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Correction to: Neurodegenerative diseases: a hotbed for splicing defects and the potential therapies

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Correction to: Translational Neurodegeneration (2021) 10:16 https://doi.org/10.1186/s40035-021-00240-7

Following publication of the original article [1], the authors would like to correct a formula from "T > C" to "C > T" in two paragraphs.

1. In the third paragraph of the section Amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD), the correct sentence should be:

However, the synonymous C>T substitution in SMN2 exon 7 alters an exonic splicing enhancer into an exonic splicing silencer, which predominantly leads to an unstable transcript missing exon 7.

2. In the fourth paragraph of the section **Splice-switching AOs**, the correct sentence should be:

The C>T substitution in SMN2 creates an exon-splicing silencer and leads to the omission of exon 7 and an unstable SMN protein that is subject to rapid ubiquitin-proteasome degradation.

In addition, the authors identified an error in Fig. 4. The correct figure is given below:

The original article [1] has been corrected.

The original article can be found online at https://doi.org/10.1186/s40035-021-00240-7.

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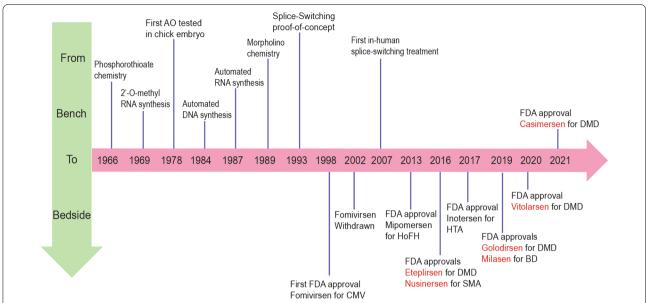


Fig. 4 Milestones of the development of antisense oligonucleotide therapeutics (excluding siRNA) from bench to bedside. Approved drugs in red are splice-switching antisense oligomers. AO: antisense oligonucleotides; FDA: US Food and Drug Administration; CMV: cytomegalovirus retinitis (in immunocompromised patients); HoFH: Homozygous familial hypercholesterolemia; DMD: Duchenne muscular dystrophy; SMA: spinal muscular atrophy; HTA: Hereditary transthyretin-mediated amyloidosis; BD: Batten disease

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Reference

1. Li, et al. Transl Neurodegener. 2021;10:16.