Thomas K Doktor

List of Publications by Year
in descending order

25	1,165	643344 15	651938 25
papers	citations	h-index	g-index

27	27	27	2360
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Pseudoexon activation in disease by nonâ€splice site deep intronic sequence variation â€" wild type pseudoexons constitute highâ€risk sites in the human genome. Human Mutation, 2022, 43, 103-127.	1.1	17
2	VEGFA-targeting miR-agshRNAs combine efficacy with specificity and safety for retinal gene therapy. Molecular Therapy - Nucleic Acids, 2022, 28, 58-76.	2.3	6
3	Vulnerable exons, like ACADM exon 5, are highly dependent on maintaining a correct balance between splicing enhancers and silencers. Human Mutation, 2022, 43, 253-265.	1.1	11
4	Essential role of CK2Î± for the interaction and stability of replication fork factors during DNA synthesis and activation of the S-phase checkpoint. Cellular and Molecular Life Sciences, 2022, 79, .	2.4	2
5	Antisense Oligonucleotide Rescue of Deep-Intronic Variants Activating Pseudoexons in the 6-Pyruvoyl-Tetrahydropterin Synthase Gene. Nucleic Acid Therapeutics, 2022, 32, 378-390.	2.0	7
6	Identification of SRSF10 as a regulator of SMN2 ISSâ€N1. Human Mutation, 2021, 42, 246-260.	1.1	15
7	Tissue-resident macrophages in omentum promote metastatic spread of ovarian cancer. Journal of Experimental Medicine, 2020, 217, .	4.2	189
8	DeepCLIP: predicting the effect of mutations on proteinâ€"RNA binding with deep learning. Nucleic Acids Research, 2020, 48, 7099-7118.	6.5	54
9	Down-regulation of CK2Î± correlates with decreased expression levels of DNA replication minichromosome maintenance protein complex (MCM) genes. Scientific Reports, 2019, 9, 14581.	1.6	5
10	Next generation sequencing of RNA reveals novel targets of resveratrol with possible implications for Canavan disease. Molecular Genetics and Metabolism, 2019, 126, 64-76.	0.5	16
11	Blocking of an intronic splicing silencer completely rescues IKBKAP exon 20 splicing in familial dysautonomia patient cells. Nucleic Acids Research, 2018, 46, 7938-7952.	6.5	15
12	Splicing factor 1 modulates dietary restriction and TORC1 pathway longevity in C. elegans. Nature, 2017, 541, 102-106.	13.7	152
13	DFI-seq identification of environment-specific gene expression in uropathogenic Escherichia coli. BMC Microbiology, 2017, 17, 99.	1.3	5
14	RNA-sequencing of a mouse-model of spinal muscular atrophy reveals tissue-wide changes in splicing of U12-dependent introns. Nucleic Acids Research, 2017, 45, 395-416.	6.5	87
15	Global identification of hnRNP A1 binding sites for SSO-based splicing modulation. BMC Biology, 2016, 14, 54.	1.7	62
16	The prevalent deep intronic c. 639 + 919 G > A GLA mutation causes pseudoexon activation and Fabry disease by abolishing the binding of hnRNPA1 and hnRNP A2/B1 to a splicing silencer. Molecular Genetics and Metabolism, 2016, 119, 258-269.	0.5	23
17	Splice-shifting oligonucleotide (SSO) mediated blocking of an exonic splicing enhancer (ESE) created by the prevalent c.903+469T>C MTRR mutation corrects splicing and restores enzyme activity in patient cells. Nucleic Acids Research, 2015, 43, 4627-4639.	6.5	28
18	Absence of an Intron Splicing Silencer in Porcine Smn1 Intron 7 Confers Immunity to the Exon Skipping Mutation in Human SMN2. PLoS ONE, 2014, 9, e98841.	1.1	4

#	Article	IF	Citations
19	TheETFDHc.158A>G Variation Disrupts the Balanced Interplay of ESE- and ESS-Binding Proteins thereby Causing Missplicing and Multiple Acyl-CoA Dehydrogenation Deficiency. Human Mutation, 2014, 35, 86-95.	1.1	32
20	A synonymous polymorphic variation in ACADM exon 11 affects splicing efficiency and may affect fatty acid oxidation. Molecular Genetics and Metabolism, 2013, 110, 122-128.	0.5	22
21	CUGBP1 and MBNL1 preferentially bind to 3â€² UTRs and facilitate mRNA decay. Scientific Reports, 2012, 2, 209.	1.6	150
22	SMN2 exon 7 splicing is inhibited by binding of hnRNP A1 to a common ESS motif that spans the 3â€² splice site. Human Mutation, 2011, 32, 220-230.	1.1	41
23	The deep intronic c.903+469T>C mutation in the MTRR gene creates an SF2/ASF binding exonic splicing enhancer, which leads to pseudoexon activation and causes the cblE type of homocystinuria. Human Mutation, 2010, 31, 437-444.	1.1	53
24	The phenylalanine hydroxylase c.30C>G synonymous variation (p.G10G) creates a common exonic splicing silencer. Molecular Genetics and Metabolism, 2010, 100, 316-323.	0.5	23
25	Seemingly Neutral Polymorphic Variants May Confer Immunity to Splicing-Inactivating Mutations: A Synonymous SNP in Exon 5 of MCAD Protects from Deleterious Mutations in a Flanking Exonic Splicing Enhancer. American Journal of Human Genetics, 2007, 80, 416-432.	2.6	140