Neuroplastic Self-Organizing Map (NPSOM): A Novel Approach for Analyzing Multiomics Data

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ABSTRACT

This paper introduces the Neuroplastic Self-Organizing Map (NPSOM), an unsupervised learning approach that enhances the traditional Self-Organizing Map (SOM) with neuroplasticity features. NPSOM incorporates mechanisms for synaptic decay, neurogenesis, and memory forgetting to improve the adaptability and performance of the SOM. By mimicking the brain's ability to strengthen certain neurons' synaptic connections and forget unnecessary information, NPSOM adapts to changing data distributions while retaining important information. The experimental results show that NPSOM outperforms MOSEGCN (best baseline) by 0.3% on the BRCA dataset while delivering similar performance on the ROSMAP dataset in terms of classification accuracy. This indicates a better ability to detect feature importance, making it a promising tool for real-world applications in machine learning and data analysis.

CCS CONCEPTS

• **Computing methodologies** → **Machine learning algorithms**;
• **Applied computing** → *Health informatics*.

KEYWORDS

Self-Organizing Map, Neuroplasticity, Feature Selection, Machine Learning

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1 INTRODUCTION

The Self-Organizing Map (SOM) is a widely used unsupervised learning algorithm for dimensionality reduction and clustering, first introduced by Teuvo Kohonen in 1982 [10]. SOMs have been extensively applied in various domains, including image processing [11], bioinformatics [18], data visualization [19], and pattern recognition [9], due to their ability to project high-dimensional data onto a low-dimensional grid while preserving the topological relationships between data points. However, traditional SOMs suffer from a significant limitation: they lack the ability to adapt to changing data distributions over time [17]. This limitation is particularly critical in real-world applications where data evolves dynamically, such as in gene expression analysis [1], epigenomic data [16], or time-series datasets [8].

To address this limitation, this paper introduces the Neuroplastic Self-Organizing Map (NPSOM), a novel approach that enhances the traditional SOM with neuroplasticity features. Neuroplasticity refers to the brain's ability to reorganize itself by forming new neural connections throughout life [12]. Inspired by this biological phenomenon, NPSOM incorporates three key mechanisms: **Synaptic Decay**, **Neurogenesis**, **Memory Forgetting**. These enhancements enable NPSOM to better capture the underlying structure of the data and improve its performance in feature selection. The model is tested on two datasets: the TCGA-BRCA dataset, which contains multiomics data related to Breast Invasive Carcinoma[15], and the ROSMAP dataset, which is a multiomics dataset used for Alzheimer's detection studies[4]. The high-dimensional nature of these datasets poses significant learning challenges, necessitating feature selection to mitigate the effects of the curse of dimensionality. They were chosen to validate the effectiveness of NPSOM in real-world applications with complex and evolving data distributions.

The NPSOM algorithm mimics the brain's ability to create new neurons (neurogenesis) and forget unnecessary information over time (synaptic decay and memory forgetting). This allows the model to adapt to new data while retaining important information, similar to how the brain forms short-term and long-term memories. By incorporating these neuroplasticity features, NPSOM can dynamically adjust to changing data distributions, making it a robust tool for real-world applications.

2 RELATED WORK

Previous research has explored various extensions and modifications to the traditional SOM to enhance its performance. One of the most notable works is the Growing Self-Organizing Map (GSOM) [5], which dynamically adjusts the size of the map based on the input data. GSOM addresses the limitation of traditional SOMs by allowing the map to grow or shrink in response to the data distribution, making it more flexible for complex datasets. However, GSOM does not incorporate mechanisms for continuous adaptation to changing data distributions over time.

Another significant contribution is the Adaptive Resonance Theory (ART) [7], which introduces mechanisms for stability and plasticity. ART networks are designed to handle the stability-plasticity

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dilemma, where the model must balance the ability to learn new information (plasticity) with the need to retain previously learned knowledge (stability) [3]. While ART networks are effective in certain applications, they do not fully address the need for continuous adaptation to evolving data distributions.

The traditional SOM algorithm [10] has been widely used for clustering and dimensionality reduction due to its simplicity and effectiveness. However, its static nature limits its ability to adapt to changing data distributions. The GSOM [5] extends the SOM by allowing the map to grow dynamically based on the input data, but it does not incorporate neuroplasticity features such as synaptic decay or memory forgetting. The NPSOM builds upon these concepts by introducing neuroplasticity features to better adapt to changing data distributions.

The concept of neuroplasticity has also been explored in other machine learning algorithms. For example, the Neurogenesis Deep Learning (NDL) framework [13] incorporates neurogenesis into deep neural networks to improve their adaptability to new data. Similarly, the Synaptic Plasticity and Neurogenesis (SPAN) model [14] combines synaptic plasticity and neurogenesis to enhance the learning capabilities of neural networks.

In summary, while previous works have made significant contributions to enhancing the traditional SOM, they do not fully address the need for continuous adaptation to changing data distributions through neuroplasticity features. NPSOM fills this gap by incorporating synaptic decay, neurogenesis, and memory forgetting, making it a more robust and adaptable tool for real-world applications.

3 METHODOLOGY

This section provides a detailed description of the Neuroplastic Self-Organizing Map (NPSOM) algorithm, including its architecture, key components, and step-by-step methodology. NPSOM builds upon the traditional SOM by incorporating neuroplasticity features to enhance its adaptability and performance in feature ranking and selection tasks.

3.1 NPSOM Architecture

The NPSOM architecture is based on the traditional SOM but includes additional components for neuroplasticity. These components enable the model to adapt to changing data distributions and improve its performance. The key components of NPSOM are:

- **Synaptic Decay:** Synaptic decay is inspired by the brain's ability to weaken or prune synapses that are no longer used. The weights in the SOM decay over time to maintain plasticity. This mechanism allows the model to adapt to new data while preventing overfitting to older data.
- **Neurogenesis:** Neurogenesis mimics the brain's ability to generate new neurons in response to learning or environmental changes. New neurons are dynamically added to the map based on the importance and activation of existing neurons. This enables the model to grow and adapt to complex data structures.
- **Memory Forgetting:** This mechanism is inspired by the brain's ability to forget unnecessary information to optimize memory and processing capacity. Neurons with low activation and importance are removed from the map to prevent overfitting. Memory forgetting helps in maintaining the model's adaptability and performance over time.

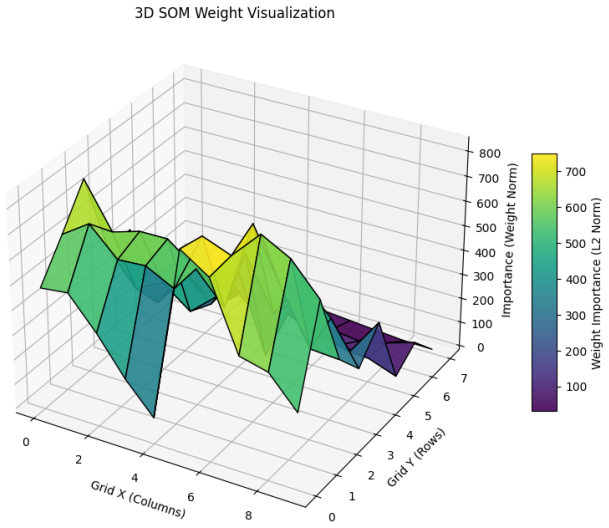

Figure 1: 3D SOM Weight Visualization showing the importance of weights across the grid of BRCA dataset.

3.2 Detailed Methodology

(1) **Initialization:** The weights of the NPSOM are initialized randomly in the range $[0, 1]$. The parameters for learning rate (η), decay rate (δ), and importance factor (α) are set. The grid size of the SOM is defined as (rows, cols). The initialization process ensures that the model starts with a diverse set of weights to avoid activation bias.

(2) **Training:** The training process involves updating the weights of the SOM based on the Best Matching Unit (BMU)[10] and applying synaptic decay. The BMU is found using the Euclidean distance:

$$\text{BMU} = \arg\min_i \|\mathbf{x} - \mathbf{w}_i\| \tag{1}$$

where $\mathbf{x}$ is the input vector and $\mathbf{w}_i$ is the weight vector of neuron i. The weights are updated using the winner-takes-all rule[10]:

$$\mathbf{w}_i(t+1) = \mathbf{w}_i(t) + \eta(t) \cdot h_{ci}(t) \cdot (\mathbf{x} - \mathbf{w}_i(t)) \tag{2}$$

where $\eta(t)$ is the learning rate at time t, and $h_{ci}(t)$ is the neighborhood function[10]:

$$h_{ci}(t) = \exp\left(-\frac{\|\mathbf{r}_c - \mathbf{r}_i\|^2}{2\sigma(t)^2}\right) \tag{3}$$

where $\mathbf{r}_c$ and $\mathbf{r}_i$ are the positions of the BMU and neuron i, respectively, and $\sigma(t)$ is the neighborhood radius at time t. The neighborhood function ensures that neurons close to the BMU are updated more than those farther away, which helps in preserving the topological relationships in the data.

(3) **Synaptic Decay:** Synaptic decay is applied to the weights to maintain plasticity:

$$\mathbf{w}_i(t+1) = \mathbf{w}_i(t) - \delta \cdot \mathbf{w}_i(t) \tag{4}$$

This mechanism allows the model to adapt to new data while preventing overfitting to older data.

(4) **Neurogenesis:** New neurons are added based on the importance map and activation count. The Importance Map is initialized as a matrix of zeros with the shape of the initial grid. The importance map $\mathcal{I}(t)$ is updated as:

$$\mathcal{I}_i(t+1) = \mathcal{I}_i(t) + \alpha \cdot h_{ci}(t) \tag{5}$$

To normalize for participation frequency, a neuron's cumulative importance is divided by the number of updates it received denoted by $\mathcal{T}(t)$ and $\mathcal{A}_i(t)$ is the activation count of neuron i at time t which is how many times that neuron has been fired up.

$$\mathcal{T}_i(t) = \left(\frac{\mathcal{I}_i(t)}{\mathcal{A}_i(t))}\right) \tag{6}$$

To determine the positions for neurogenesis, we use the following equation:

$$\mathbf{p}_{\text{new}} = \arg\max_i \mathcal{T}_i(t) \tag{7}$$

where $\mathbf{p}_{\text{new}}$ is the position of the new neuron. Neurogenesis enables the model to grow and adapt to complex data structures by dynamically adding new neurons to the map.

(5) **Memory Forgetting:** Neurons with low activation and importance are removed to prevent overfitting. The forgetting criterion is given by:

$$\text{Forget}_i = \begin{cases} 1 & \text{if } \mathcal{A}_i(t) < \theta_1 \text{ and } \mathcal{I}_i(t) < \theta_2 \\ 0 & \text{otherwise} \end{cases} \tag{8}$$

where θ_1 and θ_2 are thresholds for activation count and importance, respectively. Memory forgetting helps in maintaining the model's adaptability and performance over time by removing neurons that are no longer contributing to the learning process.

(6) **Feature Ranking via Importance:** After training, NPSOM ranks features based on their contribution to highly important and frequently activated neurons. We use $\mathcal{T}_i(t)$ as defined in Equation 6. The top 75% neurons with most $\mathcal{T}(t)$ are selected, and for each feature, the variance across their weight vectors is computed. Features with higher variance are considered more informative. Formally, the Feature Importance Score (FIS) of a feature f_j is defined as:

$$\text{FIS}(f_j) = \text{Var}\left(\{w_{ij} \mid i \in \mathcal{T}_{\text{top}}\}\right) \tag{9}$$

where w_{ij} is the weight corresponding to feature j in neuron i, and $\mathcal{T}_{\text{top}}$ denotes the set of neurons with most $\mathcal{T}$ values. Features are then ranked by sorting their FIS values in descending order:

$$\text{RankedFeatures} = \text{argsort}_j\left(\text{FIS}(f_j)\right)_{\downarrow} \tag{10}$$

3.3 Algorithm Overview

The NPSOM algorithm can be summarized as follows:

(1) **Initialization:**
- Initialize weights $\mathbf{w}_i$ randomly in the range $[0, 1]$.
- Set parameters: learning rate η, decay rate δ, importance factor α, grid size (rows, cols).

(2) **Training:**
- For each batch of data:
  - For each input vector $\mathbf{x}$:
    - Find BMU: BMU $= \arg\min_i \|\mathbf{x} - \mathbf{w}_i\|$
    - Update weights: $\mathbf{w}_i(t+1) = \mathbf{w}_i(t) + \eta(t) \cdot h_{ci}(t) \cdot (\mathbf{x} - \mathbf{w}_i(t))$
    - Update importance map: $\mathcal{I}_i(t+1) = \mathcal{I}_i(t) + \alpha \cdot h_{ci}(t)$
    - Apply synaptic decay: $\mathbf{w}_i(t+1) = \mathbf{w}_i(t) - \delta \cdot \mathbf{w}_i(t)$
  - calculate: $\mathcal{T}_i(t) = \left(\frac{\mathcal{I}_i(t)}{\mathcal{A}_i(t))}\right)$
  - Determine positions for neurogenesis:

$$\mathbf{p}_{\text{new}} = \arg\max_i \mathcal{T}_i(t)$$

  - Apply forgetting criterion:

$$\text{Forget}_i = \begin{cases} 1 & \text{if } \mathcal{A}_i(t) < \theta_1 \text{ and } \mathcal{I}_i(t) < \theta_2 \\ 0 & \text{otherwise} \end{cases}$$

(3) **Feature Ranking:**
- Calculate feature importance score:

$$\text{FIS}(f_j) = \text{Var}\left(\{w_{ij} \mid i \in \mathcal{T}_{\text{top}}\}\right)$$

- Rank features based on FIS:

$$\text{RankedFeatures} = \text{argsort}_j\left(\text{FIS}(f_j)\right)_{\downarrow}$$

4 EXPERIMENTAL RESULTS

4.1 Dataset and Evaluation Method

The NPSOM algorithm is evaluated on the BRCA dataset [15], and the ROSMAP dataset w[16], both of which are multi-omics datasets. Shape of BRCA and ROSMAP data is (875 , 2503) and (351 , 600) and all experiments are done using k-folds methodology with a total of 5 folds. It is important to note that the accuracies reported in this paper are obtained after passing the selected features from NPSOM through a simple Proto Net model for classification.

Further, we utilize the accuracies obtained from this classifier to evaluate the NPSOM preformance of the BRCA and ROSMAP datasets (Figures 2 and 3). Evaluation is conducted after NPSOM ranks the features according to their importance. Following feature ranking, a select number of features are utilized for the downstream classification task conducted using the Proto Net as mentioned above.

The NPSOM model was configured using a 5×5 grid (i.e., `grid_size = (5,5)`), a learning rate of 0.1, and a decay rate of 1×10^{-5}. The model was trained with a batch size of 10 and 5 iterations per batch per epoch, for a total of one training epoch . These hyperparameters were selected based on preliminary experimentation to balance model accuracy and computational efficiency.

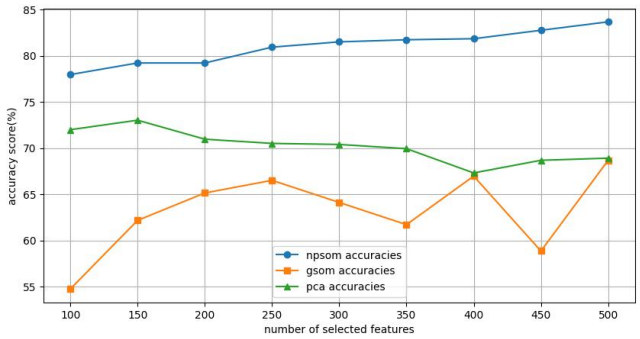

Figure 2: Accuracy plot comparing NPSOM, GSOM, and PCA across different feature counts of BRCA dataset.

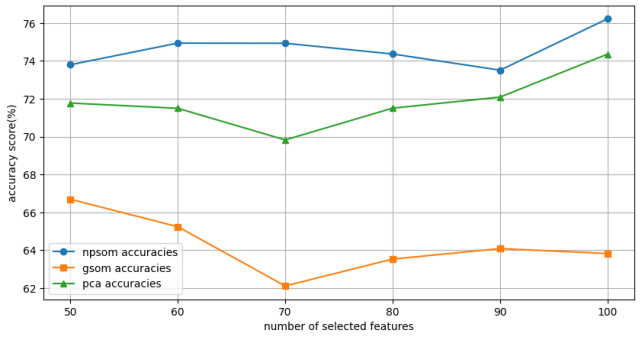

Figure 3: Accuracy plot comparing NPSOM, GSOM, and PCA across different feature counts of ROSMAP dataset.

4.2 Ablation Study

To assess the contribution of each neuroplasticity mechanism in NPSOM, we conducted an ablation study on the BRCA and ROSMAP datasets using 750 and 450 selected features

The following model variants were evaluated as seen in in Table 1 below.:

Model	BRCA Acc(%)	ROSMAP Acc (%)
Full Model	**87.07 ± 1.2**	**83.45 ± 1.6**
Without Neurogenesis	68.74 ± 4.3	72.07 ± 2.7
Without Forgetting	82.25 ± 5.2	74.32 ± 5.7
Without Synaptic Decay	83.62 ± 3.1	72.09 ± 4.4

Table 1: Accuracy comparison of NPSOM and its ablated variants on BRCA and ROSMAP datasets using 750 and 450 selected features, respectively.

The full NPSOM model achieved the highest accuracy of 87.07%, demonstrating the effectiveness of combining all neuroplasticity mechanisms. Disabling neurogenesis led to a drop in performance, with accuracy falling to 68.74%, underscoring the critical role of neurogenesis in adapting to complex data structures. Removing the forgetting mechanism resulted in an accuracy of 82.25%, suggesting its importance in mitigating overfitting. Disabling synaptic decay produced an accuracy of 83.62%, indicating that synaptic decay further aids in maintaining generalization by preventing overfitting to older data patterns.

4.3 Results

From Fig 2 and Fig 3, we see that NPSOM also surpasses other unsupervised feature selection methods like PCA , GSOM on the BRCA and ROSMAP dataset respectively. The neuroplasticity enables NPSOM to more effectively select the most important features. To further validate its robustness on diverse high-dimensional multi-omics data we compare it to SOTA methodologies with full model results from Table 1 to demonstrate its effectiveness (Table 2).

Method	BRCA Acc.	ROSMAP Acc.
KNN	0.783	0.651
RF	0.768	0.754
LASSO	0.772	0.755
XGBoost	0.791	0.764
MOGONET	0.806	0.800
SEGCN	0.840	0.792
MOSEGCN	0.867	0.830
NPSOM+Proto Net (Ours)	**0.8707 ± 0.012**	**0.8345 ± 0.016**

Table 2: Comparison of NPSOM to state-of-the-art methods [21] accuracies on BRCA and ROSMAP datasets.

5 CONCLUSION

This paper introduces the Neuroplastic Self-Organizing Map (NPSOM), a novel approach that enhances the traditional SOM with neuroplasticity features. The proposed method is evaluated on the BRCA and ROSMAP datasets, demonstrating its effectiveness in feature selection tasks and helping models tackle the curse of dimensionality. The incorporation of synaptic decay, neurogenesis, and memory forgetting allows NPSOM to mimic the brain's ability to adapt to new information while retaining important knowledge [2, 6, 12].

The experimental results show that NPSOM outperforms GSOM, PCA and SOTA methods by providing higher accuracy, making it a promising tool for real-world applications in machine learning and data analysis. The ablation study further highlights the importance of each neuroplasticity feature, demonstrating that their combined effect enables NPSOM to greatly improve its performance on high dimensional multi-omics data.

6 FUTURE WORK

One promising direction for future work is the incorporation of attention mechanisms into the NPSOM framework. Attention mechanisms have shown great success in various machine learning tasks by allowing the model to focus on relevant parts of the data [20]. By integrating attention into NPSOM, the model could potentially achieve even better results in feature selection tasks. Additionally, extending NPSOM to other types of data, such as time-series or

multimodal datasets, could open new avenues for research and application [8, 13].

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