

# Yusuke Echigoya

## List of Publications by Year in descending order

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Version: 2024-02-01

32  
papers

1,039  
citations

393982

19  
h-index

454577

30  
g-index

32  
all docs

32  
docs citations

32  
times ranked

937  
citing authors

#	ARTICLE	IF	CITATIONS
1	Effects of systemic multiexon skipping with peptide-conjugated morpholinos in the heart of a dog model of Duchenne muscular dystrophy. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 4213-4218.	3.3	94
2	Extensive and Prolonged Restoration of Dystrophin Expression with Vivo-Morpholino-Mediated Multiple Exon Skipping in Dystrophic Dogs. Nucleic Acid Therapeutics, 2012, 22, 306-315.	2.0	69
3	Long-Term Efficacy of Systemic Multiexon Skipping Targeting Dystrophin Exons 45-55 With a Cocktail of Vivo-Morpholinos in Mdx52 Mice. Molecular Therapy - Nucleic Acids, 2015, 4, e225.	2.3	67
4	Quantitative Antisense Screening and Optimization for Exon 51 Skipping in Duchenne Muscular Dystrophy. Molecular Therapy, 2017, 25, 2561-2572.	3.7	63
5	Multiple Exon Skipping in the Duchenne Muscular Dystrophy Hot Spots: Prospects and Challenges. Journal of Personalized Medicine, 2018, 8, 41.	1.1	61
6	Skipping Multiple Exons of Dystrophin Transcripts Using Cocktail Antisense Oligonucleotides. Nucleic Acid Therapeutics, 2014, 24, 57-68.	2.0	55
7	Comparison of the phenotypes of patients harboring in-frame deletions starting at exon 45 in the Duchenne muscular dystrophy gene indicates potential for the development of exon skipping therapy. Journal of Human Genetics, 2017, 62, 459-463.	1.1	53
8	Impaired regenerative capacity and lower revertant fibre expansion in dystrophin-deficient mdx muscles on DBA/2 background. Scientific Reports, 2016, 6, 38371.	1.6	47
9	Deletion of exons 3-9 encompassing a mutational hot spot in the DMD gene presents an asymptomatic phenotype, indicating a target region for multiexon skipping therapy. Journal of Human Genetics, 2016, 61, 663-667.	1.1	45
10	In Silico Screening Based on Predictive Algorithms as a Design Tool for Exon Skipping Oligonucleotides in Duchenne Muscular Dystrophy. PLoS ONE, 2015, 10, e0120058.	1.1	45
11	Current Translational Research and Murine Models For Duchenne Muscular Dystrophy. Journal of Neuromuscular Diseases, 2016, 3, 29-48.	1.1	43
12	LNA/DNA mixmer-based antisense oligonucleotides correct alternative splicing of the SMN2 gene and restore SMN protein expression in type 1 SMA fibroblasts. Scientific Reports, 2017, 7, 3672.	1.6	42
13	Inhibition of DUX4 expression with antisense LNA gapmers as a therapy for facioscapulohumeral muscular dystrophy. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 16509-16515.	3.3	40
14	Dystrophin-deficient large animal models: translational research and exon skipping. American Journal of Translational Research (discontinued), 2015, 7, 1314-31.	0.0	38
15	Exon skipping for nonsense mutations in Duchenne muscular dystrophy: too many mutations, too few patients?. Expert Opinion on Biological Therapy, 2012, 12, 1141-1152.	1.4	35
16	Exons 45-55 Skipping Using Mutation-Tailored Cocktails of Antisense Morpholinos in the DMD Gene. Molecular Therapy, 2019, 27, 2005-2017.	3.7	35
17	Mutation Types and Aging Differently Affect Revertant Fiber Expansion in Dystrophic Mdx and Mdx52 Mice. PLoS ONE, 2013, 8, e69194.	1.1	26
18	Efficacy of Multi-exon Skipping Treatment in Duchenne Muscular Dystrophy Dog Model Neonates. Molecular Therapy, 2019, 27, 76-86.	3.7	24

#	ARTICLE	IF	CITATIONS
19	DUX4 Transcript Knockdown with Antisense 2â€²-O-Methoxyethyl Gapmers for the Treatment of Facioscapulohumeral Muscular Dystrophy. <i>Molecular Therapy</i> , 2021, 29, 848-858.	3.7	24
20	Antisense PMO cocktails effectively skip dystrophin exons 45-55 in myotubes transdifferentiated from DMD patient fibroblasts. <i>PLoS ONE</i> , 2018, 13, e0197084.	1.1	22
21	Development of DG9 peptide-conjugated single- and multi-exon skipping therapies for the treatment of Duchenne muscular dystrophy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2022, 119, .	3.3	21
22	Systemic Delivery of Morpholinos to Skip Multiple Exons in a Dog Model of Duchenne Muscular Dystrophy. <i>Methods in Molecular Biology</i> , 2017, 1565, 201-213.	0.4	19
23	Molecular characterization and expression pattern of the equine lactate dehydrogenase A and B genes. <i>Gene</i> , 2009, 447, 40-50.	1.0	14
24	Amelioration of intracellular Ca <sup>2+</sup> regulation by exon-45 skipping in Duchenne muscular dystrophy-induced pluripotent stem cell-derived cardiomyocytes. <i>Biochemical and Biophysical Research Communications</i> , 2019, 520, 179-185.	1.0	14
25	A fatal case of a captive snowy owl ( <i>Bubo scandiacus</i> ) with <i>Haemoproteus</i> infection in Japan. <i>Parasitology Research</i> , 2021, 120, 277-288.	0.6	13
26	Effects of extracellular lactate on production of reactive oxygen species by equine polymorphonuclear leukocytes in vitro. <i>American Journal of Veterinary Research</i> , 2012, 73, 1290-1298.	0.3	10
27	A Dystrophin Exon-52 Deleted Miniature Pig Model of Duchenne Muscular Dystrophy and Evaluation of Exon Skipping. <i>International Journal of Molecular Sciences</i> , 2021, 22, 13065.	1.8	9
28	Molecular cloning and expression of bottlenose dolphin CD34. <i>Veterinary Immunology and Immunopathology</i> , 2011, 139, 303-307.	0.5	5
29	Molecular characterization and expression of the equine M1 and M2-pyruvate kinase gene. <i>Comparative Biochemistry and Physiology - B Biochemistry and Molecular Biology</i> , 2008, 151, 125-132.	0.7	3
30	Molecular characterization of glycogen synthase 1 and its tissue expression profile with type II hexokinase and muscle-type phosphofructokinase in horses. <i>Molecular Biology Reports</i> , 2011, 38, 461-469.	1.0	3
31	623. Dystrophin Exon 52-Deleted Pigs as a New Animal Model of Duchenne Muscular Dystrophy: Its Characterization and Potential as a Tool for Developing Exon Skipping Therapy. <i>Molecular Therapy</i> , 2016, 24, S247.	3.7	0
32	Dystrophin-deficient cardiomyocyte derived from Duchenne Muscular Dystrophy specific induced pluripotent stem cells carrying the deletion of exon 46-55 in DMD gene. <i>Journal of the Neurological Sciences</i> , 2017, 381, 859.	0.3	0