Peter J Houweling

List of Publications by Year
in descending order

49 papers	2,154 citations	236925 25 h-index	233421 45 g-index
54 all docs	54 docs citations	54 times ranked	2843 citing authors

#	Article	IF	Citations
1	The Vitamin D Receptor (VDR) Is Expressed in Skeletal Muscle of Male Mice and Modulates 25-Hydroxyvitamin D (25OHD) Uptake in Myofibers. Endocrinology, 2014, 155, 3227-3237.	2.8	165
2	Genes for Elite Power and Sprint Performance: ACTN3 Leads the Way. Sports Medicine, 2013, 43, 803-817.	6.5	158
3	Leiomodin-3 dysfunction results in thin filament disorganization and nemaline myopathy. Journal of Clinical Investigation, 2014, 124, 4693-4708.	8.2	153
4	ACTN3 genotype influences muscle performance through the regulation of calcineurin signaling. Journal of Clinical Investigation, 2013, 123, 4255-4263.	8.2	113
5	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
6	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
7	No Evidence of a Common DNA Variant Profile Specific to World Class Endurance Athletes. PLoS ONE, 2016, 11, e0147330.	2.5	96
8	Athlome Project Consortium: a concerted effort to discover genomic and other â€œomicâ€•markers of athletic performance. Physiological Genomics, 2016, 48, 183-190.	2.3	96
9	Deficiency of Î±-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
10	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
11	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
12	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
13	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
14	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
15	Evidence for ACTN3 as a genetic modifier of Duchenne muscular dystrophy. Nature Communications, 2017, 8, 14143.	12.8	58
16	How does Î±-actinin-3 deficiency alter muscle function? Mechanistic insights into ACTN3 , the â€˜gene for speedâ€™. Biochimica Et Biophysica Acta - Molecular Cell Research, 2016, 1863, 686-693.	4.1	57
17	More than a â€˜speed geneâ€™: ACTN3 R577X genotype, trainability, muscle damage, and the risk for injuries. European Journal of Applied Physiology, 2019, 119, 49-60.	2.5	55
18	Is evolutionary loss our gain? The role of ACTN3 p.Arg577Ter (R577X) genotype in athletic performance, ageing, and disease. Human Mutation, 2018, 39, 1774-1787.	2.5	50

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19	Altered Ca2+ Kinetics Associated with Î±-Actinin-3 Deficiency May Explain Positive Selection for ACTN3 Null Allele in Human Evolution. PLoS Genetics, 2015, 11, e1004862.	3.5	39
20	Î±-Actinin-3 deficiency is associated with reduced bone mass in human and mouse. Bone, 2011, 49, 790-798.	2.9	37
21	Analysis of theACTN3heterozygous genotype suggests that Î±-actinin-3 controls sarcomeric composition and muscle function in a dose-dependent fashion. Human Molecular Genetics, 2016, 25, 866-877.	2.9	35
22	Eosinophil function in adipose tissue is regulated by KrÃ¼ppel-like factor 3 (KLF3). Nature Communications, 2020, 11, 2922.	12.8	35
23	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
24	Loss of Î±-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
25	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
26	Properties of extensor digitorum longus muscle and skinned fibers from adult and aged male and female Actn3 knockout mice. Muscle and Nerve, 2011, 43, 37-48.	2.2	26
27	Î±-Actinin-3 deficiency alters muscle adaptation in response to denervation and immobilization. Human Molecular Genetics, 2014, 23, 1879-1893.	2.9	26
28	Neuronal control of bone and muscle. Bone, 2015, 80, 95-100.	2.9	25
29	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
30	Properties of regenerated mouse extensor digitorum longus muscle following notexin injury. Experimental Physiology, 2014, 99, 664-674.	2.0	17
31	The Effect of ACTN3 Gene Doping on Skeletal Muscle Performance. American Journal of Human Genetics, 2018, 102, 845-857.	6.2	17
32	The influence of Î±-actinin-3 deficiency on bone remodelling markers in young men. Bone, 2017, 98, 26-30.	2.9	14
33	Sequence analysis of the equine ACTN3 gene in Australian horse breeds. Gene, 2014, 538, 88-93.	2.2	12
34	Sarcomeric Î±-actinins and their role in human muscle disease. Future Neurology, 2009, 4, 731-743.	0.5	11
35	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
36	Exploring the relationship between Î±-actinin-3 deficiency and obesity in mice and humans. International Journal of Obesity, 2017, 41, 1154-1157.	3.4	9

#	Article	IF	Citations
37	Lifespan Analysis of Dystrophic mdx Fast-Twitch Muscle Morphology and Its Impact on Contractile Function. Frontiers in Physiology, 2021, 12, 771499.	2.8	9
38	ACTN3 genotype influences skeletal muscle mass regulation and response to dexamethasone. Science Advances, 2021, 7, .	10.3	7
39	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
40	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
41	Absence of the Z-disc protein Î±-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
42	Radiation hybrid mapping of three candidate genes for bovine neuronal ceroid lipofuscinosis: CLN3, CLN5 and CLN6. Cytogenetic and Genome Research, 2006, 115, 5-6.	1.1	2
43	Loss of Î±-actinin-3 confers protection from eccentric contraction damage in fast-twitch EDL muscles from aged mdx dystrophic mice by reducing pathological fibre branching. Human Molecular Genetics, 2022, 31, 1417-1429.	2.9	2
44	A Spotlight on T Lymphocytes in Duchenne Muscular Dystrophyâ€"Not Just a Muscle Defect. Biomedicines, 2022, 10, 535.	3.2	2
45	Response to MÃ¶rseburg etÂal.. American Journal of Human Genetics, 2022, 109, 973.	6.2	2
46	G.O.2. Neuromuscular Disorders, 2014, 24, 792-793.	0.6	1
47	The antioxidants neopterin/7,8â€dihydroneopterin: Novel biomarker and muscle protectant in Duchenne muscular dystrophy. Experimental Physiology, 2018, 103, 939-940.	2.0	1
48	RARE MYOPATHIES AND EXPERIMENTAL APPROACHES - POSTER PRESENTATIONS G.P.125 ACTN3 genotype influences skeletal muscle performance through alterations in calcineurin signaling. Neuromuscular Disorders, 2012, 22, 904.	0.6	0
49	A gene for speed: The influence of ACTN3 on muscle performance in health and disease. Neuromuscular Disorders, 2015, 25, S185.	0.6	0