

# V Arechavala-Gomez

## List of Publications by Year in descending order

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Version: 2024-02-01

58  
papers

3,268  
citations

304368

22  
h-index

301761

39  
g-index

61  
all docs

61  
docs citations

61  
times ranked

3257  
citing authors

#	ARTICLE	IF	CITATIONS
1	Antisense RNA Therapeutics: A Brief Overview. <i>Methods in Molecular Biology</i> , 2022, 2434, 33-49.	0.4	5
2	Evaluation of Exon Skipping and Dystrophin Restoration in In Vitro Models of Duchenne Muscular Dystrophy. <i>Methods in Molecular Biology</i> , 2022, 2434, 217-233.	0.4	5
3	Lessons learned from developing an oligonucleotide drug for a rare disease. , 2022, , 121-137.		0
4	Researcher's Perceptions on Publishing "Negative" Results and Open Access. <i>Nucleic Acid Therapeutics</i> , 2021, 31, 185-189.	2.0	19
5	Joining European Scientific Forces to Face Pandemics. <i>Trends in Microbiology</i> , 2021, 29, 92-97.	3.5	5
6	Special Issue "Genetic Advances in Neuromuscular Disorders: From Gene Identification to Gene Therapy" Genes, 2021, 12, 242.	1.0	0
7	Delivery of oligonucleotide-based therapeutics: challenges and opportunities. <i>EMBO Molecular Medicine</i> , 2021, 13, e13243.	3.3	181
8	Utrophin modulator drugs as potential therapies for Duchenne and Becker muscular dystrophies. <i>Neuropathology and Applied Neurobiology</i> , 2021, 47, 711-723.	1.8	22
9	Duchenne muscular dystrophy cell culture models created by CRISPR/Cas9 gene editing and their application in drug screening. <i>Scientific Reports</i> , 2021, 11, 18188.	1.6	10
10	Sharing "Negative" Results in Neuromuscular Research: A Positive Experience. <i>Journal of Neuromuscular Diseases</i> , 2021, 8, 765-767.	1.1	2
11	An Overview of Alternative Splicing Defects Implicated in Myotonic Dystrophy Type I. <i>Genes</i> , 2020, 11, 1109.	1.0	66
12	The Biomarker Potential of miRNAs in Myotonic Dystrophy Type I. <i>Journal of Clinical Medicine</i> , 2020, 9, 3939.	1.0	15
13	P.291 Overcoming barriers to establish a CRISPR/Cas9 edition protocol for human myoblasts. <i>Neuromuscular Disorders</i> , 2019, 29, S152.	0.3	0
14	P.290 Dystrophinopathic subjects with a specific mega-deletion of exons 45-55 in the DMD gene, as a template for CRISPR/Cas9 therapy in Duchenne muscular dystrophy. <i>Neuromuscular Disorders</i> , 2019, 29, S151-S152.	0.3	0
15	COST Actions: fostering collaborative research for rare diseases. <i>Lancet Neurology</i> , The, 2019, 18, 989-991.	4.9	2
16	Myoblots: dystrophin quantification by in-cell western assay for a streamlined development of Duchenne muscular dystrophy (DMD) treatments. <i>Neuropathology and Applied Neurobiology</i> , 2018, 44, 463-473.	1.8	12
17	A multicenter comparison of quantification methods for antisense oligonucleotide-induced DMD exon 51 skipping in Duchenne muscular dystrophy cell cultures. <i>PLoS ONE</i> , 2018, 13, e0204485.	1.1	14
18	Why dystrophin quantification is key in the eteplirsen saga. <i>Nature Reviews Neurology</i> , 2018, 14, 454-456.	4.9	20

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19	Delivery is key: lessons learnt from developing splice-switching antisense therapies. <i>EMBO Molecular Medicine</i> , 2017, 9, 545-557.	3.3	119
20	Myo-cytoblots: Quantification of dystrophin by in-cell western assay for a streamlined development of DMD treatments. <i>Neuromuscular Disorders</i> , 2016, 26, S159.	0.3	0
21	Selection of reference genes for normalisation of dystrophin mRNA RT-qPCR data. <i>Neuromuscular Disorders</i> , 2016, 26, S159-S160.	0.3	0
22	Stakeholder cooperation to overcome challenges in orphan medicine development: the example of Duchenne muscular dystrophy. <i>Lancet Neurology</i> , The, 2016, 15, 882-890.	4.9	77
23	Quantifying dystrophin in cell culture: A method to accelerate preclinical assessment of DMD treatments. <i>Neuromuscular Disorders</i> , 2015, 25, S254.	0.3	0
24	Splicing modulation therapy in the treatment of genetic diseases. <i>The Application of Clinical Genetics</i> , 2014, 7, 245.	1.4	33
25	Biochemical Characterization of Patients With In-Frame or Out-of-Frame DMD Deletions Pertinent to Exon 44 or 45 Skipping. <i>JAMA Neurology</i> , 2014, 71, 32.	4.5	71
26	Dystrophin quantification. <i>Neurology</i> , 2014, 83, 2062-2069.	1.5	73
27	P16 Towards a consensus on biochemical outcome measures for Duchenne muscular dystrophy clinical trials. <i>Neuromuscular Disorders</i> , 2014, 24, S11.	0.3	0
28	Antisense Oligonucleotide-Mediated Exon Skipping for Duchenne Muscular Dystrophy: Progress and Challenges. <i>Current Gene Therapy</i> , 2012, 12, 152-160.	0.9	73
29	Exon-skipping therapy for Duchenne muscular dystrophy – Authors' reply. <i>Lancet</i> , The, 2012, 379, e10-e11.	6.3	2
30	Restoration of the Dystrophin-associated Glycoprotein Complex After Exon Skipping Therapy in Duchenne Muscular Dystrophy. <i>Molecular Therapy</i> , 2012, 20, 462-467.	3.7	99
31	P01 Correlation of internally deleted dystrophin and dystrophin-associated protein expression with clinical severity in Becker muscular dystrophy. <i>Neuromuscular Disorders</i> , 2012, 22, S7.	0.3	0
32	P10 The next DMD exon skipping trial: selection of AO target. <i>Neuromuscular Disorders</i> , 2012, 22, S9-S10.	0.3	0
33	T.P.31 Biochemical and clinical variability of Becker muscular dystrophy: Predicting optimal target exons for exon skipping therapy in Duchenne muscular dystrophy. <i>Neuromuscular Disorders</i> , 2012, 22, 862.	0.3	0
34	Exon Skipping Quantification by Quantitative Reverse-Transcription Polymerase Chain Reaction in Duchenne Muscular Dystrophy Patients Treated with the Antisense Oligomer Eteplirsen. <i>Human Gene Therapy Methods</i> , 2012, 23, 336-345.	2.1	38
35	Measuring dystrophin – faster is not necessarily better. <i>Nature Reviews Neurology</i> , 2012, 8, 469-469.	4.9	1
36	P03 Exon skipping and dystrophin restoration in Duchenne muscular dystrophy patients after systemic phosphorodiamidate morpholino oligomer treatment. <i>Neuromuscular Disorders</i> , 2011, 21, S7-S8.	0.3	0

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37	Exon skipping and dystrophin restoration in patients with Duchenne muscular dystrophy after systemic phosphorodiamidate morpholino oligomer treatment: an open-label, phase 2, dose-escalation study. <i>Lancet, The</i> , 2011, 378, 595-605.	6.3	803
38	Chronic Systemic Therapy With Low-dose Morpholino Oligomers Ameliorates the Pathology and Normalizes Locomotor Behavior in mdx Mice. <i>Molecular Therapy</i> , 2011, 19, 345-354.	3.7	97
39	Dystrophin quantification and clinical correlations in Becker muscular dystrophy: implications for clinical trials. <i>Brain</i> , 2011, 134, 3547-3559.	3.7	125
40	Muscle histology vs MRI in Duchenne muscular dystrophy. <i>Neurology</i> , 2011, 76, 346-353.	1.5	134
41	Immunohistological intensity measurements as a tool to assess sarcolemma-associated protein expression. <i>Neuropathology and Applied Neurobiology</i> , 2010, 36, 265-274.	1.8	77
42	A Duchenne Muscular Dystrophy Gene Hot Spot Mutation in Dystrophin-Deficient Cavalier King Charles Spaniels Is Amenable to Exon 51 Skipping. <i>PLoS ONE</i> , 2010, 5, e8647.	1.1	102
43	Comparative analysis of antisense oligonucleotide sequences targeting exon 53 of the human DMD gene: Implications for future clinical trials. <i>Neuromuscular Disorders</i> , 2010, 20, 102-110.	0.3	44
44	The contribution of human synovial stem cells to skeletal muscle regeneration. <i>Neuromuscular Disorders</i> , 2010, 20, 6-15.	0.3	32
45	Revertant fibres and dystrophin traces in Duchenne muscular dystrophy: Implication for clinical trials. <i>Neuromuscular Disorders</i> , 2010, 20, 295-301.	0.3	80
46	P3.08 Induction of dystrophin in DMD patients by antisense oligonucleotide AVI-4658 restores the dystrophin glycoprotein complex. <i>Neuromuscular Disorders</i> , 2010, 20, 643.	0.3	0
47	O04 Results of a systemic antisense study in Duchenne muscular dystrophy. <i>Neuromuscular Disorders</i> , 2010, 20, S2.	0.3	0
48	P03 The characterisation of out of frame duplications in DMD patients. <i>Neuromuscular Disorders</i> , 2010, 20, S5.	0.3	0
49	P05 Induction of dystrophin in Duchenne muscular dystrophy patients by antisense oligonucleotide AVI-4658 restores the dystrophin-associated glycoprotein complex. <i>Neuromuscular Disorders</i> , 2010, 20, S6.	0.3	0
50	P27 Chronic long term administration of phosphorodiamidate morpholino oligomer profoundly ameliorates activity, muscle strength and phenotype in dystrophic mdx mice. <i>Neuromuscular Disorders</i> , 2010, 20, S12.	0.3	0
51	Local restoration of dystrophin expression with the morpholino oligomer AVI-4658 in Duchenne muscular dystrophy: a single-blind, placebo-controlled, dose-escalation, proof-of-concept study. <i>Lancet Neurology, The</i> , 2009, 8, 918-928.	4.9	617
52	T.P.4.09 Measuring restored dystrophin in treated muscle: An immunohistological intensity measurement method. <i>Neuromuscular Disorders</i> , 2009, 19, 615.	0.3	0
53	T.O.3 Restoration of dystrophin expression in Duchenne muscular dystrophy: A single blind, placebo-controlled dose escalation study using morpholino oligomer AVI-4658. <i>Neuromuscular Disorders</i> , 2009, 19, 659.	0.3	1
54	G.P.6.01 Establishing the parameters for clinical trials of antisense oligonucleotide therapy in Duchenne muscular dystrophy. <i>Neuromuscular Disorders</i> , 2008, 18, 773.	0.3	0

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55	Chapter 14 Familial amyotrophic lateral sclerosis. Handbook of Clinical Neurology / Edited By P J Vinken and G W Bruyn, 2007, 82, 279-300.	1.0	3
56	G.P.12.11 Do revertants increase with age in Duchenne muscular dystrophy boys?. Neuromuscular Disorders, 2007, 17, 842.	0.3	0
57	Comparative Analysis of Antisense Oligonucleotide Sequences for Targeted Skipping of Exon 51 During Dystrophin Pre-mRNA Splicing in Human Muscle. Human Gene Therapy, 2007, 18, 798-810.	1.4	144
58	T.P.1 02 A phase I/II clinical trial in Duchenne muscular dystrophy using IM and IV delivered antisense oligonucleotides: The MDEX consortium. Neuromuscular Disorders, 2006, 16, 685.	0.3	1