

**Name**

Adam Labadorf

**Email**

labadorf@bu.edu

**Institutional Affiliation**

Boston University

**Campus**

Medical Campus

**School**

Engineering

**Department**

Bioinformatics

**Position Held at Institution**

Graduate

**Poster Submissions****Poster Title**

Comparative analysis of Huntington and Parkinson disease transcriptome in post-mortem human brain identifies putative pan-neurodegenerative disease gene signature

**Authors and their Affiliation**

Adam Labadorf (BU Bioinformatics, Dept. of Neurology BUSM), Andrew Hoss (Program in Genetics and Genomics BUSM, Seung Hoan Choi (Biostatistics BUSM), Rick Myers (Dept. of Neurology BUSM)

**Please describe the extent of your work in this research**

I performed all of the sequence and statistical analysis and interpretation of the results, with guidance from the other authors.

**Abstract Submission**

- [HDvPD-GSI-Research-Symposium-abstract.docx](#)

**Would you like your abstract to be considered for an oral presentation (students and post docs only)?**

No

Huntington's Disease (HD) and Parkinson's Disease (PD) are neurodegenerative diseases that selectively affect different types of neurons in the brain yet share some common symptoms. While changes to the transcriptome in human brain have been characterized in both diseases separately, a comparative study of the two conditions may yield insight into the specific signatures of each disease as well as implicate common genes and pathways in neurodegeneration. In this study, we present a detailed analysis of the transcriptional changes in HD and PD human post-mortem prefrontal cortex compared to neuropathologically normal controls using high-throughput mRNA sequencing. Comparing the differentially expressed genes from the two conditions reveals functional gene signatures unique to each disease, a set of genes common to both, and a group of genes discovered only when the two disease datasets are analyzed together. The disease-specific signatures differ markedly in their function, transcriptional regulation, and miRNA targeting patterns as determined by pathway and geneset enrichment analysis. The common differentially expressed genes between PD and HD are enriched in the NFkB pathway, implicating the immune response in both conditions despite disease-specific differences in the degree of neurodegeneration of this brain area. Differentially expressed genes identified by contrasting controls to both diseases together are highly enriched in ribosomal components, suggesting ribosomal alterations may be a common feature of neurodegenerative disease. These results together suggest important similarities and differences between HD and PD that may be useful for designing therapies that ameliorate the pan-neurodegenerative phenotype.