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Integration Of NGS And Toxicity Prediction For Safe Drug Target Discovery In Parkinson's Disease

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Abstract
Parkinson's disease (PD) is a progressive neurodegenerative disorder characterised by the degeneration of dopaminergic neurons and abnormal protein aggregation. Building upon prior computational docking studies, this work presents an extended integrative bioinformatics framework combining structural validation, sequence analysis, molecular docking, and systems-level investigations to improve the identification of potential therapeutic targets. Protein structures associated with PD were retrieved and analysed using Bio Python, followed by structural validation using structure assessment and Verify3D to ensure model reliability. Sequence similarity and conserved regions were evaluated using Sequence similarity search, while multiple sequence alignment and phylogenetic analysis were performed using MSA and Jalview. Functional motifs and domains were identified using PROSITE, and interaction profiling was conducted using PDBsum and LigPlot. Protein-ligand docking was carried out using CB-Dock and Auto Dock Vina to identify potential binding sites and evaluate ligand affinity. Additionally, toxicity prediction of candidate compounds was performed using Toxicity Identification to assess pharmacological suitability. Protein–protein interaction (PPI) network analysis and pathway mapping were incorporated to provide a systems-level understanding of disease mechanisms. The integrated workflow enhances the reliability of structural predictions, improves functional interpretation, and refines candidate selection for therapeutic development. This study demonstrates the importance of combining multi-level computational approaches for comprehensive analysis in neurodegenerative disease research.

Keywords: *Parkinson's disease, Toxicity Prediction, System Biology, PPI network, Molecular Docking, Phylogenetic Analysis, Protein Structure Validation*

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I. Introduction

Neurodegenerative disorders are associated with a gradual decrease in neurons and their functioning, resulting in lasting damage to the central nervous system. Parkinson's disease (PD) is one of the most well-known neurological disorders (other names and types) and is estimated to affect 1% of people over the age of 50, and is, among other things, a major cause of long-term disability worldwide. PD is diagnosed by the presence of primary motor symptoms: resting tremors, rigidity, bradykinesia, and postural distortion, which fundamentally stems from the loss of dopamine-producing neurons in the substantia nigra pars compacta. At a molecular level, the development of PD is influenced by protein misfolding and/or over-aggregation, mitochondrial dysfunction, excessive oxidative stress, and defective degradation pathways. The genes that play significant roles in the development of PD include α-synuclein (SNCA), DJ-1 (PARK7), parkin (PRKN), PINK1, and LRRK2.[1–6]

While the majority of PD cases are sporadic, there is a growing body of literature describing monogenic cases of PD, which can lead to a better understanding of the disease process, with mutations in SNCA, PRKN, PINK1, DJ-1, and LRRK2 found to play a role in familial and early-onset PD. Specifically, LRRK2 (PARK8) is reported to be the most frequent cause of autosomal dominant PD in multiple patient demographics. The pathways of these genes collectively show multiple potential avenues to develop therapies for PD, including the use of mitochondrial quality control, synaptic vesicle traffic, and neuroinflammation.[7,8]

Current therapies primarily treat symptoms and try to retain patient health through the use of medications such as levodopa/carbidopa combinations, dopamine agonists, MAO-B inhibitors, COMT inhibitors and deep brain stimulation in people who are severely affected by Parkinson's disease (PD). In general, levodopa therapy is thought to be effective over a long period of time, but after prolonged treatment, patients may develop motor fluctuations that lead to dyskinesia. None of the currently available treatments has been shown to stop or reverse the neurodegeneration associated with PD. Thus, there is a great need to develop new, safer medications using systematic investigative efforts to identify safe and novel mechanisms based on understanding how to target drug compounds that are non-toxic.[4,7,9]

Next-generation sequence (NGS) is changing how we view the genetics of PD by helping us identify new rare and common variants of PD; finding new loci associated with familial PD; and clarifying options for understanding the genetic basis of sporadic PD through identifying risk factors associated with sporadic PD. Once we combine the variant and expression data obtained from NGS with protein structural data derived from bioinformatics tools, and use docking techniques as well as systems approaches, we can effectively identify proteins and pathways that will be useful for drug manufacturers to target for PD treatment. At the same time, developing in silico prediction methods for toxicity and using ADMET analysis of drug candidates have become critical to reduce the rate of failure of drug candidates for neurodegenerative diseases. The development of new bioinformatics approaches enables researchers to create bioinformatic tools to screen putative neurodegenerative drugs using biological data obtained from Bio Python, enabling structural parsing, docking, and basic systems analysis, which have already demonstrated the feasibility of such integrative approaches for neurodegenerative targets, including PD-associated proteins.[5,10–12]

The present study builds on our previous studies, including our previous integrative Bio Python and docking-based workflow for neurological targets developed, and proposes an expanded framework that incorporates a systematic integration of NGS (next generation sequencing)-driven target selection with protein structure validation, sequence and evolutionary analysis, protein-ligand docking, toxicity prediction, and protein-protein (PPI) interaction network and pathway analysis relative to PD (Parkinson's disease). This pipeline retrieves PD-associated protein structures and assesses each protein, utilising Bio Python-based structural parsing and model validation, followed by the execution of multiple sequence alignment and phylogenetic analyses to evaluate the evolutionary conservation of functional regions within PD-associated proteins. Functional motifs and domains within the target proteins are annotated, and interaction profiles are generated using structural interaction servers. The identified candidate small molecules are then in silico assessed for toxicity and pharmacokinetic predictions to determine their drug-likeness and safety, and PPI networks and pathway mapping provide a systems-level view regarding the relevance of the targets and potential for off-target effects. By integrating NGS with multi-layered structural, functional, and toxicity data, this study aims to refine the prioritisation of safe and effective drug targets for Parkinson’s disease and to illustrate a generalisable bioinformatics workflow that can be applied to other neurodegenerative disorders.[13]

II. Materials And Methods

Protein Retrieval and Preprocessing

Protein structures associated with Parkinson’s disease were retrieved from the Protein Data Bank (PDB) using their corresponding PDB IDs. Structural data were downloaded in .pdb format. BioPython (Bio.PDB module) was employed for automated parsing and preprocessing of protein structures.[14]

Structural Validation of Protein Models

To ensure structural reliability before docking, protein models were evaluated using multiple validation tools used for assessed the quality of non-bonded atomic interactions and overall structural reliability. Evaluated the compatibility between the 3D structure and its amino acid sequence (1D–3D profile). Structures with acceptable quality scores (ERRAT score > ~80% and satisfactory Verify3D profiles) were selected for further analysis. Poor-quality structures were excluded or reprocessed.[15]

Sequence Retrieval and Similarity Analysis

Protein sequences were extracted from PDB files or retrieved in FASTA format. Sequence similarity searches were conducted using BLAST against protein databases to:

- Identify homologous sequences
- Evaluate sequence conservation
- Detect functionally important regions

Top hits were selected based on sequence identity, query coverage, and E-value thresholds.[16]

Multiple Sequence Alignment (MSA)

Multiple sequence alignment was performed using Clustal Omega to identify conserved residues and structural motifs.[17]

The resulting alignments were visualised and annotated using Jalview, enabling:

- Identification of conserved and variable regions
- Detection of mutation hotspots
- Colour-coded visualisation of residue conservation

Phylogenetic Analysis

Phylogenetic analysis is a computational approach used to infer the evolutionary relationships among biological sequences, such as proteins or nucleic acids, based on their sequence similarity and divergence patterns. It relies on the principle that sequences sharing a common ancestor accumulate mutations over time and therefore retain varying degrees of similarity that can be quantitatively compared. Phylogenetic trees were constructed based on aligned sequences to analyse evolutionary relationships among homologous proteins.[8]

Tree construction methods included:

- Distance-based clustering
- Neighbour-joining approach (default in Clustal Omega output)

The resulting trees were visualised and interpreted using Jalview to assess evolutionary conservation relevant to functional domains.

Motif and Domain Identification

Functional motifs and domains were identified using the PROSITE database and sequence scanning tools.

This analysis enabled:

- Detection of conserved functional signatures
- Identification of active or binding regions
- Correlation of motifs with known biological functions

Domain architecture was further cross-validated with structural data where applicable.

Structural Visualisation and Analysis

Three-dimensional structures of proteins and protein–ligand complexes were visualized using structural bioinformatics tools. Visualisation steps included: Identification of active/binding sites, highlighting of conserved residues; structural comparison between native and docked conformations. Advanced PyMOL features such as scripting and surface rendering were used for detailed structural interpretation.

Protein-ligand interactions were analysed using PDBsum and LigPlot to generate detailed interaction maps.

The analysis included:

- Identification of hydrogen bonds
- Hydrophobic interaction mapping
- Residue-level interaction profiling

These interaction maps were used to validate docking results and identify key residues involved in ligand binding.

Toxicity Prediction

Toxicity prediction is a computational approach used to estimate the potentially harmful effects of chemical compounds on biological systems before experimental testing. It plays a critical role in early-stage drug discovery by identifying compounds that may cause adverse effects such as organ toxicity, carcinogenicity, mutagenicity, or acute toxicity. In silico toxicity prediction tools, such as ProTox-II, classify compounds into toxicity classes and estimate parameters such as median lethal dose values, commonly referred to as LD50. These tools may also provide predictions for specific toxicological endpoints, including hepatotoxicity, immunotoxicity, cytotoxicity, and potential carcinogenic effects.[11]

The following parameters were predicted:

- Toxicity class
- LD50 values
- Potential hepatotoxicity and carcinogenicity

Compounds with favourable toxicity profiles were shortlisted for further consideration.

Protein-Protein Interaction (PPI) Network Analysis

Protein–protein interaction networks were constructed to understand the biological context of target proteins in Parkinson's disease.[19]

Interaction data were analysed to:

- Identify key interacting partners
- Detect hub proteins
- Understand network topology

Pathway Analysis

Pathway analysis was performed to map proteins onto biological pathways relevant to Parkinson's disease.[20]

This step helped:

- Identify signalling pathways involved in disease progression
- Correlate molecular interactions with functional outcomes
- Provide systems-level insight into therapeutic targeting

III. Results

Identification of key target proteins

The initial screening identified a highly relevant set of proteins, such as PRKN, PINK1, SOD1, SNCA, HSPA9, MAP3K5, LRRK2, BCL2L1, DAXX, and NFE2L2 (Figure 1,2). These proteins showed high confidence interaction scores (0.969-0.999) and have very strong biological significance with respect to their function. They have an important role in the regulation of mitochondrial homeostasis, oxidative stress and apoptosis processes. For example, PRKN and PINK1 are both important regulators of mitophagy, whereas SOD1 and NFE2L2 are both important for the body's defence against oxidative stress. The presence of SNCA (a major protein in the regulation of synapses) is yet another indicator of the dataset's significance to neurodegenerative diseases, particularly Parkinson's disease.[21]

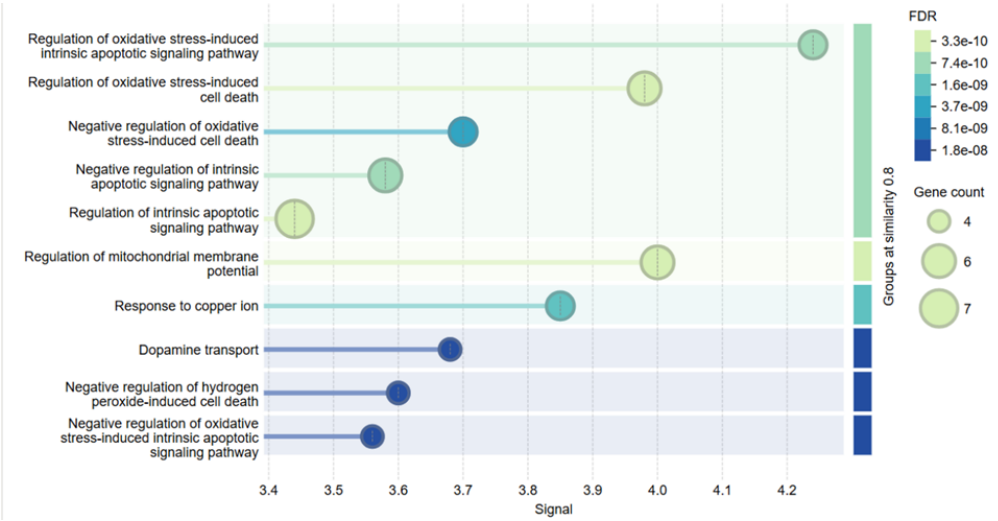

Figure1. Identification of Key proteins associated with neurodegeneration

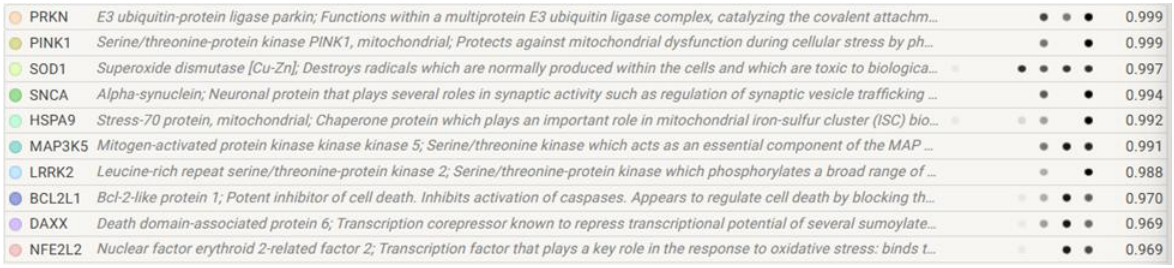

Figure 2. Predicted Functional Partner STRING

Protein-Protein Interaction Network Reveals Functional Connectivity

The PPI network (Figure 3) reinforced the high level of connectivity between these important proteins, indicating that these proteins are all likely retained to each other via common regulatory mechanisms. For example, SNCA was shown to be an important hub connecting many of the pathways associated with neuronal survival and the response to stress. SOD1 and NFE2L2, as well as MAP3K5, are all tightly connected, and together they comprise an important oxidative stress response pathway. The fact that the apoptotic regulators BCL2L1 and DAXX interact with each other and with one or more other proteins suggests that there is crosstalk between the pathways associated with stressful signals and programmed cell death. This dense network suggests that perturbation in one component could propagate through multiple pathways, contributing to disease progression.[22]

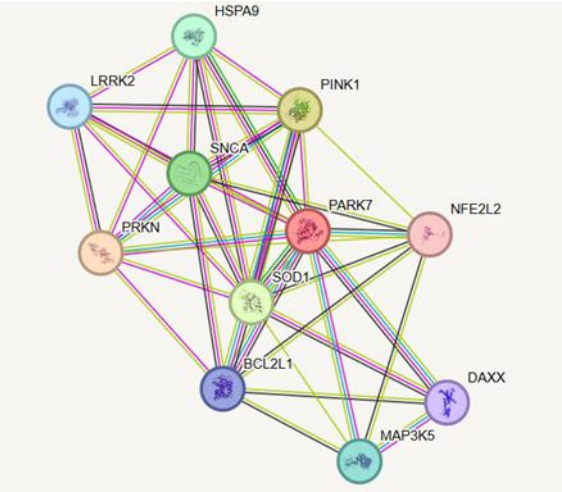

Figure 3. Protein-protein interaction network reveals functional connectivity

InterProScan Analysis of Conserved Domains and Functional Architecture

Protein 7C62 is strongly enriched in the regulation of oxidative stress-induced apoptotic (cell death) signalling (removing damaged and dead cells) and dopamine transport (Figure 4)**.** Functional pathway enrichment analysis (Figure 4) has shown an enriched representation of biological pathways involved in the regulation of oxidative stress and apoptosis. The most enriched pathways included regulation of the intrinsic apoptotic signalling pathway due to oxidative stress, regulation of mitochondrial membrane potential, and response to copper cations. Much to note is the enrichment of dopamine transport, which connects the protein set to a pathway for the development of Parkinson's disease. The signal enrichment ranged from approximately 3.4 to 4.2, with very low false discovery rates (FDRs) (10^{-8}-10^{-10}), demonstrating the strength of these findings. Overall, the findings show that the proteins found in this protein set are collectively involved in regulating redox balance, mitochondrial integrity, and neuronal survival; all of which are crucial processes involved in the progression of neurodegenerative diseases.[23]

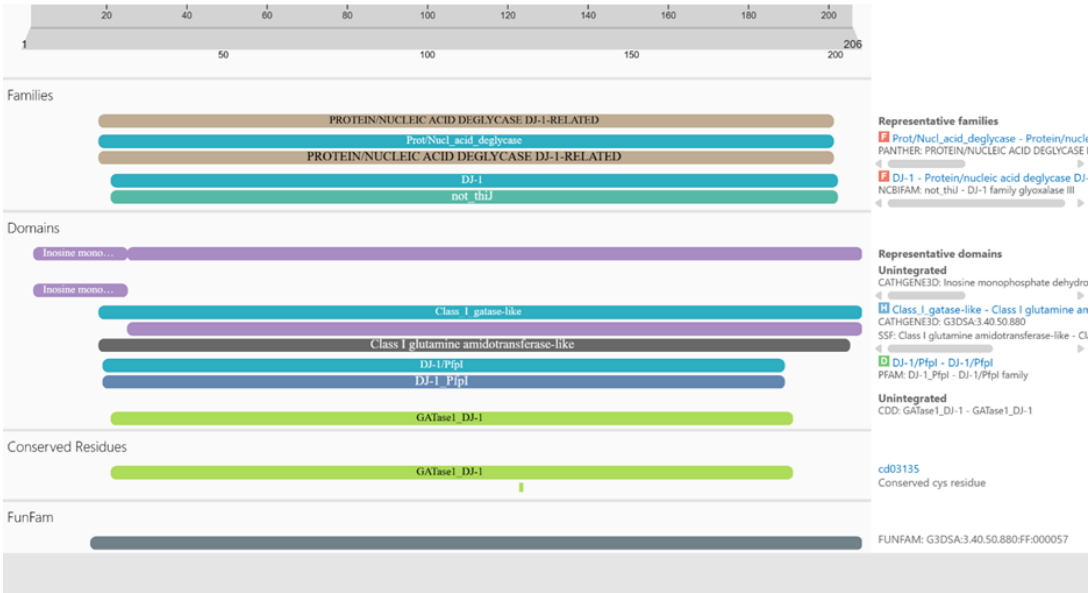

Figure 4. Sequence Analysis using INTERPROSCAN, Domain Architecture Indicates Conserved Function.

The protein analysed is strongly associated with the DJ-1/Protein-Nucleic Acid Deglycase family, as shown by multiple domain and family annotations. The presence of Class I glutamine aminotransferase-like and DJ-1/PfpI domains, along with a conserved catalytic cysteine, suggests the protein likely plays a role in cellular defence mechanisms, such as deglycation, oxidative stress response, or enzymatic detoxification. typical DJ-1 family functions, which are crucial in maintaining cellular homeostasis and may have implications in stress-related disorders or diseases.

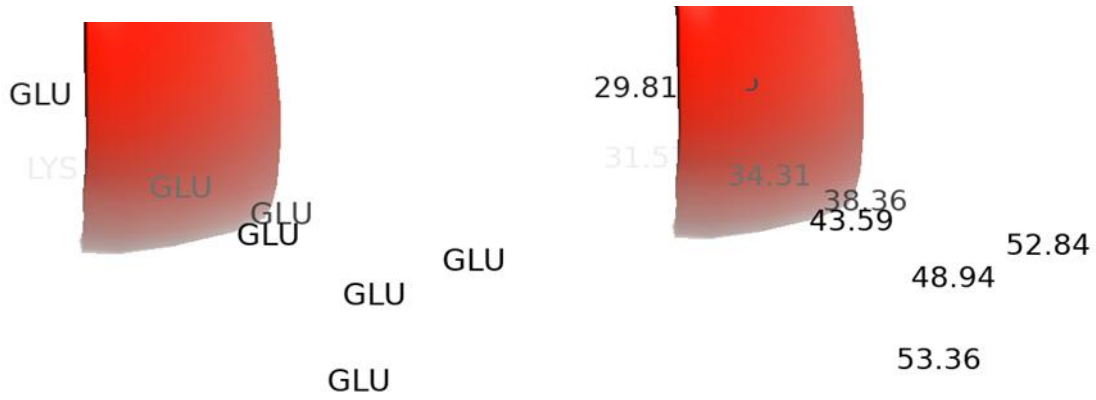

Figure 5. B factor analysis of the proteomic sample 7c62

B-factor Analysis: GLU (176 residue) if glutamine level is high in the brain, as a biological factor in the context of neurodegenerative disease, due to high glutamate, increased glutamate synthesis leads to overactivation, causing calcium overload and neuronal death.

Mitochondrial Dysfunction (oxidative stress and cell damage), increased neuroinflammation, and excess glutamine reflect high brain ammonia, leading to brain swelling.[24]

Multiple Sequence Alignment Confirms Conserved Functional Residues

Using the Jalview program (Figure 6) for multiple sequence alignment yielded a very high level of sequence conservation between homologous sequences. An important observation in the conserved regions of the homologous sequences was the frequent presence of GLU. This suggests that GLU may play a role in subunits' catalytic activity and molecular interactions.

The conservation of charged residues likely suggests that they are responsible for stability in three-dimensional structure, facilitating protein-to-protein interactions, and providing ligands with the opportunity to bind. These conserved motifs may be essential components for preserving the functional integrity of the protein.

The conservation of charged residues across the homologous sequences indicates that mutations within these motifs will influence the functionality of the protein and be involved in the development of a disease.

Toxicity Analysis

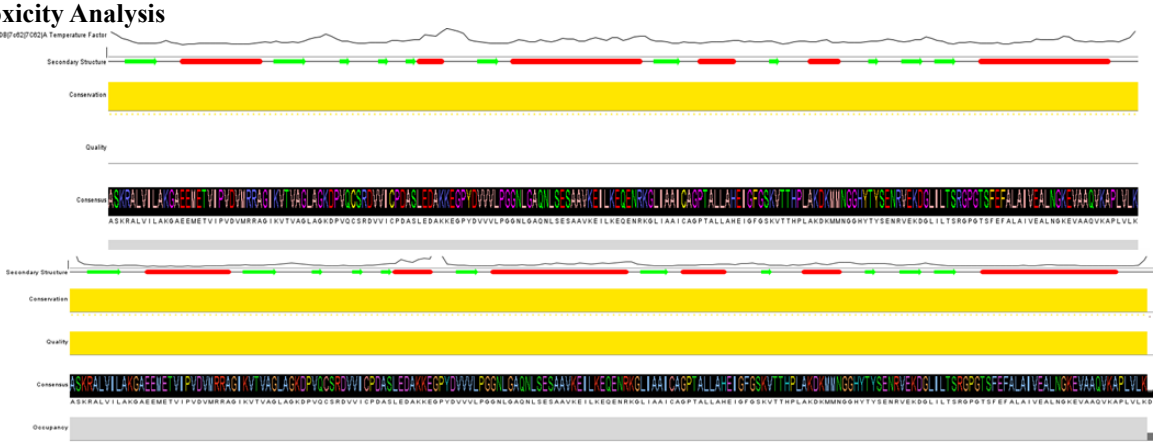

Figure 6. a) Jal view Analysis using proteomic sample with 3 Samples- 7C62, 7PA2, 6M8Z b) Jal view representation of code 7C62.

Results of an ADMET Profiling Study of Apomorphine and Gne-7915

It demonstrated several differences between these two compounds in terms of their toxicological and pharmacokinetic properties.

The toxicity of apomorphine was found to be substantially greater than that of Gne-7915, as reflected by their respective LD_{50} values of 4000 mg/kg for Apomorphine versus 1190 mg/kg for Gne-7915, placing rutin in Toxicity Class V and baicalein in Toxicity Class IV. Gne-7915 therefore has a better safety profile than baicalein (Table 1) (Figure 7).

Both compounds exhibited a high likelihood of showing neurotoxicity and respiratory toxicity with respect to their ability to produce adverse effects on the nervous and respiratory systems; however, neither compound exhibited evidence of carcinogenicity or nutritional toxicity, supporting their potential therapeutic use.

Apomorphine also exhibits limited ability to penetrate the blood-brain barrier (BBB). This limitation in the ability to reach the CNS may limit the potential therapeutic utility for the treatment of neurodegenerative diseases, thus requiring the investigation of new delivery technologies such as nanoformulations. Mechanistically, both compounds were found to have activity on PPAR-γ, a receptor implicated in producing anti-inflammatory and neuroprotective effects. Conversely, both compounds were found to lack activity against the most common neuronal receptors, namely GABA, NMDA, and AMPA, suggesting that their mechanism of action is not through direct modulation of neurotransmitter levels, but rather through alternative pathways such as regulation of oxidative stress.[5]

Oxidative stress signalling pathways and apoptotic induction were not shown o have minimal activity with either compound as they both failed to activate NRF2/ARE signal transduction, disrupt mitochondrial membrane potential, and activate p53. Thus, there appears to be the potential for both compounds to be safe in cells.The results of analyses assessing the interaction of cytochrome P450 enzymes with both compounds indicate that CYP1A2 and CYP2D6 demonstrated inactive metabolite interactions with these compounds, indicating a reduced potential for metabolic interference. CYP3A4 metabolite interactions with Gne-7915 were predicted to produce potential drug-drug interactions or altered metabolism; the prediction for Apomorphine was inactive, indicating a relative stability in its metabolic behaviour compared to Gne-7915.

Table 1. ProTox-II-based toxicity prediction of selected compounds showing activity across multiple toxicity endpoints and enabling comparative safety assessment

Target	Apomorphine	Gne-7915
LD_{50} mg/kg	1190 mg/kg	4000 mg/kg
Toxicity Class	Class IV	Class V
Neurotoxicity	Active (0.88)	Active (0.87)
Respiratory toxicity	Active (0.91)	Active (0.98)
Carcinogenicity	Inactive (0.52)	Inactive (0.62)
BBB-barrier	Inactive (0.92)	Active (0.65)
Nutritional toxicity	Inactive (0.56)	Inactive (0.97)
Peroxisome Proliferator Activated Receptor Gamma (PPAR-Gamma)	Active (0.63)	Active (0.56)
Nuclear factor (erythroid-derived 2)-like 2/antioxidant responsive element (nrf2/ARE)	Inactive (0.98)	Inactive (0.98)
Mitochondrial Membrane Potential (MMP)	Inactive (0.99)	Inactive (0.99)
Phosphoprotein (Tumour Suppressor) p53	Inactive (0.96)	Inactive (0.97)
GABA receptor (GABAR)	Inactive (0.97)	Inactive (0.90)
Glutamate N-methyl-D-aspartate receptor (NMDAR)	Inactive (0.96)	Inactive (0.96)
alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionate receptor (AMPAR)	Inactive (0.92)	Inactive (0.92)
Cytochrome CYP1A2	Inactive (0.76)	Inactive (0.97)
Cytochrome CYP2D6	Inactive (0.63)	Inactive (0.63)
Cytochrome CYP3A4	Inactive (0.65)	Active (0.71)

Apomorphine has a molecular weight in the lower end of the weight spectrum (Figure , falling close to the mode of the distribution (~300-350 g/mol). This area of the distribution contains the majority of drug-like compounds, thus indicating that apomorphine has favorable physicochemical properties for drug development. In general, lower molecular weight compounds demonstrate:

❖Increased Absorption and Permeability
❖Increased Probability of Oral Bioavailability
❖Lipinski's Rule of Five Compliance

Therefore, with respect to its molecular weight, apomorphine is located in the range considered optimal for drug-likeness and may possess favourable pharmacokinetic behaviour. On the other hand, GNE-7915 falls in the higher molecular weight range, away from the major mode of the distribution. The majority of the compounds within this molecular weight range will be significantly less abundant and may show the following characteristics:

❖Decreased Lipid Membrane Permeability
❖Decreased Rate of Bioavailability
❖Increased Requirement for Specialised Delivery Systems

However, in general, higher molecular weight compounds often exhibit higher target specificity and binding affinity, resulting in this molecular weight providing an advantage for selective, therapeutic action.[25]

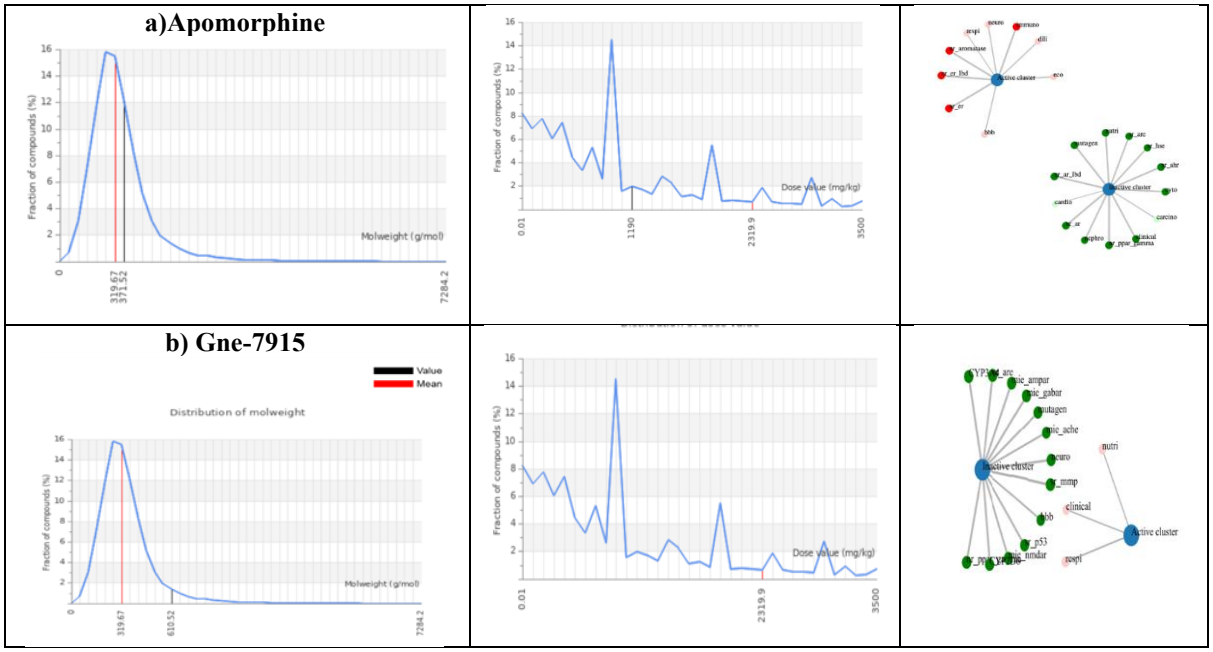

Figure 7. Comprehensive physicochemical and toxicity profiling of the studied compounds. The left panel shows the molecular weight distribution histograms, indicating a narrow distribution with mean values highlighted (red line). The middle panel shows the fraction of compounds vs dose value, and the right panel illustrates toxicity prediction networks, where compounds are clustered based on biological activity, highlighting potential toxicological endpoints (e.g., mutagenicity, carcinogenicity, and irritancy).

IV. Conclusion

The Integrated Computational Approach of Protein Network Analysis and the Analysis of Functional Enrichment, Structural Characterisation, and ADMET Profiling: Identifying Potential Therapeutic Strategies for Neurodegenerative Disorders. The protein-protein interaction (PPI) network and analysis of functional enrichment identified important roles for oxidative stress regulation, mitochondrial dysfunction, and apoptosis in neurodegenerative disease progression. This led to the identification of key target proteins that showed connectivity within these three disease processes (PRKN, PINK1, SNCA, and NFE2L2) through the formation of an interconnected regulatory system. Proteins associated with key targets were analysed structurally and by sequence, and conserved domains and residues were identified across targets. These findings support that these targets represent promising candidates for therapeutic intervention.The ADMET profile of Apomorphine and GNE-7915 revealed that both compounds had no significant differences in their safety profiles (i.e., GNE-7915 showed lower toxicity, while Apomorphine had relatively stable metabolic characteristics); however, neither compound demonstrated significant permeability across the blood-brain barrier nor displayed anticipated neurotoxicity, indicating that both may present similar barriers to application directly within the central nervous system. Overall, this study illustrates the substantial importance of targeting oxidative stress-mediated mitochondrial pathways in neurodegeneration and provides strong evidence to support Apomorphine and GNE-7915 as potential lead compounds to evaluate for worsening neurodegeneration. Going forward, the focus should be on improving bioavailability and suitable means for delivery to the brain (e.g., by means of nanoparticle-based delivery systems) in order to improve overall therapeutic effectiveness.

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