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Next-Generation Sequencing Data Analysis In DYRK1A Associated With Neurodegenerative Disease

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Abstract:
Dual-specificity tyrosine-regulated kinase 1A (DYRK1A) is an important neuronal kinase involved in various neurodevelopmental and neurodegenerative disorders, including Alzheimer's disease. In this study, we used a thorough in-silico approach that combined Next-Generation Sequencing (NGS) variant analysis with structural bioinformatics to investigate mutation-induced changes in the DYRK1A protein (PDB ID: 7O7K). We mapped NGS-derived mutations onto the crystallographic structure. Then, we compared sequences using BLAST and annotated domains with InterProScan. We validated the structure with ERRAT and used PyMOL for visualization and RMSD evaluation to examine the differences between the native and mutant models. Our analysis found several destabilizing mutations in the ATP-binding pocket and catalytic residues, which may affect the kinase's stability and activity. These findings offer insights into how structural changes could lead to DYRK1A dysfunction in neurodegenerative diseases. Overall, this computational approach provides a fast and cost-effective way to link genomic variant data with three-dimensional protein structure analysis. This can help identify mutation-specific drug targets and guide the design of future inhibitors for neurodegenerative treatments.

Keywords: *DYRK1A, Next-Generation Sequencing (NGS), Neurodegenerative Diseases, Structural Bioinformatics, RMSD Analysis, PDB 7O7K, PyMOL Visualization, In-silico Modeling.*

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I. Introduction

Neurodegenerative diseases (NDs), such as Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), are current challenge to global health. These conditions indicate progressive neuronal dysfunction and death, alongside protein misfolding, aggregation, synaptic loss, and systemic molecular disturbances. Despite extensive research over the decades, effective therapies remain scarce, partly because their causes are complex and involve subtle molecular and structural changes. Recently, the combination of high-throughput genomics and computational structural biology has opened new avenues to identify links between genetic variants and protein malfunction. In this framework, the DYRK1A (Dual-specificity tyrosine-regulated kinase 1A) gene and its protein have gained increasing interest as central to neuronal stability and as potential contributors to neurodegeneration [1, 2].

DYRK1A is a kinase with dual specificity, able to autophosphorylate on tyrosine and phosphorylate serine/threonine residues on target proteins. According to databse it plays roles in cellular processes like DNA repair, transcription regulation, cell survival, and developmental signalling which is Located on chromosome 21 within the Down syndrome critical region (DSCR), DYRK1A's dosage sensitivity is of particular interest [1, 3]. Overexpression of DYRK1A is well-documented in Down syndrome (DS), and increased DYRK1A activity is linked to early Alzheimer's disease features in DS, mainly through tau hyperphosphorylation and APP cleavage [4, 5].

Furthermore, DYRK1A is linked to classical Alzheimer's disease independently of Down syndrome, as it can phosphorylate tau at various serine/threonine residues. This activity promotes tau aggregation and the development of neuro fiber tangles which are a part of AD pathology. The connection between DYRK1A and neurodegeneration is further supported by studies showing that small-molecule inhibitors of DYRK1A can improve tau pathology, decrease amyloidogenic processing of APP, and alleviate disease symptoms in a model system [5, 6]. Thus, DYRK1A emerges as a promising therapeutic target for neurodegenerative disorders.

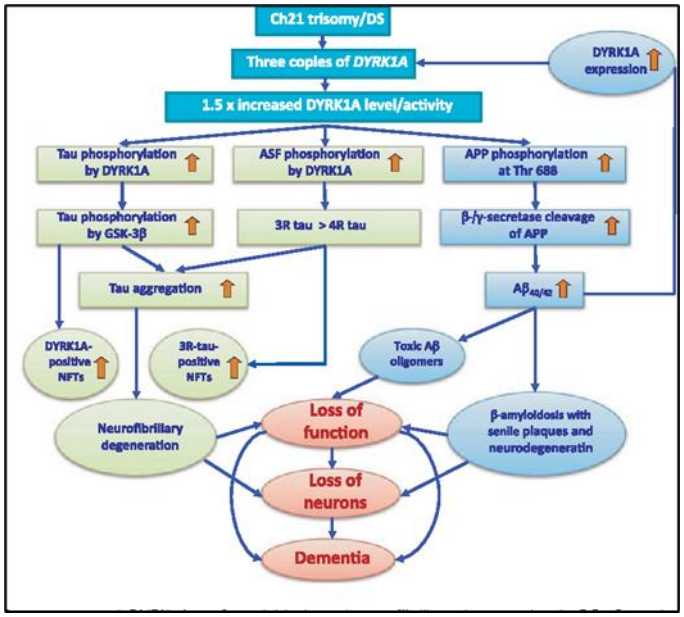

Figure 1: Mechanistic Insights into DYRK1A-Mediated Tau and APP Phosphorylation in Down Syndrome-Associated Dementia

The high-resolution structure of human DYRK1A complexed with abemaciclib, available as PDB ID 7O7K, provides an accurate atomic model for computational analysis [7]. This structure shows the kinase domain bound to a small-molecule which inhibit at the hinge region, showing detailed information on the active site layout, hydrogen-bonding network, and the ATP-binding pocket's structural features. Having access to 7O7K allows for mutation mapping on a reliable structural framework, supporting in silico mutagenesis, structural comparison, and dynamics-based analyses [8, 9].

Neurodegenerative diseases typically involve intricate interactions among common variants, rare new mutations, epigenetic changes, and environmental factors. Sequencing projects such as whole-exome sequencing (WES), whole-genome sequencing (WGS), and RNA-sequencing have identified numerous variants potentially influencing neuronal activity. However variants in kinases or signaling regulators are especially significant due to their direct role in post-translational modifications and signalling pathways [10].

NGS allows for unbiased detection of SNVs, indels, structural variants, and splice mutations [11, 12]. However, simply listing these variants is not enough; understanding their functional impact requires combining this data with computational biology. For well-studied genes like DYRK1A, the main challenge is linking variants to structural changes that could affect substrate binding, enzymatic activity, stability, or regulation [13].

In practice, NGS workflows include data retrieval, quality control, read alignment, variant calling, annotation, and downstream prioritization [14]. However, gaining functional insights from variant annotation is enhanced by incorporating structural analysis: mapping variants onto 3D models, evaluating conformational changes, predicting stability shifts (ΔΔG), and simulating dynamics or flexibility. These integrated pipelines facilitate formulating mechanistic hypotheses without requiring wet-lab experiments.

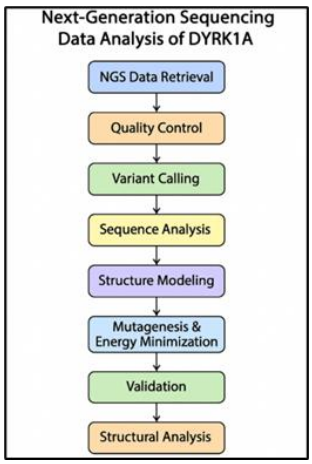

Figure 2: The Importance of Structural Mutational Analysis in DYRK1A

Certain neurodevelopmental and neurodegenerative conditions are associated with pathogenic mutations in DYRK1A, especially de novo dominant variants. Structural studies indicate these mutations might destabilize the kinase's structure, interfere with substrate binding, or affect autophosphorylation. Mutations in the activation segment or in loops responsible for coordinating ATP and Mg2+ ions can decrease catalytic activity or binding affinity. Because kinase active sites have strict structural requirements, even slight distortions can lead to significant functional impairment [15].

Mapping of these variants to PDB 7O7K, we analyze their spatial positions, the hinge, DFG motif, catalytic loop, and glycine-rich loop. Mutations close to or within these areas are likely to impact function significantly. Such effects may include increased local flexibility, changes in hydrogen-bonding patterns, steric clashes, or residue packing shifts. These changes can be detected through RMSD analyses or side-chain reorientation assessments [16, 17].

Significantly, variants that map to surface regions affecting substrate docking or allosteric regulation might change protein–protein interactions instead of the catalytic core. Consequently, structural mapping helps to show not only the direct catalytic effects but also reveals potential sites for allosteric disruption.

In summary, DYRK1A is a key kinase central to neuronal regulation, and its dysregulation is linked to various neurodegenerative pathways. The crystal structure 7O7K provides an accurate framework for structural analysis. Combining NGS variant detection with an extensive set of bioinformatics and structural tools allows us to develop mechanistic hypotheses about how mutations cause DYRK1A dysfunction without the immediate need for wet-lab experiments. This framework helps modern precision biology approaches and accelerates the translation of genotype data into a mechanistic understanding of neurodegeneration [18, 19, 20].

II. Materials And Methods

A complete in-silico pipeline for linking variants to their functions could employ various computational tools.

Step 1: Data Retrieval and Model Validation

We retrieved sequencing data for DYRK1A variants, sourced from the NCBI MMDB database and the Protein Data Bank (sample 7O7K), and downloaded FASTA files for subsequent analysis. Model Validation Tools, such as ERRAT, are used after mutagenesis or structural modelling to ensure stereochemistry, geometry, and overall structural correctness [21, 22].

Step 2: Sequence Analysis Using sequence alignment tools

We analyze sequence similarity using the BLAST sequence alignment tool. With the help of this tool, we evaluate how mutated residues are conserved throughout different species, which is crucial for determining their functional significance. Highly conserved residues are more likely to cause deleterious effects when mutated [23].

Step 3: Identifying the Domain and Motif

We utilize the Protein Signature Analysis tool to identify Domains and Motifs. We use InterProScan to annotate domain boundaries, motifs, and active-site signatures. This approach ensures that variants are understood within the context of functional modules, such as kinase domains or regulatory regions [24].

Step 4: RMSD Analysis.

The RMSD (Root Mean Square Deviation) between the wild-type and mutant models was calculated with PyMOL to evaluate structural differences. This analysis measures the overall or local conformational deviations (such as near the active site) [25].

Step 5: Visualization and Comparative Evaluation

PyMOL was employed to visualise structural changes, emphasising the ATP-binding pocket and catalytic curve. Data we retrive from RMSD, and ERRAT were combined to analyze conformational instability caused by mutations. RasMol was employed to visualize and analyze the three-dimensional structure of DYRK1A (PDB ID: 7O7K), aiding in understanding mutation-induced structural changes from Next Generation Sequencing (NGS) database. Rasmol make possible the mapping of variant residues, the examination of the ATP-binding pocket, and the comparison of wild-type and mutant forms using colour-coded RMSD [25, 26].

III. Result

MMDB Database

>pdb|7O7K|A Chain A, Dual specificity tyrosine-phosphorylation-regulated kinase 1A

SMSSHKKERKVYNDGYDDDNYDYIVKNGEKWMDRYEIDSLIGKGSFGQVVKAYDRVEQEWVAIKIIK
NKK
AFLNQAQIEVRLLELMNKHDTEMKYYIVHLKRHFMFRNHLCLVFEMLSYNLYDLLRNTNFRGVSLNLT
RK
FAQQMCTALLFLATPELSIIHCDLKPENILLCNPKRSAIKIVDFGSSCQLGQRIYQXIQSRFYRSPEVLL
GMPYDLAIDMWSLGCILVEMHTGEPLFSGANEVDQMNKIVEVLGIPPAHILDQAPKARKFFEKLPDGT
WN
LKKTKDGKREYKPPGTRKLHNILGVETGGPGGRRAGESGHTVADYLKFKDLILRMLDYDPKTRIQPYY
AL
QHSFFKKTADE

ERRAT

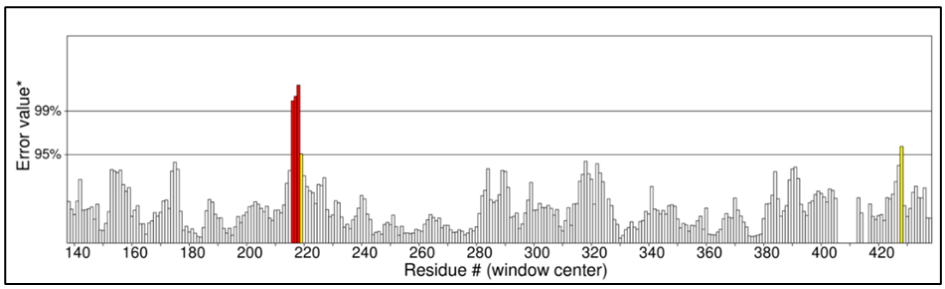

Figure 3: Structural validation scores 96% with the ERRAT server

The ERRAT server, which analyzes the statistics of non-bonded atomic interactions within the structure, was used to evaluate the overall quality of the modeled protein structure. The error values as a function of residue position are shown in the ERRAT plot. An extremely dependable and well-refined structure was indicated by the analysis's 96% overall quality factor. The majority of the residues appear to fall within the acceptable range of structural geometry, as indicated by the small regions with error values above the 95% confidence limit. This high score attests to the protein model's good stereochemical quality and suitability for additional downstream analyses, including dynamic simulations and molecular docking.

Sequence Alignment search

Description	Scientific Name	Max Score	Total Score	Query Cover	E value	Per. Ident	Acc. Len	Accession
dual specificity tyrosine-phosphorylation-regulated kinase 1A [Cyrtonyx montezumae]	Cyrtonyx monte...	766	766	99%	0.0	99.72%	756	XP_065597764.1
dual specificity tyrosine-phosphorylation-regulated kinase 1A isoform X3 [Apteryx rowi]	Apteryx rowi	766	766	99%	0.0	99.72%	725	XP_025933396.1
dual specificity tyrosine-phosphorylation-regulated kinase 1A [Rhea pennata]	Rhea pennata	766	766	99%	0.0	99.72%	756	XP_062454895.1
DYR1A kinase [Chauna torquata]	Chauna torquata	766	766	99%	0.0	99.72%	756	NXK52229.1
dual specificity tyrosine-phosphorylation-regulated kinase 1A isoform X1 [Phyllostomus discolor]	Phyllostomus di...	766	766	99%	0.0	99.72%	763	XP_028361027.1
dual specificity tyrosine-phosphorylation-regulated kinase 1A [Gallus gallus]	Gallus gallus	766	766	99%	0.0	99.72%	756	NP_989881.2
DYR1A kinase [Anseranas semipalmata]	Anseranas semi...	766	766	99%	0.0	99.72%	756	NXI70474.1
dual specificity tyrosine-phosphorylation-regulated kinase 1A isoform X1 [Apteryx rowi]	Apteryx rowi	766	766	99%	0.0	99.72%	756	XP_025933393.1
dual specificity tyrosine-phosphorylation-regulated kinase 1A isoform X1 [Cynocephalus volans]	Cynocephalus v...	765	765	99%	0.0	99.72%	754	XP_062946068.1
dual specificity tyrosine-phosphorylation-regulated kinase 1A isoform X2 [Pteronotus mesoamericanus]	Pteronotus mes...	765	765	99%	0.0	99.72%	754	XP_054434297.1
dual specificity tyrosine-phosphorylation-regulated kinase 1A isoform 2 [Homo sapiens]	Homo sapiens	765	765	99%	0.0	99.72%	754	NP_001334650.1

Figure 4: Sequence similarity and alignment using the BLAST tool

To find homologous sequences across species, the protein sequence was BLASTp-analyzed against the NCBI non-redundant database. With all of the top hits matching dual-specificity tyrosine-phosphorylation-regulated kinase 1A (DYRK1A) from different organisms, the results showed a high degree of sequence conservation. With an E-value of 0.0 and the highest sequence identity recorded at 99.72%, this indicates highly significant similarity. Cyrtonyx montezumae, Apteryx rowi, Rhea pennata, Chauna torquata, Phyllostomus discolor, Gallus gallus, Anseranas semipalmata, Cynocephalus volans, Pteronotus mesoamericanus, and Homo sapiens were found to have closely related homologs. Strong conservation of the DYRK1A kinase domain across mammalian and avian species was confirmed by the query coverage, which was consistently 99%. This high degree of similarity confirms the use of this sequence for additional structural and phylogenetic analyses and implies that the protein's structure and function are evolutionarily conserved.

RMSD and Phylogenetic Analysis
PyMOL>align 7O7K,7O7K
Match: assigning 751 x 420 pairwise scores.

MatchAlign: aligning residues (751 vs 420)...
MatchAlign: score 574.500
ExecutiveAlign: 2133 atoms aligned.
Executive: RMSD = 1.214 (1661 to 1661 atoms)

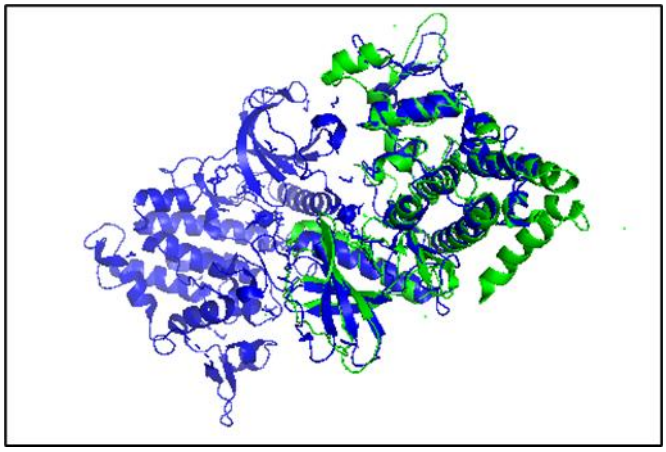

Figure 5: RMSD calculation of human proteomic sample 7O7K (blue) and 7O7J (green)

RMSD analysis of 1.2 Å between two protein structures, means that, on average, corresponding atoms deviate by 1.2 Å after superposition. Analysis suggests high structural similarity, with only minor conformational differences.

PyMOL>align 7o7j,7O7I
Match: read scoring matrix.
Match: assigning 405 x 405 pairwise scores.
MatchAlign: aligning residues (405 vs 405)...
MatchAlign: score 2138.000
ExecutiveAlign: 2967 atoms aligned.
Executive: RMSD = 0.399 (2211 to 2211 atoms)

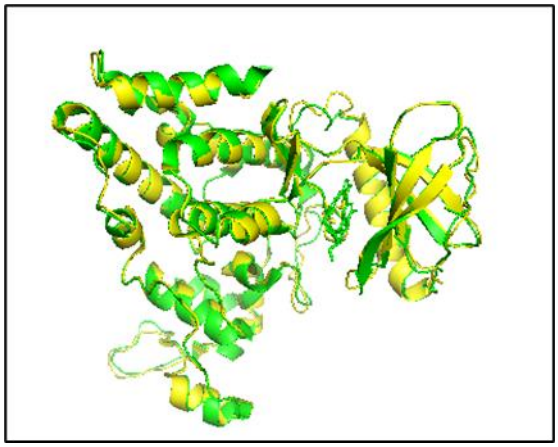

Figure 6: RMSD calculation of human proteomic sample 7O7J (green) and 7O7I (yellow)

Interpretation: In Computational analysis (Root Mean Square Deviation), a score of 0.39 Å typically indicates a perfect structural alignment between two molecular structures. The RMSD measures the average distance between corresponding atoms (usually backbone atoms) of two superimposed structures.

```
pdb|707K|A      -----SMSSHKKERKVYNDGYDDDNYDYIVKN-GEKWMDRYEIDSLIGKGSFGQVVKAYD   54
pdb|707J|A      GNPVTVVTATTGSKQNCT--TGEGDYQLVQHEVLCSMKNTYEVLDFLGRGTFGQVVKCWK   58
                      ::: . .::  .   .:.:*: : ::   .  : **: .::*:*:******.:.

pdb|707K|A      RVEQEWVAIKIIKNKKAFLNQAQIEVRLLELMNKHDTEMKYYIVHLKRHFMFRNHLCLVF   114
pdb|707J|A      RGTNEIVAIKILKNHPSYARQGQIEVSILARLSTENA-DEYNFVRAYECFQHRNHTCLVF   117
                *  :* *****:**: :: .*.**** :*  :...::  :* :*:  . * .*** ****

pdb|707K|A      EMLSYNLYDLLRNTNFRGVSLNLTRKFAQQMCTALLFLATPELSIIHCDLKPENILLCNP   174
pdb|707J|A      EMLEQNLYDFLKQNKFSPLPLKVIRPILQQVATALKKL--KSLGLIHADLKPENIMLVDP   175
                ***. ****:*::.:*  : *:: * : **:.***  *   .*.:**.*******:* :*

pdb|707K|A      KRSA--IKIVDFGSSCQLGQRIY-QXIQSRFYRSPEVLLGMPYDLAIDMWSLGCILVEMH   231
pdb|707J|A      VRQPYRVKVIDFGSASHVSKTVCSTXLQSRYYRAPEIILGLPFCEAIDMWSLGCVIAELF   235
                 *.   :*::****:.::.: :   *:***:**:**::**:*:  *********::.*:.

pdb|707K|A      TGEPLFSGANEVDQMNKIVEVLGIPPAHILDQAPKARKFFEKLP---DGTWNLKKTKDGK   288
pdb|707J|A      LGWPLYPGALEYDQIRYISQTQGLPGEQLLNVGTKSTRFFCKETDMSHSGWRLKTLEEHE   295
                 * **: ** * **:. * :. *:*  ::*: . *: :** *     .. *.**. :: :

pdb|707K|A      REYKPPG--TRK-----LHNILGVETGGPGGRRAGESGHTVADYLKFKDLILRMLDYDPK   341
pdb|707J|A      AETGMKSKEARKYIFNSLDDVAHVNTV--MDLEGSDLLAEKADRREFVSLLKKMLLIDAD   353
                 *    .  :**     *.::  *:*    . ...:     **  :* .*: :**  * .

pdb|707K|A      TRIQPYYALQHSFFKKTADE--------------------------------   361
pdb|707J|A      LRITPAETLNHPFVNMKHLLDFPHSNHVKSCFHIMDICKSHLNSCDTNNHN   404
                ** *  :*:* *.: .
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Figure 7: Clustal omega Result

Strong structural and functional conservation between the two proteins is reflected in the high degree of sequence similarity between pdb|707K|A and pdb|707J|A as determined by the Clustal Omega alignment. The alignment highlights identical and chemically similar amino acids with asterisks and colons for many conserved residues. The regions between residues 60–175 and 250–295 are the most conserved; these are probably the structural core or active site regions of the protein that are necessary for its biological function.

The N-terminal (1–50) and C-terminal (350–400) ends, on the other hand, are primarily home to variable regions, indicating that these segments may be involved in structural flexibility or interactions with other subunits. Overall, the conservation and substitution pattern shows that there is little divergence between the two sequences and that they have a common evolutionary origin.

Protein Domain Analysis

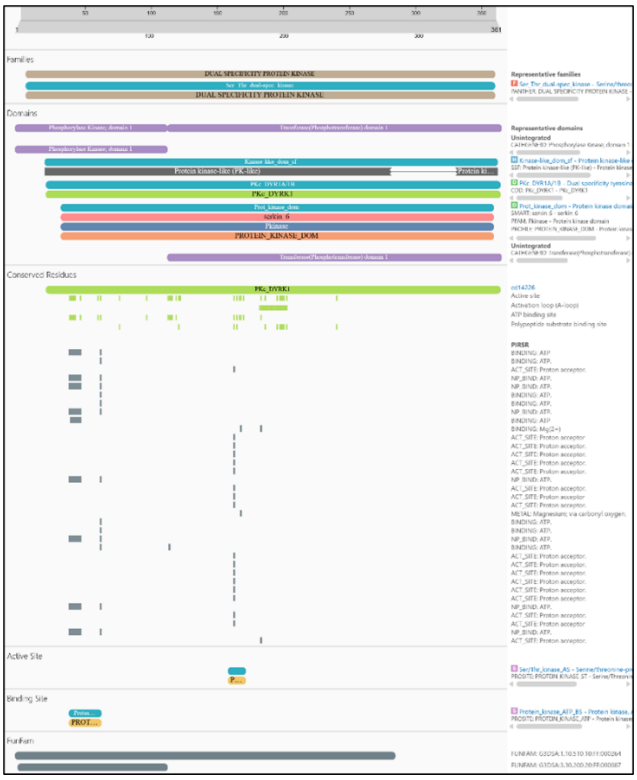

Figure 8: InterProScan results for a protein, summarising predicted domains, conserved residues, binding/active sites, and family classifications

In Protein Domain Analysis interpretation of the protein sequence, its functional domains, conserved residues, and structural features. It identifies critical regions such as active and binding sites, providing insights into the protein's molecular function. Variant positions and their predicted effects are clearly indicated, while conservation tracks illustrate the evolutionary importance of key residues. Structural annotation enriches the understanding that the protein is classified as a Dual Specificity Kinase (Ser/Thr/Tyr), which belongs to the DYRK family. This protein family features distinct kinase domains, conserved catalytic residues, and ATP-binding motifs. These functional annotations, along with predictions regarding conservation and active sites, highlight the protein's critical role in cellular signaling and regulatory pathways. This highlights its significance as a potential target for biomedical research and therapeutic development.

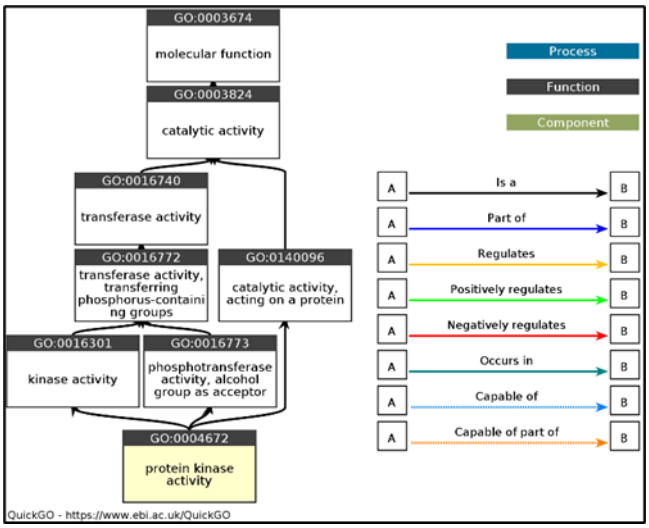

Figure 9: Ancestral analysis chart of sample 7O7K

In Figure 9, the Ancestor chart Analysis of Protein kinase activity, classified under Molecular Function, involves catalysing the phosphorylation of an amino acid residue, typically following the reaction: a protein + ATP = a phosphoprotein + ADP.

Protein Structure Visualization Program

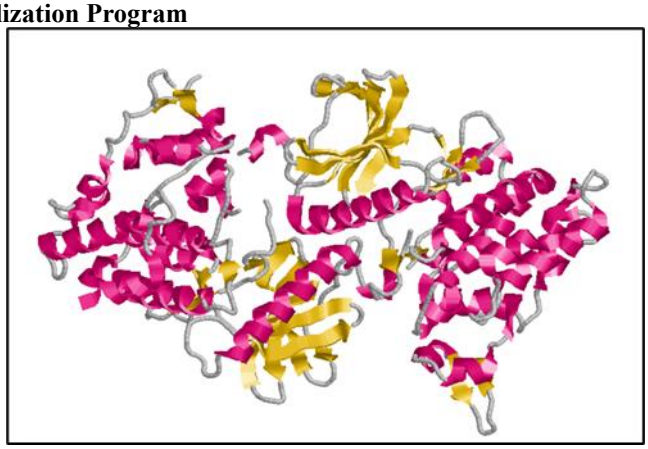

Figure 10: Secondary structure analysis sample 7O7K presented in yellow colour shows beta helix, and magenta colour shows alpha helix.

Yellow arrows are indicating beta sheets and magenta ribbons are representing alpha helices. The ribbon indicates the special framework of these elements, suggesting an understanding of the protein folding, their stability, and important regions involved in molecular interactions.

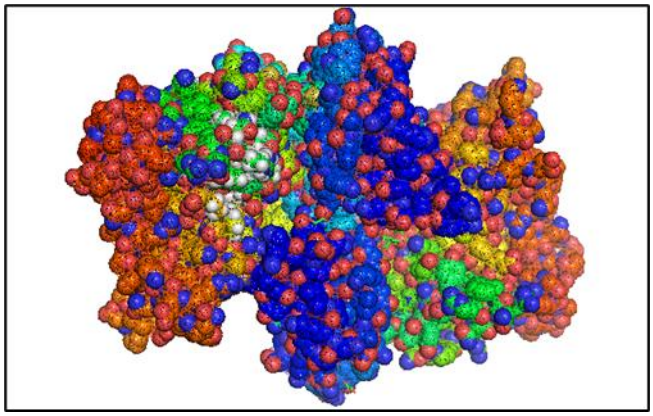

Figure 11: Active site identification analysis with homology model

To illustrate, a prediction of the active site in the homology model of sample 7O7K reveals an evolutionarily conserved catalytic pocket that structurally aligns with functional homologues is shown in Figure 11. The space-filling model highlights the essential residues which form the active site, showing potential ligand binding interactions. These structural insights aid functional annotation and enable further drug-related studies or mutational analysis.

IV. Conclusion

This research introduces a fully integrated in-silico workflow that combines NGS variant analysis with structural bioinformatics. The goal is to explore how mutations in DYRK1A relate to neurodegenerative diseases. We used the crystallographic structure of DYRK1A (PDB ID: 7O7K) as a reference. Multiple computational tools, such as BLAST, InterProScan many other pipeline were used to map, validate, and visualize the structural changes caused by the variants. Structure model score of 96% confirmed the reliability of the modeled structure. RMSD analyses showed small but important conformational changes in the ATP-binding and catalytic regions, suggesting a potential loss of enzymatic stability. Sequence conservation and domain analyses demonstrated that several mutations occur at crucial and evolutionarily conserved sites, indicating their likely pathogenic significance. Overall, these findings stress DYRK1A's central role in neurodegeneration. They also highlight the power of computational genomics in revealing structure-function relationships. The approach presented here offers a replicable and cost-effective way to prioritize disease-associated mutations. It provides a solid foundation for structure-based drug design aimed at creating selective DYRK1A inhibitors to address neurodegenerative diseases.

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