receptor Binding. Molecular Cell, 2008, 29, 157-168.	9.7	247
9	Functional Modeling of the ACVR1 (R206H) Mutation in FOP. Clinical Orthopaedics and Related Research, 2007, 462, 87-92.	1.5	86
10	The BMP7/ActRII Extracellular Domain Complex Provides New Insights into the Cooperative Nature of Receptor Assembly. Molecular Cell, 2003, 11, 605-617.	9.7	248