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Transcriptional Analysis of ER Stress and Autophagy Proteins in Peripheral Blood of Sporadic Amyotrophic Lateral Sclerosis Patients

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ABSTRACT: Amyotrophic Lateral Sclerosis (ALS) is a motor neurone disease that involves protein misfolding and aggregation. Majority (90%) of the cases are sporadic (sALS) and <10% are familial (fALS). Aggregates of mutant SOD1, TDP-43 and FUS are implicated in both fALS and sALS. Failure in cellular machinery involved in maintaining cellular proteostasis contributes to protein misfolding disorders. Further, investigations related to neurodegenerative diseases are done using cerebrospinal fluid. It is difficult to get consent from patients for these invasive procedures and especially for diseases like ALS which have no treatment. Several studies suggest transcriptional alterations in peripheral blood in neurodegenerative disorders. We are comparing the status of ER quality control machinery and protein degradation (autophagy and proteasome) pathways in sporadic ALS patients and age-matched healthy individuals. Our pilot data shows that Grp78, Grp94, ATG5, ATG12, Parkin and Ubqln2 are differentially expressed in ALS patients compared to controls. At present, we are analyzing the expression of these genes in more number of samples. The results will be elaborated in the meeting.

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