MP exhibits notable advantages in the clinical study and has high anti-PD potential in toxin-induced PD models. MP has strong anti-PD action because of its high L-DOPA content. Although several Parkinson's research has so far demonstrated that MPE has significant anti-parkinsonian effects, the possible mechanisms behind these benefits have not yet been thoroughly investigated (27). In order to understand the complex connections between herbs and illnesses at a systematic level and to adhere to a systematic and holistic approach, network pharmacology integrates bioinformatics, systems biology, and pharmacology. To investigate the possible bioactive components of MP, we used two OB and DL. Studies on the pharmacokinetics of MP have been few thus far. It has shown that the constituents of MP (mainly Beta-sitosterol and, Levodopa) and, SCFAs (mainly acetate and propionate) target dopaminergic synapses which may be useful in the treatment of PD. We identified 4 active compounds from MPE and 120 possible targets in total for our study. We also showed that our herb is a synergistic one with multi-component, multi-target, and multi-pathway properties. The compound-target network revealed that the primary elements were SCFAs (propionate and acetate) and MP (beta-sitosterol, levodopa). In addition, the PPI network revealed data on co-expression and homology of proteins as well as information about the origin of the interactions. The biological network as a whole, comprising targets including PPARA, HDAC1, EGFR, HMGCR, ACHE, TOP2A, MAOA, SI, PCNA, GCG, COMT, CDK2, MGAM, OPRM1, APEX1, RXRA, ESR2, NR1H3, MCL1, SLC18A2, FABP3, KDM6B, and UCP3, was significantly impacted by MP, according to our PPI study. The findings of the

enrichment also suggested that the mechanism by which MP prevents Parkinson's disease (PD) may be related to the coordinated regulation of multiple pathways, including the cAMP signaling pathway, the neuroactive ligand-receptor interaction, the calcium signaling pathway, the dopaminergic synapse, and other interconnected pathways.

We also investigated the neuroprotective effects of MPE on locomotor function, anxiety-like behavior, and antioxidant capacity in mice with ROT-induced PD at two doses: 100 mg/kg and 200 mg/kg. MPE's neuroprotective activity was also evaluated against ROT-induced neurotoxicity in SH-SY5Y cells. MPE exhibits antioxidant and neuroprotective properties in mice and SH-SY5Y cells, modulating the expression of alpha-SYN, OH-1, Parkin-1, p-NF-κB, and GLP-1 in PD illness. We looked at the effectiveness of MPE in the developed model of PD and discovered that it lowered the phenotypic and pathological features of the condition. MPE's anti-oxidative and anti-aggregative properties support dopaminergic neurons' survival, proliferation, and differentiation.

As an addendum to previous research on medications against PD, this study used a network method to detail how MP chemicals modify several pathways against PD. Additionally, we showed that MP has a significant impact on several PD-related targets. We fervently hope that our study will contribute to the use of network pharmacology in elucidating the possible mechanisms of anti-parkinsonism herbs and offer insights into how herbs operate in concert to treat other complicated illnesses. From a critical perspective, this study does have certain shortcomings, though. Further tests (Western blot, immunofluorescence on cell lines analysis) are required to firmly confirm our findings, as this work was based only on data analysis. Furthermore, more research is required to confirm the possible benefits of MP for gut health and neuroprotection, even if this has only been

mentioned in a small amount of literature. In the meanwhile, studies on the pharmacokinetics of MP against PD are required to demonstrate its properties.

Future Directions: As mentioned above there are some limitations in this study briefly network pharmacology have shown various enriched genes like PPARA, HDAC1, EGFR, HMGCR, ACHE, TOP2A, MAOA, SI, PCNA, GCG, COMT, CDK2, MGAM, OPRM1, APEX1, RXRA, ESR2, NR1H3, MCL1, SLC18A2, FABP3, KDM6B, UCP3, DRD3, and DRD4. These genes will have to be evaluated for a proper understanding of the mechanism behind MP in the treatment of PD.