Peter J Houweling

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Loss of α-actinin-3 confers protection from eccentric contraction damage in fast-twitch EDL muscles from aged <i>mdx</i> dystrophic mice by reducing pathological fibre branching. Human Molecular Genetics, 2022, 31, 1417-1429.	2.9	2
2	A Spotlight on T Lymphocytes in Duchenne Muscular Dystrophy—Not Just a Muscle Defect. Biomedicines, 2022, 10, 535.	3.2	2
3	Response to Mörseburg etÂal American Journal of Human Genetics, 2022, 109, 973.	6.2	2
4	Absence of the Z-disc protein α-actinin-3 impairs the mechanical stability of Actn3KO mouse fast-twitch muscle fibres without altering their contractile properties or twitch kinetics. Skeletal Muscle, 2022, 12, .	4.2	3
5	Loss of α-actinin-3 during human evolution provides superior cold resilience and muscle heat generation. American Journal of Human Genetics, 2021, 108, 446-457.	6.2	32
6	<i>ACTN3</i> genotype influences skeletal muscle mass regulation and response to dexamethasone. Science Advances, 2021, 7, .	10.3	7
7	Generating an iPSC line (with isogenic control) from the PBMCs of an ACTA1 (p.Gly148Asp) nemaline myopathy patient. Stem Cell Research, 2021, 54, 102429.	0.7	3
8	Dystrophin-negative slow-twitch soleus muscles are not susceptible to eccentric contraction induced injury over the lifespan of the mdx mouse. American Journal of Physiology - Cell Physiology, 2021, 321, C704-C720.	4.6	11
9	Lifespan Analysis of Dystrophic mdx Fast-Twitch Muscle Morphology and Its Impact on Contractile Function. Frontiers in Physiology, 2021, 12, 771499.	2.8	9
10	Evaluating modified diets and dietary supplement therapies for reducing muscle lipid accumulation and improving muscle function in neurofibromatosis type 1 (NF1). PLoS ONE, 2020, 15, e0237097.	2.5	5
11	Eosinophil function in adipose tissue is regulated by Krüppel-like factor 3 (KLF3). Nature Communications, 2020, 11, 2922.	12.8	35
12	Mice with myocyte deletion of vitamin D receptor have sarcopenia and impaired muscle function. Journal of Cachexia, Sarcopenia and Muscle, 2019, 10, 1228-1240.	7.3	79
13	More than a â€~speed gene': ACTN3 R577X genotype, trainability, muscle damage, and the risk for injuries. European Journal of Applied Physiology, 2019, 119, 49-60.	2.5	55
14	The Effect of ACTN3 Gene Doping on Skeletal Muscle Performance. American Journal of Human Genetics, 2018, 102, 845-857.	6.2	17
15	ls evolutionary loss our gain? The role of <i>ACTN3</i> p.Arg577Ter (R577X) genotype in athletic performance, ageing, and disease. Human Mutation, 2018, 39, 1774-1787.	2.5	50
16	Branched fibers from old fast-twitch dystrophic muscles are the sites of terminal damage in muscular dystrophy. American Journal of Physiology - Cell Physiology, 2018, 314, C662-C674.	4.6	23
17	The antioxidants neopterin/7,8â€dihydroneopterin: Novel biomarker and muscle protectant in Duchenne muscular dystrophy. Experimental Physiology, 2018, 103, 939-940.	2.0	1
18	No association between ACTN3 R577X and ACE I/D polymorphisms and endurance running times in 698 Caucasian athletes. BMC Genomics, 2018, 19, 13.	2.8	65

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19	Evidence for ACTN3 as a genetic modifier of Duchenne muscular dystrophy. Nature Communications, 2017, 8, 14143.	12.8	58
20	The influence of α-actinin-3 deficiency on bone remodelling markers in young men. Bone, 2017, 98, 26-30.	2.9	14
21	Exploring the relationship between α-actinin-3 deficiency and obesity in mice and humans. International Journal of Obesity, 2017, 41, 1154-1157.	3.4	9
22	No Evidence of a Common DNA Variant Profile Specific to World Class Endurance Athletes. PLoS ONE, 2016, 11, e0147330.	2.5	96
23	ACTN3 R577X and ACE I/D gene variants influence performance in elite sprinters: a multi-cohort study. BMC Genomics, 2016, 17, 285.	2.8	106
24	How does α-actinin-3 deficiency alter muscle function? Mechanistic insights into ACTN3 , the â€~gene for speed'. Biochimica Et Biophysica Acta - Molecular Cell Research, 2016, 1863, 686-693.	4.1	57
25	Athlome Project Consortium: a concerted effort to discover genomic and other "omic―markers of athletic performance. Physiological Genomics, 2016, 48, 183-190.	2.3	96
26	Analysis of the <i>ACTN3</i> heterozygous genotype suggests that α-actinin-3 controls sarcomeric composition and muscle function in a dose-dependent fashion. Human Molecular Genetics, 2016, 25, 866-877.	2.9	35
27	Neuronal control of bone and muscle. Bone, 2015, 80, 95-100.	2.9	25
28	Altered Ca2+ Kinetics Associated with α-Actinin-3 Deficiency May Explain Positive Selection for ACTN3 Null Allele in Human Evolution. PLoS Genetics, 2015, 11, e1004862.	3.5	39
29	Recent studies of ovine neuronal ceroid lipofuscinoses from BARN, the Batten Animal Research Network. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 2279-2286.	3.8	29
30	Vitamin D Receptor Ablation and Vitamin D Deficiency Result in Reduced Grip Strength, Altered Muscle Fibers, and Increased Myostatin in Mice. Calcified Tissue International, 2015, 97, 602-610.	3.1	110
31	A gene for speed: The influence of ACTN3 on muscle performance in health and disease. Neuromuscular Disorders, 2015, 25, S185.	0.6	0
32	Properties of regenerated mouse extensor digitorum longus muscle following notexin injury. Experimental Physiology, 2014, 99, 664-674.	2.0	17
33	The Vitamin D Receptor (VDR) Is Expressed in Skeletal Muscle of Male Mice and Modulates 25-Hydroxyvitamin D (250HD) Uptake in Myofibers. Endocrinology, 2014, 155, 3227-3237.	2.8	165
34	α-Actinin-3 deficiency alters muscle adaptation in response to denervation and immobilization. Human Molecular Genetics, 2014, 23, 1879-1893.	2.9	26
35	Sequence analysis of the equine ACTN3 gene in Australian horse breeds. Gene, 2014, 538, 88-93.	2.2	12

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37	Leiomodin-3 dysfunction results in thin filament disorganization and nemaline myopathy. Journal of Clinical Investigation, 2014, 124, 4693-4708.	8.2	153
38	Evidence Based Selection of Commonly Used RT-qPCR Reference Genes for the Analysis of Mouse Skeletal Muscle. PLoS ONE, 2014, 9, e88653.	2.5	69
39	Genes for Elite Power and Sprint Performance: ACTN3 Leads the Way. Sports Medicine, 2013, 43, 803-817.	6.5	158
40	ACTN3 genotype influences muscle performance through the regulation of calcineurin signaling. Journal of Clinical Investigation, 2013, 123, 4255-4263.	8.2	113
41	RARE MYOPATHIES AND EXPERIMENTAL APPROACHES - POSTER PRESENTATIONS C.P.125 ACTN3 genotype influences skeletal muscle performance through alterations in calcineurin signaling. Neuromuscular Disorders, 2012, 22, 904.	0.6	Ο
42	α-Actinin-3 deficiency is associated with reduced bone mass in human and mouse. Bone, 2011, 49, 790-798.	2.9	37
43	Properties of extensor digitorum longus muscle and skinned fibers from adult and aged male and female <i>Actn3</i> knockout mice. Muscle and Nerve, 2011, 43, 37-48.	2.2	26
44	Deficiency of α-actinin-3 is associated with increased susceptibility to contraction-induced damage and skeletal muscle remodeling. Human Molecular Genetics, 2011, 20, 2914-2927.	2.9	95
45	Sarcomeric α-actinins and their role in human muscle disease. Future Neurology, 2009, 4, 731-743.	0.5	11
46	A new large animal model of CLN5 neuronal ceroid lipofuscinosis in Borderdale sheep is caused by a nucleotide substitution at a consensus splice site (c.571 + 1G >>> A) leading to excision of exon 3. Neurobiology of Disease, 2008, 29, 306-315.	4.4	64
47	Neuronal ceroid lipofuscinosis in Devon cattle is caused by a single base duplication (c.662dupG) in the bovine CLN5 gene. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2006, 1762, 890-897.	3.8	33
48	A missense mutation (c.184C>T) in ovine CLN6 causes neuronal ceroid lipofuscinosis in Merino sheep whereas affected South Hampshire sheep have reduced levels of CLN6 mRNA. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2006, 1762, 898-905.	3.8	68
49	Radiation hybrid mapping of three candidate genes for bovine neuronal ceroid lipofuscinosis: <i>CLN3, CLN5 </i> and <i>CLN6</i> . Cytogenetic and Genome Research, 2006, 115, 5-6.	1.1	2