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A Commentary on MicroRNA and Gene Expression Changes in Parkinson's Disease Patients with Blood Leukocytes

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Commentary

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ABOUT THE STUDY

Parkinson's Disease (PD) is a devastative late life disease with many unknown molecular mechanisms. Upon disease neurological diagnosis the majority of brain dopaminergic neurons have already been diminished. Notably, there are several similar pathologies (e.g. essential tremor, Progressive Supranuclear Palsy (PSP), Dementia with Lewy Bodies, Multiple System Atrophy (MSA) which complexes the disease accurate diagnosis. Deep Brain Stimulation (DBS) neurosurgery can significant ameliorate the disease motor symptoms including tremor (typically present in about 15% of patients). Blood leukocyte RNA from PD patients and age and gender matched Healthy Control (HC) volunteers can be explored by analysing microarray and total/single cell RNA sequencing data for detection of altered genes (e.g. differentially expressed/alternatively spliced/altered microRNAs). Additionally, target genes for microRNAs may be detected using targeted genomic databases. Here, describes the microarray study of microRNA changes in PD patients prior to and following DBS both on and following 1 hour off electrical stimulation cessation and as compared with healthy control volunteers.

MicroRNAs are short 21 nucleotide long genes which regulate target genes expression patterns through binding to the 3 Untranslated Regions (UTRs) typically. Several genes were identified as involved in the disease so far (e.g., Park, SNCA, MAPT and PINK1). Our PD blood leukocytes microarray study detected reversible changes in several microRNAs expression patterns prior to and following DBS. Some microRNAs were found as highly expressed (e.g let-7a). The samples had health committee approval (approval number 6-07.09.07 code 2507). Prior to this study detected expression and alternative splicing expression changes in PD blood leukocytes and as compared to age and gender matched Healthy Control (HC) volunteers (Soreq).

The RNA Integrity Values (RIN) was measured for the samples using bioanalyzes for sample quality/integrity assurance. The quantified several microRNAs by detection of the expressed ones in exon arrays data from patients and by mapping RNA-Seq (SOLiD platform) data to the human genome. Analysis of total RNA-Seq SOLiD data also allowed detection of expression changes in long non-coding RNAs (lncRNAs). A total of patients and control 3 samples were studied in microRNAs microarrays study (1 sample was re-scanned). Box plots verified the data statistical quality and 846 probes were analyses overall. MicroRNAs are short 21 nucleotide long genes which regulate target genes expression patterns through binding to the 3' Untranslated Regions (UTRs) typically. Several genes were identified as involved in the disease so far (e.g., Park, SNCA, MAPT and PINK1). In recent microarray study detected reversible changes in several microRNAs expression patterns prior to and following DBS. Some microRNAs were found as highly expressed (e.g let-7a). The samples had health committee approval (approval number 6-07.09.07 code 2507). Prior to this study detected the expression and alternative splicing expression changes in PD blood leukocytes and as compared to age and gender matched Healthy Control (HC) volunteers (Soreq). The RNA Integrity Values (RIN) was measured for the samples using bioanalyzes for sample quality/integrity assurance. Also quantified several microRNAs by detection of the expressed ones in exon arrays data from patients and by mapping RNA-Seq (SOLiD platform) data to the human genome. Analysis of total RNA-Seq SOLiD data also allowed detection of expression changes in long non-coding RNAs (lncRNAs). A total of patients and control 3 samples were studied in microRNAs microarrays study (1 sample was re-scanned). Box plots verified the data statistical quality. 846 probes were analyses overall.

CONCLUSION

The results indicate that the brain effects of DBS in PD yield leukocyte detectable RNA changes in both long and small RNAs and point at possible disease and treatment-responsive candidates for future therapeutic interference for PD and potentially other nervous system diseases that are responsive to DBS treatment. A better understanding of PD underlying molecular mechanisms will allow us to develop targeted genomic disease treatment options.

DATA DEPOSITION

GEO accession number GSE23676 (exon arrays), GSE40915 (small RNA seq).

CONFLICT OF INTEREST

The author declares no conflict of interest.

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