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Impact of activation functions on dendritic neuron models in medical datasets

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Abstract

This study evaluates the dendritic neuron model (DNM) as an innovative approach for analysing medical datasets with balanced and imbalanced class distributions, encompassing breast cancer Wisconsin, hepatitis, heart failure clinical records (HFCR), anemia, and heart disease. The objective is to optimize DNM generalization through combinations of sigmoid, tanh, ReLU, and ELU activation functions, with a focus on detecting minority classes critical for clinical applications. Experiments employ stratified K-fold cross-validation to ensure robust evaluation. The sigmoid-ReLU combination excels on imbalanced datasets, achieving an accuracy of 0.9807 ± 0.0166 on breast cancer Wisconsin and an F1-score of 0.9179 ± 0.0453 on hepatitis, demonstrating robust handling of class imbalance. On balanced datasets, ReLU-ReLU achieves a perfect accuracy of 1.0000 ± 0.0000 on anemia, while ReLU-sigmoid stands out on heart disease with a ROC-AUC of 0.9026 ± 0.0576, reflecting effective class discrimination. Conversely, ELU-tanh exhibits limitations on HFCR, with an accuracy of 0.7092 ± 0.0740, indicating challenges with extreme class imbalance. This research provides theoretical insights into activation function dynamics in DNM and practical contributions for clinical decision support systems, with broad potential for heterogeneous medical data analysis.

Keywords: Dendritic neuron model, Activation functions, Medical, Clinical decision, Imbalance

1. Introduction

Machine learning has revolutionized medical data analysis, enabling predictive models that significantly improve diagnostic accuracy, prognostic precision, and clinical decision-making (Bhavsar et al., 2020; Garg & Mago, 2021). The proliferation of diverse medical data, including electronic health records, imaging datasets, and genomic profiles, has intensified the demand for models capable of addressing complex data characteristics, such as imbalanced class distributions (Sankar Ganesh & Sripriya, 2020; Yang, Khorshidi, & Aickelin, 2024). Medical datasets, such as those for breast cancer Wisconsin, hepatitis, and heart failure clinical records (HFCR), often exhibit significant class imbalances, with the minority class typically representing critical conditions that require high sensitivity for timely interventions (Araf, Idri, & Chairi, 2024; Khan, Chaudhari, & Chandra, 2024; Sachdeva, Bathla, Rani, & others, 2023). Conventional machine learning models struggle with these imbalances, often favoring the majority class and compromising the detection of critical minority classes essential for clinical applications.

Traditional neural networks, despite their success across various domains, face challenges such as slow convergence, susceptibility to overfitting, and reliance on optimal activation function selection (Dureja & Pahwa, 2019; Osman & Aljahdali, 2020; Rajkomar, Oren, Chen, & others, 2018; Sharma, Sharma, & Athaiya, 2020; Ying, 2019). Activation functions are pivotal in shaping training dynamics, influencing gradient stability, and enabling the capture of complex, non-linear data patterns. Suboptimal choices can exacerbate performance issues in heterogeneous medical datasets characterized by diverse

class distributions and intricate feature relationships, making the selection of appropriate activation functions critical for enhancing model performance in medical applications (Esteva, Kuprel, Novoa, & others, 2017).

The dendritic neuron model (DNM) introduces an innovative approach inspired by the biological structure of neurons (Chen et al., 2024). Unlike conventional neural networks with linear layer architectures, DNM employs non-linear signal processing at the dendritic layer before final computation at the soma, facilitating adaptive and hierarchical data representation (Gao et al., 2019; Jia et al., 2022). This mechanism enables DNM to effectively capture complex data relationships, making it particularly suitable for medical datasets with uneven class distributions or interdependent features (Xu et al., 2021). However, DNM performance is highly dependent on the choice of activation functions at the dendritic and soma layers, which govern convergence efficiency, gradient stability, and overall model generalization.

reported accuracies of 98.25% for convolutional neural networks (CNNs), 97.37% for gated recurrent units (GRU), 96.49% for feedforward neural networks (FNN), and 95.61% for long short-term memory (LSTM) networks (Jony & Arnob, 2024). However, the computational intensity of deep learning models like CNNs and LSTMs poses challenges in resource-constrained clinical settings, underscoring the need for efficient models like DNM, optimized with appropriate activation functions.

This study aims to enhance the accuracy and robustness of machine learning models in medical applications by systematically exploring activation function combinations—sigmoid, tanh, ReLU, and ELU—in DNM. By addressing class imbalance challenges and improving model generalization, it seeks to strengthen clinical decision support systems, enhancing diagnostic efficiency and patient outcomes. Additionally, it advances the theoretical understanding of activation function dynamics in non-conventional neural architectures, providing

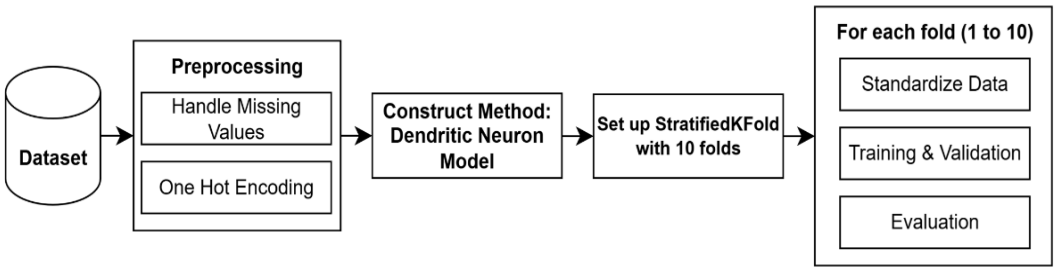

Figure 1. Research flow

Although DNM has shown promise in tasks like pattern recognition and non-medical data classification, its application to medical datasets remains underexplored (Ji, Tang, Zhao, Zheng, & Todo, 2022). Most studies utilize standard activation functions, such as sigmoid or ReLU, with limited investigation into alternatives like tanh or ELU, which may offer advantages for datasets with varied class distributions (Li, Ouyang, & Wang, 2016). Prior research has primarily focused on synthetic or non-medical datasets, leaving DNM's efficacy on complex medical datasets—marked by class imbalances or high-dimensional features—largely unaddressed. Comprehensive evaluations of activation function combinations and their impact on key metrics, such as accuracy, precision, recall, F1-score, and ROC-AUC, as well as their influence on training dynamics like gradient stability and convergence speed, are critical yet scarce in the medical context (Naidu, Zuva, & Sibanda, 2023).

Comparative studies highlight the importance of robust evaluation methodologies in medical data analysis. For instance, Khatun et al. reported accuracies of 92.42% for random forest, 85.00% for logistic regression, and 83.75% for support vector machine (SVM) on the hepatitis dataset using a manual 80:20 train-test split, which may introduce bias and limit generalizability (Khatun, Umam, Razzak, & others, 2025). Similarly, Diana Nurlaily et al. achieved accuracies of 79.3% for logistic regression and 81.94% for SVM on the same dataset, also using manual splits (Nurlaily, Irfandi, Santoso, Qomariyah, & Wibowo, 2022). On the breast cancer Wisconsin dataset, Jony and Arnob

insights into adapting DNM for complex medical data and laying the groundwork for efficient, adaptive models in healthcare and beyond.

2. Research Methods

This study was conducted on medical datasets characterized by both balanced and imbalanced class distributions, enabling a comprehensive evaluation of model performance across diverse data scenarios. Figure 1 illustrates the research workflow, which begins with a meticulous pre-processing stage to ensure data quality. Pre-processing involves handling missing values by imputing the median for numerical variables and the mode for categorical variables, followed by one-hot encoding to convert categorical features into a machine-readable format. Standardization is performed using Standard Scaler within each fold to prevent data leakage, ensuring that scaling parameters are derived solely from the training data in each cross-validation iteration. Five dendritic neuron models (DNM) with distinct activation function combinations were trained using a stratified K-fold cross-validation scheme with 10 folds. This approach was chosen for its ability to maintain consistent class distribution across folds, thereby providing a more robust and reliable performance estimation compared to a single manual train-test split, which may introduce bias due to uneven data partitioning. Model evaluation within each fold utilized a comprehensive set of metrics—accuracy, precision, recall, F1-score, and ROC-AUC—to thoroughly assess the model's generalization capability across diverse medical

datasets. These metrics collectively ensure a balanced evaluation, capturing both overall performance and the model's ability to handle critical minority classes, which is particularly vital in medical applications where accurate detection of rare conditions can significantly impact clinical outcomes.

2.1. Datasets

This study employs multiple medical datasets with varying class distributions, encompassing both imbalanced and balanced conditions. Datasets with imbalanced class distributions are presented in Table 1, while those with balanced class distributions are shown in Table 2. The selection of these dataset groups aims to consistently evaluate model performance across diverse class distribution characteristics.

Table 1: Overview of imbalanced datasets

Dataset	Instances	Features	Number of Classes	Class Proportion (%)
Breast cancer Wisconsin (diagnostic)	569	9	2	0: 62.74%, 1: 37.26%
Hepatitis	155	19	2	1: 79.35%, 0: 20.65%
Heart failure clinical records (HFCR)	299	12	2	0: 67.89%, 1: 32.11%

Table 2: Overview of balanced datasets

Dataset	Instances	Features	Number of Classes	Class Proportion (%)
Anemia	1421	5	2	0: 56.37%, 1: 43.63%
Heart disease	303	13	2	0: 54.13%, 1: 45.87%

2.2. Dendritic neuron model

The dendritic neuron model (DNM) represents an innovative approach to artificial neuron modeling, drawing inspiration from the biological architecture of neurons (Ji et al., 2022; Jia et al., 2022; Tang, Chen, et al., 2024). This model emphasizes the role of dendrites as complex initial signal processing units, integrating and modulating signals before they are processed by the soma as the final computation center. Through this mechanism, DNM effectively captures non-linear and adaptive interactions in data, making it highly relevant for machine learning applications, particularly tasks involving hierarchical or dynamic data representations (Gao et al., 2019; Jia et al., 2022). The structure of the model is detailed in Figure 2.

The computational process of DNM begins with the summation of inputs against dendritic weights, as formulated:

$$S_d = x \cdot W_d \quad (1)$$

The dendritic signal is then transformed through a non-linear activation function, as shown:

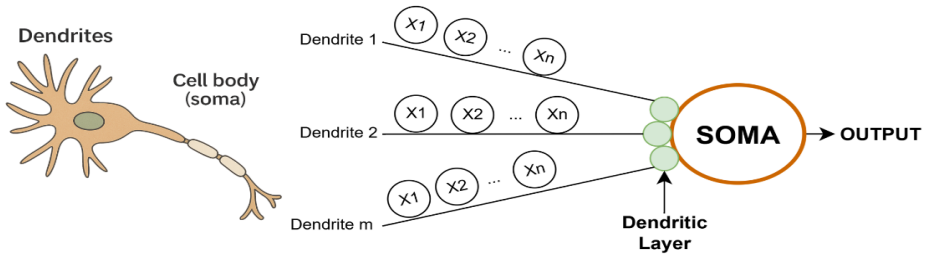

Figure 2: Structure of the dendritic neuron model

$$A_d = \varphi_d(S_d) \quad (2)$$

Inputs are also projected through kernel weights to produce a linear output, as indicated:

$$O = x \cdot W_k \quad (3)$$

This linear output is modulated by the dendritic signal based on:

$$O_m = O \odot A_d \quad (4)$$

The final stage involves adding a bias term and applying the soma activation function, as formulated:

$$y = \varphi_s(O_m + b) \quad (5)$$

Training of the parameters W_k, W_d, and b employs forward and backward propagation mechanisms based on gradient descent (Tang, Ji, et al., 2024). The detailed training steps are presented in Algorithm 1.

Algorithm 1: Dendritic neuron model training

Input: Vector $\mathbf{x} \in \mathbb{R}^d$, units u
Output: Vector $\mathbf{y} \in \mathbb{R}^u$

Parameters: Kernel weights $\mathbf{W}_k \in \mathbb{R}^{dxu}$, dendritic weights $\mathbf{W}_d \in \mathbb{R}^{dxu}$, bias $\mathbf{b} \in \mathbb{R}^u$

1. **Initialize** $\mathbf{W}_k$, $\mathbf{W}_d$ with random distribution, $\mathbf{b}$ as zeros.
2. **Compute** dendritic sum: $\mathbf{S}_d = \mathbf{x} \cdot \mathbf{W}_d$
3. **Apply** dendritic activation: $\mathbf{A}_d = \varphi(\mathbf{S}_d)$, where ϕ_d is the dendritic activation function
4. **Compute** neural output: $\mathbf{O} = \mathbf{x} \cdot \mathbf{W}_k$
5. **Modulate**: $\mathbf{O}_m = \mathbf{O} \odot \mathbf{A}_d$

.

6. **Apply** bias and soma activation: $\mathbf{y} = \varphi_s(\mathbf{O}_m + \mathbf{b})$, where φ_s is the soma activation function
7. **Return y**
8. **Update** $\mathbf{W}_k$, $\mathbf{W}_d$, **b** via backpropagation with appropriate loss

Upon completion of training, the prediction phase is conducted without weight updates, focusing on dendritic computation, modulation, and soma activation. This procedure is summarized in Algorithm 2.

Algorithm 2: Dendritic neuron model prediction

Input: Vector $\mathbf{x} \in \mathbb{R}^d$, units u
Output: Vector $\mathbf{y} \in \mathbb{R}^u$

Parameters: Kernel weights $\mathbf{W}_k \in \mathbb{R}^{dxu}$, dendritic weights $\mathbf{W}_d \in \mathbb{R}^{dxu}$, bias $\mathbf{b} \in \mathbb{R}^u$

1. **Load** trained parameters $\mathbf{W}_k$, $\mathbf{W}_d$, and **b**
2. **Compute** dendritic sum: $\mathbf{S}_d = \mathbf{x} \cdot \mathbf{W}_d$
3. **Apply** dendritic activation: $\mathbf{A}_d = \varphi(\mathbf{S}_d)$, where φ_d is the dendritic activation function
4. **Compute** neural output: $\mathbf{O} = \mathbf{x} \cdot \mathbf{W}_k$
5. **Modulate**: $\mathbf{O}_m = \mathbf{O} \odot \mathbf{A}_d$
6. **Apply** bias and soma activation: $\mathbf{y} = \varphi_s(\mathbf{O}_m + \mathbf{b})$, where φ_s is the soma activation function
7. **Return y**

For a comprehensive understanding of the core components of DNM, details regarding weights, biases, and activation functions are provided in Table 3.

Table 3: Core computational components of the dendritic neuron model

Component	Symbol	Dimension	Functional Role	Dependency (Determinants)
Kernel weights	$\mathbf{W}_k$	$\mathbb{R}^{dxu}$	Perform linear projection from the input space to the neuronal output	Defined by the number of input features (d) and output units (u)
Dendritic weights	$\mathbf{W}_d$	$\mathbb{R}^{dxu}$	Modulate input signals before integration at the soma	Determined by d and u; continuously optimized during training
Bias	**b**	$\mathbb{R}^u$	Shifts the neuron's response to improve flexibility	Determined by the number of output units (u)
Dendritic activation	φ_d	–	Applies non-linear transformation to dendritic input sums	Fixed by model design; influences modulation strength
Soma activation	φ_s	–	Applies non-linear transformation at the soma to yield the final output	Fixed by model design; governs output range and learning dynamics

2.3. Experiments and evaluation

To assess the performance of the Dendritic Neuron Model (DNM), experiments were conducted using 10-fold cross-validation to ensure generalizable results across the dataset (Bates, Hastie, & Tibshirani, 2024; Lumumba, Sang, Njoka, Musyimi, & Kavita, 2024). The experiments compared combinations of activation functions applied to the dendritic and soma layers, specifically sigmoid, tanh, ReLU, and ELU (Dureja & Pahwa, 2019; Li et al., 2016; Sharma et al., 2020; Shi, Chen, & Wang, 2021). The formulas for these activation functions are presented below.

$$f(x) = 1/(1 + e^{-x}) \quad (6)$$

$$f(x) = (e^{x} - e^{-x})/(e^{x} + e^{-x}) \quad (7)$$

$$f(x) = \max(0, x) \quad (8)$$

$$f(x) = x \text{ if } x > 0; \alpha(e^{x} - 1) \text{ if } x \leq 0 \quad (9)$$

In the above formulas, x represents the input to the activation function, e is the natural logarithm base (approximately 2.718), and α (typically set to 1) in the ELU function controls the scale of negative outputs.

The combinations of activation functions tested are summarized in Table 4, with the first function applied to the dendritic layer and the second to the soma layer.

Table 4: Activation function combinations

Combination	Dendritic Layer	Soma Layer
sigmoid-tanh	Sigmoid	Tanh
ReLU-ReLU	ReLU	ReLU
sigmoid-ReLU	Sigmoid	ReLU
ELU-tanh	ELU	Tanh
ReLU-sigmoid	ReLU	Sigmoid

To thoroughly evaluate the performance of the dendritic neuron model (DNM), several metrics were utilized, including accuracy, precision, recall, F1-score, and ROC-AUC. These metrics collectively offer a comprehensive understanding of the model's effectiveness across diverse dimensions, particularly for medical datasets with varied class distributions (Garg & Mago, 2021; Naidu et al., 2023). Accuracy measures the proportion of correct predictions and is calculated as:

$$\text{Accuracy} = \frac{TP+TN}{TP+TN+FP+FN} \quad (10)$$

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Precision quantifies the proportion of positive predictions that are correct, defined as:

$$\text{Precision} = \frac{\text{TP}}{\text{TP+FP}} \quad (11)$$

Recall represents the proportion of actual positive cases correctly identified, given by:

$$\text{Recall} = \frac{\text{TP}}{\text{TP+FN}} \quad (12)$$

F1-score balances precision and recall through their harmonic mean, expressed as:

$$\text{F1-score} = 2 \times \frac{\text{Precision} \times \text{Recall}}{\text{Precision+Recall}} \quad (13)$$

ROC-AUC assesses the model's ability to distinguish between classes across all classification thresholds, defined as:

$$\text{ROC-AUC} = \int_0^1 \text{TPR(t)} \times \text{FPR}'\text{(t)}, \text{dt} \quad (14)$$

In these formulas, TP (true positive) represents the number of correct positive predictions, TN (true negative) indicates correct negative predictions, FP (false positive) denotes incorrect positive predictions, and FN (false negative) signifies incorrect negative predictions. The true positive rate (TPR) corresponds to recall, while the false positive rate (FPR) is computed as FP / (FP + TN), with t representing the classification threshold.

The F1-score is particularly vital for imbalanced datasets, which are common in medical applications such as breast cancer, hepatitis, and heart failure datasets, where the minority class often represents critical conditions (Araf et al., 2024; Yang et al., 2024). Unlike accuracy, which may be misleading in imbalanced settings due to bias toward the majority class, the F1-score effectively balances precision and recall. This ensures robust evaluation of the model's capability to detect the minority class while maintaining overall performance, which is crucial in healthcare where missing critical diagnoses (false negatives) can have severe consequences (An, Rahman, Zhou, & Kang, 2023; Shortliffe & Sepúlveda, 2018). Similarly, ROC-AUC is essential in medical contexts as it evaluates the model's discriminative ability across varying thresholds, ensuring reliable performance for both balanced and imbalanced datasets. Together, these metrics provide a robust framework for assessing DNM's performance, addressing the unique challenges of medical data analysis and facilitating the development of effective clinical decision support systems.

3. Results and Discussion

This study evaluates the performance of the dendritic neuron model (DNM) across medical datasets with diverse class distributions, including breast cancer Wisconsin (Diagnostic), hepatitis, heart failure clinical records (HFCR), anemia, and heart disease. Experiments employed stratified 10-fold cross-validation to ensure reliable and robust performance evaluation, enhancing confidence in model generalization compared to single manual splits, which may introduce bias and yield inconsistent performance across different split configurations. The focus was on assessing the impact of activation function combinations—sigmoid, tanh, ReLU, and ELU—on key metrics: accuracy, precision, recall, F1-score, and ROC-AUC. These metrics provide a comprehensive assessment of DNM's ability to address the complexities of medical data, particularly class imbalances and non-linear feature interactions. Results, detailed in Tables 5 and 6 and visualized in Figures 3 and 4, offer insights into the interplay between activation functions, dataset characteristics, and model performance, with significant implications for clinical decision support systems.

The sigmoid-ReLU combination demonstrated superior performance on imbalanced datasets, achieving an accuracy of 0.9807 ± 0.0166 and an F1-score of 0.9735 ± 0.0234 on breast cancer Wisconsin, and an F1-score of 0.9179 ± 0.0453 on hepatitis. This indicates robust handling of class imbalance, likely due to sigmoid's smooth, bounded output stabilizing gradients at the dendritic layer, mitigating vanishing gradient issues, while ReLU's non-saturating nature at the soma accelerates convergence. These results align with prior findings that combining smooth and non-smooth activation functions enhances performance on skewed distributions by balancing gradient flow and sparsity. In contrast, the ELU-tanh combination exhibited the weakest performance on HFCR, with an accuracy of 0.7092 ± 0.0740 and a recall of 0.3722 ± 0.1149. ELU's sensitivity to negative inputs may amplify noise in highly imbalanced datasets (32.11% minority class), while tanh's constrained output range (-1, 1) limits adaptability to extreme class disparities, leading to poor minority class detection. The ReLU-sigmoid combination showed strong performance on HFCR (accuracy of 0.8228 ± 0.0615, F1-score of 0.7009 ± 0.1067), suggesting that sigmoid at the soma enhances model flexibility through smooth output scaling, though its precision (0.7657 ± 0.1133) reflects variability in positive class predictions, likely due to the dataset's pronounced imbalance.

Comparative studies contextualize these findings. On the hepatitis dataset, Khatun et al. reported accuracies of 92.42% for Random Forest, 85.00% for Logistic Regression, and 83.75% for SVM using an 80:20 manual split, while Nurlaily et al. achieved 79.3% for Logistic Regression and 81.94% for SVM (Khatun et al., 2025; Nurlaily et al., 2022). The sigmoid-ReLU DNM configuration (accuracy of 0.8700 ± 0.0724, F1-score of 0.9179 ± 0.0453) outperforms these models, benefiting from stratified 10-fold cross-validation, which enhances reliability and generalization by mitigating bias inherent in manual splits. On breast cancer Wisconsin, Jony and Arnob reported a CNN accuracy of 98.25%, followed by GRU (97.37%), FNN (96.49%), and LSTM (95.61%) (Jony & Arnob, 2024). While DNM's sigmoid-ReLU configuration (accuracy of 0.9807 ± 0.0166) is competitive, its lower computational complexity compared to deep learning models makes it more suitable for resource-constrained clinical environments.

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On balanced datasets, ReLU-ReLU and sigmoid-ReLU achieved perfect performance on anemia (accuracy of 1.0000 ± 0.0000, F1-score of 1.0000 ± 0.0000), reflecting

Table 5: Dendritic neuron performance on imbalanced datasets with different activation functions

Activation	Dataset	Accuracy	Precision	Recall	F1-Score	ROC-AUC
sigmoid-tanh	Breast cancer Wisconsin (Diagnostic)	0.9772 ± 0.0193	0.9772 ± 0.0228	0.9619 ± 0.0555	0.9683 ± 0.0288	0.9947 ± 0.0088
	Hepatitis	0.8508 ± 0.0776	0.9062 ± 0.0623	0.9090 ± 0.0585	0.9060 ± 0.0482	0.8707 ± 0.0829
	Heart failure clinical records (HFCR)	0.7960 ± 0.0605	0.7015 ± 0.1186	0.6556 ± 0.1136	0.6734 ± 0.0982	0.8556 ± 0.0757
ReLU-ReLU	Breast cancer Wisconsin (Diagnostic)	0.9772 ± 0.0176	0.9776 ± 0.0297	0.9619 ± 0.0467	0.9687 ± 0.0251	0.9948 ± 0.0083
	Hepatitis	0.8579 ± 0.0805	0.9149 ± 0.0665	0.9090 ± 0.0787	0.9091 ± 0.0542	0.8122 ± 0.1441
	Heart failure clinical records (HFCR)	0.7861 ± 0.0856	0.6950 ± 0.1627	0.6056 ± 0.1531	0.6431 ± 0.1500	0.8395 ± 0.0941
sigmoid-ReLU	Breast cancer Wisconsin (Diagnostic)	0.9807 ± 0.0166	0.9822 ± 0.0292	0.9667 ± 0.0429	0.9735 ± 0.0234	0.9952 ± 0.0081
	Hepatitis	0.8700 ± 0.0724	0.9156 ± 0.0582	0.9250 ± 0.0692	0.9179 ± 0.0453	0.8350 ± 0.1082
	Heart failure clinical records (HFCR)	0.7960 ± 0.0737	0.7084 ± 0.1208	0.6367 ± 0.1653	0.6617 ± 0.1307	0.8395 ± 0.0923
ELU-tanh	Breast cancer Wisconsin (Diagnostic)	0.9421 ± 0.0360	0.9502 ± 0.0566	0.8963 ± 0.0933	0.9183 ± 0.0532	0.9837 ± 0.0150
	Hepatitis	0.8637 ± 0.0630	0.8824 ± 0.0413	0.9583 ± 0.0768	0.9164 ± 0.0427	0.8222 ± 0.1527
	Heart failure clinical records (HFCR)	0.7092 ± 0.0740	0.6135 ± 0.2221	0.3722 ± 0.1149	0.4500 ± 0.1303	0.6568 ± 0.1012
ReLU-sigmoid	Breast cancer wisconsin (Diagnostic)	0.9772 ± 0.0208	0.9865 ± 0.0283	0.9526 ± 0.0563	0.9680 ± 0.0306	0.9939 ± 0.0120
	Hepatitis	0.8704 ± 0.0705	0.8997 ± 0.0472	0.9417 ± 0.0651	0.9191 ± 0.0465	0.8768 ± 0.0935
	Heart failure clinical records (HFCR)	0.8228 ± 0.0615	0.7657 ± 0.1133	0.6556 ± 0.1301	0.7009 ± 0.1067	0.8652 ± 0.0718

Table 6: Dendritic neuron performance on balanced datasets with different activation functions

Activation	Dataset	Accuracy	Precision	Recall	F1-Score	ROC-AUC
sigmoid-tanh	Anemia	0.9993 ± 0.0021	0.9984 ± 0.0048	1.0000 ± 0.0000	0.9992 ± 0.0024	1.0000 ± 0.0000
	Heart disease	0.8183 ± 0.0478	0.8293 ± 0.0942	0.7775 ± 0.0735	0.7973 ± 0.0502	0.8868 ± 0.0515
ReLU-ReLU	Anemia	1.0000 ± 0.0000	1.0000 ± 0.0000	1.0000 ± 0.0000	1.0000 ± 0.0000	1.0000 ± 0.0000
	Heart disease	0.8053 ± 0.0594	0.7849 ± 0.0812	0.8060 ± 0.0842	0.7916 ± 0.0625	0.8594 ± 0.0457
sigmoid-ReLU	Anemia	1.0000 ± 0.0000	1.0000 ± 0.0000	1.0000 ± 0.0000	1.0000 ± 0.0000	1.0000 ± 0.0000
	Heart disease	0.8052 ± 0.0506	0.8005 ± 0.0786	0.7775 ± 0.0974	0.7839 ± 0.0613	0.8807 ± 0.0464
ELU-tanh	Anemia	0.9951 ± 0.0070	0.9968 ± 0.0064	0.9919 ± 0.0149	0.9943 ± 0.0082	1.0000 ± 0.0001

DNM's ability to leverage uniform class distributions (56.37% vs. 43.63%). ReLU's non-saturating property promotes sparsity and prevents vanishing gradients, enabling rapid convergence and precise class separation. The sigmoid-tanh combination also performed

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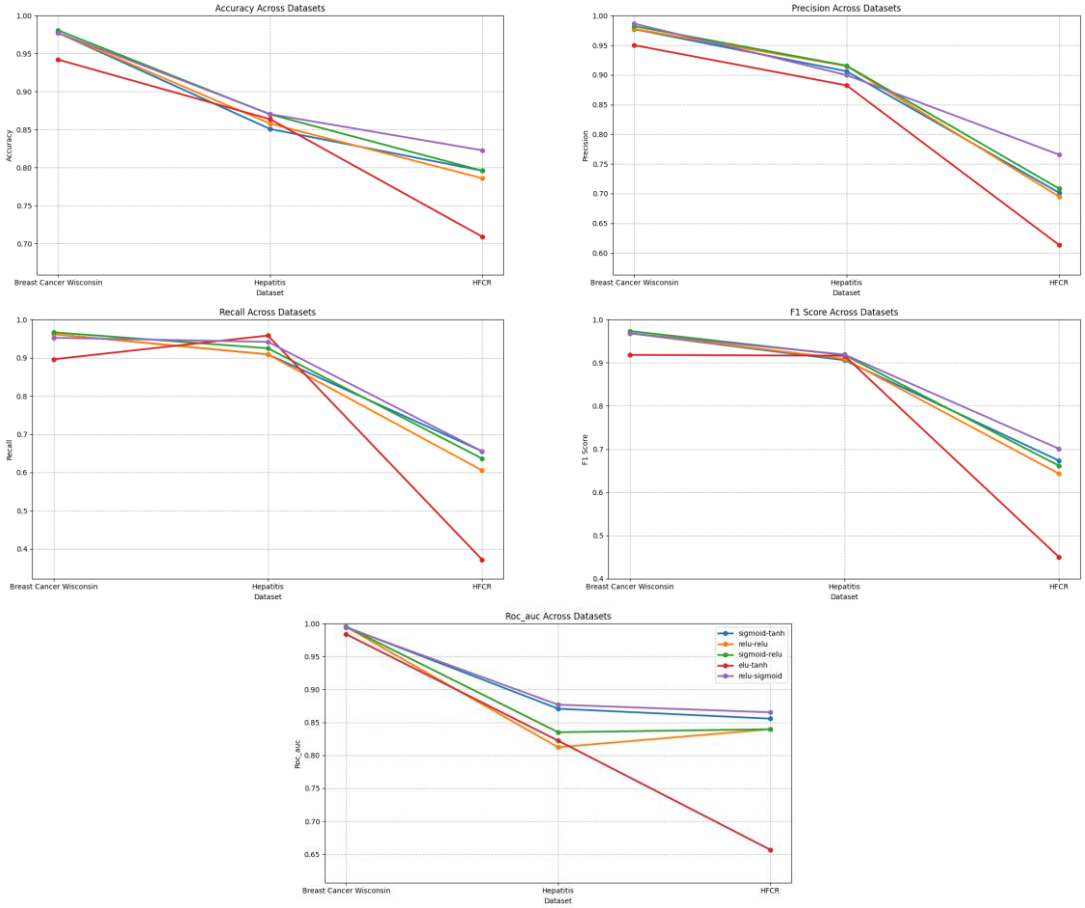

Figure 3: Experimental results comparison using imbalanced datasets

exceptionally well on anemia (accuracy of 0.9993 ± 0.0021), indicating that smooth activation functions are effective when class proportions are nearly equal. For heart disease, ReLU-sigmoid outperformed others (accuracy of 0.8181 ± 0.0550, ROC-AUC of 0.9026 ± 0.0576), with sigmoid at the soma enhancing discriminative capability through smooth output scaling, particularly suitable for datasets with complex feature interactions and moderate class balance (54.13% vs. 45.87%). Conversely, ELU-tanh showed the lowest performance on heart disease (accuracy of 0.7524 ± 0.0517, F1-score of 0.7208 ± 0.0661), likely due to ELU's negative value handling disrupting dendritic modulation and tanh's limited output range hindering adaptability to nuanced class boundaries.

The results underscore the pivotal role of activation function selection in DNM's performance. The sigmoid-ReLU combination's success on imbalanced datasets stems from sigmoid's ability to stabilize gradients at the dendritic layer and ReLU's efficiency at the soma, making it ideal for datasets like breast cancer Wisconsin (37.26% minority class) and hepatitis (20.65% minority class), where minority class detection is critical. The high F1-score (0.9179 ± 0.0453 on hepatitis) highlights DNM's sensitivity to minority classes, essential for clinical applications where false negatives can have severe consequences. In contrast, ELU-tanh's poor performance on HFCR suggests that ELU's handling of negative inputs introduces instability in highly imbalanced datasets, amplified by tanh's constrained output range, resulting in low recall (0.3722 ± 0.1149). This contrasts with studies on non-medical datasets where ELU and tanh offer stability, indicating dataset-specific challenges.

Compared to traditional models, DNM's performance is competitive. On hepatitis, DNM's sigmoid-ReLU configuration outperforms Random Forest (92.42%), Logistic Regression (85.00%), and SVM (83.75%), benefiting from the robust evaluation of stratified 10-fold cross-validation, which enhances confidence in model generalization compared to manual splits prone to bias. On breast cancer Wisconsin, DNM's accuracy (0.9807 ± 0.0166) rivals CNN (98.25%), with the added advantage of lower computational complexity, making it more practical for clinical settings.

These findings highlight DNM's flexibility in handling medical datasets, with activation function choice significantly influencing performance. Sigmoid-ReLU excels in imbalanced settings, balancing gradient stability and convergence efficiency, while ReLU-ReLU and ReLU-sigmoid are optimal for balanced datasets due to their simplicity and discriminative power. However, ELU-tanh's underperformance indicates the need for further optimization, such as adaptive activation functions or hybrid architectures, to address extreme imbalances. The reliance on precise parameter tuning poses a challenge, as

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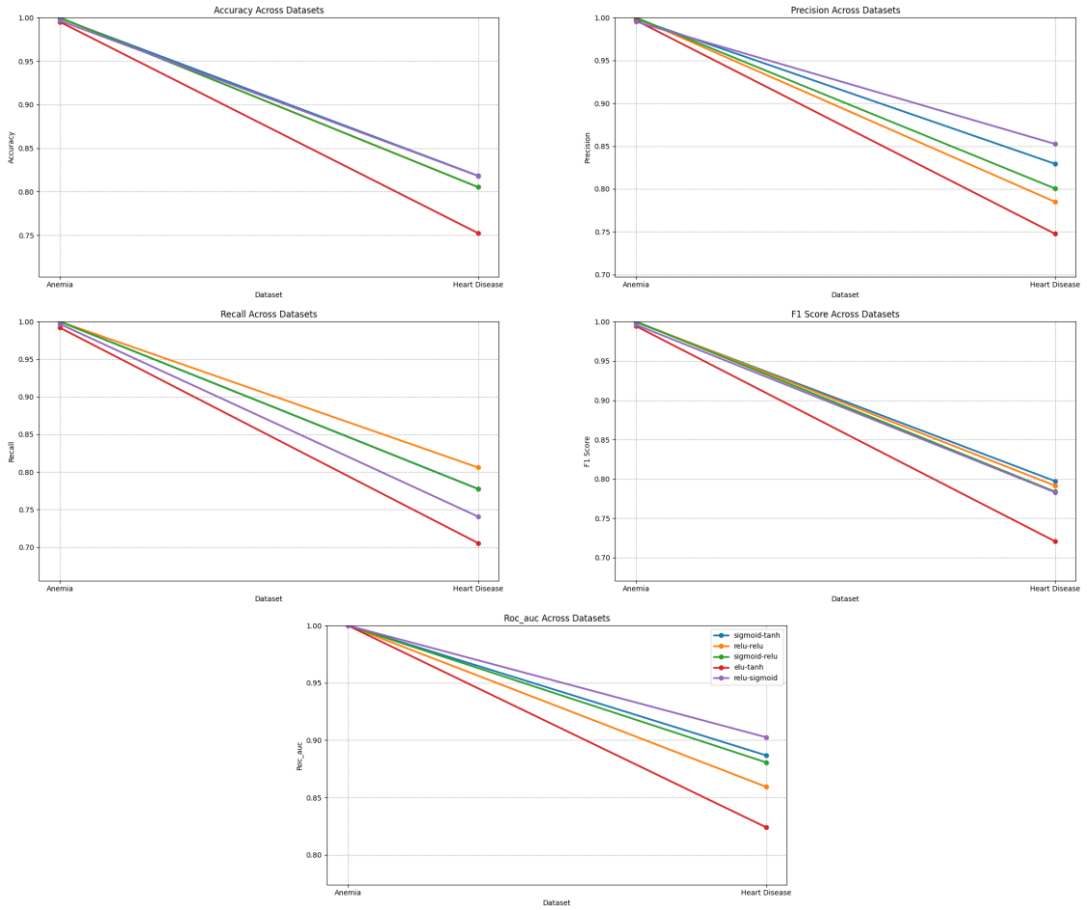

Figure 4: Experimental results comparison using balanced datasets

suboptimal configurations can degrade performance, particularly in high-dimensional datasets like heart disease.

The use of stratified 10-fold cross-validation ensures reliable evaluation, reducing bias compared to manual splits and providing greater confidence in model generalization. However, limitations include potential overfitting in small datasets like hepatitis (155 instances) and the need for techniques like SMOTE to enhance minority class detection. Future research should explore adaptive activation functions, ensemble methods, or DNM architectural modifications to improve robustness and scalability for clinical applications.

Practically, DNM's high sensitivity to minority classes supports its potential for clinical decision support systems, enabling early diagnosis of conditions like breast cancer and hepatitis. Its computational efficiency compared to deep learning models enhances feasibility in resource-limited healthcare settings. Theoretically, this study advances understanding of activation function dynamics in non-conventional neural architectures, highlighting the interplay between dendritic and soma processing in addressing complex medical data. These insights provide a foundation for developing adaptive and efficient machine learning models across diverse domains.

4. Conclusion

This study comprehensively demonstrates the dendritic neuron model (DNM)'s remarkable flexibility and efficacy in analyzing medical datasets characterized by diverse class distributions, underscoring its potential as a robust tool in clinical decision support systems. The performance of DNM is highly contingent on the strategic selection of activation functions, which significantly influence its ability to handle varying dataset characteristics. Specifically, the sigmoid-ReLU combination emerges as optimal for imbalanced datasets, such as the Breast Cancer Wisconsin and Hepatitis datasets, achieving a high accuracy of 0.9807 ± 0.0166 and an F1-score of 0.9179 ± 0.0453. This success is attributed to sigmoid's ability to stabilize gradients at the dendritic layer, coupled with ReLU's efficiency in promoting rapid convergence at the soma, particularly enhancing the detection of critical minority classes. For balanced datasets, such as the Anemia dataset, the ReLU-ReLU configuration delivers exceptional performance, achieving a perfect accuracy of 1.0000 ± 0.0000, capitalizing on ReLU's effectiveness in scenarios with uniform class distributions. Similarly, the ReLU-sigmoid combination excels on the Heart Disease dataset, yielding a ROC-AUC of 0.9026 ± 0.0576, where sigmoid's role at the soma enhances complex class separation, thereby improving diagnostic precision. Conversely, the ELU-tanh combination underperforms on the Heart Failure Clinical Records (HFCR) dataset, with a lower accuracy of 0.7092 ± 0.0740 and a recall of 0.3722 ± 0.1149, likely due to ELU's sensitivity to negative values, which is not adequately mitigated by tanh in highly imbalanced settings. Despite its strengths, DNM exhibits limitations, including its dependence on meticulous hyperparameter optimization and challenges in accurately detecting minority classes in severely imbalanced datasets.

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These constraints highlight the need for further research to enhance DNM's robustness, potentially through the development of adaptive activation functions, integration of ensemble learning techniques, or architectural refinements to the DNM framework. These findings not only deepen the theoretical understanding of activation function dynamics in non-conventional neural architectures but also establish a practical foundation for leveraging DNM in real-world clinical applications. By improving diagnostic accuracy, optimizing clinical decision-making processes, and enabling robust analysis of heterogeneous medical data across diverse domains, DNM holds significant promise for advancing precision medicine and supporting healthcare professionals in delivering more effective patient care.

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