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We are writing to thank the Blazeman Foundation for ALS Research for your support towards our publication listed below. Using a new technique recently developed at Brandeis, we identified new targets for the ALS gene TDP-43 that may explain why it causes defects in the growth of neurons.

TDP-43 dysfunction restricts dendritic complexity by inhibiting CREB activation and altering gene expression. Herzog JJ, Xu W, Deshpande M, Rahman R, Suib H, Rodal AA, Rosbash M, Paradis S. Proc Natl Acad Sci U S A. 2020 May 11. pii: 201917038. doi: 10.1073/pnas.1917038117.

Copied below is an excerpt from the article:

"TDP-43 is an RNA-binding protein closely associated with neurodegenerative diseases, such as amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD). We have previously shown that TDP-43 dysfunction leads to reduced dendritic complexity. In this study, we apply the recently developed genetic approach TRIBE (targets of RNA-binding proteins identified by editing) to identify the RNAs that are bound by TDP-43 in neurons. Of the many TDP-43 target RNAs identified, we observed that mRNAs encoding proteins related to the CREB signaling pathway are enriched. Next, we were able to rescue the dendritic complexity defect caused by TDP-43 dysfunction by restoring CREB signaling. Our study may provide pathways for therapeutic intervention in TDP-43−associated neurodegenerative diseases."

Thank you so much for your support of our work,

Sincerely,

Avital A. Rodal