Yoshitsugu Aoki

List of Publications by Year
in descending order

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		236612	214527
79	2,418	25	47
papers	citations	h-index	g-index

81	81	81	2775
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Identification of Muscle-Specific MicroRNAs in Serum of Muscular Dystrophy Animal Models: Promising Novel Blood-Based Markers for Muscular Dystrophy. PLoS ONE, 2011, 6, e18388.	1.1	178
2	Bodywide skipping of exons 45â€“55 in dystrophic mdx52 mice by systemic antisense delivery. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 13763-13768.	3.3	139
3	C9orf72 and RAB7L1 regulate vesicle trafficking in amyotrophic lateral sclerosis and frontotemporal dementia. Brain, 2017, 140, 887-897.	3.7	126
4	In-frame Dystrophin Following Exon 51-Skipping Improves Muscle Pathology and Function in the Exon 52â€“Deficient mdx Mouse. Molecular Therapy, 2010, 18, 1995-2005.	3.7	118
5	Systemic administration of the antisense oligonucleotide NS-065/NCNP-01 for skipping of exon 53 in patients with Duchenne muscular dystrophy. Science Translational Medicine, 2018, 10, .	5.8	111
6	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
7	Identification of Disease Specific Pathways Using in Vivo SILAC Proteomics in Dystrophin Deficient mdx Mouse. Molecular and Cellular Proteomics, 2013, 12, 1061-1073.	2.5	88
8	Three novel serum biomarkers, miR-1, miR-133a, and miR-206 for Limb-girdle muscular dystrophy, Facioscapulohumeral muscular dystrophy, and Becker muscular dystrophy. Environmental Health and Preventive Medicine, 2014, 19, 452-458.	1.4	86
9	Restoring Dystrophin Expression in Duchenne Muscular Dystrophy: Current Status of Therapeutic Approaches. Journal of Personalized Medicine, 2019, 9, 1.	1.1	84
10	Extracellular microRNAs are dynamic non-vesicular biomarkers of muscle turnover. Nucleic Acids Research, 2013, 41, 9500-9513.	6.5	83
11	Self-Assembly into Nanoparticles Is Essential for Receptor Mediated Uptake of Therapeutic Antisense Oligonucleotides. Nano Letters, 2015, 15, 4364-4373.	4.5	80
12	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
13	Long-Term Efficacy of Systemic Multiexon Skipping Targeting Dystrophin Exons 45â€“55 With a Cocktail of Vivo-Morpholinos in Mdx52 Mice. Molecular Therapy - Nucleic Acids, 2015, 4, e225.	2.3	67
14	Quantitative Antisense Screening and Optimization for Exon 51 Skipping in Duchenne Muscular Dystrophy. Molecular Therapy, 2017, 25, 2561-2572.	3.7	63
15	Peptide-conjugate antisense based splice-correction for Duchenne muscular dystrophy and other neuromuscular diseases. EBioMedicine, 2019, 45, 630-645.	2.7	61
16	Highly efficient in vivo delivery of PMO into regenerating myotubes and rescue in laminin-Î±2 chain-null congenital muscular dystrophy mice. Human Molecular Genetics, 2013, 22, 4914-4928.	1.4	59
17	Recent advances in innovative therapeutic approaches for Duchenne muscular dystrophy: from discovery to clinical trials. American Journal of Translational Research (discontinued), 2016, 8, 2471-89.	0.0	57
18	Antisense PMO Found in Dystrophic Dog Model Was Effective in Cells from Exon 7-Deleted DMD Patient. PLoS ONE, 2010, 5, e12239.	1.1	56

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19	Anti-inflammatory drugs for Duchenne muscular dystrophy: focus on skeletal muscle-releasing factors. Drug Design, Development and Therapy, 2016, Volume 10, 2745-2758.	2.0	54
20	Relationship between diffusion tensor imaging and brain morphology in patients with myotonic dystrophy. Neuroscience Letters, 2006, 407, 234-239.	1.0	50
21	Development of Multiexon Skipping Antisense Oligonucleotide Therapy for Duchenne Muscular Dystrophy. BioMed Research International, 2013, 2013, 1-8.	0.9	45
22	Deletion of exons 3â^'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
23	Endogenous Multiple Exon Skipping and Back-Splicing at the DMD Mutation Hotspot. International Journal of Molecular Sciences, 2016, 17, 1722.	1.8	35
24	Exons 45â€“55 Skipping Using Mutation-Tailored Cocktails of Antisense Morpholinos in the DMD Gene. Molecular Therapy, 2019, 27, 2005-2017.	3.7	35
25	Synthesis of 2â€²-O-[2-(N-Methylcarbamoyl)ethyl]ribonucleosides Using Oxa-Michael Reaction and Chemical and Biological Properties of Oligonucleotide Derivatives Incorporating These Modified Ribonucleosides. Journal of Organic Chemistry, 2011, 76, 3042-3053.	1.7	32
26	Pharmacological activation of SERCA ameliorates dystrophic phenotypes in dystrophin-deficient mdx mice. Human Molecular Genetics, 2021, 30, 1006-1019.	1.4	28
27	Serum Osteopontin as a Novel Biomarker for Muscle Regeneration in Duchenne Muscular Dystrophy. American Journal of Pathology, 2016, 186, 1302-1312.	1.9	27
28	Mutation Types and Aging Differently Affect Revertant Fiber Expansion in Dystrophic Mdx and Mdx52 Mice. PLoS ONE, 2013, 8, e69194.	1.1	26
29	Mutation-independent Proteomic Signatures of Pathological Progression in Murine Models of Duchenne Muscular Dystrophy. Molecular and Cellular Proteomics, 2020, 19, 2047-2068.	2.5	25
30	Truncated dystrophin ameliorates the dystrophic phenotype of mdx mice by reducing sarcolipin-mediated SERCA inhibition. Biochemical and Biophysical Research Communications, 2018, 505, 51-59.	1.0	24
31	Efficacy of Multi-exon Skipping Treatment in Duchenne Muscular Dystrophy Dog Model Neonates. Molecular Therapy, 2019, 27, 76-86.	3.7	24
32	Antisense PMO cocktails effectively skip dystrophin exons 45-55 in myotubes transdifferentiated from DMD patient fibroblasts. PLoS ONE, 2018, 13, e0197084.	1.1	22
33	Scavenger Receptor Class A1 Mediates Uptake of Morpholino Antisense Oligonucleotide into Dystrophic Skeletal Muscle. Molecular Therapy - Nucleic Acids, 2019, 14, 520-535.	2.3	22
34	Investigating the role of dystrophin isoform deficiency in motor function in Duchenne muscular dystrophy. Journal of Cachexia, Sarcopenia and Muscle, 2022, 13, 1360-1372.	2.9	22
35	Systemic Delivery of Morpholinos to Skip Multiple Exons in a Dog Model of Duchenne Muscular Dystrophy. Methods in Molecular Biology, 2017, 1565, 201-213.	0.4	19
36	Application of Urine-Derived Stem Cells to Cellular Modeling in Neuromuscular and Neurodegenerative Diseases. Frontiers in Molecular Neuroscience, 2019, 12, 297.	1.4	19

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37	Emerging Oligonucleotide Therapeutics for Rare Neuromuscular Diseases. Journal of Neuromuscular Diseases, 2021, 8, 869-884.	1.1	19
38	Modelling Duchenne muscular dystrophy in MYOD1-converted urine-derived cells treated with 3-deazaneplanocin A hydrochloride. Scientific Reports, 2019, 9, 3807.	1.6	18
39	Multi-exon Skipping Using Cocktail Antisense Oligonucleotides in the Canine X-linked Muscular Dystrophy. Journal of Visualized Experiments, 2016, , .	0.2	16
40	Solid-Phase Synthesis of Difficult Purine-Rich PNAs through Selective Hmb Incorporation: Application to the Total Synthesis of Cell Penetrating Peptide-PNAs. Frontiers in Chemistry, 2017, 5, 81.	1.8	15
41	Supramolecular Assembly of Aminoethyleneâ€Lipopeptide PMO Conjugates into RNA Spliceâ€Switching Nanomicelles. Advanced Functional Materials, 2019, 29, 1906432.	7.8	14
42	Potential Therapies Using Myogenic Stem Cells Combined with Bio-Engineering Approaches for Treatment of Muscular Dystrophies. Cells, 2019, 8, 1066.	1.8	14
43	Amelioration of intracellular Ca2+ regulation by exon-45 skipping in Duchenne muscular dystrophy-induced pluripotent stem cell-derived cardiomyocytes. Biochemical and Biophysical Research Communications, 2019, 520, 179-185.	1.0	14
44	Exon Skipping Therapy Using Phosphorodiamidate Morpholino Oligomers in the mdx52 Mouse Model of Duchenne Muscular Dystrophy. Methods in Molecular Biology, 2018, 1687, 123-141.	0.4	13
45	eSkip-Finder: a machine learning-based web application and database to identify the optimal sequences of antisense oligonucleotides for exon skipping. Nucleic Acids Research, 2021, 49, W193-W198.	6.5	13
46	The nSMase2/Smpd3 gene modulates the severity of muscular dystrophy and the emotional stress response in mdx mice. BMC Medicine, 2020, 18, 343.	2.3	12
47	Brain Dp140 alters glutamatergic transmission and social behaviour in the mdx52 mouse model of Duchenne muscular dystrophy. Progress in Neurobiology, 2022, 216, 102288.	2.8	12
48	Immortalized Canine Dystrophic Myoblast Cell Lines for Development of Peptide-Conjugated Splice-Switching Oligonucleotides. Nucleic Acid Therapeutics, 2021, 31, 172-181.	2.0	9
49	Oligonucleotide-Based Therapy for FTD/ALS Caused by theC9orf72Repeat Expansion: A Perspective. Journal of Nucleic Acids, 2013, 2013, 1-11.	0.8	8
50	Age-Dependent Echocardiographic and Pathologic Findings in a Rat Model with Duchenne Muscular Dystrophy Generated by CRISPR/Cas9 Genome Editing. International Heart Journal, 2020, 61, 1279-1284.	0.5	8
51	Accelerometric outcomes of motor function related to clinical evaluations and muscle involvement in dystrophic dogs. PLoS ONE, 2018, 13, e0208415.	1.1	6
52	Size-tunable PEG-grafted copolymers as a polymeric nanoruler for passive targeting muscle tissues. Journal of Controlled Release, 2022, 347, 607-614.	4.8	6
53	Dystrobrevin alpha gene is a direct target of the vitamin D receptor in muscle. Journal of Molecular Endocrinology, 2020, 64, 195-208.	1.1	5
54	Lipidomic Analyses Reveal Specific Alterations of Phosphatidylcholine in Dystrophic Mdx Muscle. Frontiers in Physiology, 2021, 12, 698166.	1.3	5

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55	Development of Therapeutic RNA Manipulation for Muscular Dystrophy. Frontiers in Genome Editing, 0, 4, .	2.7	5
56	In Vivo Evaluation of Single-Exon and Multiexon Skipping in mdx52 Mice. Methods in Molecular Biology, 2018, 1828, 275-292.	0.4	4
57	Exon Skipping in Directly Reprogrammed Myotubes Obtained from Human Urine-Derived Cells. Journal of Visualized Experiments, 2020, , .	0.2	4
58	In Vivo Evaluation of Multiple Exon Skipping with Peptide-PMOs in Cardiac and Skeletal Muscles in Dystrophic Dogs. Methods in Molecular Biology, 2018, 1828, 365-379.	0.4	3
59	Exon Skipping Using Antisense Oligonucleotides for Laminin-Alpha2-Deficient Muscular Dystrophy. Methods in Molecular Biology, 2018, 1828, 553-564.	0.4	3
60	Novel EGFP reporter cell and mouse models for sensitive imaging and quantification of exon skipping. Scientific Reports, 2020, 10, 10110.	1.6	3
61	Severe cardiac involvement with preserved truncated dystrophin expression in Becker muscular dystrophy by +1G>A DMD splice-site mutation: a case report. Journal of Human Genetics, 2020, 65, 903-909.	1.1	3
62	Highly sensitive screening of antisense sequences for different types of DMD mutations in patients' urine-derived cells. Journal of the Neurological Sciences, 2021, 423, 117337.	0.3	3
63	Oligonucleotide therapies: the future of amyotrophic lateral sclerosis treatment?. Neurodegenerative Disease Management, 2015, 5, 93-95.	1.2	2
64	In Vitro Multiexon Skipping by Antisense PMOs in Dystrophic Dog and Exon 7-Deleted DMD Patient. Methods in Molecular Biology, 2018, 1828, 151-163.	0.4	2
65	Autoimmune response and its longâ€term consequences after exonâ€skipping therapy in a Duchenne muscular dystrophy mouse model. Journal of Pathology, 2019, 249, 271-273.	2.1	2
66	Development of outcome measures according to dystrophic phenotypes in canine X-linked muscular dystrophy in Japan. Experimental Animals, 2021, 70, 419-430.	0.7	2
67	G.P.85. Neuromuscular Disorders, 2014, 24, 820.	0.3	1
68	Development of LNA Gapmer Oligonucleotide-Based Therapy for ALS/FTD Caused by the C9orf72 Repeat Expansion. Methods in Molecular Biology, 2020, 2176, 185-208.	0.4	1
69	P3.04 Skipping of exons 6 and 8 of the DMD gene has been achieved in myogenic cells from an exon-7 deleted DMD patient: direct application of antisense sequences found in study with canine muscular dystrophy. Neuromuscular Disorders, 2010, 20, 641-642.	0.3	0
70	Challenges for antisense oligonucleotide-based therapeutics, in particular for exon 51-skipping in Duchenne muscular dystrophy. , 2011, , .		0
71	741. Development of LNA Gapmer Oligonucleotide Based Therapy for FTD/ALS Caused by the C9orf72 Repeat Expansion. Molecular Therapy, 2016, 24, S292.	3.7	0
72	Comparative high resolution proteomic analysis of dystrophic mouse models reveals a core dystrophic proteome and the impact of aging. Neuromuscular Disorders, 2017, 27, S167-S168.	0.3	0

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73	Dystrophin-deficient cardiomyocyte derived from Duchenne Muscular Dystrophy specific induced pluripotent stem cells carrying the deletion of exon 46-55 in DMD gene. Journal of the Neurological Sciences, 2017, 381, 859.	0.3	0
74	A symptomatic male carrier of Duchenne muscular dystrophy with Klinefelter's syndrome mimicking Becker muscular dystrophy. Neuromuscular Disorders, 2021, 31, 666-672.	0.3	0
75	Fine Tuning of Phosphorothioate Inclusion in 2â€²-O-Methyl Oligonucleotides Contributes to Specific Cell Targeting for Splice-Switching Modulation. Frontiers in Physiology, 2021, 12, 689179.	1.3	0
76	Synthesis and biological properties of 2'-O-modified oligoribonucleotide derivatives. , 2011, , .		0
77	New Approach for Antisense Oligonucleotide-Mediated Exon Skipping in Duchenne Muscular Dystrophy. Journal of Advanced Computational Intelligence and Intelligent Informatics, 2012, 16, 521-526.	0.5	0
78	Characterizing Exon Skipping Efficiency in DMD Patient Samples in Clinical Trials of Antisense Oligonucleotides. Journal of Visualized Experiments, 2020, , .	0.2	0
79	Editorial: Challenges and Opportunities for Neuromuscular Disease Modelling Using Urine-derived Stem Cells. Frontiers in Physiology, 2022, 13, 848220.	1.3	0