

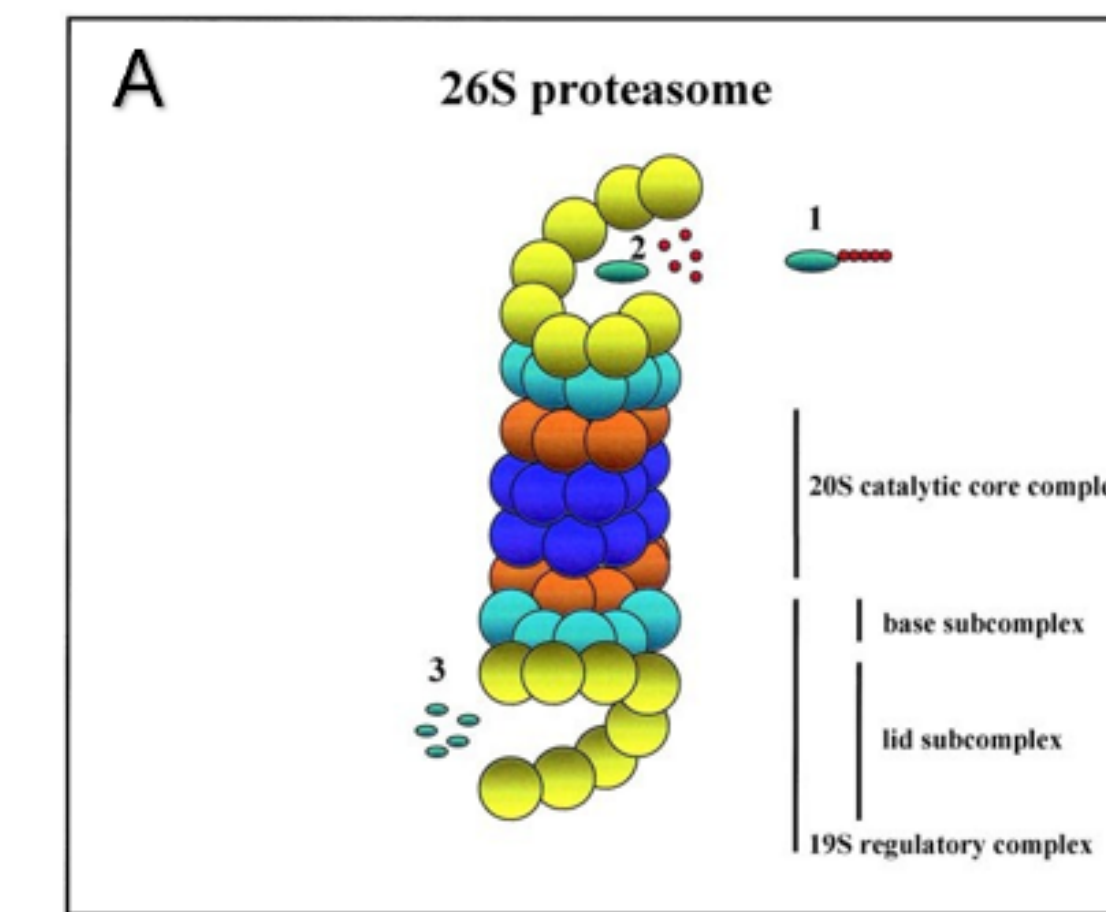
INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive lung disorder characterized by airflow limitation, inflammation, and tissue remodeling. At the molecular level, COPD is associated with **cellular stress, mitochondrial dysfunction, and impaired protein turnover**.

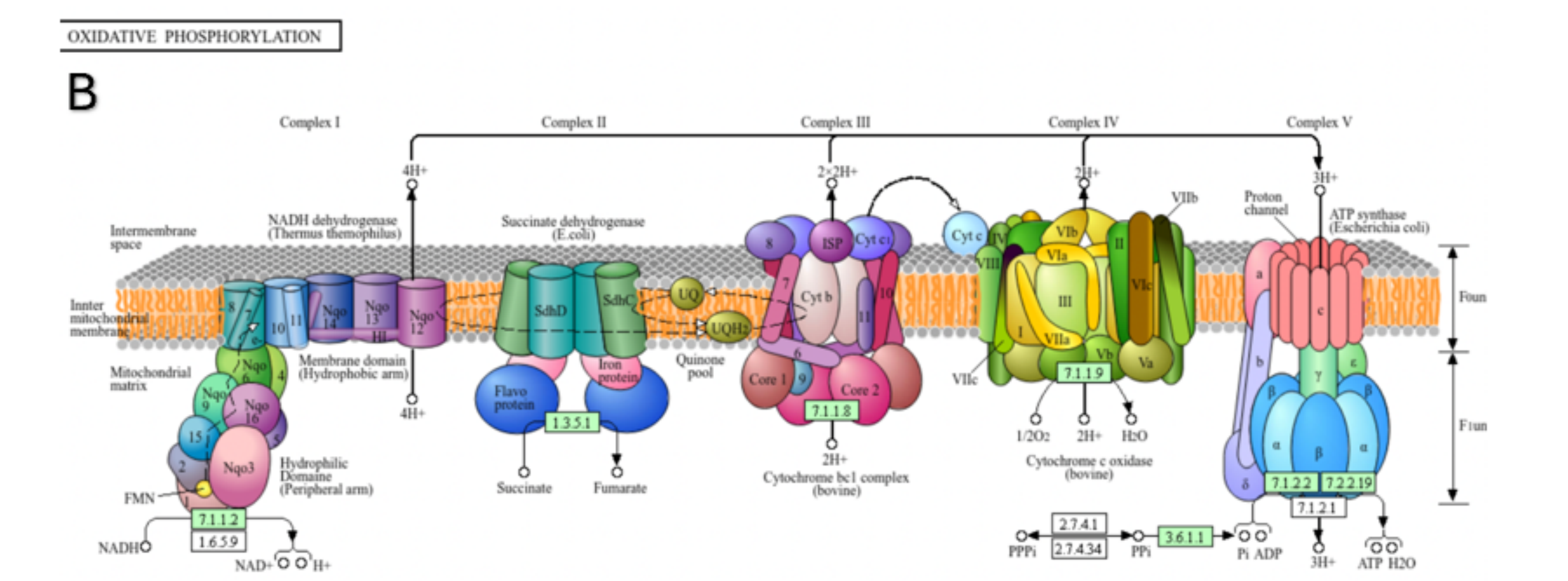
We aim to identify **key genes and pathways dysregulated in COPD lung tissue compared to control non-COPD lung tissue**, with a focus on two functional categories:

- 1. Proteasome-related genes:** mediators of protein degradation, whose dysfunction can lead to accumulation of damaged proteins and increased cellular stress.
- 2. Oxidative phosphorylation (OXPHOS) genes:** critical components of mitochondrial energy metabolism, often downregulated under oxidative stress conditions.

Proteasome and Oxidative phosphorylation

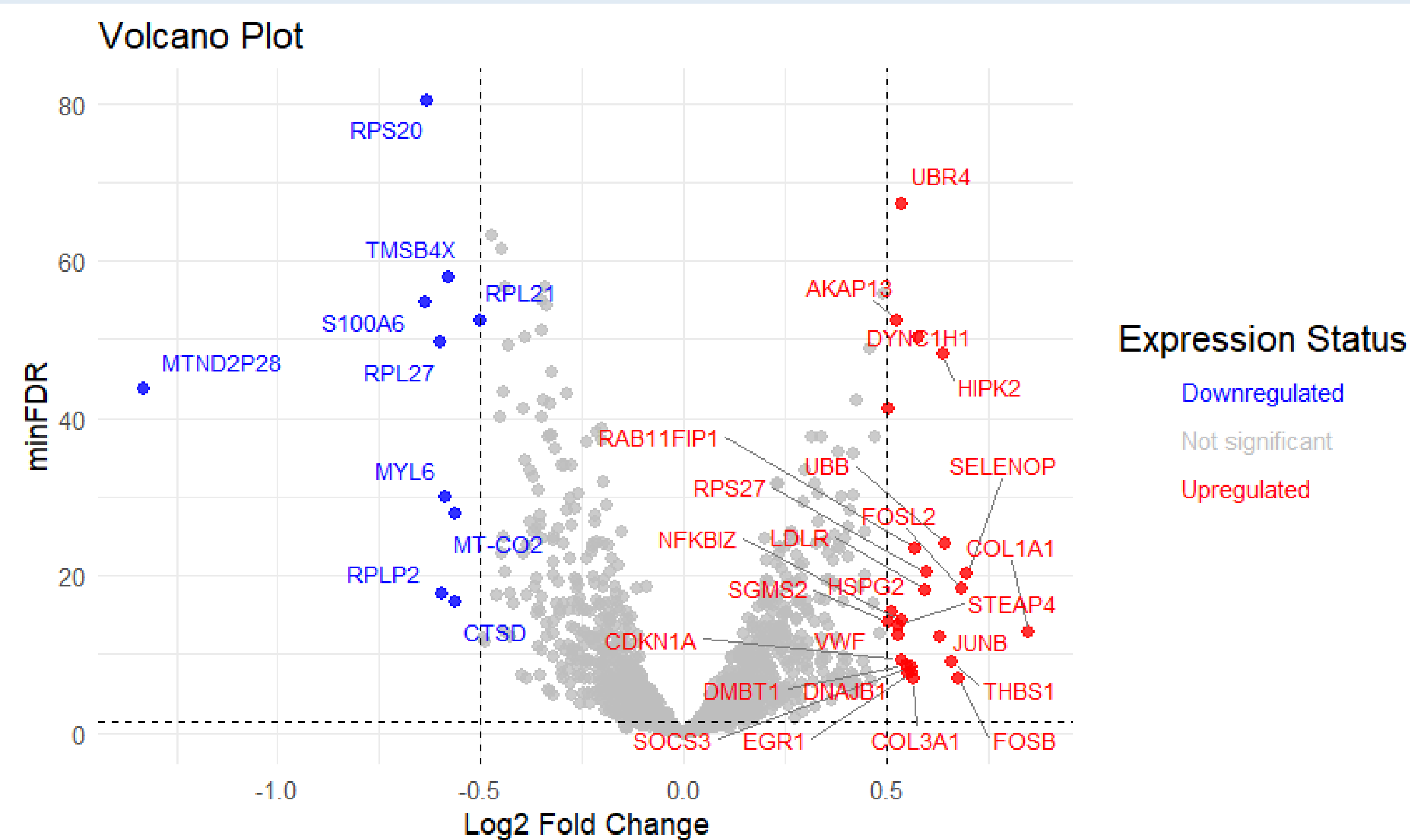
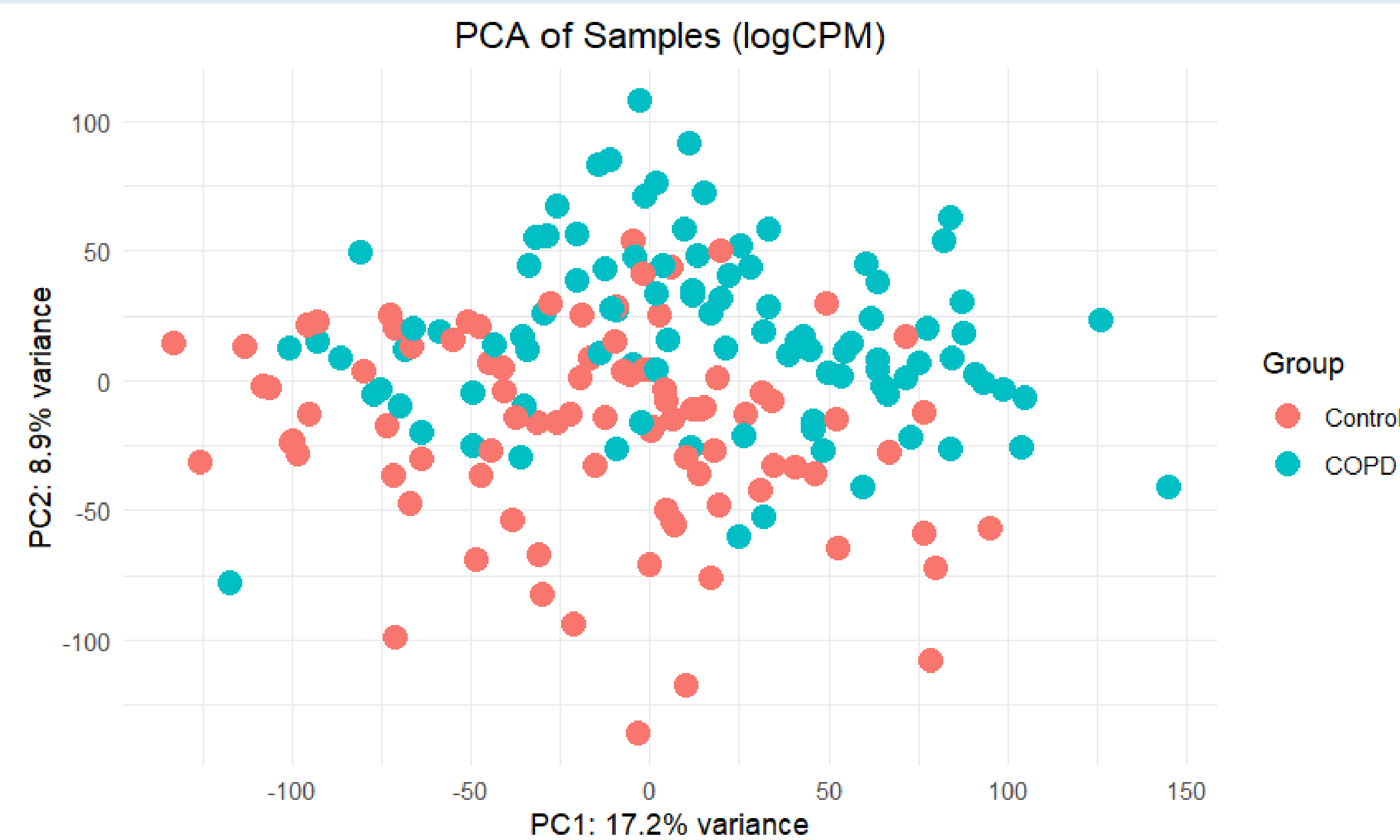


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(KEGG PATHWAY: Oxidative Phosphorylation - Homo Sapiens (Human), n.d.)

RESULTS



Discussion

- Oxidative phosphorylation genes differentially regulated in COPD
 - ATP5B1** couples proton translocation to ATP synthesis in the mitochondrial membrane, enabling efficient energy production. Its downregulation in COPD likely causes a reduction in ATP production and disrupts pH regulation, impairing lung cell repair and accelerating disease progression.
 - MT-ATP6** forms a proton channel in the mitochondrial inner membrane, driving ATP synthase rotation and enabling ATP production. Its downregulation in COPD could impair oxidative phosphorylation, by reducing ATP synthesis, and increasing ROS, potentially worsening lung cell damage and disease progression.
 - MT-ND4L** is a transmembrane subunit of complex I (NADH dehydrogenase). Transfers electrons from NADH to ubiquinone. Upregulation could be a possible compensatory response to impaired electron transport chain function.
 - COX7C** is part of the mitochondrial respiratory chain complexes. It catalyzes electron transfer from cytochrome c to oxygen. Downregulation may indicate mitochondrial dysfunction, reduced ATP, and increased ROS.
- Proteasome genes upregulated in COPD lung tissue compared to the control
 - STAT3** is a transcription factor that activates cytokines and growth factors. It is involved in cell survival, proliferation, inflammation and immune response.
 - EGFR** is a receptor tyrosine kinase, activated by growth factors. It controls cell proliferation, differentiation, survival and tissue repair.
 - LRRK2** is a serine/threonine kinase that functions in vesicle trafficking, autophagy and immune responses, and cytoskeleton dynamics

Conclusion

There are several significant changes in the proteasome pathway show an upregulated gene expression. This is a possible contribution to the progression of COPD. COPD may also be associated with broad mitochondrial dysfunction in several components of the electron transport chain, leading to energy deficits, oxidative stress, and eventual apoptosis.

ACKNOWLEDGEMENTS

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U.S. National Library of Medicine. (n.d.). **MT-ATP6** mitochondrially encoded ATP synthase 6 [Homo sapiens (human)] - Gene - NCBI. National Center for Biotechnology Information. <https://www.ncbi.nlm.nih.gov/gene/4508>

Our code:
<https://docs.google.com/document/d/19XzReP-Wf7chz4YwTThQnKzRTAemhNXs3wcFFkgRv/edit?usp=sharing>