

Yoshitsugu Aoki

List of Publications by Year in descending order

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Version: 2024-02-01

79
papers

2,418
citations

236612

25
h-index

214527

47
g-index

81
all docs

81
docs citations

81
times ranked

2775
citing authors

#	ARTICLE	IF	CITATIONS
1	Investigating the role of dystrophin isoform deficiency in motor function in Duchenne muscular dystrophy. <i>Journal of Cachexia, Sarcopenia and Muscle</i> , 2022, 13, 1360-1372.	2.9	22
2	Editorial: Challenges and Opportunities for Neuromuscular Disease Modelling Using Urine-derived Stem Cells. <i>Frontiers in Physiology</i> , 2022, 13, 848220.	1.3	0
3	Brain Dp140 alters glutamatergic transmission and social behaviour in the mdx52 mouse model of Duchenne muscular dystrophy. <i>Progress in Neurobiology</i> , 2022, 216, 102288.	2.8	12
4	Size-tunable PEG-grafted copolymers as a polymeric nanoruler for passive targeting muscle tissues. <i>Journal of Controlled Release</i> , 2022, 347, 607-614.	4.8	6
5	Development of outcome measures according to dystrophic phenotypes in canine X-linked muscular dystrophy in Japan. <i>Experimental Animals</i> , 2021, 70, 419-430.	0.7	2
6	Immortalized Canine Dystrophic Myoblast Cell Lines for Development of Peptide-Conjugated Splice-Switching Oligonucleotides. <i>Nucleic Acid Therapeutics</i> , 2021, 31, 172-181.	2.0	9
7	Pharmacological activation of SERCA ameliorates dystrophic phenotypes in dystrophin-deficient <i>mdx</i> mice. <i>Human Molecular Genetics</i> , 2021, 30, 1006-1019.	1.4	28
8	Highly sensitive screening of antisense sequences for different types of DMD mutations in patients' urine-derived cells. <i>Journal of the Neurological Sciences</i> , 2021, 423, 117337.	0.3	3
9	eSkip-Finder: a machine learning-based web application and database to identify the optimal sequences of antisense oligonucleotides for exon skipping. <i>Nucleic Acids Research</i> , 2021, 49, W193-W198.	6.5	13
10	Emerging Oligonucleotide Therapeutics for Rare Neuromuscular Diseases. <i>Journal of Neuromuscular Diseases</i> , 2021, 8, 869-884.	1.1	19
11	A symptomatic male carrier of Duchenne muscular dystrophy with Klinefelter's syndrome mimicking Becker muscular dystrophy. <i>Neuromuscular Disorders</i> , 2021, 31, 666-672.	0.3	0
12	Fine Tuning of Phosphorothioate Inclusion in 2'-O-Methyl Oligonucleotides Contributes to Specific Cell Targeting for Splice-Switching Modulation. <i>Frontiers in Physiology</i> , 2021, 12, 689179.	1.3	0
13	Lipidomic Analyses Reveal Specific Alterations of Phosphatidylcholine in Dystrophic Mdx Muscle. <i>Frontiers in Physiology</i> , 2021, 12, 698166.	1.3	5
14	Mutation-independent Proteomic Signatures of Pathological Progression in Murine Models of Duchenne Muscular Dystrophy. <i>Molecular and Cellular Proteomics</i> , 2020, 19, 2047-2068.	2.5	25
15	The nSMase2/Smpd3 gene modulates the severity of muscular dystrophy and the emotional stress response in mdx mice. <i>BMC Medicine</i> , 2020, 18, 343.	2.3	12
16	Exon Skipping in Directly Reprogrammed Myotubes Obtained from Human Urine-Derived Cells. <i>Journal of Visualized Experiments</i> , 2020, , .	0.2	4
17	Novel EGFP reporter cell and mouse models for sensitive imaging and quantification of exon skipping. <i>Scientific Reports</i> , 2020, 10, 10110.	1.6	3
18	Severe cardiac involvement with preserved truncated dystrophin expression in Becker muscular dystrophy by +1G>A DMD splice-site mutation: a case report. <i>Journal of Human Genetics</i> , 2020, 65, 903-909.	1.1	3

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19	Dystrobrevin alpha gene is a direct target of the vitamin D receptor in muscle. <i>Journal of Molecular Endocrinology</i> , 2020, 64, 195-208.	1.1	5
20	Age-Dependent Echocardiographic and Pathologic Findings in a Rat Model with Duchenne Muscular Dystrophy Generated by CRISPR/Cas9 Genome Editing. <i>International Heart Journal</i> , 2020, 61, 1279-1284.	0.5	8
21	Characterizing Exon Skipping Efficiency in DMD Patient Samples in Clinical Trials of Antisense Oligonucleotides. <i>Journal of Visualized Experiments</i> , 2020, , .	0.2	0
22	Development of LNA Gapmer Oligonucleotide-Based Therapy for ALS/FTD Caused by the C9orf72 Repeat Expansion. <i>Methods in Molecular Biology</i> , 2020, 2176, 185-208.	0.4	1
23	Autoimmune response and its long-term consequences after exon-skipping therapy in a Duchenne muscular dystrophy mouse model. <i>Journal of Pathology</i> , 2019, 249, 271-273.	2.1	2
24	Peptide-conjugate antisense based splice-correction for Duchenne muscular dystrophy and other neuromuscular diseases. <i>EBioMedicine</i> , 2019, 45, 630-645.	2.7	61
25	Supramolecular Assembly of Aminoethylene-Lipopeptide PMO Conjugates into RNA Splice-switching Nanomicelles. <i>Advanced Functional Materials</i> , 2019, 29, 1906432.	7.8	14
26	Restoring Dystrophin Expression in Duchenne Muscular Dystrophy: Current Status of Therapeutic Approaches. <i>Journal of Personalized Medicine</i> , 2019, 9, 1.	1.1	84
27	Exons 45-55 Skipping Using Mutation-Tailored Cocktails of Antisense Morpholinos in the DMD Gene. <i>Molecular Therapy</i> , 2019, 27, 2005-2017.	3.7	35
28	Potential Therapies Using Myogenic Stem Cells Combined with Bio-Engineering Approaches for Treatment of Muscular Dystrophies. <i>Cells</i> , 2019, 8, 1066.	1.8	14
29	Amelioration of intracellular Ca ²⁺ 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
30	Modelling Duchenne muscular dystrophy in MYOD1-converted urine-derived cells treated with 3-deazaneplanocin A hydrochloride. <i>Scientific Reports</i> , 2019, 9, 3807.	1.6	18
31	Scavenger Receptor Class A1 Mediates Uptake of Morpholino Antisense Oligonucleotide into Dystrophic Skeletal Muscle. <i>Molecular Therapy - Nucleic Acids</i> , 2019, 14, 520-535.	2.3	22
32	Application of Urine-Derived Stem Cells to Cellular Modeling in Neuromuscular and Neurodegenerative Diseases. <i>Frontiers in Molecular Neuroscience</i> , 2019, 12, 297.	1.4	19
33	Efficacy of Multi-exon Skipping Treatment in Duchenne Muscular Dystrophy Dog Model Neonates. <i>Molecular Therapy</i> , 2019, 27, 76-86.	3.7	24
34	Systemic administration of the antisense oligonucleotide NS-065/NCNP-01 for skipping of exon 53 in patients with Duchenne muscular dystrophy. <i>Science Translational Medicine</i> , 2018, 10, .	5.8	111
35	Exon Skipping Therapy Using Phosphorodiamidate Morpholino Oligomers in the mdx52 Mouse Model of Duchenne Muscular Dystrophy. <i>Methods in Molecular Biology</i> , 2018, 1687, 123-141.	0.4	13
36	Accelerometric outcomes of motor function related to clinical evaluations and muscle involvement in dystrophic dogs. <i>PLoS ONE</i> , 2018, 13, e0208415.	1.1	6

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37	Truncated dystrophin ameliorates the dystrophic phenotype of mdx mice by reducing sarcolipin-mediated SERCA inhibition. <i>Biochemical and Biophysical Research Communications</i> , 2018, 505, 51-59.	1.0	24
38	In Vivo Evaluation of Single-Exon and Multiexon Skipping in mdx52 Mice. <i>Methods in Molecular Biology</i> , 2018, 1828, 275-292.	0.4	4
39	In Vivo Evaluation of Multiple Exon Skipping with Peptide-PMOs in Cardiac and Skeletal Muscles in Dystrophic Dogs. <i>Methods in Molecular Biology</i> , 2018, 1828, 365-379.	0.4	3
40	Exon Skipping Using Antisense Oligonucleotides for Laminin-Alpha2-Deficient Muscular Dystrophy. <i>Methods in Molecular Biology</i> , 2018, 1828, 553-564.	0.4	3
41	In Vitro Multiexon Skipping by Antisense PMOs in Dystrophic Dog and Exon 7-Deleted DMD Patient. <i>Methods in Molecular Biology</i> , 2018, 1828, 151-163.	0.4	2
42	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
43	C9orf72 and RAB7L1 regulate vesicle trafficking in amyotrophic lateral sclerosis and frontotemporal dementia. <i>Brain</i> , 2017, 140, 887-897.	3.7	126
44	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
45	Effects of systemic multiexon skipping with peptide-conjugated morpholinos in the heart of a dog model of Duchenne muscular dystrophy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, 4213-4218.	3.3	94
46	Quantitative Antisense Screening and Optimization for Exon 51 Skipping in Duchenne Muscular Dystrophy. <i>Molecular Therapy</i> , 2017, 25, 2561-2572.	3.7	63
47	Comparative high resolution proteomic analysis of dystrophic mouse models reveals a core dystrophic proteome and the impact of aging. <i>Neuromuscular Disorders</i> , 2017, 27, S167-S168.	0.3	0
48	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
49	Solid-Phase Synthesis of Difficult Purine-Rich PNAs through Selective Hmb Incorporation: Application to the Total Synthesis of Cell Penetrating Peptide-PNAs. <i>Frontiers in Chemistry</i> , 2017, 5, 81.	1.8	15
50	Anti-inflammatory drugs for Duchenne muscular dystrophy: focus on skeletal muscle-releasing factors. <i>Drug Design, Development and Therapy</i> , 2016, Volume 10, 2745-2758.	2.0	54
51	Endogenous Multiple Exon Skipping and Back-Splicing at the DMD Mutation Hotspot. <i>International Journal of Molecular Sciences</i> , 2016, 17, 1722.	1.8	35
52	741. Development of LNA Gapmer Oligonucleotide Based Therapy for FTD/ALS Caused by the C9orf72 Repeat Expansion. <i>Molecular Therapy</i> , 2016, 24, S292.	3.7	0
53	Multi-exon Skipping Using Cocktail Antisense Oligonucleotides in the Canine X-linked Muscular Dystrophy. <i>Journal of Visualized Experiments</i> , 2016, , .	0.2	16
54	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. <i>Journal of Human Genetics</i> , 2016, 61, 663-667.	1.1	45

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55	Serum Osteopontin as a Novel Biomarker for Muscle Regeneration in Duchenne Muscular Dystrophy. <i>American Journal of Pathology</i> , 2016, 186, 1302-1312.	1.9	27
56	Recent advances in innovative therapeutic approaches for Duchenne muscular dystrophy: from discovery to clinical trials. <i>American Journal of Translational Research (discontinued)</i> , 2016, 8, 2471-89.	0.0	57
57	Oligonucleotide therapies: the future of amyotrophic lateral sclerosis treatment?. <i>Neurodegenerative Disease Management</i> , 2015, 5, 93-95.	1.2	2
58	Self-Assembly into Nanoparticles Is Essential for Receptor Mediated Uptake of Therapeutic Antisense Oligonucleotides. <i>Nano Letters</i> , 2015, 15, 4364-4373.	4.5	80
59	Long-Term Efficacy of Systemic Multiexon Skipping Targeting Dystrophin Exons 45-55 With a Cocktail of Vivo-Morpholinos in Mdx52 Mice. <i>Molecular Therapy - Nucleic Acids</i> , 2015, 4, e225.	2.3	67
60	Three novel serum biomarkers, miR-1, miR-133a, and miR-206 for Limb-girdle muscular dystrophy, Facioscapulohumeral muscular dystrophy, and Becker muscular dystrophy. <i>Environmental Health and Preventive Medicine</i> , 2014, 19, 452-458.	1.4	86
61	G.P.85. <i>Neuromuscular Disorders</i> , 2014, 24, 820.	0.3	1
62	Extracellular microRNAs are dynamic non-vesicular biomarkers of muscle turnover. <i>Nucleic Acids Research</i> , 2013, 41, 9500-9513.	6.5	83
63	Development of Multiexon Skipping Antisense Oligonucleotide Therapy for Duchenne Muscular Dystrophy. <i>BioMed Research International</i> , 2013, 2013, 1-8.	0.9	45
64	Highly efficient in vivo delivery of PMO into regenerating myotubes and rescue in laminin-Î±2 chain-null congenital muscular dystrophy mice. <i>Human Molecular Genetics</i> , 2013, 22, 4914-4928.	1.4	59
65	Identification of Disease Specific Pathways Using in Vivo SILAC Proteomics in Dystrophin Deficient mdx Mouse. <i>Molecular and Cellular Proteomics</i> , 2013, 12, 1061-1073.	2.5	88
66	Mutation Types and Aging Differently Affect Revertant Fiber Expansion in Dystrophic Mdx and Mdx52 Mice. <i>PLoS ONE</i> , 2013, 8, e69194.	1.1	26
67	Oligonucleotide-Based Therapy for FTD/ALS Caused by the C9orf72 Repeat Expansion: A Perspective. <i>Journal of Nucleic Acids</i> , 2013, 2013, 1-11.	0.8	8
68	Bodywide skipping of exons 45-55 in dystrophic mdx52 mice by systemic antisense delivery. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2012, 109, 13763-13768.	3.3	139
69	Extensive and Prolonged Restoration of Dystrophin Expression with Vivo-Morpholino-Mediated Multiple Exon Skipping in Dystrophic Dogs. <i>Nucleic Acid Therapeutics</i> , 2012, 22, 306-315.	2.0	69
70	New Approach for Antisense Oligonucleotide-Mediated Exon Skipping in Duchenne Muscular Dystrophy. <i>Journal of Advanced Computational Intelligence and Intelligent Informatics</i> , 2012, 16, 521-526.	0.5	0
71	Synthesis of 2'-O-[2-(N-Methylcarbamoyl)ethyl]ribonucleosides Using Oxa-Michael Reaction and Chemical and Biological Properties of Oligonucleotide Derivatives Incorporating These Modified Ribonucleosides. <i>Journal of Organic Chemistry</i> , 2011, 76, 3042-3053.	1.7	32
72	Challenges for antisense oligonucleotide-based therapeutics, in particular for exon 51-skipping in Duchenne muscular dystrophy. , 2011, , .		0

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73	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
74	Synthesis and biological properties of 2'-O-modified oligoribonucleotide derivatives. , 2011, , .		0
75	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
76	In-frame Dystrophin Following Exon 51-Skipping Improves Muscle Pathology and Function in the Exon 52-Deficient mdx Mouse. Molecular Therapy, 2010, 18, 1995-2005.	3.7	118
77	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
78	Relationship between diffusion tensor imaging and brain morphology in patients with myotonic dystrophy. Neuroscience Letters, 2006, 407, 234-239.	1.0	50
79	Development of Therapeutic RNA Manipulation for Muscular Dystrophy. Frontiers in Genome Editing, 0, 4, .	2.7	5