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Research Article

Deep Learning in Genomic Sequencing: Advanced Algorithms for HIV/AIDS Strain Prediction and Drug Resistance Analysis

Asma Khazaal Abdulsahib[1,*], Geehan Sabah Hassan[2], Faris M. Alwan[3], Israa Ibraheem Al_Barazanchi[4]

[1] *College of Education for Human Science Ibn Rushed, University of Baghdad, Baghdad, Iraq.*
[2] *Continuing Education Center, University of Baghdad*
[3] *College of Administration & Economic, University of Baghdad*
[4] *College of Engineering, University of Warith Al-Anbiyaa, Karbala, Iraq*

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ABSTRACT

Genome sequencing has significantly improved the understanding of HIV and AIDS through accurate data on viral transmission, evolution and anti-therapeutic processes. Deep learning algorithms, like the Fined-Tuned Gradient Descent Fused Multi-Kernal Convolutional Neural Network (FGD-MCNN), can predict strain behaviour and evaluate complex patterns. Using genotypic-phenotypic data obtained from the Stanford University HIV Drug Resistance Database, the FGD-MCNN created three files covering various antiretroviral medications for HIV predictions and drug resistance. These files include PIs, NRTIs and NNRTIs. FGD-MCNNs classify genetic sequences as vulnerable or resistant to antiretroviral drugs by analyzing chromosomal information and identifying variants. A patient's HIV strain can be classified as susceptible or resistant to 17 different treatments. The FGD-MCNN transforms DNA genotype and HIV data into mathematical metrics, providing valuable insights into treatment-resistant HIV strains through pooling analysis. With remarkable accuracy, the FGD-MCNN deep learning system predicts HIV medication resistance using behavioral and genome-wide data from the HIV database. DNA patterns can be classified as resistant or susceptible by 17 antiretroviral drugs, providing valuable information for treatment planning and medical judgment. The model's parameter values illustrate the connections between neurons and the complex webs observed in the data have been examined. This study improves treatment effectiveness and expands the knowledge of HIV/AIDS.

1. INTRODUCTION

Millions of individuals around the world are infected with acquired immunodeficiency syndrome (AIDS) and Human Immunodeficiency Virus (HIV). Common ways of transmitting the virus include mother-to-child transmission, infected needles, unprotected sexual contact and contact with tainted blood [1]. It targets CD4 cells inside the immune system. Stages of disease development that are characterized by opportunistic infections or malignancies and a severely weakened immune system are acute infection, clinical delay and AIDS. The goal of antiretroviral therapy (ART) is to stop the virus from reproducing as much as possible so that the immune system can recover, and AIDS cannot develop [2]. Examples of preventative strategies include voluntary HIV testing, sterile needle availability, safe sexual practices, pre-exposure prophylaxis and the elimination of stigma [3]. To combat the epidemic, governments, corporations and civil society groups must work together. There must be a persistent political commitment as well as more financing for infrastructure and medical research. There is additional emphasis on how successful treatment is. It illustrates how lowering the viral load to undetectable levels by antiretroviral treatment (ART) can extend life and lower the rate of transmission [4]. HIV mutations and treatment resistance have been linked; certain mutations result in resistance even in non-receivers of the medicines. HIV is a crucial substance for HIV research and treatment as well as maintenance. Two methods are available for assessing drug resistance: genotype assessments, which determine the risk of HIV infection and phenotypic testing, which measures drug resistance directly by giving antiretroviral agents to patients who can be harmful to themselves and wild-type attachment strains. Based on the genetic composition of the strains, HIV predicts medication resistance using statistical methods [5]. Next-generation sequencing (NGS) has revolutionized DNA sequencing methods, particularly for HIV drug resistance. Its high sensitivity allows for the simultaneous sequencing of millions of DNA pieces, providing vast data. NGS helps detect minority resistant variants (MRVs) at frequencies below 20%, improving patient outcomes and guiding treatment decisions.

*Corresponding author. Email: asma.khazaal@ircoedu.uobaghdad.edu.iq

Appropriate external quality assessment methods are needed to ensure accuracy and consistency [6]. There are two primary forms of HIV, or human immunodeficiency virus: HIV-1 and HIV-2. Most infections worldwide are caused by the most prevalent and ubiquitous variety, HIV-1. Based on genetic variations in the envelope and polymerase genes, it has several subtypes, groupings and recombinant forms. HIV-2 is less widespread, mostly found in West Africa, although it spreads more slowly, and it is not as transmissible. If untreated, both strains have the potential to develop AIDS; however, HIV-2 advances more slowly. Comprehending these classifications and variations is imperative in the advancement of efficacious measures for prevention, diagnosis and therapy, in addition to epidemiological monitoring as well as tracking patterns in HIV infections worldwide [7]. Figure 1 depicts the structure of HIV (Source: https://biosci.mcdb.ucsb.edu/immunology/Immunodeficiencies/HIV-structure.htm).

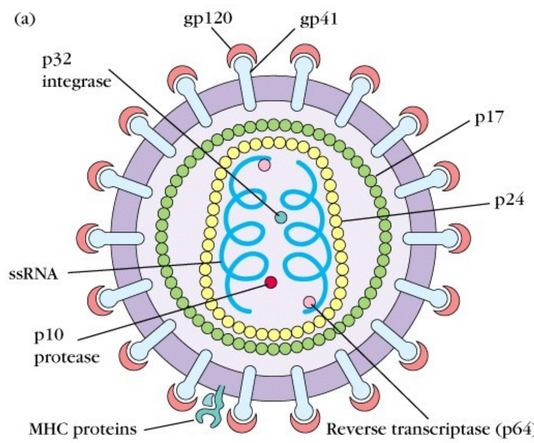


Fig. 1. HIV Structure

HIV is a particular type of virus that spreads to people when they encounter infected body fluids such as blood, semen, vaginal secretions, or breast milk. Despite this, HIV is not a disease that affects everyone, and many people are not affected globally. Antiretroviral medications, which target several stages of the HIV replication lifecycle, are crucial for controlling HIV infection. Entry inhibitors, referred to as fusion inhibitors, stop the HIV from fusing with the CD4 cell membrane of the host. The reverse transcription process, which converts HIV RNA to DNA, is the target of reverse transcriptase inhibitors. They are sometimes referred to "Protease Inhibitors (PIs), Nucleoside Reverse Transcriptase Inhibitors (NRTIs) and Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs)". Integrase inhibitors stop HIV DNA from integrating into the host's genome, which stops the virus from replicating. Protease inhibitors suppress the development of mature and infectious virus particles by acting at the end of the HIV replication cycle. To obtain the highest level of viral suppression and avoid medication resistance, combination treatment entails the use of several categories of drugs [8]. Deep learning is used for medication resistance examination and HIV/AIDS strain prediction using genomic sequencing. The process involves gathering genotype-phenotype data from Stanford HIV DB, extracting features and classifying the data using a fine-tuned gradient descent fused multi-kernal neural network (FGD-MCNN). The next part of this study is section 2: Related works, section 3: Material and methods, section 4: Result and section 5: Conclusion.

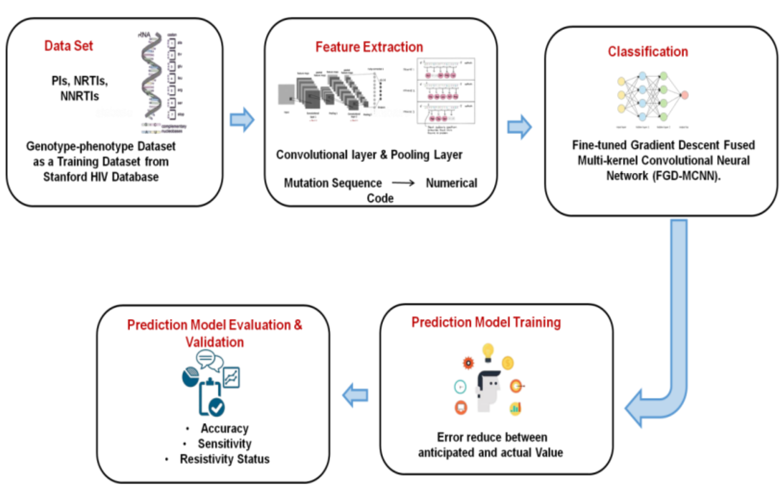


Fig. 2. Workflow Model

2. RELATED WORKS

The paper [9] presented a novel method for predicting HIV strains using generative topographic mapping, achieving an average balanced accuracy of 0.89 ± 0.01, with potential applications in healthcare informatics and sequence space exploration. The paper [10] examined how artificial intelligence (AI) can be used to improve treatment methods and prevent antimicrobial resistance through medication discovery, treatment optimization, and system design coupled with outcome prediction. The study [11] used artificial neural network (ANN) models to estimate HIV-1 protease inhibitors' resistance potential, accurately predicting drug resistance tendencies with sensitivity, specificity, accuracy and Matthews correlation coefficient values. Researchers [12] used machine learning techniques and Random Forest to predict HIV resistance using various descriptor types, finding prediction performance more sensitive to specific medications than the descriptor used. Researchers [13] had developed new antiretroviral drugs to address safety and effectiveness concerns, targeting HIV reverse transcriptase along with an early virus-host interaction. This can be achieved by predicting HIV resistance using clinical and biochemical data, benefiting treatment optimization and drug creation. The study [14] bioinformatics techniques used machine learning and deep learning, aided by biotechnology advancements and high-throughput sequencing, were revolutionizing computational biology research and biomedical informatics by identifying disease patterns, forecasting disease progression as well as enhancing precision medicine research in genomics. The research [15] analyzed that HIV drug resistance mutations could lead to limited treatment options coupled with viral collapse, necessitating accurate measurement of medication resistance frequency for health policy development and patient care. Bioinformatics innovations can aid this study. Using algorithms based on genetics, neural networks, probabilistic research, unpredictable modeling and recognition of patterns to address computational problems along with the application domain expertise, the study [16] investigated machine learning techniques in HIV/AIDS diagnosis, screening, treatment and vaccine development [17-20].

3. MATERIAL METHODS

Deep learning is applied to medication resistance examination and HIV/AIDS strain prediction using genomic sequencing. There are several phases in the process, which include gathering and preparing the genotype-phenotype data from the Stanford HIV DB. This data is subjected to feature extraction by a Convolutional layer and pooling layer processed and feature selection involves mutations and mixtures of mutation codons that are converted into an integer vector. The vector is used for classifying the data using fine-tuned gradient Descent fused multi-kernal convolution neural network (FGD-MCNN) that is employed to identify the error or resistance level between the pre-drug and post-drug stages in the training model, evaluating it, as well as validate it. Figure 2 depicts the research model of this study [21-27].

3.1 Data Collection

The genotypic-phenotypic (Figure 3) dataset comes from the "HIV Drug Resistance Database at Stanford University", (Source: https://i-base.info/guides/changing/resistance-tests) which has been collecting information from global HIV medication resistance programs for more than ten years, as shown in Figure 4. The collection contains the phenotypic values of 17 antiretroviral medications together with the sequencing of HIV strains from different subtypes. More than 20,000 phenotypic findings are accessible for examination. But, before neural networks can be trained using this dataset's raw data, it must be processed.

3.2 Strain Prediction and Drug Resistance Analysis using Fine-Tuned Gradient Descent Fused Multi-Kernal Convolutional Neural Network (FGD-MCNN)

To train neural network models to predict HIV drug resistance, a genotypic-phenotypic dataset is created utilizing the "Stanford University HIV Drug Resistance Database (HIVDB)". PI, NNRTI and NRTI are the three different files that make up the dataset. Predicting drug resistance in HIV therapy is a significant use of statistical learning techniques. By using genomic data analysis, these techniques help to clarify the relationships between treatment results and mutation patterns. Using the Multi-Kernal convolutional neural network (MCNN) layers, one can classify genomic sequences as susceptible or resistant to antiretroviral medications and find notable alterations that affect phenotypic fold resistance levels in the convolutional layer. These strategies support the creation of successful HIV treatment and management plans as well as their implementation. The files include abbreviations for the pertinent amino acids, which function as markers for the mutations discovered in the sequences. These mutations representation is converted into matching code numbers before that is fed into the neural network model for training. The data at codons (1 to 250 for RTIs and 1 to 70 for PIs) has been supplemented with the predictor factors that will be fed into the neural network. To be able to handle mixture mutations, each amino acid involved in the mixture at a given codon is taken as well and each integer vector is added together to create a composite integer vector in the pooling layer. Using a modified genotypic-phenotypic dataset, the creation and development of neural network models for HIV medication resistance prediction, the non-linear correlations between mutations in genomic sequences and associated phenotypic fold resistance levels are captured by fine-tuned gradient Descent fused multi-kernal convolutional neural network (FGD-MCNN). There are several levels in the architecture, including input, concealed and output layers. Each HIV medication's phenotypic factor resistance data is transformed to binary values, where 0 denotes vulnerability to the drug and 1 denotes drug resistance. Figure 5 depicts the classification model for HIV/AIDS strain prediction and drug resistance analysis

(a) Genotype: Genotype tests look to see how the structure of a sample of HIV May have changed

(b) Phenotype: Phenotype tests see whether HIV drugs still work to control type of HIV

Fig. 3. HIV Drug Resistance Test

3.3 Fine-tuned gradient Descent optimization (FGDO)

The neural network's weights indicate the way neurons in its various layers are connected. Weights are adjusted during training by the FGDO method, which considers the error that is determined at the output layer. The paces at which weights are changed during training to reduce the error between anticipated and actual values are determined by the rate of learning parameter. The models have different hidden layer depths, and the number of iterations needed to reach convergence depends on which antiviral medication is being used. Users can add genetic information about an individual's HIV strain and classify it as either vulnerable to or resistant to 17 antiretroviral medications using an approach called the input file. It allows users to choose a specific variation for training models. Pseudo-code 1 will explain the optimization method.

Pseudocode 1: FGD Optimization

$\boldsymbol{Input}(Training\ Set\ t\ \sqsubset Q, Learning\ Rate\ \eta, Rate, Factor\ \lambda, m^{\circ}\ of$
$latent\ factors\ Q)$
$Randomly\ Initialize\ matrices\ O\ and\ R\ ;$
$m = 0$
$\boldsymbol{While}\ not\ (Convergence)\boldsymbol{do}$
$Randomly\ Shuffle\ Observed\ Entries\ in\ T;$
$\boldsymbol{for}\ each\ (v, j) \in T\ \boldsymbol{do}$
$\boldsymbol{f_{v,j} = (q_{vj} - \sum_{l=1}^{L} o_{vl}.r_{lj})}$
$\boldsymbol{for\ each\ r\ \in \{1 \ldots l\} do\acute{o}_{vl} = o_{vl} + \eta\ .(f_{v,j}.r_{lj} - \lambda.o_{vl})}$
$\boldsymbol{for\ each\ r\ \in \{1 \ldots l\} do\acute{r}_{lj} = r_{lj} + \eta\ .(f_{v,j}.o_{vl} - \lambda.o_{lj})}$
$\boldsymbol{for\ each\ r\ \in \{1 \ldots l\} doo_{vl} = \grave{o}_{vl}\ and\ r_{lj} = \grave{r}_{lj}}$
end for
$m = m + 1$
end while
Output *(O,R)*

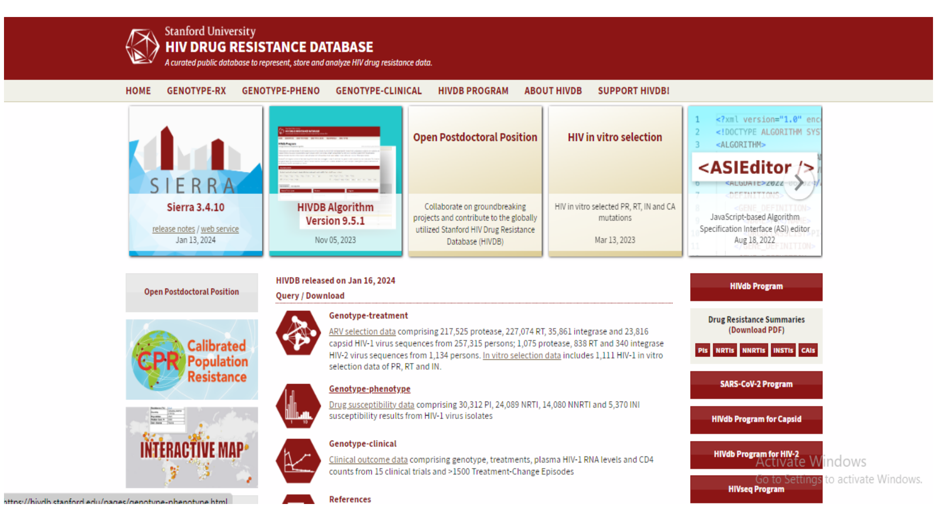


Fig. 4. Stanford Data Base (Source: [HIV Drug Resistance Database (stanford.edu)](https://hivdb.stanford.edu))

A dropdown list with the options P, Q, R, S, T and U and X allows users to choose a variant from inside the input file's variant Information section shown in Figure 6. If a value other than X is chosen, the models are trained using the genotype-phenotype data from the subtype's master dataset. If a value is chosen, the system uses the filtered data to train the neural network model, removing the genotypic sequences of variant S and the phenotypic resistance values that go along with them from the original data set.

4. RESULTS AND DISCUSSION

The HIV Drug Resistance Predictor Tool is a tool intended to forecast an individual HIV strain's susceptibility or resistance to 17 antiretroviral drugs. Utilizing a dataset of genotype-phenotype correlations from sources such as the Stanford HIVDB internal database, it employs 17 neural network models for training. Users can provide input data that contains the subtype and mutation information needed by the utility. Following the analysis of the input data, a Drug Resistance Report is produced that summarizes the predictions of the neural network models for each medication based on the mutations present in the input HIV strain. This report assists doctors in determining the best medication regimen based on the drug susceptibility of the HIV strain. The application is a R script that can take in Excel sheet data, transform it into a neural network training and building format, optimize the networks for training and classify HIV strains according to how sensitive or resistant they are to drugs that treat HIV. A set of measures based on the confusion matrix is used to assess the models' performance shown in Figure 7.

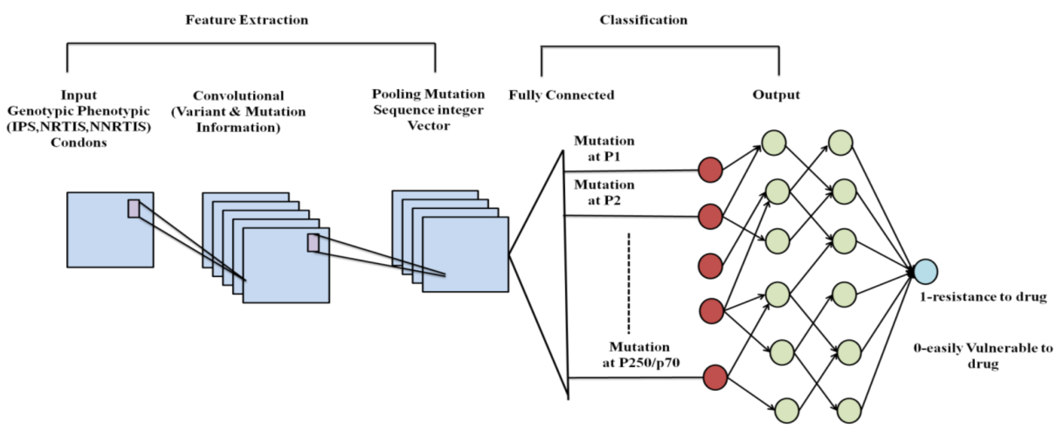


Fig. 5. Classification Model

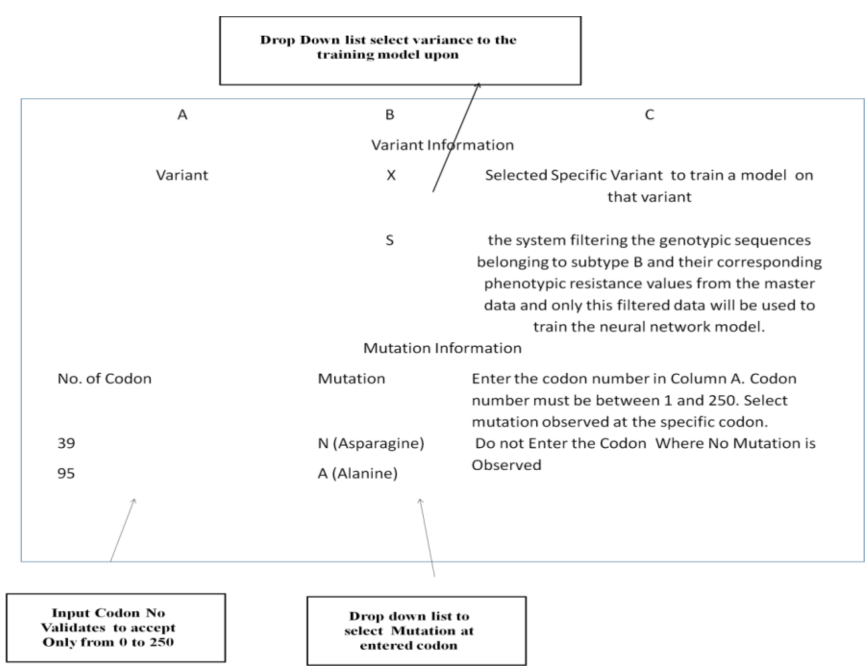


Fig. 6. Input Data File

TABLE I. PREDICTION AND PIS DRUG RESISTANCE OUTCOMES

Drug Name	No. of Training Sequence	Fold Resistance cut-off	Model Accuracy	Model Sensitivity	Prediction	Remark
FPV-Fosamprenavir	19348	6	90.03	90.15	Easily Vulnerable	Model Valid
NFV-Nelfinavir	1947	4	89.48	92.5	Resistant	Model Valid
ATV-Atazanavir	8670	3	88.11	89.45	Easily Vulnerable	
DRV-Darunavir	9834	3	95.34	91.63	Easily Vulnerable	Model Valid
SVQ-Saquinavir	687	10	97.04	58.43	Easily Vulnerable	Model Valid
IDV-Indinavir	1835	4	91.28	90.48	Easily Vulnerable	Model Valid
LPV-Lopinavie	1205	2	94.85	97.36	Easily Vulnerable	Model Valid
TPV-Tipranavir	958	9	89.48	73.42	Easily Vulnerable	Model Valid

TABLE II. PREDICTION AND NRTIS DRUG RESISTANCE OUTCOMES

Drug Name	No. of Training Sequence	Fold Resistance cut-off	Model Accuracy	Model Sensitivity	Prediction	Remark
AZT-Zidovudine	1839	3	84.39	83.37	Easily Vulnerable	Model Valid
D4T-Stavudine	1923	1.5	93.47	84.38	Easily Vulnerable	Model Valid
TDT-Tenofovir Disoproxil Fumarate	1176	1.5	77.78	82.75	Easily Vulnerable	Model Valid
ABC- Abacavir	1937	3	80.93	79.32	Easily Vulnerable	Model Valid
3TC-Lamivudine	2746	3	85.38	77.54	Easily Vulnerable	Model Valid
DDI-Didanosine	1220	1.5	75.05	74.19	Easily Vulnerable	Model Valid

TABLE III. PREDICTION AND NNRTIS DRUG RESISTANCE OUTCOMES

Drug Name	No. of Training Sequence	Fold Resistance cut-off	Model Accuracy	Model Sensitivity	Prediction	Remark
Reverse Transcriptase Inhibitor Drug						
EFV-Efavirenz	1839	3	84.93	87.85	Easily Vulnerable	Model Valid
NVP-Nevirapine	1943	3	87.23	85.39	Easily Vulnerable	Model Valid
ETR-Etravirine	867	3	72.11	85.29	Easily Vulnerable	Model Valid

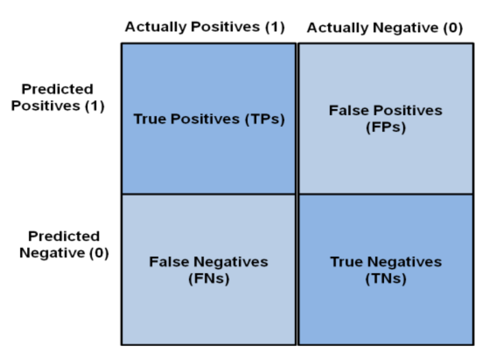


Fig. 7. Confusion Matrix

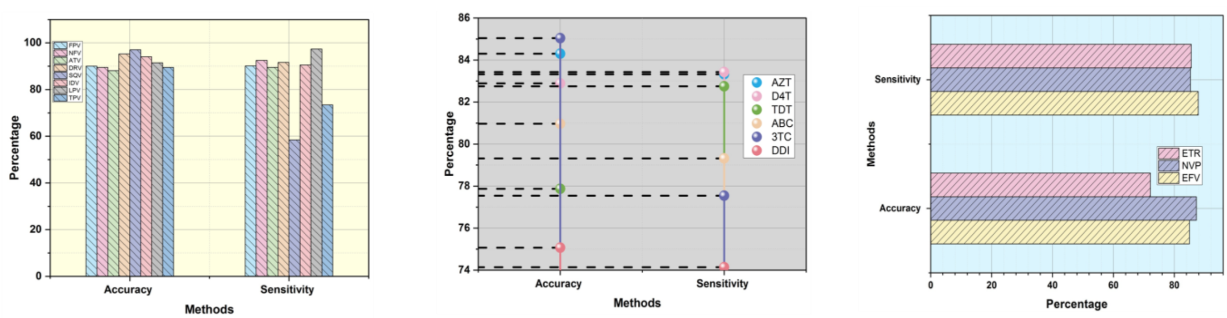
The model's total accuracy in designating easily affected sequences as vulnerable and resistant sequences as resistant are known as accuracy.

$$\text{Accuracy} = \frac{\text{TP+TN}}{\text{TP+TN+FP+FN}} \tag{1}$$

The frequency with which the model predicts resistance to antiretroviral medication is measured by sensitivity.

$$\text{Sensitivity} = \frac{\text{TP}}{\text{TP+FN}} \tag{2}$$

The neural network models built to predict HIV medication resistance to antiretroviral medicines are included in the table 1 for PIs Drug Resistance Outcomes, table 2 for NRTIs Drug Resistance Outcomes and table 3 for NNTRIs Drug Resistance Outcomes along with their predictions and performance metrics. The number of sequence and fold resistance cut-off values are taken from the Stanford DB. The graphic shows the neural network models and performance indicators that were developed to predict HIV drug resistance to antiretroviral drugs in the Figure 8.



(A) PIs Drug Resistance Performance

(B) NRTIs Drug Resistance Performance

(C) NNRTIs Drug Resistance Performance

Fig. 8. Performance Graph for A) PIs Drug Resistance; B) NRTIs Drug Resistance; C) NNRTIs Drug Resistance

5. CONCLUSION

HIV/AIDS strain prediction by genomic sequencing and drug resistance assessment are two areas where deep learning techniques are being used. To create a reliable HIV medication resistance predictor tool, this study shows how to combine feature extraction, classification, optimization and data gathering methodically. Comprehensive genotype-phenotype datasets obtained from the Stanford HIV DRDB can be used to train neural network models for drug resistance prediction to 17 antiretroviral medications. The procedure of feature extraction allows genomic sequences to be classified as resistant or susceptible by converting mutation patterns into numerical numbers. FGD-MCNN is used to create the classification model, which captures non-linear relationships between phenotypic fold resistance levels and mutations in genomic sequences; FGD-MCNN drives the optimization process, which works to reduce the error between the actual and anticipated values. The HIV Drug Resistance Predictor Tool helps doctors to create individualized treatment plans by giving them important information on the drug vulnerability of different HIV strains. The accuracy and sensitivity are higher than the prediction model.

Conflicts of Interest

The author declares no conflicts of interest.

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