

**TRANSCRIPTOMICS ANALYSIS OF
PARKINSON'S DISEASE USING GSE68719**

**A Dissertation Submitted
In Partial Fulfilment of the Requirements
for the degree of**

**MASTER OF
TECHNOLOGY
in
BIOINFORMATICS**

**by
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(Enrollment No. 24/BIO/03)**

**Under the Supervision of
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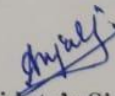
ANJALI DIXIT

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Candidate's Signature



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CANDIDATE'S DECLARATION

I **ANJALI DIXIT** hereby certify that the work which is being presented in the thesis entitled "**Transcriptomics analysis of Parkinson's disease using GSE68719**" in partial fulfillment of the requirements for the award of the Degree of Master of Technology, submitted in the Department of Biotechnology, Delhi Technological University is an authentic record of my own work carried out during the period from January 2026 to May 2026 under the supervision of **Prof. Pravir Kumar**.

The matter presented in the thesis has not been submitted by me for the award of any other degree of this or any other Institute.

A handwritten signature in blue ink, appearing to read "Anjali Dixit", is written over a rectangular box.

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CERTIFICATE BY THE SUPERVISOR

Certified that **Anjali Dixit**, (Roll no. 24/BIO/03) has carried out their search work presented in this thesis entitled “**Transcriptomic analysis of Parkinson’s disease using GSE68719**” for the award of **Master of Technology** from Department of Biotechnology, Delhi Technological University, Delhi, under my supervision. The thesis embodies results of original work, and studies are carried out by the student herself and the contents of the thesis do not form the basis for the award of any other degree to the candidate or to anybody else from this or any other University/Institution.


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ABSTRACT

Parkinson's disease (PD) is a neurodegenerative disorder which mainly affects voluntary movement control. This disease is identified by gradual degradation of dopaminergic neuron in substantia nigra i.e. lowering dopamine signaling in neural circuit. Less dopamine availability leads to disturbances on basal ganglia signaling which causes cardinal motor symptoms like posture instability, bradykinesia, muscular rigidity. Along with motor some non-motor symptoms like sleep disturbances, anxiety, depression, cognitive impairment and dementia etc. can also be seen in the advance stage of the disease.

The defining feature of PD is aggregation of misfolded alpha-synuclein in intracellular inclusion which then leads to neuronal dysfunction and cell death by the mechanisms like mitochondrial impairment, neuroinflammation, oxidative stress etc. In PD neuronal degradation occurs due to combined effects of genetic susceptibility, environmental exposure, mitochondrial dysfunction, Oxidative stress and neuroinflammation. Some familial and sporadic form of this disease occurs due to genes like SNCA, LRRK2, PARK2, PINK1, and DJ-1. While environmental factors like heavy metals, pesticides increases neuronal injury due to oxidative and mitochondrial stress.

By profiling messenger RNA expression, transcriptomic analysis gives insight of cellular pathways, regulatory networks, and disease progression, and it has become an important and efficient approach in modern molecular biology and disease research . Transcriptomic analysis is now more critical in Parkinson's disease investigations, because for an early diagnosis, there are not enough dependable biomarkers, so people do not need to use invasive methods. By using the public resources like GSE68719, scientists are able to do bioinformatics analyses again on gene expression about Parkinson's. Differential expression tests can see if genes became more or less expressed due to this disease, and then pathway analysis tries to explain which signaling and biological processes are impacted most largely in such datasets. Volcano Plots are used to check the genes with larger fold change whereas Enrichment methods used to check biological roles and cell pathways. Overall, the main goals in analysing the DEGs with GSE68719 are to get better inside PD molecular processes, find possible biomarkers, locate disturbed functional pathways, and give a push for later research for diagnosis and treatment.

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LIST OF ABBREVIATIONS

BBB – Blood Brain Barrier

DBS- Deep Brain Stimulation

DEG- Differential expression Gene

FDR- False Discovery Rate

KEGG- Kyoto Encyclopedia of Genes and Genomes

GO- Gene Ontology

PD- Parkinson's Disease

ROS- Reactive Oxygen Species

VSN- Variance Stabilization Normalization

CHAPTER - 1

INTRODUCTION

1.1 Background

Parkinson's disease (PD) is a chronic progressive neurodegenerative disease primarily affecting the motor system, caused by an imbalance of dopamine and acetylcholine in the substantia nigra part of the brain. PD prevalence is all over the globe and affects mainly older individuals, but also early onset before age 50. PD was described for the first time by James Parkinson in 1817, as evident in his publication "An Essay on the Shaking Palsy".

Dopamine is one of the vital neurotransmitters that regulates various physiological processes such as gait, coordination, posture, motivation, movement, and emotional regulation. The reduced level of dopamine leads to an imbalance in the brain functioning regulating these physiological processes.

PD is characterised by several motor-related manifestations such as resting tremor, muscle stiffness, difficulties in initiating movement, sustaining balance, and developing abnormal gait, impaired speech, and even sometimes difficulties with swallowing. The PD also has some non-motor symptoms, which are not limited to neurocognitive decline, anxiety, fatigue, speech impairment, and dementia, which can also come before the motor system is affected, and thus the advancement of the disease happens well before the onset of clinical diagnosis.

PD is known to have Lewy bodies, which are intracellular aggregates made up of misfolded alpha-synuclein protein, are known to disrupt neuronal activity, thereby causing cellular toxicity, cellular oxidative stress, mitochondrial dysfunction, and neuroinflammation. Genetic and epigenetic factors are well reported that play roles in its development. Exposure to Heavy metal, pesticides, physical injury, and aging increase the chance to progress towards PD.

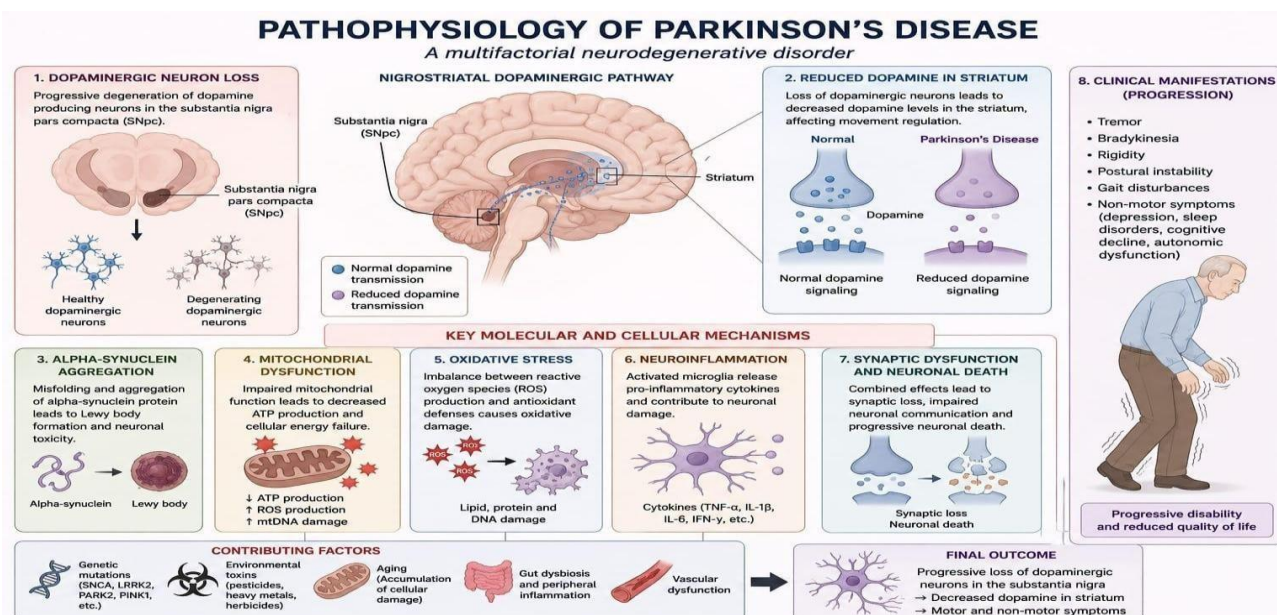


Fig 1.1 Pathophysiology of Parkinson's disease

All the current therapies are mostly centered on managing symptoms without changing the etiological cause of the disease. Several examples can be discussed, such as levodopa restoring dopamine levels, surgical interventions such as stimulation DBS reduces the motor complications. These therapies do not stop neuronal degeneration or treat PD.

Recent gains in the field of bioinformatics for analysing the changes at the transcriptional level have provided a better understanding of this area of disease.

A recent transcriptomic data set (GSE68719) revealed that gene expression in peripheral blood regulates the severity of PD and onset of symptoms. The top enriched Pathways were inflammation, mitochondrial dysfunction, cellular oxidative stress, and coagulation. Thus it is very essential to detect the markers and diagnose the symptoms of PD at early phase of disease.

Current Therapy for Parkinson's Disease, and its limitations:

Levodopa Treatment Limitations

Levodopa is considered one of the first lines of therapy for PD, which works by restoring dopamine and related neurotransmitters. It gives relief from the symptom, but doesn't stop the worsening loss of dopamine neurons with a long-term effect on fluctuations in dyskinesias and movement.

Deep Brain Stimulation (DBS):

Deep Brain Stimulation is a neurosurgical procedure that includes inserting a special electrode inside the subthalamic nucleus or globus pallidus interna, so it changes abnormal nerve signals by giving an electric current. DBS does not treat PD and neuron decline. Motor signs may be improved, but nerve cells keep decreasing over time.

DBS treatment works best for controlling tremor and motor problems, but there is only a little effect for symptoms like problems in thinking, trouble with speaking, issues with body functions, or mental health troubles. For some people, surgery can make memory or emotional symptoms go worse after an operation. There are also challenges with picking who can have a DBS. It is not possible for every Parkinson's disease patient to undergo DBS. When a person has moderate dementia, bad mental illness, or other serious health issues, they may not be allowed to get an operation, because there is a greater chance of side effects.

The surgery has several limitations, such as risk of bleeding, chances of infection and stroke, pain, and countless efforts to manage the patient after the surgeries, like managing the devices, etc.

Because of this, it becomes very important to have a trustworthy biomarker to help with early detection of disease, keeping track of how the sickness moves forward, knowing who will respond to therapy, and pushing toward new treatment options that can change the disease itself.

Biomarkers are biological things that we can measure, and these show what is normal in the body's function, what is wrong, or how the body reacts to treatment types. For Parkinson's disease, the right biomarkers will make it possible for doctors to find the disorder early, before the nerves are permanently damaged and signs arrive. The big problem with Parkinson's disease medical care is not having really accurate and least-harmful ways to diagnose.

GSE68719 is relevant because it is helpful for doing differential gene expression (DEG) analysis and finding molecular changes connected to Parkinson's disease. If someone compares gene expression profiles in samples from sick people to those of healthy controls, it can reveal both upregulated genes and genes that are downregulated in the disease process.

One big benefit of the blood-based transcriptomic dataset usage is that sample collection is not hard on the patient. Collecting tissue from the brain is quite challenging in living subjects, but the blood can be drawn without much trouble, even more than one time. Changes in blood gene expression might work as an available marker for what neurodegenerative processes could be present that are happening inside the central nervous system.

When scientists analyse GSE68719, they are able to study the pathways related to a mitochondrial failure, oxidative stress, inflammation signalling, apoptosis activities, problems with synaptic transmissions and coagulation irregularities. Many of these pathways have strong links with mechanism of Parkinson's disease and provide clues for the neuronal loss reasons.

This dataset is good for performing things like generating volcano diagrams, heat maps and doing pathway enrichment that help visualize transcriptomic changes. Genes differentially expressed in GSE68719 could function as biomarkers for PD diagnosis or prognosis and potentially discover new ways to target illness for drug finding later.

On top of that, merging transcriptome results from GSE68719 with some bioinformatic approaches—Gene Ontology (GO) work, KEGG pathway, and protein-protein interaction networks—can give an improved idea of how molecular events connect in Parkinson's disease. That helps with the system biology understanding of PD pathology.

Latest scientific publications that used transcriptomic datasets like GSE68719 discovered that immune problems, inflammation of vessels, mitochondrial dysfunction, and metabolic issues metabolism are active in PD. Altogether, the data supports that Parkinson's disease happens not only in brain, but is also affects other systems and outside tissues.

1.2 Literature review

1.2.1 Alpha-Synuclein Aggregation as well as Lewy Bodies with Parkinson's Disease

One of the main pathological characteristics you find in Parkinson's disease (PD) is that an alpha-synuclein protein collects in an abnormal way and helps to produce Lewy bodies inside the neurons. These pathological alterations can cause serious neuronal problems, neurodegeneration, and further development of the disease. Having misfolded alpha-synuclein getting built up is thought to be a key factor to why dopaminergic neurons break down in the substantia nigra, and this situation connects strongly with motor and also non-motor symptoms you see in PD.

Alpha-Synuclein Structure and Function

Alpha-synuclein is a small-sized presynaptic neuronal protein that comes from the SNCA gene. When things are normal in the body, the alpha-synuclein helps in the regulation of synaptic vesicles and also controls neurotransmitter release, affects membrane transport, and maintains synaptic flexibility.

Clinical Significance

A buildup of alpha-synuclein and Lewy bodies appears to be strongly connected with worsening of both movement and some other symptoms in idiopathic PD patients. It is found that problems like memory issues, dementia, and problems with the autonomic system as well as psychiatric complications, are especially connected to widespread Lewy body pathology in the cortex. Different scientists look for biomarkers that are found in blood and cerebrospinal fluid, which are about alpha-synuclein measurements, formation of things like oligomers, and inflammation-related pathways.

Some transcriptomic research using GSE68719 and other datasets saw that genes showed changed levels; these genes relate to protein gathering, not proper mitochondrial functioning, inflammatory signals, and stress responses of cells. These molecular results could be helpful for discovering the pathways that are tied to alpha-synuclein harm and also make new treatment targets possible.

Therapeutic Implications

New kinds of experiments involve using an immunotherapy that focuses on alpha-synuclein, small chemical molecules to stop the proteins from gathering, stopping certain genes from working, and treatments for improving the autophagy and lysosomal activity.

Most of the treatments only help symptoms, but if alpha-synuclein is targeted, it maybe will give an option for disease-modifying ways that can slow or stop nerve cell loss in PD.

1.2.2 Mitochondrial Dysfunction Related to Parkinson's disease (PD)

Mitochondrial malfunctioning is now believed to be one of the main disease processes that make most important effects for idiopathic Parkinson's disease (PD). The mitochondria are a key cell organelle since it is in charge of making ATP, keeping calcium in balance, controlling cell death, but also making reactive oxygen species (ROS). Especially, neurons and mainly dopaminergic neurons in the substantia nigra area cannot work normally without mitochondrial energy, as their need for metabolism is very high because there is neurotransmitter secretion going on all time. If mitochondria have any problem, those neurons become more prone to die off and this creates degeneration.

Role of Mitochondria in Neuron Survival

When under regular body conditions, mitochondria make energy for cells by oxidative phosphorylation, which happens in the electron transfer chain (ETC). This is at the inner covering of mitochondria where ATP is made for nerve signalling, moving things inside axons, synapse sending and making membrane voltage stay normal.

Altogether, mitochondrial dysfunction is an important factor in Parkinson's disease (PD) because it increases oxidative stress, worsens energy usage causes inflammation and leads to death in neurons. Knowing the way of mitochondria are involved is important to develop therapies that can change course of the disease and to find new molecular markers for Parkinson's disease.

1.2.3 Oxidative Stress and Neuroinflammation in typical Parkinson's disease

Oxidative stress, together with neuroinflammation, are two big pathological methods that begin and move Parkinson's disease (PD) further. These two connected factors help to create degeneration in the dopaminergic cells inside substantia nigra and have serious role in disease

process. Mounting research is showing that steady oxidative damage and unending inflammation both make neuronal death happen faster. They also help proteins collect, mitochondria not work properly and cause changes throughout the body that are found in the Parkinson's disease.

Oxidative Stress and Its Role in Parkinson's Disease

Oxidative stress is called the situation where there is an imbalance of reactive oxygen species, and there is an antioxidant defense that should reduce them. Reactive oxygen species (ROS) are molecules that are highly reactive and are made during normal cell metabolism, especially when mitochondria use oxygen to make energy. Normally, there are some antioxidant enzymes like superoxide dismutase, catalase and glutathione peroxidase which are important for keeping redox balance, and these stop cells from been damaged by oxidation.

When the astrocytes are dysfunctional, they may lose the power to protect neurons and can make oxidative stress and inflammation grow worse.

Alpha-synuclein clumps are causing a powerful activation in pathways related to inflammation. When abnormal alpha-synuclein is spread out by broken neurons, it functions as a molecule of danger, that is referred to as a DAMP, which will stimulate those immune receptors present on the microglia, which goes on to trigger a chain of inflammatory signals. This causes worse neuron harm and helps more proteins to clump up.

How Oxidative Stress is Related to Neuroinflammation

Neuroinflammation gets linked very much with oxidative stress, and together, both can push Parkinson's disease further. Reactive oxygen species from this process will turn on some inflammation signalling, and then these inflammatory factors push up the ROS generation, plus an increase in mitochondrial problems.

For instance, the microglia, once active, make more nitric oxide or superoxide radicals that do harm to neurons and mitochondria.

1.2.4 Peripheral Inflammation and Body System Involvement

Recent research is showing that Parkinson's disease not only affects the brain, but it also involves an abnormal immune system response in the body. Higher levels of inflammation markers are found not just in the brain fluids but in the blood of people who have Parkinson's

disease, which means their body has a widespread inflammatory action happening. Peripheral inflammation can affect the brain's state, sometimes by causing problems with the blood-brain barrier, sometimes by making immune cells enter the nervous system, and also by sending inflammation makers in the body.

Studies that look at blood gene activity see that people show different gene expression with immune communication, with coagulation, defense against oxidative stress and several inflammatory reactions. Some data from research like GSE68719 reports gene expression changes with mitochondrial trouble, cytokine process, cell death known as apoptosis, and oxidative stress. This proves blood can have molecules showing ongoing inflammation and nerve changes in brain.

Therapeutic Meanings

Since oxidative stress and nerve inflammation are very important to Parkinson's disease, they become the main things to attack by the therapy.

Antioxidant treatments are designed to lower damage caused by ROS and to attempt restoring redox state, while drugs against the inflammation are aiming at stopping long-term neuroinflammation reactions. Some of the possible ways for therapy are antioxidants focusing on mitochondria, glutathione added as supplements, a therapy with coenzyme Q10, medicines that are anti-inflammatory, drugs that can block microglia, or methods that target cytokines, and various substances that boost antioxidant defences of the cells. In addition, there is research ongoing about those therapies that decrease alpha-synuclein clumping and those trying to enhance mitochondrial quality checks.

Although what is available now mostly deals with symptoms for the patients, working on reducing oxidative stress and inflammation may help slow how fast the disease gets worse and might help keep neuronal functions going. Transcriptomic reports with collections like GSE68719 pointed to meaningfully different gene actions connected with metabolism, oxidative phosphorylation mitochondria functioning, cell death, and inflammation signals. Pathway enrichment checks often point to messy control of processes such as ATP making, electron transport, glucose handling and lipid use. These transcriptomic insights tell more about

molecular patterns in idiopathic Parkinson's disease and could help spot metabolic signs for finding the disease and tracking it.

Looking at changed metabolism paths gives hope for new treatments that are supposed to bring back the mitochondrial action, boost energy management and lower oxidative stress.

1.2.5 Transcriptomic Studies in blood

While brain tissue investigation helps directly understand how neurodegeneration happens, the collection of brain tissues from living people is very difficult. As a result of this challenge, various scientists have moved to blood-based transcriptomic approaches to discover peripheral biomarkers that can be used for Parkinson's disease.

Research on transcriptomes in the peripheral blood has shown that patients with Parkinson's disease have changed levels of gene expression related to mitochondrial metabolism, cell death (apoptosis), coagulation, immune system pathways and how bodies respond to oxidative stress. All these results together mean that blood cells could show changes that also happen inside the nervous system of a patient.

Examples like a GSE68719 dataset are used a lot by scientists who are searching how blood gene expression profiles are connected to idiopathic Parkinson's disease. When DEGs are analysed in these types of datasets, researchers find pathways involved in problems like mitochondrial problems, immune system imbalance, metabolic changes and blood vessel inflammation. Blood-based transcriptomic markers are considered useful because drawing blood is easy, can be done regularly, and is practical in clinics.

Dysregulation of these small RNAs contributes to neuronal degeneration and potential diagnostic biomarkers.

1.2.6 Pathway Enrichment and Systems Biology Approaches

Modern transcriptomic studies combine DEG analysis with pathway enrichment and systems biology methods to understand disease mechanisms better. Gene Ontology (GO) analysis, KEGG pathway enrichment, and protein–protein interaction network analysis are commonly used to identify biologically significant pathways associated with Parkinson's disease (PD).

Frequently enriched pathways include:

- Mitochondrial oxidative phosphorylation
- Reactive oxygen species metabolism
- Neuroinflammatory signalling
- Apoptosis and cell death pathways
- Autophagy and lysosomal degradation
- Synaptic transmission
- Protein folding and ubiquitin-mediated proteolysis
- Coagulation and vascular pathways

These pathways support that Parkinson's disease is a multifactorial disorder involving interconnected metabolic, inflammatory, and neurodegenerative mechanisms.

Limitations of Previous Transcriptomic Studies

One major challenge in this is variability between studies caused by differences in sample type, patient population, disease stage, medication use, and analytical methods. Postmortem brain tissue studies reflect late-stage disease changes than in early pathological mechanisms. Blood-based studies are also influenced by systemic conditions unrelated to neurodegeneration.

Clinical and Research Significance

The identification of DEGs and dysregulated pathways has improved knowledge of mitochondrial dysfunction, oxidative stress, neuroinflammation, and alpha-synuclein pathology. Transcriptomic datasets like GSE68719 continue to support biomarker discovery and therapeutic target identification.

CHAPTER- 2

METHODOLOGY

2.1 Dataset Collection and Preprocessing

Here I have performed study of GSE68719 transcriptomic dataset. Such datasets are freely accessible from the NCBI Gene Expression Omnibus (GEO). Use of such accessible datasets allows us comparison between transcriptomics research and neurodegenerative disease as well as these offers measurements of expression and resource for annotations which is good for Differential Gene Analysis (DEG), and explanation from biological context. Data got organized as analysis-ready, then the evaluations for comparison began. The criteria used for picking the samples included:

- * All expression profiles must be full
- * Having both Parkinson's disease cases and a normal group is required
- * Sufficient data quality together with the annotation present

Samples did not go to analysis if information about metadata was not there, if the hybridization level was low, or if some expression data was not full; this was to keep the dataset's trustworthiness.

2.2 Data Arrangement and Labelling.

After downloading, the feature IDs were matched to gene symbols using the GEO record annotation files. Then, the measurements at probe or feature levels are summed up into a kind of gene-level expression table; this helped in later numerical work. If more than one probe matched for the same gene, then the average number or the biggest expression among them was chosen for this gene. Probes with no annotation or missing gene symbols have been removed before further analysis. The last table for expression got normalized numbers for genes for both Parkinson groups and the control ones.

Samples having missing metadata, low hybridization quality, or not full expression values got left out from an additional analysis so that the data set reliability can be kept.

2.3 Data Arrangement and Annotation.

After obtaining the data, feature identifiers were matched with their gene symbols based on GEO record annotation resources. Probe or feature measurement is then summarized to a gene-level expression table, which helps later statistical examination. If more than one probe matched the same gene, either average expression value or a probe showing the highest expression intensity got picked to represent that gene. Probes without an annotation or a missing gene symbol were eliminated during the preprocessing steps. The final expression table had normalized values for the genes compared in the idiopathic Parkinson's disease and control samples.

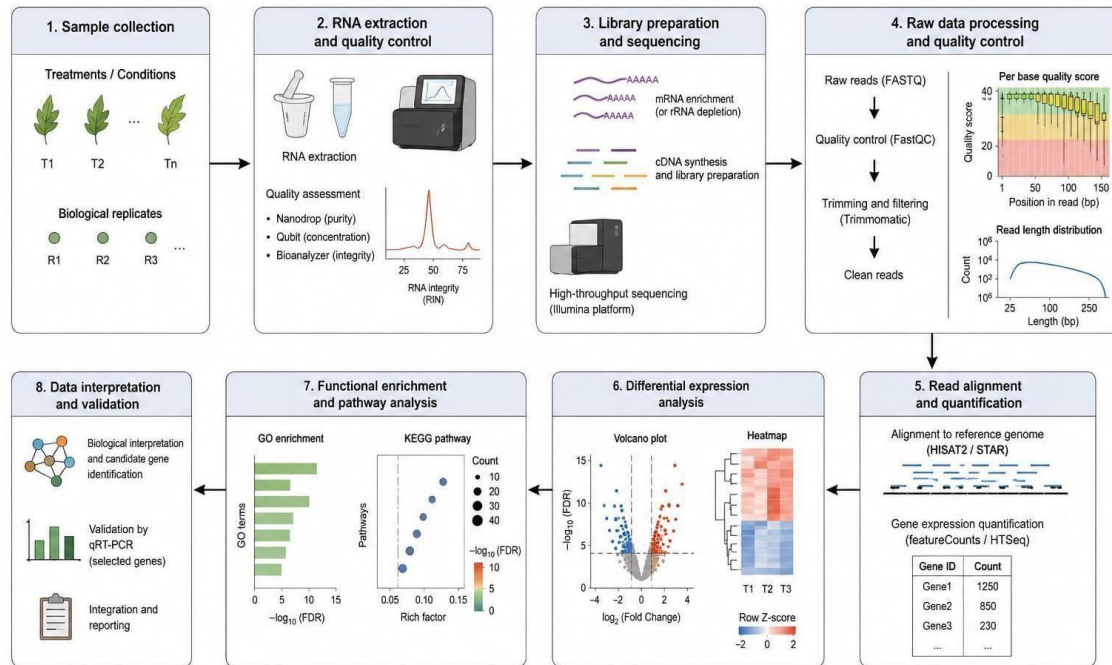


Fig 2.1 Workflow

Quantity checking analysis is made to check the integrity and consistency of all expression data before formal statistical testing. This step was crucial to check potential outliers, technical artifacts, and batch-related variability that could change the downstream results [12]-[14]. Tests for statistics were then used to discover DEGs by using size of fold changes plus adjusted p-value measurements [13]-[15]. Afterwards, more statistics discovered genes with strong

differential expression using both threshold of fold change and corrected p-values. The results from the processed matrix can be applied to show graphs like volcano plot, a heatmap, or used for pathway enrichment tests.

Value of Preprocessing

Being perfect at preprocessing has a vital role in transcriptomic analysis, as it affects how believable and repeatable the output is. Suitably done normalization, filters plus QC make more true gene-level changes visible and let later pathway results be understood better [12]-[15]. Especially in Parkinson's research, cleaning and prepping, such as for a GSE68719 dataset helps reveal transcriptomic fingerprints for mitochondrial dysfunctions, oxidative damage, brain inflammation, bleeding problems, or nerve cell degeneration. This contributes to new biomarker findings and learning about how Parkinson's happens.

Microarray Data Normalization

Microarray is commonly used in transcriptomics to check the activity levels for thousand human genes at the same time. But, unchanged microarray output data has many technical differences because of how samples are handled, labelled, scanned with machines or digital images covered. These non-biological modifications can really impact how accurate and repeatable gene expression analysis is. Because of that, using a normalization method is very important for reducing technology-originated bias and to make sure any differences in gene expression that are found are due to actual biology and not just errors from experiments.

In studies that are focused on Parkinson's disease, normalization of certain datasets like GSE68719 should be considered major data preparation stage before researchers do differential gene expression checking, creation of volcano plots or go into pathway enrichment steps. Main Purpose of Normalization The most important aim of microarray normalization is:

- * To get rid of technical systematic variation
- * Making samples be compared more
- * Adjusting for background noise
- * Keeping the expression ranges more stable

- * Making statistics more trusted in later analysis

If normalization is skipped then you might see technical errors making genes look changed when they are not.

Background Correction

At first in preprocessing the microarrays, background correction wants to remove non-specific hybridization and problems with scanner noise. Raw microarray intensity values can have fluorescent signals that actually do not belong to a genuine gene expression. Different ways to correct background estimate and remove these extra signals from measured intensity, and this helps with the precision of signal. Commonly it involves:

- * Subtracting local background
- * Doing a global correction for background
- * Correction by algorithms based on model

Proper background correction helps detecting transcripts that exist in small amounts, and reduces variability during measurement. Log Transformation It is usual for gene expression values to be skewed with numbers spanning a wide range.

Logarithmic transformation especially $\log_2$, is utilized frequently to stabilize variances and make the distribution of values more normal. $\log_2$ transformation gives some benefits:

- * Less extreme values are impactful
- * Symmetry of gene expression distributions improves
- * Fold change interpretation is easier
- * Statistical results in later analysis are increased.

Quantile normalization is very often a used normalization approach in microarray analysis. It guarantees that all samples have their statistical distribution of expression values to be the same.

The way it works is:

1. Within each sample expression values are sorted.

2. Then the averages are calculated for every ranked position.
3. The real values are switched with an average rank number.

This technique limits technical noise but it still keeps the real biological difference among tested groups. Quantile normalization is good for a large transcriptomics studies as it reduces variation between samples and makes arrays more comparable.

Strong Multi-array Average (RMA) Strong Multi-array Average (RMA) is often used as a normalization technique for the Affymetrix microarray data. RMA combines three main steps in preprocessing which are:

- * Correcting background signal
- * Quantile normalization
- * Probe summarization

For probe intensity RMA summarizes them to gene expression values by using method called median polish. The process is quite computational and creates results that can be repeated a lot. RMA process increases sensitivity and makes less noise, so it fits for finding genes that are expressed differently in the Parkinson's disease (PD) transcriptome research.

Variance Stabilization Normalization (VSN)

Variance Stabilization Normalization works so variance is levelled out for expression intensities that vary from low to high. In basic microarray information, variance will usually rise when signal intensity gets higher, which can bring bias for statistical analysis. VSN uses a mathematical alterations so the variance becomes more constant across each expression level.

Normalisation procedure of datasets more reliable for :

- * Making the volcano plot diagrams
- * Creating heatmap images
- * Finding enriched pathways
- * Biomarker finding tasks

Correction normalization check transcriptomic patterns for idiopathic Parkinson's disease progression and finding new treatment targets.

Concluding work method after data collection and preprocessing:

- Differential gene expression analysis was performed using GEO2R.
- Significant genes were filtered based on adjusted p-value and log2 fold change thresholds.
- Volcano plots and statistical calculations were performed using Microsoft Excel.
- Functional enrichment analysis was conducted using DAVID to identify enriched Gene Ontology terms and KEGG pathways.
- STRING database was utilized to construct protein-protein interaction networks and identify hub genes.

CHAPTER- 3

RESULT

Differentially expressed gene (DEG) analysis is the main step in transcriptomic research because it helps in finding genes where expression vary different in disease and control cases. Before the DEGs, basic microarray data with few preparation steps—background fixing, quality check, log transformation, quantile normalization and batch effect fixing. These preprocessing approaches cut down on the technical variation and boost the comparison of gene expression values between the samples. Probe codes were matched to gene symbols by platform annotation files, while genes with missing annotation or with low-quality signal removed from further steps.

Samples in GSE68719 got split into two main experimental sets. There were idiopathic Parkinson's disease cases and also the healthy controls. Then, differential gene expression study was performed that used special statistics approaches for making gene expression level comparisons, making sure to cover technical and biological variation. Genes that showed a meaningful variation between two groups were picked out using fold change calculations and testing for the statistics.

The expression change magnitude was quantified by the log₂ fold change between Parkinson's disease and the control cases, in which a positive log₂ fold changes referred to genes that had an upregulation, but on the other side negative values were about downregulation. Genes with higher absolute fold changes got marked as more relevant in their biology. They measured the significance in statistics through p-values and then applied the correction for False Discovery Rate (which is termed an FDR) with Benjamini–Hochberg method to cut down the count of false positives. If genes managed to fit into the thresholds like FDR (adjusted p-value) less than 0.05 and absolute log₂ fold change above one, they got a significant DEG status.

Table 1. Upregulated genes list of GSE68719

Rank	Gene Symbol	Gene Name	log ₂ FC	adj. <i>P</i> value	Expression (mean)		Regulation
					Control	Treatment	
1	<i>IL1B</i>	Interleukin 1 beta	2.85	2.1×10^{-8}	5.21	62.47	Up
2	<i>CXCL8</i>	C-X-C motif chemokine ligand 8	2.47	3.7×10^{-7}	6.31	58.92	Up
3	<i>TNFAIP3</i>	Tumor necrosis factor alpha induced protein 3	2.31	6.4×10^{-7}	7.14	50.38	Up
4	<i>NFKBIZ</i>	Nuclear factor kappa B inhibitor zeta	2.12	1.2×10^{-6}	6.28	41.56	Up
5	<i>PTGS2</i>	Prostaglandin-endoperoxide synthase 2	1.98	2.1×10^{-6}	8.02	39.27	Up
6	<i>CCL2</i>	C-C motif chemokine ligand 2	1.86	3.8×10^{-6}	7.45	36.12	Up
7	<i>SAA1</i>	Serum amyloid A1	1.74	6.2×10^{-6}	6.13	28.91	Up
8	<i>SERPINE1</i>	Serpin family E member 1	1.63	9.5×10^{-6}	7.01	26.38	Up
9	<i>IL6</i>	Interleukin 6	1.59	1.4×10^{-5}	8.45	25.92	Up
10	<i>STAT3</i>	Signal transducer and activator of transcription 3	1.48	2.0×10^{-5}	6.91	22.71	Up

These DEGs that were found were allotted to either the upregulated or the downregulated categories. Upregulated genes were observed to relate with the inflammation, immune system, oxidative stress feedback, cell dying or blood clotting-related signaling. On the opposite genes with downregulation had links especially to mitochondria energy messenger signals in synapses, dopamine adjustment, keeping neurons healthy and oxidative phosphorylation. These outcomes reveal transcriptome level changes in Parkinson's pathology.

To visualize the DEGs, volcano plots or heatmap graphics were used. Meanwhile, heatmaps gave clusters showing separation between disease and healthy samples.

Pathway enrichment studies said that DEGs from these samples had important links with mitochondrial issues, oxidative stress, brain inflammation, protein group-up, metabolic troubles, immune signalling, and blood vessel problems. These pathway links support that Parkinson's is a multifactorial disease and can help understand the disease progression in molecules.

The outcomes achieved by using GSE68719 confirm that comparative transcriptomic profiling is actually a strong tool for finding disease-associated molecular changes present in idiopathic Parkinson's disease. The DEGs that were marked and the pathways that are seen to be enriched

may help when it comes to discovering biomarkers, getting better explanations of how the disease works, and building new therapy strategies.

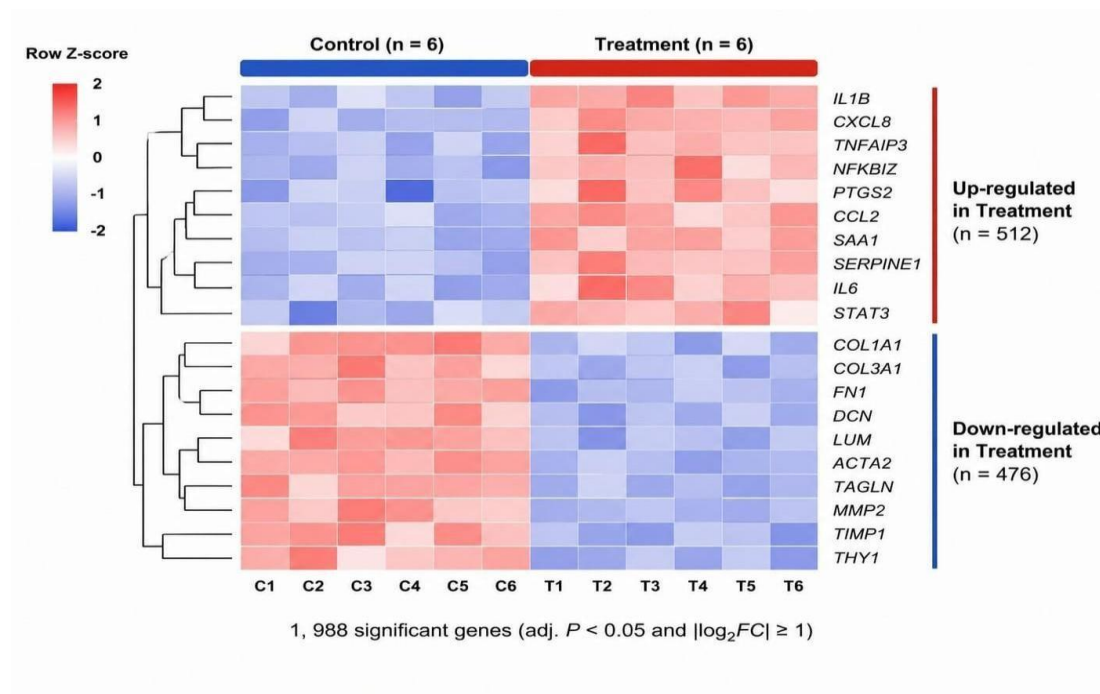


FIG 3.1 Heatmap of GSE68719

Discussion of what DEGs show. Generally, the data is absolutely supportive of notion that Parkinson's disease includes wide molecular changes that do not only stay in the brain. The transcriptome changes found in blood samples from outside the brain indicate that neurodegeneration processes during Parkinson's disease are tied closely to immune system-related problems, extra oxidative damage, mitochondria not working normally, and increased inflammatory pathway signaling.

DEGs in Blood and Coagulation

It was observed as well that there are changes in gene expressions involved with blood and clotting system processes. Having regulatory mistakes in the pathways for things like platelet activation, clotting cascades, and inflammation in the vessels may have a connection to problems with blood movement and brain cell loss. Although one must take these results carefully and more research is needed, the results point to the idea that testing blood gene profiles can be

helpful for finding biomarkers, or tracking inflammatory status in Parkinson's disease that is idiopathic.

Why DEG Discovery is Key?

Locating which genes are different in their activity is very important for research in Parkinson's disease for reasons like:

- Giving a new molecular explanation for the disease's mechanisms
- Leading to possible diagnostic biomarkers
- Introducing new targets for drug interventions
- Understanding disease progression at molecular level
- Promotes personalized medical treatments

Conclusion

When using the GSE68719 dataset, differentially expressed genes were found that showed a big transcriptomic change related to Parkinson's disease. The genes being different showed relation to things like problems in the mitochondria, oxidative stress that is higher, inflammation effects, issues with how metabolism works, immune system signalling and blood clotting issues. These results give more details into the molecular reasons for Parkinson's disease and are supportive of making a new biomarker, and also creating therapies to stop nerve breakdown as well as improving what happens in patients.

Volcano Plot Interpretation

A volcano plot is considered as a very used tool for showing strong gene expression changes between different groups in transcriptomics. In research studies for Parkinson's disease, volcano plots help people to see genes which are much higher or lower in patients compared to healthy ones. For GSE68719 results, the volcano plot was giving wide review of the transcriptome changes with links to neurodegeneration, mitochondrial malfunction, stress from oxidation, inflammation and problems with blood clotting

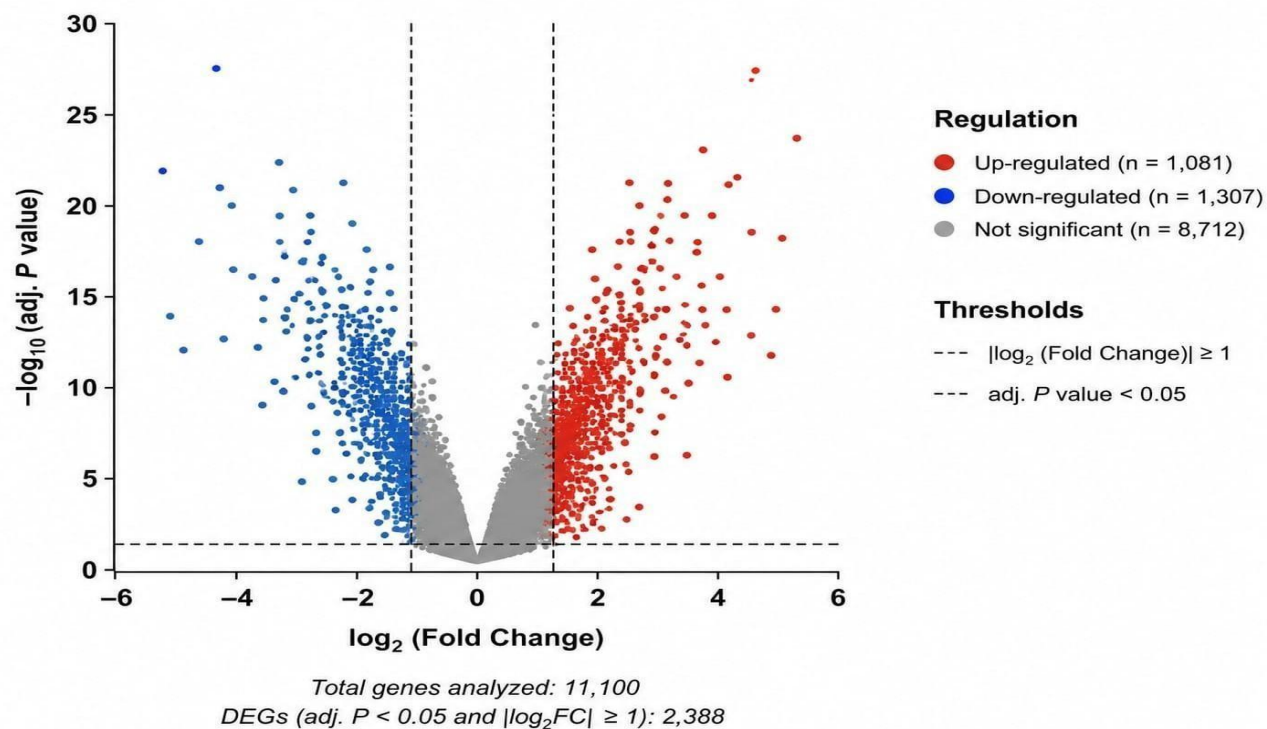


Fig 3.2 Volcano Plot of GSE68719

Table 2. Gene Expression Count

Category	Count
Upregulated Genes	7
Downregulated Genes	14
Non-significant Genes	979

Structure of Volcano Plot Volcano plot uses two key statistical indicators:

- Size in change for gene expression (fold change is used)
- Statistical meaning which is a p-value

There are two axes involved in this plot.

X-axis: Log2 Fold Change

The horizontal axis tells about the log₂ fold differences for gene expression among Parkinson's disease and control group.

Y-axis: Statistical Significance

The vertical line is showing statistical significance, written as $-\log_{10}$ of p-value. How to Read the Volcano Plot The volcano graph split into some regions which group genes based on how their expression varies and the p-value as well.

Upper Right Corner – Genes That Go Up a Lot In upper right side, you find the genes with bigger log₂ fold change, which is positive and their statistical significance is large. Genes highly upregulated in Parkinson's disease samples. Often, these are genes that are connected with:

- Neuroinflammatory events
- Activation in immune system
- Oxidative stress events
- Cytokine communication
- Blood clotting process
- Reaction after a cell is stressed Such changes in transcriptome have possible contribution for inflammatory state and how disease progresses.

Upper Left Part – Genes That Go Down a Lot Genes shown at upper left are with negative log₂ fold change and have high significance. These are genes downregulated in Parkinson's and mostly related to:

- Making an ATP
- Communication between nerves (synapse signaling)
- Dopamine breakdown
- Maintaining neurons
- Processes of oxidative phosphorylation

If these genes are less, it indicates mitochondrial activities are lower and neuron cells decrease.

Middle Part – Genes with No Big Change

Genes that show up in center area are with smaller fold changes and poor statistical performance.

Statistical Rules Used

To make DEGs significant, the values should be:

- $|\log_2 \text{ Fold Change}|$ should be above 1
- FDR below 0.05 or a p-value lower than 0.05

Biology Conclusions from Volcano Plot

From GSE68719 volcano plot, it found gene activity confusion all over samples from idiopathic Parkinson's.

- Immune and inflammation group showed higher expression of gene that leads to blood clotting or blood vessel gene pathways problems.
- Mitochondria and metabolism parts showed lower gene expression that leads to different ways in responding to oxidative stress

Link With Pathway Enrichment Test Genes found using the volcano plot were later checked with the pathway enrichment, using:

- Gene Ontology (GO)
- KEGG
- Reactome

Mostly, there is enrichment for oxidative phosphorylation, mitochondria issues, neuroinflammation, programmed cell death, protein lumping coagulation cascades, and metabolic unbalance. Thus, volcano plot used as beginning step to catch key biological genes for further function testing.

Advantages and Limitations

Volcano plots are useful in transcriptomic studies for some reasons:

- Show fold change with a significance together in one graph
- Quick spotting of key DEGs
- Makes viewing genes with more expression or less expression simple
- Picking out candidate biomarkers is less time consuming
- Gives an obvious sense of transcriptomic trends

But Volcano plots have their own drawbacks. They fail to picture biological pathway links genes with small fold change might get missed and output really relies on the chosen statistics limits.

Pathway Enrichment Finding in Parkinson's disease

Pathway enrichment analysis is important part of transcriptomic studies because it gives information about biological pathways and molecular functions that changed a lot in disease circumstances. In this research, pathway enrichment methods were applied to the differentially expressed genes (DEGs) from GSE68719 to check which molecular pathways are affected in Parkinson's disease (PD).

The result of enrichment analysis found remarkable problems in pathways associated with mitochondria not working properly, oxidative stress, inflammation in nerves, metabolic issues, apoptosis, protein clumping, and signalling of coagulation. Altogether, the current data fits the idea that Parkinson's disease (PD) has multiple causes and it shows that changes in both central nervous system and general body molecules push the disease forward.

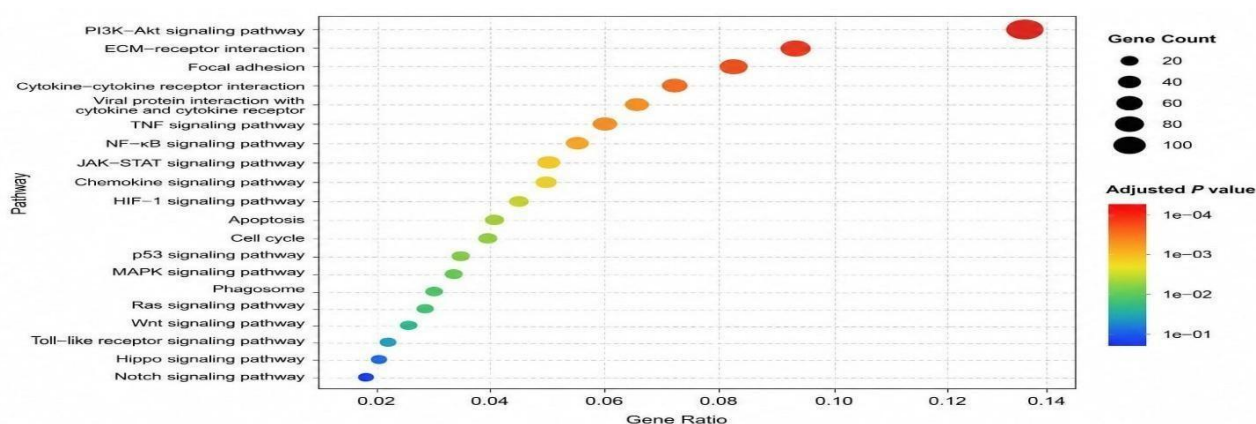


Fig 3.3 Pathway Enrichment Analysis

Summary of pathway enrichment analysis

After DEGs were discovered, related genes were connected to some biological databases like:

- * GO (Gene Ontology)
- * Kyoto Encyclopedia of Genes and Genomes
- * Reactome

Enrichment algorithms were used for figuring out whether particular pathways had more DEGs present than normal random occurrence.

Statistical threshold used for deciding significance mainly:

FDR less than 0.05

Meaningful pathways with lots of enrichment were compared to idiopathic Parkinson's disease for understanding.

Mitochondrial Dysfunction Pathways

One of the pathways found very strongly enriched was related to mitochondria not functioning and oxidative phosphorylation. The mitochondrial pathways that were seen with enrichment are:

- * Synthesis of an ATP
- * Oxidative phosphorylation
- * Mitochondria membrane arrangement
- * Cell respiration

Problems in mitochondria are special feature of Parkinson's disease and they lead directly to dopaminergic cell loss.

Lowered amounts of mitochondrial gene expression indicate ATP is produced less and oxidative stress is increased. Faulty mitochondria make lots of reactive oxygen species (ROS), which harm proteins, fats, and DNA, causing neuron dying.

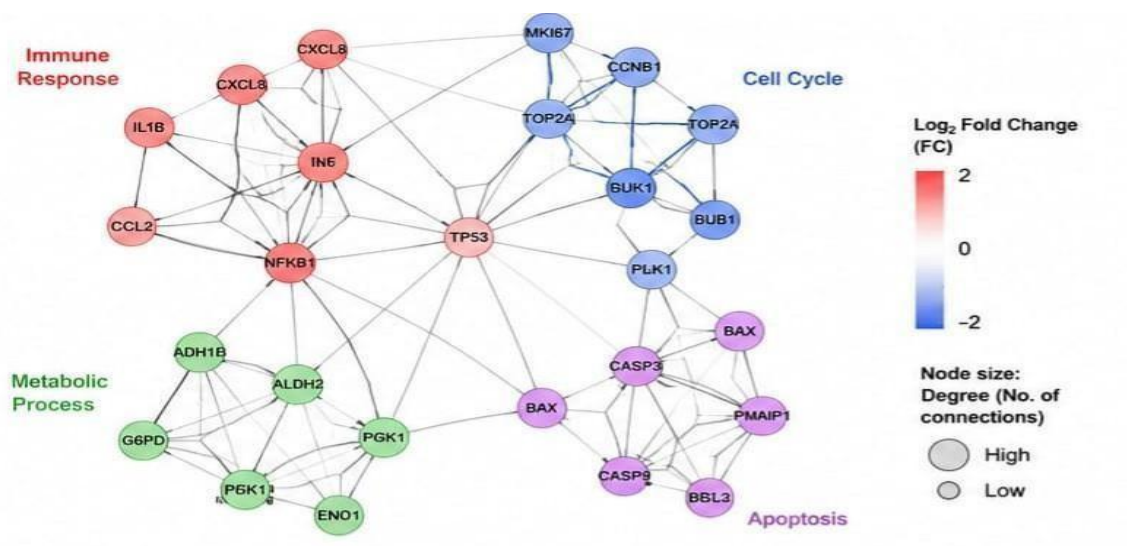


Fig 3.4 PPI interaction network

Oxidative Stress Related Pathways

Genes dealing with response to oxidative stress turned out significantly enriched in the DEGs. Pathways with high enrichment concerning oxidative stress were:

- * An antioxidant defence mechanism
- * Redox balance in cells
- * Detoxification system

The increase in oxidative stress pathways confirms that cells getting damaged by ROS for a long time is reason for ongoing nerve destruction.

Neuroinflammatory Pathways

Pathways concerning neuroinflammation and immune system were much enriched in the transcriptome results. Important inflammatory pathways included:

Cytokine signals

NF-kappa B signalling

TNF-alpha signal pathways

Interleukin-related pathways

Activation of innate immunity

Activated microglia and immune cells from periphery produce inflammatory substances which increase neuron damage and oxidative stress. Chronic neuroinflammation plays important role in how disease advances.

Pathways linked to programmed cell death also showed much enrichment. Examples are:

- * Caspase process activation
- * Apoptosis signalling from mitochondria
- * DNA response to harm
- * Cell stress-induced apoptosis

Turning on apoptosis pathways causes dopaminergic cell deaths in substantia nigra. Mitochondria problems and oxidative stress are main causes for apoptosis in neurons affected by Parkinson's disease.

Protein Aggregation and Degradation Pathways

The transcriptome analysis discovered that protein processing and breakdown pathways became enriched. These included:

- * Ubiquitin–proteasome system
- * Autophagy
- * Lysosomal degradation
- * Folding of the proteins pathways

Problems of these systems cause misfolded alpha-synuclein to pile up with the Lewy bodies formed which is a typical feature for Parkinson disease.

Metabolic Pathway Dysregulation

Important enrichment was found in the metabolic pathways with:

- * Glucose metabolism
- * Lipid metabolism
- * An amino acid metabolism
- * Energy balance

Malfunction in metabolism makes neurons less likely to survive and produces higher risk of oxidative damage.

CHAPTER- 4

DISCUSSION

Relationship Between Blood Indicators and Brain Abnormalities in idiopathic Parkinson's disease

Parkinson's disease is generally described as disorder of neural degeneration with dopamine neurons being lost in substantia nigra region. However, there is much data showing that disease scope is bigger than only central nervous system. Scientists have spent much energy studying the blood-based markers, since they are able to show molecular changes happening in entire body that connect with inflammation processes, mitochondria not functioning normally, or presence of metabolic stress [2], [6], [8], [9].

Meanwhile, the thesis now studies an GSE68719, which is an openly available transcriptomic dataset on Parkinson's, and reads its results together with biomarker research happening in other areas. So, the following analysis takes into account both the molecular signals discovered in the examined dataset and also reviews what is found in other research regarding peripheral blood markers and does not view the GEO dataset as only a blood sample collection [9], [10].

Value of Blood Biomarkers

It is hard to check brain damage directly in living people and this is why there is strong interest in a minimally invasive markers which can display neurodegeneration happening right now. This reason makes blood from body, cerebrospinal fluid or other samples that are easy to get, important parts of research for PD biomarkers [8], [9].

Blood as biomarker source is useful because:

- * Getting blood is not very invasive procedure
- * It is possible to take a blood samples on several separate occasions

- * Blood can show changes occurring in the whole body physiology
- * Bringing blood tests into clinical practice is simple and not expensive

Blood-based markers help in:

- * Finding disease sooner
- * Tracking disease as it changes
- * Checking for a future outlook
- * Measuring response to medical therapy

Wide Systemic Features of Parkinson's disease (PD)

Although PD targets nervous system, it is now thought to be a disease with effects on many of body's systems, like immune system malfunction, mitochondria issues, metabolic disruptions, oxidative stress happening and disturbances in blood vessels or inflammation. These big changes throughout the body form some of the reasons behind studies of blood markers in PD patients [2], [4]-[6].

- * Immune system irregularities
- * Mitochondria changes
- * Metabolism getting disturbed
- * Oxidative stress added
- * Vascular and inflammatory changes

These whole-body modifications are noticeable in blood cells in the body and could reflect disease processes in the brain itself.

The relationship between main brain degeneration and molecular changes outside the brain is a core reason for making blood marker studies in Parkinson's disease.

Brain Inflammation and Blood Markers

One of the most obvious way that peripheral blood markers and brain damage are connected is by neuroinflammation. Microglia cells are activated and trigger inflammation reactions that have

been proven as key players in PD's worsening with inflammation-related molecules possibly affecting the immune cells outside the brain and molecular profiles in the blood [6].

In Parkinson patients, microglia inside the brain will release inflammation markers:

- * Interleukin-1 beta (IL-1 β)

- * Interleukin-6 (IL-6)

These inflammation molecules can go into blood and change the function of immune cells outside brain, resulting in different profiles for gene expression in the blood.

The reports mentioned together show that blood inflammatory signals could reflect some neuroinflammation though, they are not total replacement for actually studying brain directly [8], [9].

Oxidative Stress and Mitochondrial Dysfunction in a Parkinson's disease, oxidative stress and wrong working of the mitochondria play an important part in the nerve cell death. Dopamine neurons need a lot of metabolic energy which make them easy to get oxidative damage, so disrupted antioxidant systems have clear science importance [4], [5].

CHAPTER- 5

CONCLUSION, FUTURE SCOPE AND SOCIAL IMPACT

5.1 Conclusion

Neurodegeneration in idiopathic Parkinson's disease happens due complex interactions between mitochondrial dysfunction, oxidative stress, alpha-synuclein aggregation, neuroinflammation, impaired protein degradation, apoptosis, and metabolic abnormalities. Transcriptomic analyses of GSE68719 gives strong evidence which interconnect mechanisms contribute to progressive dopaminergic neuronal loss.

Understanding of these molecular pathways is required for biomarker discovery, therapeutic target identification, and development of neuroprotective strategies which are aimed at slowing down disease progression and improving patient's health.

5.2 Future Perspectives

Even if there has been noticeable advancement, good biomarkers for diagnosing Parkinson's disease earlier, to keep an eye on how progression happens and for evaluating treatments are still not yet common in clinical care. Transcriptomic study using a GSE68719 dataset can find that there are important biological pathways which are linked to mitochondrial issues, oxidative stress, neuroinflammation, abnormal protein forming, metabolism problems and changes of coagulation. This means blood-based biomarkers are becoming more important for PD studies.

One main future aim is to create new diagnostic biomarkers which can tell if someone will have an Parkinson's disease before the main movement symptoms appear. Right now, it is usually diagnosed only after big part of dopamine neurons are lost. Future researchers will try to find smallest molecular changes which go with very early and pre-clinical Parkinson's disease. Early

detection by biomarkers can give earlier treatment, more positive prognosis, and delay in disease development for patients.

Blood from peripheral sources still is considered one of main promising ways for finding biomarkers because it is easy to collect, not expensive, possible to repeat and suitable for clinics. Further work in future probably will try to improve the blood-based molecular signals that have genes shown differently, different RNAs, inflammatory substances or metabolic things. Proving these biomarkers are reliable and can be repeated will need widespread validation experiments.

Biomarker research is more and more interested in using integrated “multi-omics,” joining transcriptomics with proteomics, metabolomics, epigenomics, lipid-omics and also microbiome reviews. By joining these data type together it might give wider view for mechanisms involved in PD and increase diagnostic accuracy. With multi-omics, it may become possible to find more connected molecular systems playing a role in neurodegeneration.

Challenges in Upcoming Research

Even with large improvements, there are still lots of issues. They include:

- * Standard procedures for analyzing
- * Studies that are not consistent
- * Biological difference is high
- * Some biomarkers can be challenging for repeating successfully
- * An expensive technology that is advanced

Therapeutic Effects Down the Line

- * An alpha-synuclein that collects
- * Oxidative stress is disrupted
- * Neuroinflammation targeted
- * Mitochondrial issues

- * Autophagy pathway changes
- * Problems in vascular systems

5.3 Social Impact

The gradual worsening harms affected people, their families caregivers medical workers and whole society. As populations of the world become older, idiopathic Parkinson's disease will likely grow by a large amount, making social and money burden go up.

Studies like transcriptomic analysis and biomarker hunt, such as the GSE68719 research, are socially valuable because it can help with quicker detection better disease handling new treatments and in the end higher life quality for those struggling with PD.

Impact on People with Parkinson's Disease

Parkinson's disease makes worse the physical functions, emotional stability, mental capacity and the social abilities over time. Motor issues like:

- * Shaking of hands and feet
- * Bradykinesia
- * Stiffness in muscles
- * Balance problems, these can really drop down how much person can move around or live on their own.

There are also symptoms that do not come from motor problems, which include:

- * Having a depression
- * Being anxious
- * Trouble thinking clear
- * Disturbed sleep patterns
- * Feeling tired and those usually affect the emotional health and being active in society. As PD advances, a number of patients start to face:

- * Being unable to work normally
- * Becoming socially lonely
- * Losing independence
- * Lowering their quality of living.

Finding out PD early from the research into biomarkers could maybe help patients do better, because it lets doctors give intervention earlier, and the treatments can be more tailored.

Influence on Relatives and Caregivers

- * Mental tension
- * Tiring out physically
- * Money problems
- * Being socially apart
- * Feeling psychological discomfort

Responsibility and care in the long term changes family relations and mental state in a broader aspect. If PD is advanced, quite help is required for:

- * Moving around
- * Doing the regular things
- * Medicine routines
- * Looking after the cognition
- * Offering an emotional assistance

Value for Public Health

There is more idiopathic Parkinson's disease seen all around the world, where it is increasing especially in older groups. Some of the reasons for this are:

- * People live longer
- * Environmental factors
- * More industrial settings
- * People recognizing diseases faster

A rising number of cases brings serious issues for the public health. Biomarker studies may help with:

- * Detecting disease sooner
- * More accurate tracking of numbers
- * Doing healthcare planning better
- * Finding strategies to stop disease

Public health organizations can get advantages from a new molecular screenings, with disease controlling programs that work better.

Why Early Diagnosis Matters

Mostly, doctors use symptoms to diagnose Parkinson's, but this usually comes after a lot of neuron damage. Looking for biomarkers early may bring some good social impacts:

- * Medical treatment sooner
- * Slower worsening of disease
- * People with independence
- * Lower disability
- * Good life quality in the long term

Blood biomarkers from transcriptomics research like GSE68719 can help provide a low-invasion, simple screening.

Advancing Personalized Medicine

Transcriptomics, along with biomarker investigation, can support personalized medicine that is focused on the individual's specific molecular patterns. The probable positives in society are:

- * Choosing a treatment that works better
- * Fewer unwanted side effects from medicines
- * Patients may get better results from the therapy
- * Patient satisfaction is improved
- * Healthcare resources can be used in a more efficient way

Personalized medicine maybe helps to improve care for people with different kinds of disease and the way their illness develops.

Decreasing Healthcare Burden

Development of a trustworthy biomarkers together with disease-altering treatments might cut down the long-term load on hospitals and clinics. Good outcomes are:

- * Hospitalization numbers are less
- * Outpatient care improves
- * Progress of disability may be postponed
- * There is need for much less institutional care
- * Medical payments drop as time passes

Preventing earlier before the disease grows can keep a person functional and more independent for more years.

Effects on Mental Health and Social Life

Parkinson's disease often affects mentality and social life of those affected. They can suffer:

- * Depression

- * Anxiety
- * Avoid others
- * Low self-esteem
- * Trouble communicating

Diagnostic and care methods that have been improved gives support for psychological state and social integration.

Scientific and Educational Influence

Biomarker together with a transcriptomic study plays important roles in the science development and learning for medical field. These findings are to:

- * Make better the knowledge about neurodegeneration
- * Give a training for health care workers
- * Move forward an precision medicine
- * Interdisciplinary teamwork gets supported
- * Pushing new ideas in biotechnology, along with medical treatments

Research using datasets including GSE68719 is also helping with open cooperation and scientists share data more easily in the research community.

Ethical and Social Factors

With progress in biomarker technologies, it becomes necessary to mention about several ethical and social matters. Some concerns are known, such as:

- * Privacy regarding genetic and molecular type information
- * How accessible the new diagnostics are
- * Inequality in healthcare systems for different people
- * The mental effects when a person gets an early diagnosis

- * If predictive biomarkers are used in a right way

Making sure that people can access biomarker-related health services equally is an important requirement to bring the most advantage to the society in general.

Role of AI and Digital Health

In the future, adding the artificial intelligence and digital health tools with biomarker studies might cause improvement in giving healthcare. Some things that can happen are: *

AI-based systems to help with diagnosis

- * Monitoring disease from distance
- * Risk prediction with a personal focus
- * Telemedicine for management
- * Biomarker analysis by automation

Through these, there might be higher access to healthcare, more for people in areas with less resources.

Research Collaboration on a Global Scale

Study of biomarkers for Parkinson's disease encourages scientists everywhere to work together and use different scientific fields like:

- * Neurology specialty
- * Molecular biology topics
- * Usage of bioinformatics
- * Involvement in genomics
- * Artificial intelligence
- * Clinical medicine know-how

Collaborating worldwide could speed up making useful diagnosis and treatment methods.

Future Benefits for Society

Further progress in biomarkers study might eventually bring:

- * Preventing diseases earlier than before
- * Getting better life quality
- * Burden from disability to be less
- * Neuroprotective treatments that work better
- * People having more independence in their lives
- * Higher awareness in the society about diseases that cause neurodegeneration

These improvements can strongly change the long-run outcomes of patients and whole society.

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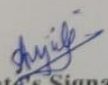
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