Masayuki Nakamori

List of Publications by Year
in descending order

61	3,456	159585 30	144013 57
papers	citations	h-index	g-index

65	65	65	3247
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Targeting nuclear RNA for in vivo correction of myotonic dystrophy. Nature, 2012, 488, 111-115.	27.8	435
2	Altered mRNA splicing of the skeletal muscle ryanodine receptor and sarcoplasmic/endoplasmic reticulum Ca2+-ATPase in myotonic dystrophy type 1. Human Molecular Genetics, 2005, 14, 2189-2200.	2.9	247
3	Pentamidine reverses the splicing defects associated with myotonic dystrophy. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 18551-18556.	7.1	234
4	Splicing biomarkers of disease severity in myotonic dystrophy. Annals of Neurology, 2013, 74, 862-872.	5.3	215
5	Splicing misregulation of SCN5A contributes to cardiac-conduction delay and heart arrhythmia in myotonic dystrophy. Nature Communications, 2016, 7, 11067.	12.8	155
6	Muscle weakness in myotonic dystrophy associated with misregulated splicing and altered gating of CaV1.1 calcium channel. Human Molecular Genetics, 2012, 21, 1312-1324.	2.9	146
7	Expanded CTG repeat demarcates a boundary for abnormal CpG methylation in myotonic dystrophy patient tissues. Human Molecular Genetics, 2011, 20, 1-15.	2.9	129
8	Design of a Bioactive Small Molecule That Targets the Myotonic Dystrophy Type 1 RNA via an RNA Motifâ€“Ligand Database and Chemical Similarity Searching. Journal of the American Chemical Society, 2012, 134, 4731-4742.	13.7	129
9	A slipped-CAG DNA-binding small molecule induces trinucleotide-repeat contractions in vivo. Nature Genetics, 2020, 52, 146-159.	21.4	110
10	Bidirectional transcription stimulates expansion and contraction of expanded (CTG)â€¢(CAG) repeats. Human Molecular Genetics, 2011, 20, 580-588.	2.9	99
11	Molecular mechanisms responsible for aberrant splicing of SERCA1 in myotonic dystrophy type 1. Human Molecular Genetics, 2007, 16, 2834-2843.	2.9	92
12	Disrupted prenatal RNA processing and myogenesis in congenital myotonic dystrophy. Genes and Development, 2017, 31, 1122-1133.	5.9	80
13	Muscleblind-Like 1 Knockout Mice Reveal Novel Splicing Defects in the Myotonic Dystrophy Brain. PLoS ONE, 2012, 7, e33218.	2.5	79
14	Actinomycin D Specifically Reduces Expanded CUG Repeat RNA in Myotonic Dystrophy Models. Cell Reports, 2015, 13, 2386-2394.	6.4	74
15	Reducing Levels of Toxic RNA with Small Molecules. ACS Chemical Biology, 2013, 8, 2528-2537.	3.4	71
16	From dynamic combinatorial â€˜hitâ€™ to lead: in vitro and in vivo activity of compounds targeting the pathogenic RNAs that cause myotonic dystrophy. Nucleic Acids Research, 2012, 40, 6380-6390.	14.5	69
17	Stabilization of Expanded (CTG)â€¢(CAG) Repeats by Antisense Oligonucleotides. Molecular Therapy, 2011, 19, 2222-2227.	8.2	65
18	Amido-bridged nucleic acid (AmNA)-modified antisense oligonucleotides targeting Î±-synuclein as a novel therapy for Parkinsonâ€™s disease. Scientific Reports, 2019, 9, 7567.	3.3	65

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19	Detection of Slipped-DNAs at the Trinucleotide Repeats of the Myotonic Dystrophy Type I Disease Locus in Patient Tissues. PLoS Genetics, 2013, 9, e1003866.	3.5	62
20	A Kir3.4 mutation causes Andersenâ€“Tawil syndrome by an inhibitory effect on Kir2.1. Neurology, 2014, 82, 1058-1064.	1.1	59
21	Rational Design of Bioactive, Modularly Assembled Aminoglycosides Targeting the RNA that Causes Myotonic Dystrophy Type 1. ACS Chemical Biology, 2012, 7, 1984-1993.	3.4	57
22	Oral administration of erythromycin decreases <scp>RNA</scp> toxicity in myotonic dystrophy. Annals of Clinical and Translational Neurology, 2016, 3, 42-54.	3.7	55
23	Endoplasmic reticulum stress in myotonic dystrophy type 1 muscle. Acta Neuropathologica, 2007, 114, 527-535.	7.7	52
24	Lomofungin and dilomofungin: inhibitors of MBNL1-CUG RNA binding with distinct cellular effects. Nucleic Acids Research, 2014, 42, 6591-6602.	14.5	46
25	Nucleic Acidâ€“Based Therapeutics for Parkinson's Disease. Neurotherapeutics, 2019, 16, 287-298.	4.4	45
26	Altered mRNA splicing of dystrophin in type 1 myotonic dystrophy. Muscle and Nerve, 2007, 36, 251-257.	2.2	42
27	Aberrant Myokine Signaling in Congenital Myotonic Dystrophy. Cell Reports, 2017, 21, 1240-1252.	6.4	40
28	Large expansion of CTGâ€¢CAG repeats is exacerbated by MutSÎ² in human cells. Scientific Reports, 2015, 5, 11020.	3.3	37
29	Myotonic dystrophy type 1 patient-derived iPSCs for the investigation of CTG repeat instability. Scientific Reports, 2017, 7, 42522.	3.3	34
30	Epigenetic changes and non-coding expanded repeats. Neurobiology of Disease, 2010, 39, 21-27.	4.4	32
31	Biological Efficacy and Toxicity of Diamidines in Myotonic Dystrophy Type 1 Models. Journal of Medicinal Chemistry, 2015, 58, 5770-5780.	6.4	31
32	Alternative splicing of clathrin heavy chain contributes to the switch from coated pits to plaques. Journal of Cell Biology, 2020, 219, .	5.2	31
33	The Role of Alpha-Dystrobrevin in Striated Muscle. International Journal of Molecular Sciences, 2011, 12, 1660-1671.	4.1	27
34	A Dimeric 2,9â€Diaminoâ€1,10â€phenanthroline Derivative Improves Alternative Splicing in Myotonic Dystrophy Typeâ€…1 Cell and Mouse Models. Chemistry - A European Journal, 2018, 24, 18115-18122.	3.3	27
35	Furamidine Rescues Myotonic Dystrophy Type I Associated Mis-Splicing through Multiple Mechanisms. ACS Chemical Biology, 2018, 13, 2708-2718.	3.4	26
36	Human Genomic Safe Harbors and the Suicide Gene-Based Safeguard System for iPSC-Based Cell Therapy. Stem Cells Translational Medicine, 2019, 8, 627-638.	3.3	26

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37	Improvement of Anti-Hu-associated Paraneoplastic Sensory Neuropathy after Chemoradiotherapy in a Small Cell Lung Cancer Patient.. Internal Medicine, 2001, 40, 1140-1143.	0.7	21
38	Scaled-down genetic analysis of myotonic dystrophy type 1 and type 2. Neuromuscular Disorders, 2009, 19, 759-762.	0.6	21
39	Combination Treatment of Erythromycin and Furamidine Provides Additive and Synergistic Rescue of Mis-splicing in Myotonic Dystrophy Type 1 Models. ACS Pharmacology and Translational Science, 2019, 2, 247-263.	4.9	20
40	A CTG repeat-selective chemical screen identifies microtubule inhibitors as selective modulators of toxic CUG RNA levels. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 20991-21000.	7.1	20
41	FAN1 exo- not endo-nuclease pausing on disease-associated slipped-DNA repeats: A mechanism of repeat instability. Cell Reports, 2021, 37, 110078.	6.4	19
42	Identification of restriction endonucleases sensitive to 5-cytosine methylation at non-CpG sites, including expanded (CAG)n/(CTG)n repeats. Epigenetics, 2011, 6, 416-420.	2.7	13
43	Sacsin-related ataxia caused by the novel nonsense mutation Arg4325X. Journal of Neurology, 2006, 253, 1372-1373.	3.6	12
44	CAG repeat-binding small molecule improves motor coordination impairment in a mouse model of Dentatorubralâ€“pallidoluysian atrophy. Neurobiology of Disease, 2022, 163, 105604.	4.4	11
45	Modulating RNA secondary and tertiary structures by mismatch binding ligands. Methods, 2019, 167, 78-91.	3.8	10
46	The Dimeric Form of 1,3â€Diaminoisoquinoline Derivative Rescued the Misâ€splicing of Atp2a1 and Clcn1 Genes in Myotonic Dystrophy Typeâ€…1 Mouse Model. Chemistry - A European Journal, 2020, 26, 14305-14309.	3.3	10
47	Macroscopic and microscopic diversity of missplicing in the central nervous system of patients with myotonic dystrophy type 1. NeuroReport, 2018, 29, 235-240.	1.2	8
48	Straightjacket/Î±2Î´3 deregulation is associated with cardiac conduction defects in myotonic dystrophy type 1. ELife, 2019, 8, .	6.0	8
49	Replacement of the myotonic dystrophy type 1 CTG repeat with 'non-CTG repeat' insertions in specific tissues. Journal of Medical Genetics, 2011, 48, 438-443.	3.2	7
50	Targeting Expanded Repeats by Small Molecules in Repeat Expansion Disorders. Movement Disorders, 2021, 36, 298-305.	3.9	7
51	Alphaâ€synuclein dynamics in induced pluripotent stem cellâ€derived dopaminergic neurons from a Parkinsonâ€™s disease patient (PARK4) with SNCA triplication. FEBS Open Bio, 2021, 11, 354-366.	2.3	7
52	Renal dysfunction can be a common complication in patients with myotonic dystrophy 1. Journal of the Neurological Sciences, 2016, 368, 266-271.	0.6	6
53	Cell type-specific abnormalities of central nervous system in myotonic dystrophy type 1. Brain Communications, 2022, 4, .	3.3	6
54	The myotonic dystrophy health index: Japanese adaption and validity testing. Muscle and Nerve, 2019, 59, 577-582.	2.2	5

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55	Cellular Senescence and Aging in Myotonic Dystrophy. International Journal of Molecular Sciences, 2022, 23, 2339.	4.1	5
56	Myotonic Dystrophy. , 2016, , 39-61.		4
57	Expanded CUG Repeat RNA Induces Premature Senescence in Myotonic Dystrophy Model Cells. Frontiers in Genetics, 2022, 13, 865811.	2.3	4
58	A hidden ancestral legacy trumped. Nature, 2011, 478, 46-47.	27.8	0
59	Therapeutic Development in Myotonic Dystrophy. , 2018, , 203-214.		0
60	Therapeutic approach for myotonic dystrophy: Recent advances in translational research. Neurology and Clinical Neuroscience, 0, , .	0.4	0
61	Pharmacotherapy alleviates pathological changes in human direct reprogrammed neuronal cell model of myotonic dystrophy type 1. PLoS ONE, 2022, 17, e0269683.	2.5	0