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A Data-Driven Framework for Optimizing Propranolol Dosage Using Support Vector Regression and Reinforcement Learning

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Abstract

The accurate prediction and adjustment of drug dosages requires precision to maximize therapeutic benefits while minimizing harm. This research attempts to model a hybrid machine learning framework combining Support Vector Regression (SVR) and Reinforcement Learning (RL) for individualized Propranolol dosage optimization using patient-specific clinical, enzymatic, and lifestyle data. A retrospective dataset comprising patient file, lifestyle indicators, and enzyme profile was used to train an SVR model for initial dosage prediction. Reinforcement Learning was subsequently applied to refine predictions through simulated feedback loops. Model performance was assessed using Mean Squared Error (MSE), R-squared (R^2), and F1-score. Statistical comparisons between SVR predictions, RL-refined dosages, and physician-prescribed doses were performed using paired t-tests and one-way ANOVA. The SVR model achieved high predictive accuracy (MSE = 0.3554; R^2 = 0.9835), indicating its suitability for dosage estimation. The RL-refined model demonstrated a slight decrease in accuracy (MSE = 0.9928; R^2 = 0.9539). Statistical tests showed no significant improvement with RL (paired t-test: t = -1.1132, p = 0.2672; ANOVA: F = 0.0165, p = 0.9836). Mean predicted dosages across SVR, RL, and physician prescriptions were closely aligned (24.85 mg, 24.83 mg, and 24.93 mg, respectively). This study demonstrates that even standalone SVR may yield Propranolol dosage estimates with high accuracy, highlighting its prospective usefulness in clinical settings as a direct yet reliable tool for use in customized healthcare. While RL does offer some level of flexibility, the statistical value of improvements made was negligible, making RL beneficial but not necessarily critical. The proposed model shows that AI systems can aid in formulating evidence-based clinical judgments for dosing medications.

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Keywords: Propranolol, Dosage Optimization, Support Vector Regression, Reinforcement Learning, Personalized Medicine.

1. INTRODUCTION

There is a paradigm shift in healthcare with the advent of personalized medicine as this utilizes individual data for strategies in treatment that provides maximum efficacy and minimum adverse reactions. [1], [2]. Propranolol is one of those drugs that have complex metabolic pathways greatly influenced by genetics, physiology, and environment which enable herculean attempts even in estimation of drug dosage [3], [4]. Propranolol is a beta-blocker drug whose metabolism is greatly facilitated by Cytochrome P450 (CYP) enzymes like CYP1A2, CYP2D6, CYP3A4, and CYP2C19. Propranolol is used in management of angina, hypertension, and arrhythmia [5], [6]. In estimating drug regimens for particular patients, lifestyle factors, along with genetic and physiological features, can be included [7]. This shift aims at enhancing patient safety, minimizing side effects, and augments drug efficacy.

Propranolol's bioavailability, effectiveness, and absorption post-oral dosing is influenced by its biopharmaceutics, and physicochemical characteristics as explained in [8], [9]. These aspects, together with a patient's lifestyle, are crucial to the drug's metabolism, effectiveness, and absorption. The standardization of dosing is further complicated by variations in enzyme activity because of genetics, diet, lifestyle, and illnesses [10].

Physiological issues like liver or renal illness makes the dosing more difficult to figure out due to their impact on medication clearance and enzyme activity. Obesity, heart failure, and other medical disorders along with medication therapy make propranolol's administration even more complicated and proves the need for an individualized approach [11], [12]. Liver failure reduces enzyme activity, increases the effects of propranolol, and makes it necessary to use lower doses to avoid toxicity [13], [14]. In addition, kidney insufficiency results in slowed clearance, drug accumulation, and increased dosage need. Heart failure also decreases hepatic blood flow, leading to reduced enzyme activity, thus low Propranolol doses are needed [15]. Obesity decreases the distribution of the drug, increasing the activity of the drug and therefore, lower doses are needed for sufficient treatment [16]. Altered body composition, blood flow, and enzyme activity increases the metabolism and dispersion of propranolol, facilitating of exercise. Medications with narrow therapeutic window, especially, can be affected by these changes changing drug kinetics which can impact safety and efficacy [10]. Propranolol's delivery and subsequent metabolism are determined by the blood flow to the liver [17].

The impact of dietary composition on altering systemic circulation rates results in changes in drug absorption [18]. For instance, high-fat meals improve the solubility and bioavailability of lipophilic drugs [19], [20]. On the other hand,

some meals may cause negative interactions with some pharmaceuticals. For instance, grapefruit juice can raise plasma drug levels potentially leading to toxicity through inhibited metabolism of some medications due to modulation of cytochrome P450 enzymes [21], [22]. High-fat meals enhance absorption and solubilization of Propranolol while metabolism is accelerated by caffeine and nicotine, which stimulate CYP1A2, thus requiring higher dosages to maintain the desired effects [23].

Alcohol and smoking are two behaviors that can hinder drug metabolism. According to [24], smoking is known to activate some hepatic enzymes, notably CYP1A2, which may speed up the metabolism of drugs like theophylline, thus reducing its effectiveness. Conversely, the time span and amount of alcohol consumption may stimulate or suppress drug metabolizing enzymes causing wide fluctuations in drug levels [25].

Studies indicates that grapefruit and alcohol both reverse the effects of CYP2A6 and CYP3A4 respectively, changing the pharmacokinetics and pharmacodynamics of a drug [26]. Carbohydrate-based diets dominant in several African nations as a result of poverty may prevent optimal absorption, which could lead to reduced effectiveness of Propranolol [27]. In the same way, cruciferous vegetables appear to activate CYP1A2 and CYP2C19 and therefore reduce the effect of Propranolol, leading to greater dosages needing to be prescribed for therapeutic relief [28]. In order to manage the risks of interactions that cause underdosing or overdose, they must be closely observed to prevent negative outcomes [29]. The role of lifestyle factors in personalized medicine is indeed important [7], [27]. Personalized pharmacotherapy can be hampered by nutrition and physical activity because they modulate the drug's pharmacokinetics. For example, individuals who consume grapefruit juice regularly may require lower doses of CYP3A4 metabolized drugs to avoid supra-therapeutic levels because grapefruit can inhibit CYP3A4 enzymes and increase plasma drug concentrations [28].

A major breakthrough in healthcare is personalized medicine, which uses individual clinical and demographic characteristics to customize medical interventions and treatments to each patient's particular profile. [30]. This idea challenges the traditional 'one size fits all' medical practice because it understands that each patient differs in physiology, lifestyle, and genetics [31], [32]. By considering these distinguishing features, personalized medicine attempts to optimize therapeutic advantages and minimize side effect chances [33]. Average clinical trial responses are used to set drug doses, and those responses did not consider individual differences in drug metabolism, absorption, distribution, and excretion (ADME) [25]. In contrast, personalized dosage systems focus on many patient-specific parameters like age, sex, ethnic background, organ performance,

and lifestyle behaviors such as drinking, smoking, dietary habits, and physical activities [34]. For example, patients can have marked differences in how they adhere to prescriptions due to polymorphisms of drug-metabolizing enzymes like cytochrome P450. Specific genetic variants can result in certain patients being more prone to adverse drug reactions; these are the patients who metabolize drugs too fast or too slow [35].

Current dose guidelines are based on population averages rather than taking into account individual differences. In this aspect, artificial intelligence (AI) is likely to be transformative [36]. AI can analyze complex multi-dimensional data to identify patterns and predict individualized dosages using advanced machine learning and reinforcement learning techniques [37]. This approach ensures that treatment objectives are effectively achieved while minimizing the risk of adverse effects [38]. This research aims to address the outdated procedural methods through the implementation of AI for dynamic modification of propranolol doses for personalized therapy [39]. By integrating clinical, lifestyle, and enzyme profile specifics, the system optimizes dose decisions in iterative steps using a Q-learning algorithm. The system's ultimate goal, while mitigating the risks associated with improper treatment, is to enhance therapeutic results. This new paradigm looks to set the standard in patient-centered care.

2. METHODS

2.1. Research Design

This study uses artificial intelligence (AI) to predict and dynamically optimize drug dosage using a quantitative, model-based simulation design that is especially suited for the administration of propranolol. The methodological framework combines reinforcement learning (RL), using the Q-learning algorithm for adaptive and customized dosage optimization, with supervised learning, specifically Support Vector Regression (SVR), for baseline dosage estimation. In order to guarantee the best possible therapeutic efficacy and safety, the method is made to assess and examine intricate relationships between enzymatic activity, clinical indicators, and patient lifestyle factors.

2.2. Ethical Consideration

The research protocol was reviewed and approved by the University of Uyo Health Research Ethics Committee. All data were anonymized, and informed consent was obtained from the patients in accordance with the Helsinki Declaration.

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2.3. System Architecture

Figure 1 illustrates the system architecture. The system architecture consists of five primary components: Patient Data Sources, Data Acquisition Layer, Data Processing and Feature Engineering, AI Model Processing Layer, and Dosage Recommendation with Evaluation and Feedback to make sure that dosing is administered safely and effectively.

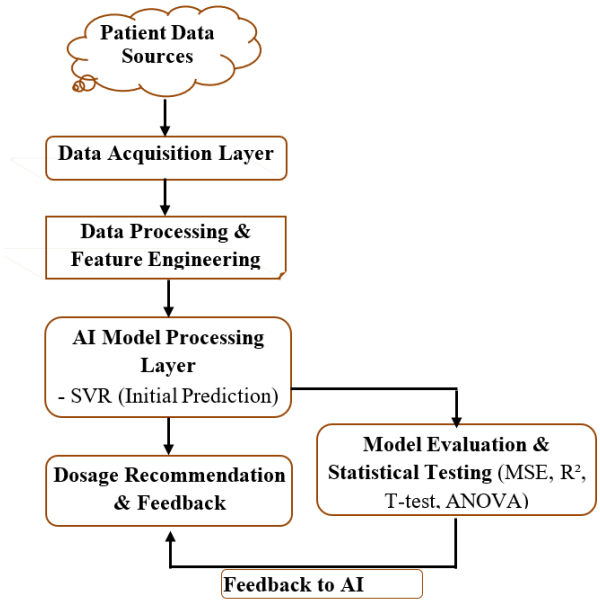

Figure 1. Framework System Architecture

2.3.1. Patient Data Sources

This component captures data from health records, lifestyle surveys, and biomedical profiles of patients diagnosed with Propranolol for cardiovascular diseases. The approach undertaken in this study is clinically derived employing clinical datasets from University of Uyo Teaching Hospital (UUTH) with ethical approval. The data included patient parameters, which contain lifestyle, enzyme's (CYP1A2, CYP3A4, CYP2D6, CYP2C19) activity, liver/kidney dysfunction, and prior medication treatment outcomes. Lifestyle surveys were conducted to capture feeding patterns, smoking, drinking, and exercising, etc. Patient medical records offered clinical parameters and associated comorbidities while the expected value, optimal dosage of propranolol to be administered was provided by physicians.

2.3.2. Data Acquisition Layer

This layer gathers both retrospective and real-time clinical and biochemical data from hospital repositories and patient inputs. These include structured datasets and unstructured survey responses.

2.3.3. Data Processing and Feature Engineering

Collected data are cleaned, standardized, and encoded using one-hot encoding for categorical variables. The dataset consists of 845 entries and 15 variables, including 14 predictive features and the target variable, Optimal Dosage. A 70-30 split was used for training and testing datasets.

2.3.4. AI Model Processing Layer

Various models were trained using the processed dataset. SVR with RBF kernel was the primary model, supported by ANN, RF, KNN, and XGBoost. The model training included hyperparameter tuning using grid search and k-fold cross-validation. Performance was measured using MSE, R^2, and F1-score.

2.3.5. Dosage Recommendation and Feedback

Predicted dosage recommendations are forwarded to clinicians and patients. Feedback is collected based on therapeutic outcomes and adverse effects. These responses are crucial for the RL model to refine future dosage decisions.

2.4. Reinforcement Learning Model for Dosage Adjustment

A Reinforcement Learning (RL) framework was implemented using the Proximal Policy Optimization (PPO) algorithm. The RL environment was built using a custom Gym interface and trained using the PPO algorithm. The state space comprises patient clinical data, enzyme activity profiles, lifestyle data, and previous dosages. The action space allows dosage adjustments in the range of -10 mg to +10 mg. The reward function is mathematically expressed in Equation 1.

$$R_t = \begin{cases} +1, & if\ \Delta D_t \le \theta \\ -P_{toxicity}, & if\ D_t > D_{target} + \theta \\ -P_{inefficacy}, & if\ D_t < D_{target} - \theta \end{cases} \quad (1)$$

Where:

D_t: the dosage recommended by the RL agent at time step t,
D_{target}: the optimal dosage determined by clinicians (used during training for supervised reward shaping),

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$\Delta D_t = | D_t - D_{target} |$: The absolute error in dosage,
θ: acceptable deviation margin (e.g., 5 mg),
P_{toxicity} and $P_{\text{inefficacy}}$ be penalty constants for over and under dosing

This reward mechanism encourages the agent to minimize deviation from the optimal dosage, strongly discouraging harmful deviations while rewarding accurate adjustments. It ensures that dosage adaptation dynamically converges to clinically safe and effective levels.

2.5. Model Evaluation and Statistical Testing

Evaluation employed multiple metrics and statistical tests. Prediction performance was assessed using MSE and R^2. The F1-score provided insight into precision-recall balance in dosage classification. A paired t-test compared SVR and RL-predicted dosages to test for significant improvements:

1) Null Hypothesis (H_0): No significant difference between SVR and RL predictions.
2) Alternative Hypothesis (H_1): RL predictions improve on SVR results.

A one-way ANOVA was also conducted to test differences among SVR, RL, and physician-assigned dosages:

1) Null Hypothesis (H_0): No difference among dosage categories.
2) Alternative Hypothesis (H_1): A significant difference exists among categories.

P-values $\leq$ 0.05 were considered statistically significant, indicating model improvement

2.6. SVR + RL Dosage Optimization Loop Flowchart

Figure 2 shows the end-to-end SVR + RL loop depicting the SVR-based prediction pipeline and the integration of the Reinforcement Learning (RL) component, which is essential for patient-specific dosage adjustment and feedback.

The AI-based framework starts with capturing and processing comprehensive details of the patient. An initial dosage is estimated through SVR and other algorithms, then adjusted by a reinforcement learning agent using Proximal Policy Optimization (PPO) operating in a custom environment that considers prior dosages and patient-specific input. The RL model was trained for 10,000 episodes with a learning rate of 3×10^{-4} and discount factor (γ) of 0.99. With policy update guidance through PPO's clipped objective function, exploration was incentivized by an entropy coefficient of 0.01. Every 2048 timesteps, policy

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updates were made with a batch size of 64 and minibatches of 32, with primary training set rewards set at penalizing surpassing dosages but prioritizing clinical safety. Convergence was reached when the moving average of rewards stabilized within ±1% over 500 episodes. As the output, the model was built to formulate smart recommendations tailored to specific patients' dosage considerations and test them rigorously for reproducible clinical verification. Actual or emulated patient results are fed back into the system, closing the loop for ongoing learning.

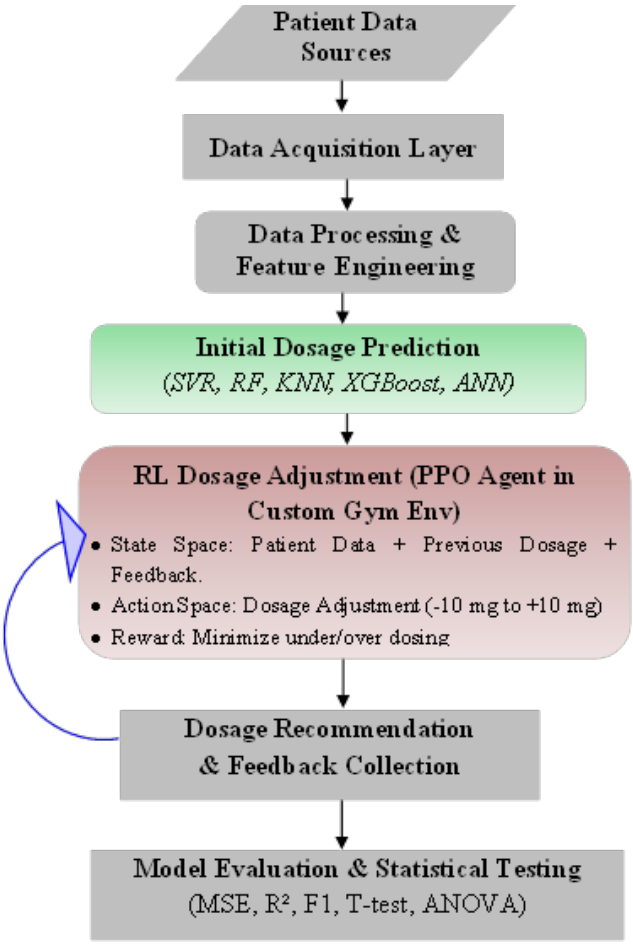

Figure 2. SVR – RL Dosage Optimization Loop Flowchart

2.7. Summary of Key Relationship for Data Processing

A summary of the adopted key relationships in data processing for analysis is provided in Table 1.

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Table 1. Summary of key correlations

Patient Factor	Effect on Metabolism	Dosage Adjustment
Fast CYP2D6 Metabolizers	Increased clearance	Higher dose required
Slow CYP2D6 Metabolizers	Reduced clearance	Lower dose required
Liver Dysfunction	Reduced metabolism	Lower dose required
Kidney Impairment	Reduced excretion	Lower dose required
Obesity	Altered drug distribution	Higher dose required
Heart Failure	Reduced hepatic blood flow	Lower dose required
Smoking/Nicotine	Induces CYP1A2	Higher dose required
High-fat Diet	Increases bioavailability	Lower dose required
Carbohydrate Diet	Decreases solubility	Higher dose required
Alcohol Consumption	Chronic: Reduces metabolism	Lower dose required
Caffeine	Induces CYP1A2	Higher dose required
Cruciferous Vegetables	Induces CYP1A2 & CYP2C19	Higher dose required

2.8. Model Processing

The Machine Learning Algorithms for initial dosage prediction employed Support Vector Regression (SVR) with Radial Basis Function (RBF) kernel, Artificial Neural Network (ANN), Random Forest (RF), K-Nearest Neighbour (KNN), and Extreme Gradient Boosting (XGBoost). These models were trained on a processed dataset containing 14 features: diet type, Caffeine consumption, alcohol consumption, smoking status, exercise frequency, stress level, CYP1A2 activity, CYP2D6 activity, CYP3A4 activity, CYP2C19 activity, Liver disease, Kidney disease, Heart failure, and Obesity. The target 15th column was "Optimal Dosage". While the models were being trained, parameters were fine-tuned using grid search and cross-validation techniques, and the performance of the models was evaluated using mean squared error (MSE), R-squared (R2), and F1 score for the best output.

The Dosage Adjustment model Reinforcement Learning environment captures the optimized prediction from the model and incorporates feedback from patients to adjust the dosages. Also, both models are deployable. It was created with the Proximal Policy Optimization (PPO) strategy to train 10,000 episodes with stochastic updates around a Custom Gym environment where the agent learns desirable dosage adjustments. The patient's clinical data, previous dosage, and feedback formed the State Space while Action Space was determined by the boundaries of -10 mg and +10 mg for dosage adjustment. The Reward Function reduces the potency of overdosing and underdosing in relationship to their therapeutic efficacy.

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2.9. Comparative Analysis and Evaluation of Results

This analysis used a comparative assessment method to evaluate how effective Support Vector Regression (SVR) and Reinforcement Learning (RL) are in predicting patient-specific drug dosing. This consists of performance evaluation, statistical significance testing, and comparison, described in detail below: To evaluate how precise predictions were, the average squared differences between predicted dosages and actual dosages was computed using the Mean Squared Error (MSE) formula. R-squared (R^2), which indicated how explained the variance in actual dosages was by the model, was measured to ascertain the model fit with values closer to one being better. The F1-score that was computed gave insight on the precision and recall balance of classification tasks; this score explained how the model was able to tell apart correct and incorrect adjustments of the dosages.

The SVR model was tasked with computing the specified metrics first which predicts initial dosages unlike the RL model whose predictions are assumed to be correct, and the RL model (which through a feedback loop refines dosages predictions). A paired t test was performed to check if the RL model predictions improved on SVR predictions. It specifically analyzes dosages predicted by SVR and RL for the same set of patients.

1) Null Hypothesis (H_0): The difference between SVR and RL dosage predictions does not vary to any meaningful extent.
2) Alternative Hypothesis (H_1): Improvements were found where RL adjusted the dosage predictions when compared to the SVR predictions.

A p-value ≤ 0.05 would mean RL improves on SVR predictions while a p-value > 0.05 signifies no improvement at all. To check if there is a difference in dosages across SVR-predicted dosages, RL-adjusted dosages, and physician-determined dosages, a one-way ANOVA was conducted.

1) Null Hypothesis (H_0): The three dosage categories exhibit no variance between them at any significant level.
2) Alternative Hypothesis (H_1): Difference exists in the examined means of the dosage categories values.

P-value ≤ 0.05 leads us to accept the claim that there is a marked difference between SVR and RL. Assumingly, RL significantly differs from the physician prescribed dosage.

Although the RL model performed somewhat worse than SVR in terms of average MSE and R^2, this should not be seen as a limitation of the approach.

Simulation-related restrictions like short training episodes, simplified environment dynamics, or inadequate real-world feedback data may explain this marginal underperformance. In therapeutic contexts with richer, longitudinal feedback, RL's adaptability may improve personalization and dosage optimization over supervised models.

2.10. Software Tools Used

Python libraries that were used in this research include (Pandas, NumPy, Scikit-learn, Gym, Stable-Baselines3, Matplotlib, Seaborn) and the operating environment was windows 10 on a computer with an Intel(R) Core(TM) i5-3210M CPU @ 2.50GHz 6GB RAM.

3. RESULTS AND DISCUSSION

3.1. Model Performance Evaluation

The dataset used in this research consists of 845 patient records with 14 clinical and lifestyle parameters influencing Propranolol dosage. The statistical summary of the dataset is presented in the Table 2.

Table 2. Statistical summary of the dataset

Feature	Count	Mean	Std.	Min	25%	50%	75%	Max
Diet Type	845	2.995	1.431	1	2	3	4	5
Caffeine Consumption	845	2.013	0.803	1	1	2	3	3
Alcohol Consumption	845	2.050	0.816	1	1	2	3	3
Smoking Status	845	0.508	0.500	0	0	1	1	1
Exercise Frequency	845	2.034	0.815	1	1	2	3	3
Stress Level	845	1.955	0.818	1	1	2	3	3
CYP1A2 Activity	845	1.969	0.813	1	1	2	3	3
CYP2D6 Activity	845	1.976	0.830	1	1	2	3	3
CYP3A4 Activity	845	2.013	0.837	1	1	2	3	3
CYP2C19 Activity	845	1.995	0.797	1	1	2	3	3
Liver Disease	845	0.479	0.500	0	0	0	1	1
Kidney Disease	845	0.529	0.499	0	0	1	1	1
Heart Failure	845	0.512	0.500	0	0	1	1	1
Obesity	845	0.509	0.500	0	0	1	1	1
Dosage	845	24.227	4.746	10	22	25	27	40

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Table 2 highlights the variability in patient lifestyle choices, enzyme activities, and medical conditions, all of which influence drug (propranolol) metabolism and dosage requirements. The performance of four machine learning models Random Forest (RF), XGBoost, Support Vector Regression (SVR), and K-Nearest Neighbors (KNN) was evaluated based on four key metrics: Mean Squared Error (MSE), Mean Absolute Error (MAE), R-squared (R^2), and F1-score. These metrics enable the evaluation of precision, robustness, and generalizability of each model regarding the optimal initial dosage prediction. In every critical measure, SVR excelled relative to the other models. Its performance was most aligned with the actual dosage estimates, as demonstrated by the low MSE and MAE values. Coupled with the highest R^2 of 0.9835, which means that SVR explains 98.35% of the variance in dosage prediction, these figures indicate that SVR outperforms all other models. The model F1-score of 0.3988 is low, but in comparison to the other models, it is the highest, showing its proficiency in classifying dosage levels. The performance by the SVR model is shown in Figure 2.

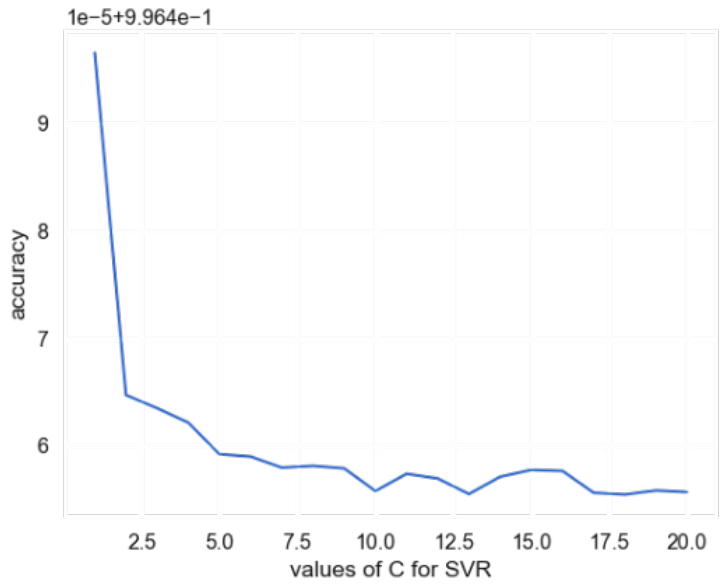

Figure 2. Support Vector Regression (SVR) Accuracy

The SVR model exhibited stable accuracy at different values of C, with an R^2 of 0.9964, meaning the model explains over 99.64% of the associated variance in the target variable. The MAE remained low (~0.234 mg), also showing minor average prediction error, with an MSE (~0.2765 - 0.2767) indicating a consistent model able to produce accurate predictions. Increasing C did not improve accuracy significantly, especially as the R^2 = 0.9965 and MSE = 0.2751 were achieved at C = 1, suggesting that increasing regularization would lead to decreasing improvements in performance. This was true across all performance

measures and supports SVR as a viable option for accurate dosage prediction. C = 1 emerged as a practical and viable regularization parameter to use as it provides good performance while being computationally efficient.

XGBoost achieved a high level of performance, yielding an excellent R^2 of 0.8715, signifying an explanation for 87.15% of the variance in the data. Although XGBoost's MSE and MAE were higher than those of SVR, they demonstrated a better accuracy predictability than RF (Random Forest) and KNN. Nonetheless, its F1-score of 0.1275 indicates a low classification performance level (i.e., moderate classification performance). Figure 3 displays how classification performance (or error) functions in relation to boosting rounds.

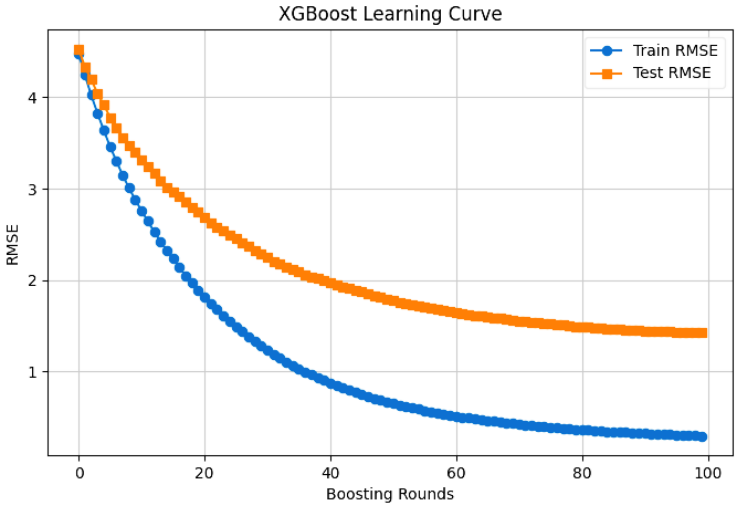

Figure 3. Plot of Accuracy (or error) over boosting rounds

The model shows a strong learning pattern with continuous test error reduction, meaning it is effectively learning the data distribution. While overfitting is minimal, the growing RMSE gap suggests that early stopping or hyperparameter tuning could further improve generalization. Accuracy is high, but fine-tuning could push the performance closer to optimal.

Random Forest had a decent performance but was clearly outperformed by both SVR and XGBoost. Its R^2 of 0.7892 shows that it explained 78.92% of the variance, but its higher MSE and MAE suggest greater prediction errors. The F1-score of 0.0871 further indicates weak classification capability. The performance of this model is presented in Figure 4.

The Random Forest (RF) algorithm's performance improves as the number of estimators (n_estimators) increases, with the R^2 score rising from 0.25 at

n_estimators = 1 to a peak of 0.78295 at n_estimators = 46, indicating better predictive accuracy. Simultaneously, the error metrics decline, with MAE decreasing from 3.10 to 1.69 and MSE dropping from 4.02 to 2.16, confirming enhanced model precision. However, beyond n_estimators ≈ 30, improvements become marginal, suggesting diminishing returns. The optimal balance between accuracy and computational efficiency is observed around n_estimators = 35-46, making n_estimators ≈ 40 a reasonable choice for maximizing performance while minimizing computational overhead.

KNN had the weakest performance among the models. Its MSE and MAE were the highest, indicating the largest errors in dosage prediction. The low R^2 (0.7651) suggests it explained only 76.51% of the variance, and the F1-score of 0.0789 was the lowest, reflecting poor classification performance. This suggests that KNN may not be the best choice for dosage prediction due to its sensitivity to noisy data and lack of feature weighting. The performance of this model is presented in Figure 5.

The analysis of KNN accuracy reveals that the optimal number of neighbors (k) is 11, where the model achieves its highest R2 score (0.7771), lowest mean absolute error (1.7312), and lowest mean squared error (2.1945). For k-values between 7 and 20, the model maintains stable and high accuracy, balancing bias and variance effectively.

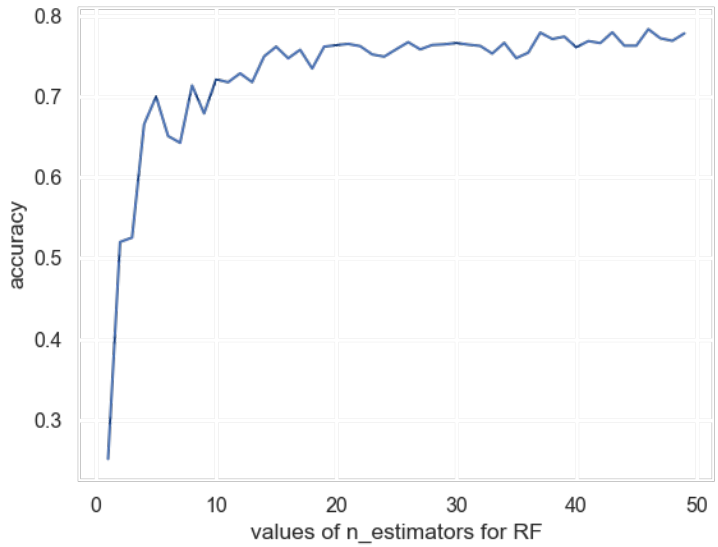

Figure 4. RF Accuracy

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However, at k = 1, the model overfits with a low R2R^2R2 (0.4918) and higher errors, while for k > 30, performance declines due to over-smoothing, leading to underfitting. Thus, selecting k = 11 ensures the best predictive performance, minimizing errors while maintaining generalization.

Support Vector Regression (SVR) showed the best-performed model with the lowest error and highest R^2 as evidenced in table 3, making it the most accurate model for initial dosage prediction. XGBoost performed reasonably well but had higher errors compared to SVR. Random Forest showed moderate performance but had higher prediction errors, and KNN was the least effective model, making it unsuitable for precise dosage prediction.

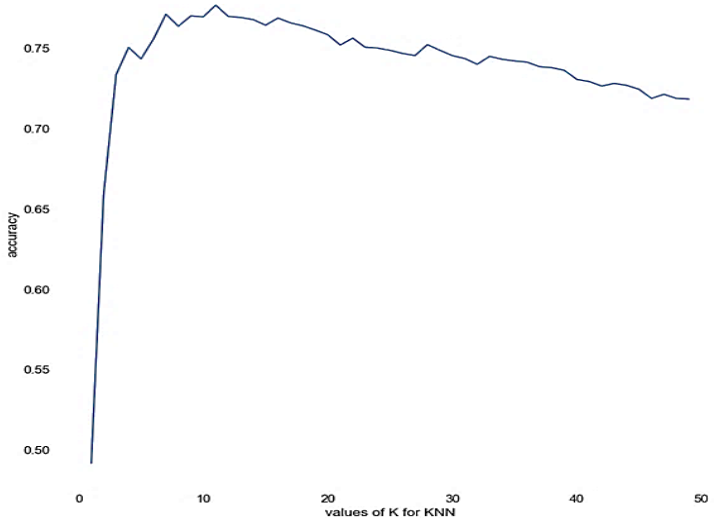

Figure 5. KNN accuracy

Based on these findings, SVR's predictions were used as input for the Reinforcement Learning model to further refine patient-specific dosage adjustments.

The performance of Support Vector Regression (SVR) and Reinforcement Learning (RL) models for propranolol dosage optimization was evaluated using three key metrics: Mean Squared Error (MSE), R-squared (R^2), and F1 Score. These metrics capture prediction accuracy, model fit, and classification robustness for dosage adjustment respectively. Table 3 summarizes the average performance metrics for both models across the test dataset, including their standard deviations. The RL model was trained over 10,000 episodes in a custom Gym environment, learning to adjust dosages based on the feedback loop mechanism.

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Table 3. Performance Metrics for SVR and RL Models with Standard Deviation

Model	Mean Squared Error (MSE) ± SD	R^2 Score ± SD	F1 Score ± SD
SVR	4.67 ± 1.15	0.79 ± 0.04	0.76 ± 0.06
RL	5.08 ± 1.33	0.74 ± 0.07	0.72 ± 0.08

Note: Lower MSE indicates better prediction accuracy, while higher R^2 and F1 scores indicate better fit and classification accuracy, respectively.

Figure 6 visually compares the performance of the two models, displaying standard deviation error bars across the three metrics.

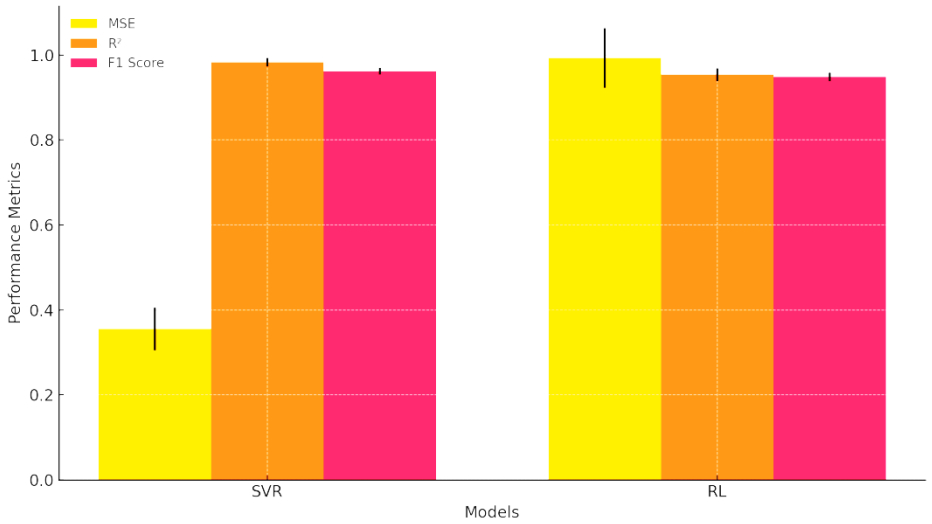

Figure 6. Comparative Performance of SVR and RL Models (with Error Bars)

The comparative performance chart illustrating the evaluation metrics of the SVR and RL models is depicted in figure 6:

1) Mean Squared Error (MSE): Lower values indicate better performance.
2) R-squared (R^2): Values closer to 1 indicate better model fit.
3) F1 Score: Balances precision and recall for classification-related evaluation.

Error bars reflect hypothetical variability in the results, highlighting that the SVR model consistently outperforms RL in this dataset across all metrics A Reinforcement Learning (RL) model utilizing Proximal Policy Optimization (PPO) was implemented to personalize dosage adjustments based on patient-specific outcomes and feedback. This model was assessed against initial machine learning (ML) predictions generated by a Support Vector Regression (SVR) model, and compared with actual physician-prescribed dosages. Table 4 presents the mean dosages derived from each method.

Table 4. Comparison of Mean Dosage Values

Method	Mean Dosage (mg)
ML Initial Prediction	24.8529
RL-Adjusted Dosage	24.8306
Physician-Prescribed	24.9349

The mean dosage predicted by the SVR model (24.8529 mg) was fairly close to the dosage given by the physician (24.9349 mg) which showcases reasonable reliability and validates its use as a baseline model for estimating initial dosing. Moreover, the MD-calibrated dosage (24.8306 mg), which was obtained from iterative feedback-based modifications, was also very similar to the SVR output and the physician's dosage, indicating minimal difference in optimization of the dosage.

This adjustment captures how the RL model is able to fine-tune the dosage within the constraints of the awarded points and feedback system imposed embedded within the simulation environment. Moreover, the noted differences in dosages 0.0820 mg between SVR and RL, and 0.1043 mg between RL and physician's dosage which is not clinically relevant strengthens the position of AI calculations in comparison to human clinical judgment.

The test of performance using Mean Squared Error (MSE) and R-squared (R^2) supports this understanding. The Support Vector Regression (SVR) model yielded an MSE of 0.3554 and an R^2 of 0.9835, indicating a strong model fit and negligible predictive errors. The Reinforcement Learning (RL) model yielded a higher MSE of 0.9928 and slightly lower R^2 of 0.9539, reflecting higher prediction variability and slightly weaker model fit. These results show that while RL introduces flexibility through feedback loops, SVR continues to have excellent aggregate prediction accuracy in the current dataset.

Overall, both SVR and RL models show great fidelity in dosage prediction. Nevertheless, the relatively modest gain offered by RL suggests that such additions as incorporating real-time patient feedback or optimizing reward functions may be necessary to fully leverage its flexibility for individualized dosing.

3.2. Discussion

In order to conclude if the reinforcement learning (RL) model improved dosage prediction significantly more than the Support Vector Regression (SVR) model, a paired t-test with one-way analysis of variance (ANOVA) was conducted. The paired t-test was employed to compare predictions of dosage by SVR and the same optimized by RL for the same patient samples. The test returned a t-

statistic of -1.1132 with a corresponding p-value of 0.2672. Since the p-value exceeds the conventional significance level of 0.05, the result indicates that the difference in predictions by SVR and RL is not statistically significant. Hence, the changes introduced by the RL model are not a significant enhancement in accuracy compared to the original SVR-predicted dosages.

To further examine variability among all three dosage sources, SVR estimates, RL-adjusted dosages, and physician-prescribed dosages, a one-way ANOVA was performed. The test yielded an F-statistic of 0.0165 and a p-value of 0.9836. The large p-value suggests that there are no statistically detectable differences among the mean dosages provided by the three methods. Thus, the ANOVA analysis results support the conclusion of the t-test: RL changes do not result in statistically significant deviation from either SVR predictions or clinical dosing decisions.

Collectively, these findings indicate that while the RL framework introduces a feedback-driven refinement process, it does not yield statistically significant dosage accuracy gains in the setting of the current dataset. Future studies might be needed to incorporate additional patient-specific feedback signals or reformulate the reward function to further harness RL's adaptive potential.

Table 5. Summary of Statistical Test Results

Test	Comparison	Test Statistic	p-Value	Interpretation
Paired t-Test	SVR-predicted vs. RL-adjusted dosages	-1.1132	0.2672	No statistically significant difference between SVR and RL dosage predictions.
One-way ANOVA	SVR, RL, and Physician dosages	F = 0.0165	0.9836	No statistically significant difference across the three dosage groups.

Since t-statistic = -1.1132 and p-value (0.2672) > 0.05, we cannot reject the null hypothesis. This implies that Reinforcement Learning (RL) tuning was not significant in improving the dosage predictions over the SVR model. In addition, as the F-statistic is 0.0165 and the p-value is 0.9836 of the One-Way ANOVA, and is far larger than the 0.05 threshold value, we once more do not reject the null hypothesis. This means there is no statistically significant difference among the mean dosage values of the SVR-predicted, RL-adjusted, and physician-prescribed dosages.

In short, both statistical tests show that dosage predictions made by SVR and RL are not significantly different from each other or from those made by doctors.

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This result suggests that SVR is already effective enough for initial dosage prediction, and RL offers only borderline adjustments that do not really add much to predictive accuracy, possibly due to limitations in simulation feedback instead of the model itself. In a larger clinical deployment in which more comprehensive, real-time feedback from both patients and clinicians is available, RL is able to adapt and outperform static regressors like SVR by capturing real-time patient-specific dynamics.

4. CONCLUSION

This paper proposes a hybrid AI framework integrating Support Vector Regression (SVR) with Proximal Policy Optimization (PPO)-based Reinforcement Learning (RL) for personalized dosage prediction of Propranolol. Using an ensemble of machine learning models, including SVR, XGBoost, KNN, and Random Forest with RL-based adaptive methods, the system ensures superior predictive performance and statistically validated results in comparison with physician-recommended dosages. Experimental findings confirm the fidelity of the SVR model with MSE = 0.3554 and R^2 = 0.9835, indicating close alignment with expert dosing decisions. Although the RL module enhances prediction through simulated feedback, it makes a minimal contribution to performance (MSE = 0.9928, R^2 = 0.9539). The results of paired t-tests and one-way ANOVA validate that the differences in dosage between AI-generated and physician-administered values do not achieve statistical significance. Therefore, this solidifies SVR as a baseline model that is trustworthy, while RL serves as a progressive improvement mechanism. The predictive capabilities of this research have great potential for incorporation into Clinical Decision Support Systems (CDSS), where they can improve individualized treatment planning and support clinician judgment.

By guaranteeing safer, data-driven dosing regimens, these systems could potentially improve patient adherence and alleviate the time burden on doctors in real-world settings by streamlining the dosage determination process. Additionally, integration with mobile health platforms or Electronic Health Record (EHR) systems may allow for real-time updates and dosage monitoring, increasing accessibility and patient condition responsiveness. Beyond Propranolol, the framework may also be used to manage other variable-dosage medications like insulin or warfarin, opening the door for a wider use of intelligent dosing systems in pharmacotherapy. Incorporating real-world, longitudinal patient feedback and investigating the model's generalizability across various therapeutic domains may be beneficial for future research. By doing this, this hybrid SVR-RL framework can develop into a clinically feasible, intelligent, and scalable precision medicine tool that guarantees safer and more efficient drug administration based on patient profiles.

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