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Dynamic analysis of synaptic loss and synaptic compensation in the process of associative memory ability decline in Alzheimer's disease

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ABSTRACT

The cognitive decline caused by Alzheimer's disease (AD) has a great impact on the life of patients and their families. Modern medicine has shown that loss of synaptic function is one of the causes of AD, and synaptic compensation compensates for cognitive abilities of the human brain. However, there are no studies on the internal mechanism of synaptic loss and synaptic compensation affecting human cognitive ability. In order to solve this problem, we propose here a three-layer neural network with multiple associative memory abilities, which is one of the main cognitive abilities. Based on synaptic plasticity, models of synaptic loss and synaptic compensation are established to study the pathogenesis of the degeneration of associative memory and explore feasible treatment approaches by setting different degrees of loss and compensation. Our simulation results show that the model can describe the associative memory ability at different stages of AD, which is of great significance for paramedics to determine the stage of disease and develop effective treatment strategies.

1. Introduction

Alzheimer's disease (AD) is a progressive neurodegenerative disease. It has become one of the greatest health care challenges of the 21st century because of the uncertainty of its pathogenesis and the lack of specific drugs to treat it [1–3]. At present, studies on AD mainly focus on biomarkers and brain magnetic resonance imaging. Mattsson found that plasma neurofilament light(NFL) can be used to monitor the efficacy of disease treatment drugs [4]. Sahar carried out a dynamic

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analysis on EEG of motor function changes in patients with AD [5]. These studies have made some progress in accurately distinguishing the stages of AD. However, Alzheimer's disease is a heterogeneous disease, and its pathogenesis is very complex. Therefore, it is feasible to study the pathogenic factors of Alzheimer's disease from the perspective of dynamic disease.

The main clinical feature of Alzheimer's disease is the decline of human brain cognitive ability [6,7]. As one of the cognitive abilities of human brain, associative memory is usually divided into self-associative, heteroassociative and multidirectional associative memory. Self-associative memory refers to the recovery from the damaged input pattern to the intact complete pattern itself. Heteroassociative memory refers to other memory patterns obtained by input pattern, while multidirectional associative memory obtains multiple memory patterns related to it by a single input pattern. In recent years, HNNs(Hopfield neural networks), BAMNNs(Bidirectional Associative Memory Neural Networks) and MAMNNs(Multidirectional Associative Memory Neural Networks) have been proposed to simulate single, bidirectional and multidirectional associative memory [8–10]. They are all classical dynamic models studied in associative memory neural networks. However, the current studies on associative memory mainly focus on the stability and dynamic behavior of neural networks [8–13], but there are few studies on brain diseases only. Therefore, from the perspective of dynamics, based on the network structure of MAMNNs, this paper constructe a neural network model with multiple associative memory abilities to explore the degree of decline of self-associative, heteroassociative and multi-directional associative memory in different stages of Alzheimer's disease from the perspective of dynamics.

Synaptic loss is the main neurobiological basis of cognitive dysfunction in Alzheimer's disease[14]. Adam measured widespread synaptic loss due to AD using [^{11}C]UCB-J PET [15]. In patients with mild cognitive impairment, synaptic loss can be detected, at the same time, synapses are also compensating for their own function [16]. It has been proved that amyloid beta protein($A\beta$) oligomerization can damage synaptic plasticity and neuronal activity, but its mechanism is still unclear. As the basis of learning and memory at the cellular level, synaptic plasticity can well explain the effects of synaptic loss and synaptic compensation on human learning and memory [17]. Chiayu explored multiple forms of long-term GABAgic synaptic plasticity and demonstrated that multiple parallel forms of plasticity contribute to increased stability and flexibility throughout the life cycle of an organism [18]. Kelly explored the synaptic plasticity of hippocampal CA1 and CA2 regions in connection with the learning and memory ability of human brain [19]. Therefore, based on synaptic plasticity, this paper studies the effects of different degrees of synaptic loss and synaptic compensation on human brain associative memory.

Gamma synchronous oscillation is a common phenomenon in the nervous system. The existing results have proved that gamma oscillation is the basis of higher brain functions (such as perception, attention and memory), and is very sensitive to metabolic and oxidative stress [20–22]. In 2020, Sitong Wang proposed that gamma neural oscillation analysis plays an important role in the early diagnosis of AD [23]. Based on the above rule, we intend to analyze the gamma-oscillation behavior of neural networks to identify the associative memory ability of the human brain. Then, based on the analysis results of associative memory, the disease stage of AD is judged.

Therefore, on the basis of previous work, we use dynamic modeling methods to create synaptic loss and synaptic compensation models. By analyzing the synchronous oscillation of neurons in neural networks, the effects of synaptic loss and compensation on the ability of multi-directional associative memory are studied.

The main research contents of this paper can be summarized as follows.

1. By establishing a neural network spiking model with multiple associative memories, the pathogenesis of AD is analyzed from the perspective of associative memories.
2. To simulate the different stages of AD, a synaptic loss model is established which determines the stages of AD through associative memory ability.
3. In order to seek for the treatment of AD, a synaptic compensation model is established on the basis of the synaptic loss model to study the recovery of associative memory ability of the neural network under different stages of compensation intensity.

The contents of the remaining chapters are as follows. In Section 2, the methods of constructing neural network model, synaptic loss model and synaptic compensation model are introduced in detail. In Section 3, simulation experiments are carried out to test the models we proposed. In Section 4, we summarize the work and give conclusions.

2. Methods

2.1. Structure of the neural network with multiple associative memory abilities

We propose a three-layer neural network model with multiple associative memory abilities. The neural network model is composed of 260 neurons, among which the number of neurons in the first, second and third layers is 80, 100 and 80 respectively. Neurons are connected by synapses, satisfying the principle of full interconnection between layers and non-interconnection within layers, and each neuron considers other external current input. The structure of the neural network is as follows. The Izhikevich neuron model can mimic the spiking of various neurons by adjusting parameters, which is suitable for a large-scale neuronal network construction [24–27]. So we use this neuron model to simulate real neurons in

the human brain. The structure of the neuron model is as follows:

$$\begin{aligned} v' &= 0.04v^2 + 5v + 140 - u + I, \\ u' &= a(bv - u), \\ \text{if } v &\geq 30mv,\ v \leftarrow c,\ u \leftarrow u + d, \end{aligned} \tag{1}$$

where v represents the membrane voltage of neurons, u the recovery variable of membranes, which is used to describe the activation of potassium ion current and the inactivation of sodium ion current. u provides negative feedback to v. When the pulse reaches 30 millivolts, the membrane voltage and the recovery variables are reset. The synaptic current or the injected direct current flow is given through the variable I. a describes the time range of the recovery variable u; b the sensitivity of the recovery variable u to the membrane voltage v; c the resting value of the membrane voltage v caused by the fast high threshold potassium ion conductance and d the resting value of the recovery variable u caused by slow, high threshold conductance of sodium and potassium ions.

2.2. Synaptic model

The classic rule describing synaptic plasticity is the Hebbian learning rule [28–31]. Here we use an improved Hebbian learning rule to simulate the weight changes of synapses. The synaptic model is shown as follows:

$$w_{ij} = \frac{\sigma}{Na_0(1-a_0)} \sum_{\mu=1}^{p} \left(\xi_i^\mu - a_0\right)\left(\xi_j^\mu - a_0\right), \tag{2}$$

in which σ is the connection strength, related to the degree of the synaptic loss. $N = 260$ represents the total number of neurons in the neural network. a_0 is the mean value of the memory sequence, p the total number of the memory patterns, μ the serial number of the memory patterns, and ξ_i^μ, ξ_j^μ (i,j=1,2,3) are the memory patterns of neurons in the ith and jth layer.

Synaptic loss does not only reduce synaptic plasticity, but also affects the associative memory ability. By setting the degree of synaptic loss $\Delta\sigma$, $(0 \leq \Delta\sigma < 1)$, we simulate the decline of the associative memory ability with synaptic loss. The synaptic weight in the synaptic loss situation can be updated as follows.

$$w_{ij} = \frac{\sigma - \Delta\sigma}{Na_0(1-a_0)} \sum_{\mu=1}^{p} \left(\xi_i^\mu - a_0\right)\left(\xi_j^\mu - a_0\right). \tag{3}$$

On this basis, synaptic compensation can compensate the normal function of the nervous system caused by synaptic loss to a certain extent. The compensation strategy is implemented by multiplying the synaptic weight by the common factor $c = f(\Delta\sigma)$ and it is defined as

$$c = 1 + \frac{\Delta\sigma k}{1 - \Delta\sigma}, \tag{4}$$

in which $k(0 < k \leq 1)$ is the compensation intensity. The synapse weight after the compensation is as follows.

$$w_{ij} = c\frac{\sigma - \Delta\sigma}{Na_0(1-a_0)} \sum_{\mu=1}^{p} \left(\xi_i^\mu - a_0\right)\left(\xi_j^\mu - a_0\right). \tag{5}$$

2.3. Neural network model

Combined with the established neuronal and synaptic models, the final three-layer neural network model is determined as follows.

The membrane voltage of a single neuron in the first layer of the neural network is as follows.

$$\begin{cases} v_{1i}'(t) & = 0.04v_{1i}^2(t) + 5v_{1i}(t) + 140 - u_{1i}(t) + \gamma_1 I_{1i}(t) \\ & \quad + \sum_{j=1}^{q} W_{2ij}\left(v_j - \theta_j\right) + \sum_{l=1}^{k} W_{3il}(v_t - \theta_l) + \xi_{1i}(t), \\ u_{1i}'(t) & = a(bv_{1i}(t) - u_{1i}(t-1)), \\ v_{1i} \geq 30 & = \begin{cases} v_{1i} = c, \\ u_{1i} = u_{1i} + d'. \end{cases} \end{cases} \tag{6}$$

The membrane voltage of a single neuron in the second layer of the neural network is as follows.

$$\begin{cases} v_{2j}'(t) & = 0.04v_{2j}^2(t) + 5v_{2j}(t) + 140 - u_{2j}(t) + \gamma_2 I_{2j}(t) \\ & \quad + \sum_{i=1}^{p} W_{1ji}(v_i - \theta_i) + \sum_{l=1}^{k} W_{3jl}(v_l - \theta_l) + \xi_{2j}(t), \\ u_{2j}'(t) & = a\left(bv_{2j}(t) - u_{2j}(t-1)\right), \\ v_{2j} \geq 30 & = \begin{cases} v_{2j} = c, \\ u_{2j} = u_{2j} + d'. \end{cases} \end{cases} \tag{7}$$

Table 1
Table of relevant parameters of neural network.

Parameter	Value
a	0.02
b	0.06
c	−65
d	2
p	80
q	100
k	80
s	1
μ	1
γ	20

The membrane voltage of a single neuron in the third layer of the neural network is as follows.

$$\begin{cases} v'_{3l}(t) & = 0.04v_{3l}^2(t) + 5v_{3l}(t) + 140 - u_{3l}(t) + \gamma_3 I_{3l}(t) \\ & \quad + \sum_{i=1}^{p} W_{1li}(v_i - \theta_i) + \sum_{j=1}^{q} W_{2lj}\left(v_j - \theta_j\right) + \xi_{3l}(t), \\ u'_{3l}(t) & = a(bv_{3l}(t) - u_{3l}(t-1)), \\ v_{3l} \geq 30 & = \begin{cases} v_{3l} = c, \\ u_{3l} = u_{3l} + d', \end{cases} \end{cases} \tag{8}$$

in which $\xi_i(t)\xi_m(t') = D\delta_{im}\delta(t-t')$, $\xi_j(t)\xi_n(t') = D\delta_{jn}\delta(t-t')$, $\xi_l(t)\xi_k(t') = D\delta_{lk}\delta(t-t')$, where i, j, k are the serial numbers of neurons in the first, second, and third layers respectively, and m, n, l are the serial numbers of neurons in the first, second, and third layers apart from neuron i, j, and k respectively. $v_i(t)$ is the membrane voltage of the ith neuron at time t. $u_i(t)$ is the recovery variable of neuronal membrane voltage, which provides negative feedback for membrane potential. γ_i is the current intensity of the external current of the ith neuron. $I_i = x_i\theta(t)$, $(x_i \in 0, 1)$ is the external input current of the ith neuron. $\xi(t)$ is autocorrelation Gaussian white noise with the intensity D. θ is the neuron threshold, which ranges from -70mv to -65mv. $\Theta(t)$ is a Heaviside function. $x_i = 1$ means the neuron has an external current input, $x_i = 0$ means no input.

3. Results

In this section, we use the parameter values as shown in Table 1 to study the voltage variations of the neuron membrane in the neural network within 40ms. It reveals the changes of associative memory in the human brain under normal and different degree of synaptic loss and synaptic compensation. According to the above, different degrees of synaptic loss are judged corresponding to the disease stage of AD, so as to achieve the purpose of analyzing the pathogenesis of AD from a pathological aspect.

To make our analysis of the associative memory ability of neural networks more accurate, we create a relationship between the phenomenon of synchronous oscillation and associative memory ability. The ability of associative memory is evaluated by judging the synchronous oscillation behavior in the current network. The initial memory pattern stored in the neural network is: $\xi_1(30 \leq i \leq 50, \xi_{1i} = 1; else, \xi_{1i} = 0)$, the second layer can be written as $\xi_2\left(30 \leq i \leq 70, \xi_{2j} = 1; else, \xi_{2j} = 0\right)$, the third $\xi_3(40 \leq i \leq 60, \xi_{3l} = 1; else, \xi_{3l} = 0)$. The stored memory exists in the form of synchronous oscillation of neurons within a certain range. For example, the memory stored in the first layer exists in the form of synchronous oscillation from the 30th to the 50th neurons.

3.1. Associative memory ability under normal conditions

3.1.1. Self-association and heteroassociative memory ability

In order to test the self-associative ability of the neural network, a damaged memory pattern is introduced into the first layer of the neural network. If the final output is consistent with the stored memory, then the self-associative ability of the neural network is proved. In addition, in order to test the ability of the heteroassociative memory, we only input the memory pattern of the first and second layers. If the output of the last third layer is consistent with the storage memory, we can conclude that the neural network has the ability of heteroassociative memory. In our simulations, we define the input pattern as follows: the first layer is $\xi_1(35 \leq i \leq 45, \xi_{1i} = 1; else, \xi_{1i} = 0)$. The second layer remains unchanged, and the third layer is $\xi_3 = 0$. Fig. 1 shows the membrane voltage scatter plots of the first and third layers of the neurons.

Different colored points in Fig. 2 correspond to different values of neuronal membrane voltages. It can be seen from Fig. 2(a) that the synchronous oscillation of the first layer of neurons occurs within the range of neurons 30–50. Therefore, it can be concluded that the neural network has the ability of self-associating memory, which can associate the damaged memory with the complete memory. Although there is no input memory sequence in the third layer, neurons 40–60 in

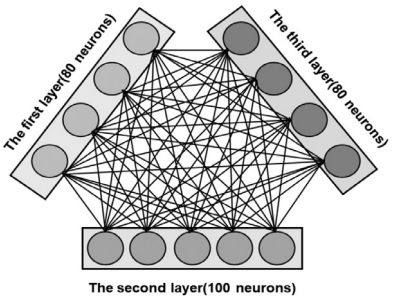


Fig. 1. The structure of the neural network.

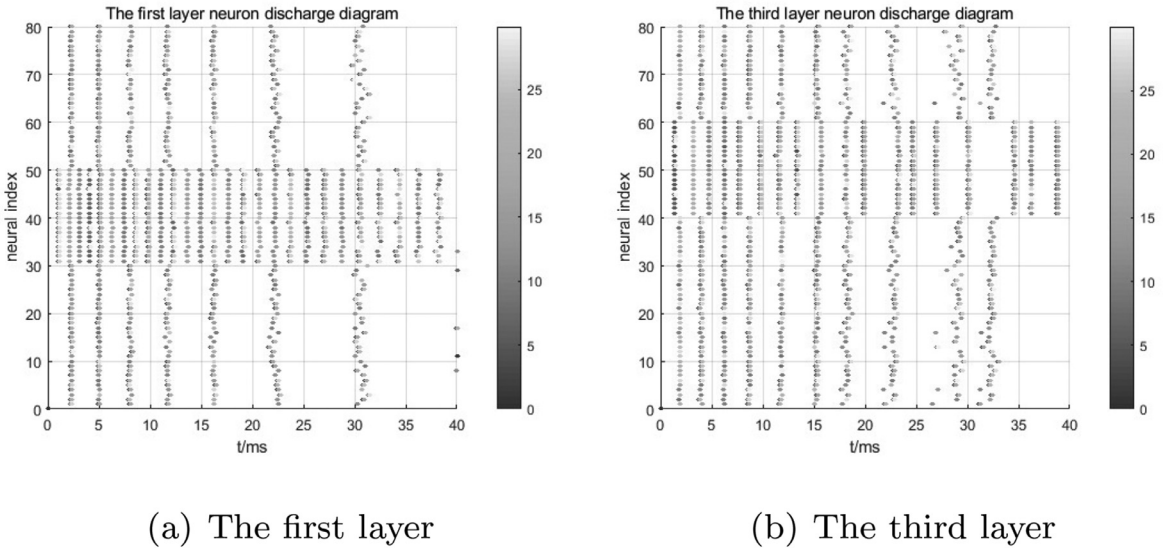


Fig. 2. Voltage scatter diagram of the first and third layers of neuron membrane. Note that the 30th–50th neurons in the first layer and the 40th–60th neurons in the third layer show obviously the synchronous oscillation phenomenon.

the third layer still oscillate synchronously, so the neural network we established can realize the ability of heteroassociative memory.

3.1.2. Multidirectional associative memory ability

The multidirectional associative memory ability of the neural network can be tested by inputting a memory pattern only to the first layer. At this point, the input memory pattern of the first layer is updated to $\xi_1 = 0$, the second layer is the same as before, and the third layer is updated to $\xi_3 = 0$. Fig. 3 shows the scatter plot of the membrane voltage of the second and third layer neurons.

Within 40ms, when only the second layer neurons of neural network were input with memory pattern, the synchronous oscillation of neurons in the third and third layers still occurred within a certain range. The membrane voltage values of the 30th–50th neurons in the first layer are consistent within 40ms, and the peak spiking behavior is denser than other ranges, so it can be concluded that the synchronous oscillation phenomenon occurs in the first layer. The same phenomenon occurred from the 40th to the 60th neurons of the third layer. The simulation results show that the neural network can simulate the multidirectional associative memory. Without losing generality, the 40th and the 70th neuron in the first layer of the neural network are selected to analyze the discharge law of neurons in the verification of self-association and heteras-sociation abilities. In Fig. 4, both neurons showed obvious peak and cluster spiking behaviors within 40ms, and the spiking behavior of the 40th neuron was significantly different from that of the 70th neuron, and the discharge frequency and peak value of the 40th neuron were significantly higher than those of the 70th neuron. From another point of view, the difference between the neurons with synchronous oscillation and those without synchronous oscillation is proved.

The range of gamma rhythms is approximately 25–100hz. For the ability of self association and heteroassociative Memory, the number of discharges per 200ms in Fig. 5 is 9 and 14, and the discharge frequency is 45hz and 70hz, respectively, for the ability of multi-directional associative memory, the number of discharges is 17 and 11, and the discharge frequency is 85 hz and 55 hz. This shows that all oscillation bands in this study are gamma oscillations, which is closely linked with the human brain learning and memory ability.

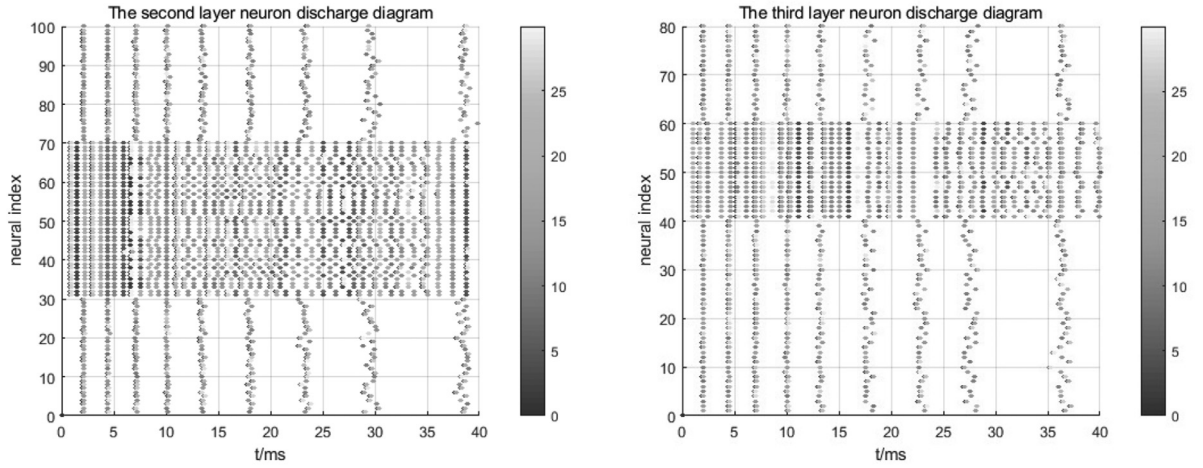


Fig. 3. Voltage scatter plots of the first and third layers of neuron membranes.Note that there are obvious synchronous oscillations between neuron 30th–50th in the first layer and neuron 40–60 in the third layer.

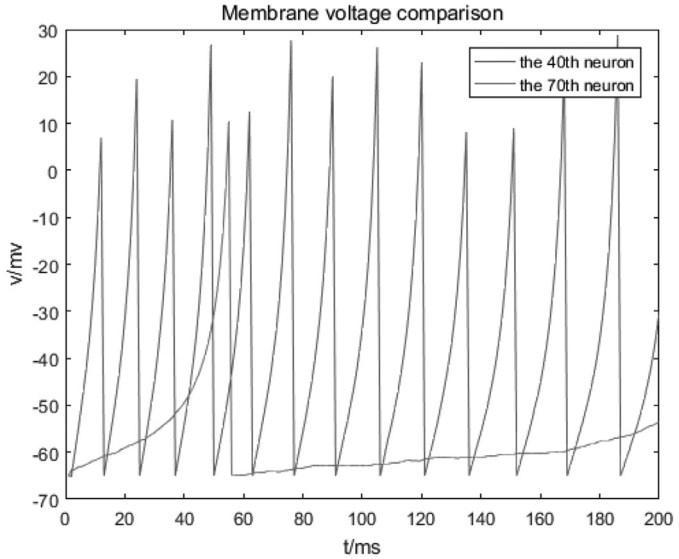


Fig. 4. Changes of membrane voltage of neurons 40 and 70 in the first layer of the neural network.

3.2. Associative memory ability under synaptic loss

Different degrees of synaptic loss will lead to different degrees of associative memory degradation, leading to different degrees of AD. Hence, establishing a dynamic model of synaptic loss to simulate the associative memory ability becomes an effective way to explore the pathogenesis of Alzheimer's disease. We tested the associative memory capacity of the neural network under the three conditions of synaptic loss degree $\Delta\sigma = 0.3, 0.5.0.8$. To maintain consistency, the memory pattern of the input neural network is the same as under normal conditions.

3.2.1. Self-association and heteroassociative memory ability

The membrane voltage changes of the first and third layers of neurons within 0-40ms are shown in Fig. 6.

We can see from above the above figture that, with the increase of synaptic loss intensity, the oscillatory behavior of the 30th–50th neurons in the first layer within 40ms gradually become consistent with that of neurons in other ranges. This phenomenon indicates that the memory stored in the first layer is gradually disappearing. The self-association ability of the first layer decreases with the strength of synaptic loss, which corresponds to the phenomenon that damaged memories are

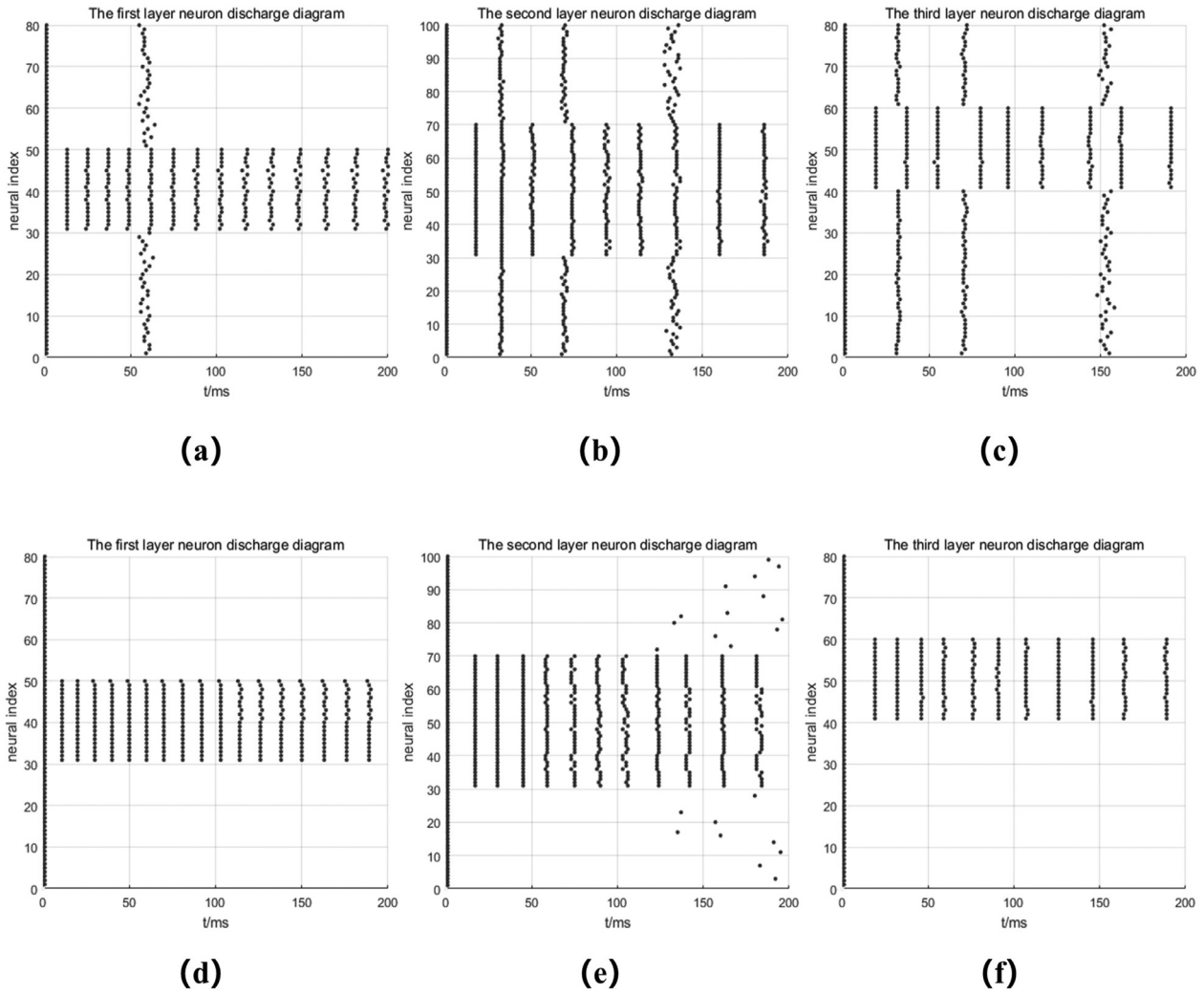


Fig. 5. The scatterplot of neuron spiking in each layer in neural network. (a)(b)(c) is the scatter diagram of the peak spiking time of neurons in each layer under the condition of self-associative and heteroassociative ability test; (d)(e)(f) is the scatter diagram of neuron peak spiking moment of each layer under the condition of multidirectional associative memory ability test.

hard to remember. Similarly, in the absence of any memory pattern input in the third layer, the synchronous oscillation of neurons in a certain range still occurs in the third layer, which is similar to the synchronous oscillation of neurons in the first layer. With the increase of the strength of synaptic loss, the synchronous oscillation effect weakens. Therefore, with the enhancement of synaptic loss intensity, the self-associative and heterassociative memory abilities of the neural network are gradually lost.

3.2.2. Multidirectional associative memory ability

The membrane voltage changes of the first and third layers of neurons within 40ms are shown in Fig. 7. Under the condition that only the second layer output memory pattern, the synchronous oscillation of neurons occurred in the first and third layers. However, with the increase of the intensity of synaptic loss, the oscillatory behaviors of the 30–50 neurons in the first layer gradually tend to be consistent with those in other ranges, and the oscillatory behaviors of the neurons in the third layer also tend to be consistent. This indicates that it is difficult to associate with the memory pattern already stored in each layer through the multi-direction associative memory ability of the neural network, and the multi-direction associative memory ability of the neural network decreases. With the increase of synaptic loss intensity, the multidirectional associative memory ability of neural network gradually decreases.

The effect of synaptic loss on the voltage of a single neuron's discharge membrane is shown in the Fig. 8. Taking self-associative and heterassociative memory ability as an example, it can be concluded that with the increase of synaptic loss intensity, the frequency of neurons within the range of memory pattern decreased gradually, while the firing frequency of the neurons outside the range of memory pattern increased gradually, and they tended to be consistent gradually. The

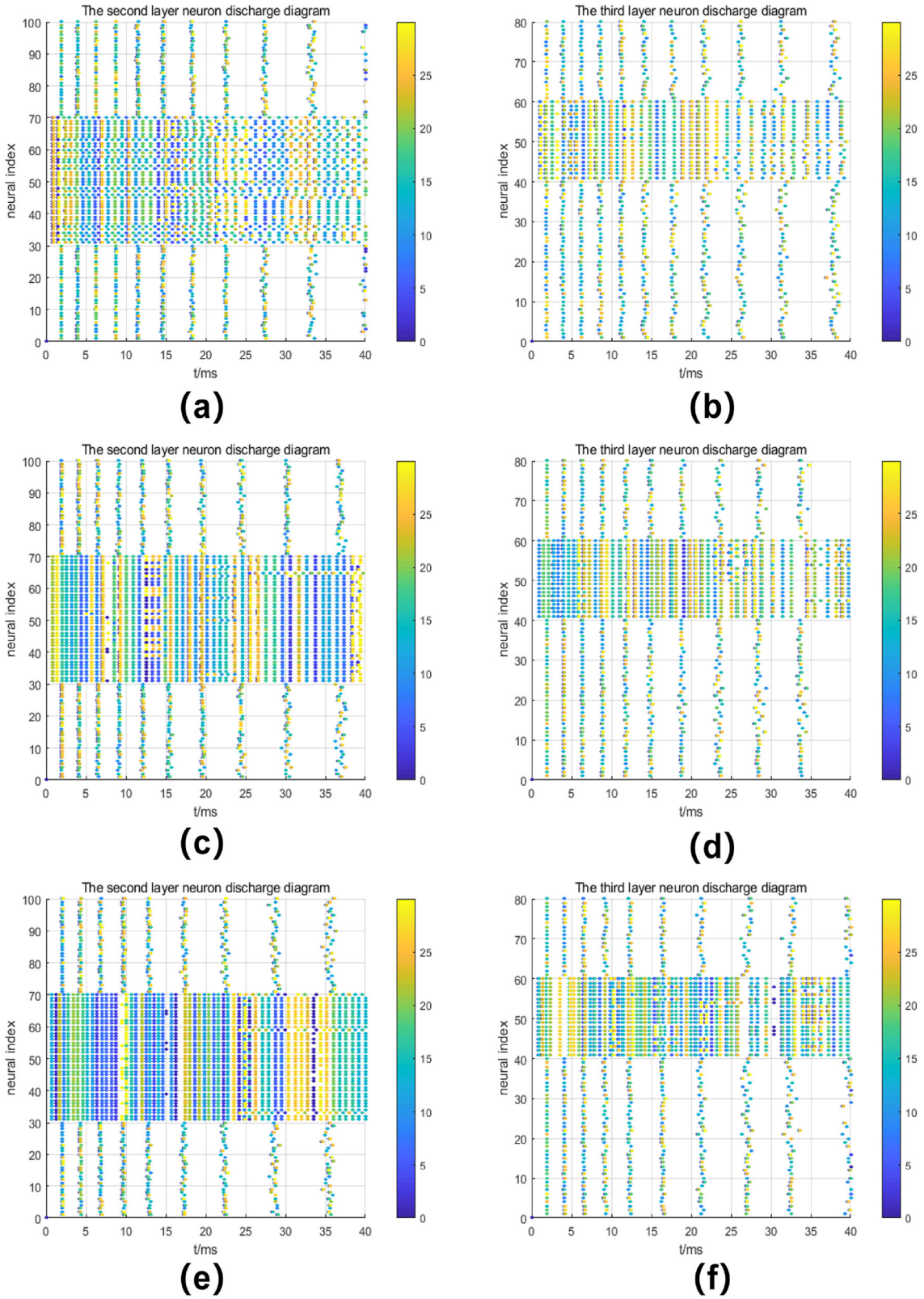


Fig. 6. The membrane voltage scatter plots of the first layer and the third layer neurons with different degrees of synaptic loss. (a)(c)(e) is the voltage scatter diagram of the first layer of the neuron membrane and the loss degree $\Delta\sigma = 0.3, 0.5, 0.8$; (b)(d)(f) is the membrane voltage scatter plot of the third layer of neurons and the loss degree $\Delta\sigma = 0.3, 0.5, 0.8$ respectively.

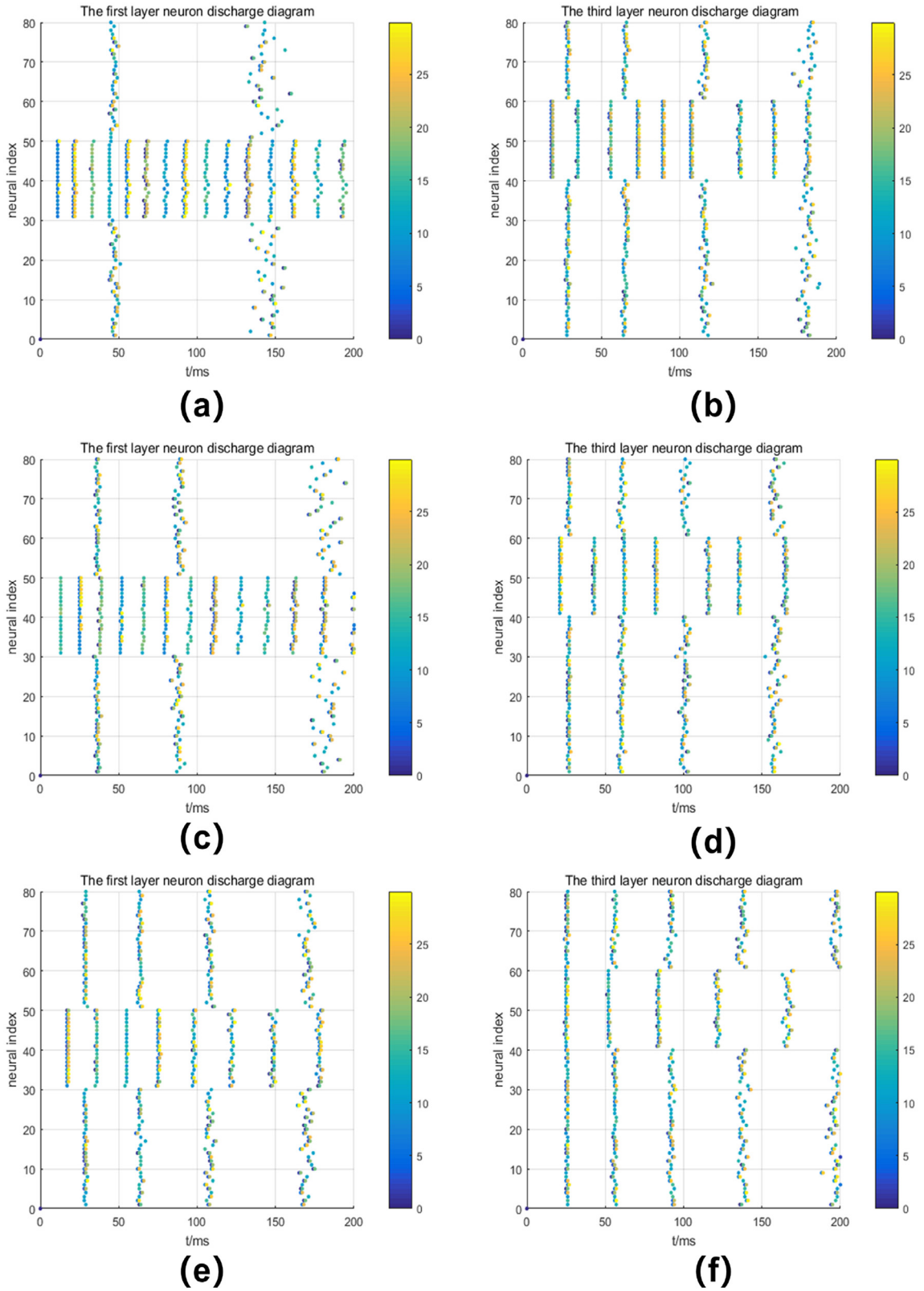
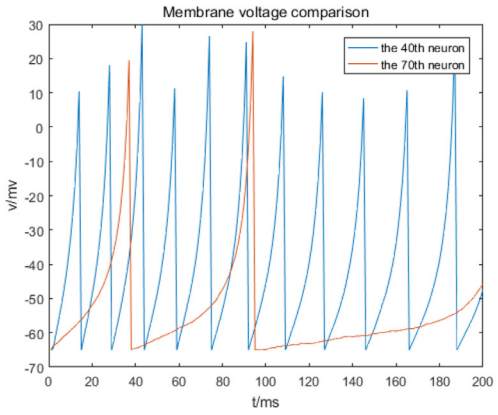
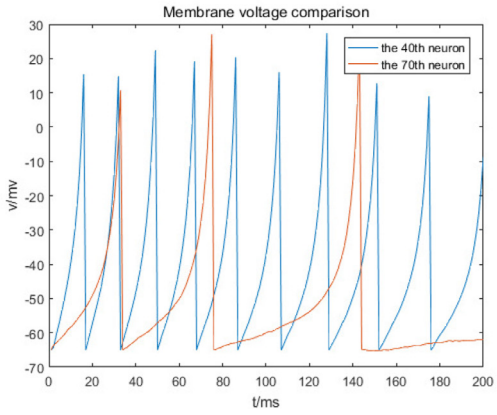


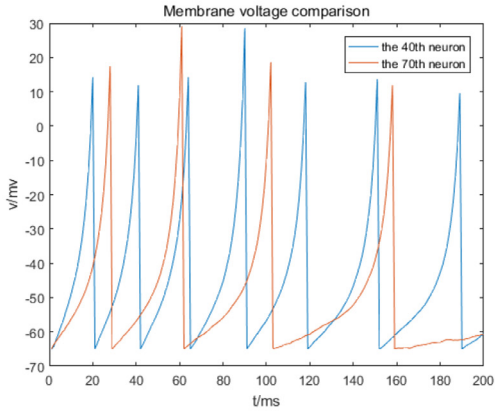
Fig. 7. The membrane voltage scatter plots of the first layer and the third layer neurons with different degrees of synaptic loss. (a)(c)(e) is the voltage scatter diagram of the first layer of the neuron membrane and the loss degree $\Delta\sigma = 0.3, 0.5, 0.8$; (b)(d)(f) is the membrane voltage scatter plot of the third layer of neurons and the loss degree $\Delta\sigma = 0.3, 0.5, 0.8$ respectively.



(a) $\Delta\sigma = 0.3$



(b) $\Delta\sigma = 0.5$



(c) $\Delta\sigma = 0.8$

Fig. 8. Changes of membrane voltage of neurons 40th and 70th in the first layer with $\Delta\sigma = 0.3, 0.5.0.8$.

synchronous oscillation of neurons in the memory pattern was weakened and the associative memory ability gradually disappeared.

Fig. 9 shows the associative memory performance of the neural network under different conditions of synaptic loss. The abscissa is the strength of the connection between the synapses, and the ordinate is the similarity between the stable state of the network and the stored memory. With the decrease of the synaptic connection strength, the associative memory performance of the neural network showed an obvious trend of decline, and basically lost associative memory ability when the connection strength dropped to 0.3.

3.3. Associative memory ability under synaptic compensation

It can be seen from the previous section that when the intensity of synaptic loss is 0.8, the network basically loses the association ability. Synaptic compensation can restore the associative memory ability of neural network. The neural network with $\Delta\sigma = 0.8$ was selected to test the association ability of the neural network with synaptic compensation under three compensation intensities of $k = 0.3, 0.5, 0.8$ respectively.

3.3.1. Self-association and heteroassociative memory ability

The changes of membrane voltage in the first and third layers of neurons under different synaptic compensation intensities within 40 ms are shown in Fig. 10.

Under the condition of $\Delta\sigma = 0.8$, with the enhancement of synaptic compensation intensity, the synchronous oscillation behavior of neurons in the first and third layers of the neural network becomes more and more obvious. The self-associative

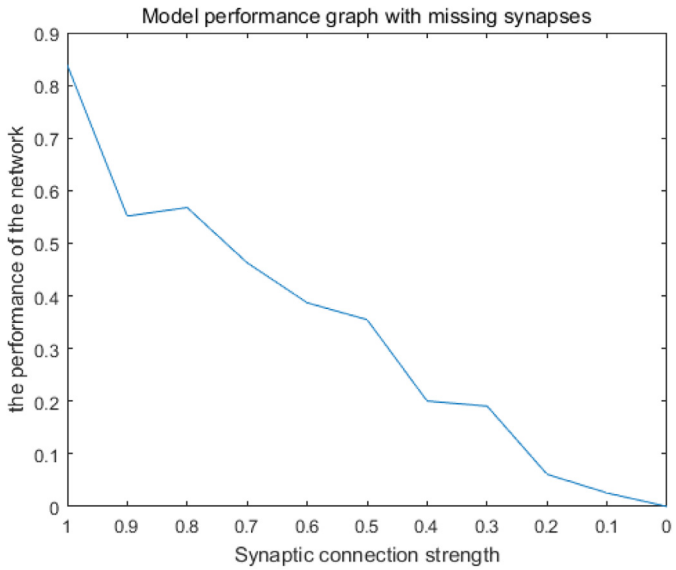


Fig. 9. The influence of synaptic connection strength on associative memory ability of the neural network.

and heteroassociative memory ability of the neural network recovered gradually, and basically recovered for $k = 0.8$. The neural network can well simulate the effects of synaptic compensation when recovering the self-associative and heteroassociative memory after synaptic loss.

3.3.2. Multidirectional associative memory ability

The changes of membrane voltage in the first and third layers of neurons under different synaptic compensation intensities within 40 ms are shown in Fig. 11.

Under the condition of the $\Delta\sigma = 0.8$, with the increase of synaptic compensation strength k, the synchronous oscillation behavior of neurons in the first and third layers of the neural network becomes more and more obvious. The multidirectional associative memory ability of the neural network gradually recovered, and basically recovered when $k = 0.8$. It shows that the neural network can simulate the recovery of associative memory by synaptic compensation after different degrees of synaptic loss.

When the synaptic loss intensity is 0.8, as shown in Fig. 12. With the gradual increase of the synaptic compensation intensity, the spiking frequency of neurons within the memory mode gradually increases and the synchronous oscillation effect is more obvious. The spiking frequency of neurons outside the memory pattern decreases gradually. The associative memory ability of neural network recovers gradually. When testing the multi-directional associative memory ability of neural network, the 40th and 70th neurons in the first layer of neural network were selected, and their membrane voltage change law can verify the rule: with the increase of synaptic strength of compensation, the associative memory neural network ability recover gradually, side validation, synaptic compensation ability of inhibition of associative memory deterioration in patients with AD.

4. Conclusion

In order to solve the problem that the pathogenesis of Alzheimer's disease is not clear and there is no specific drug treatment, we set up a neural network model with multiple associative memory abilities to simulate Alzheimer's disease with different degrees from the perspective of the degeneration of associative memory caused by Alzheimer's disease. Considering that synaptic loss is the cause of the decline of associative memory ability of neural network, and a certain degree of synaptic compensation can improve the associative memory ability after synaptic loss. Synaptic loss and synaptic compensation models are constructed to simulate the normal, synaptic loss and synaptic compensation of self-association, heteroassociation and multidirectional associative memory in human brain. The results clearly show that the neural network is biointerpretable and can simulate many kinds of associative memory abilities of the human brain under healthy and non healthy conditions. The establishment of the network provides ideas for the development of intelligent algorithms with high robustness, and lays a good foundation for the early prediction and symptomatic treatment of AD.

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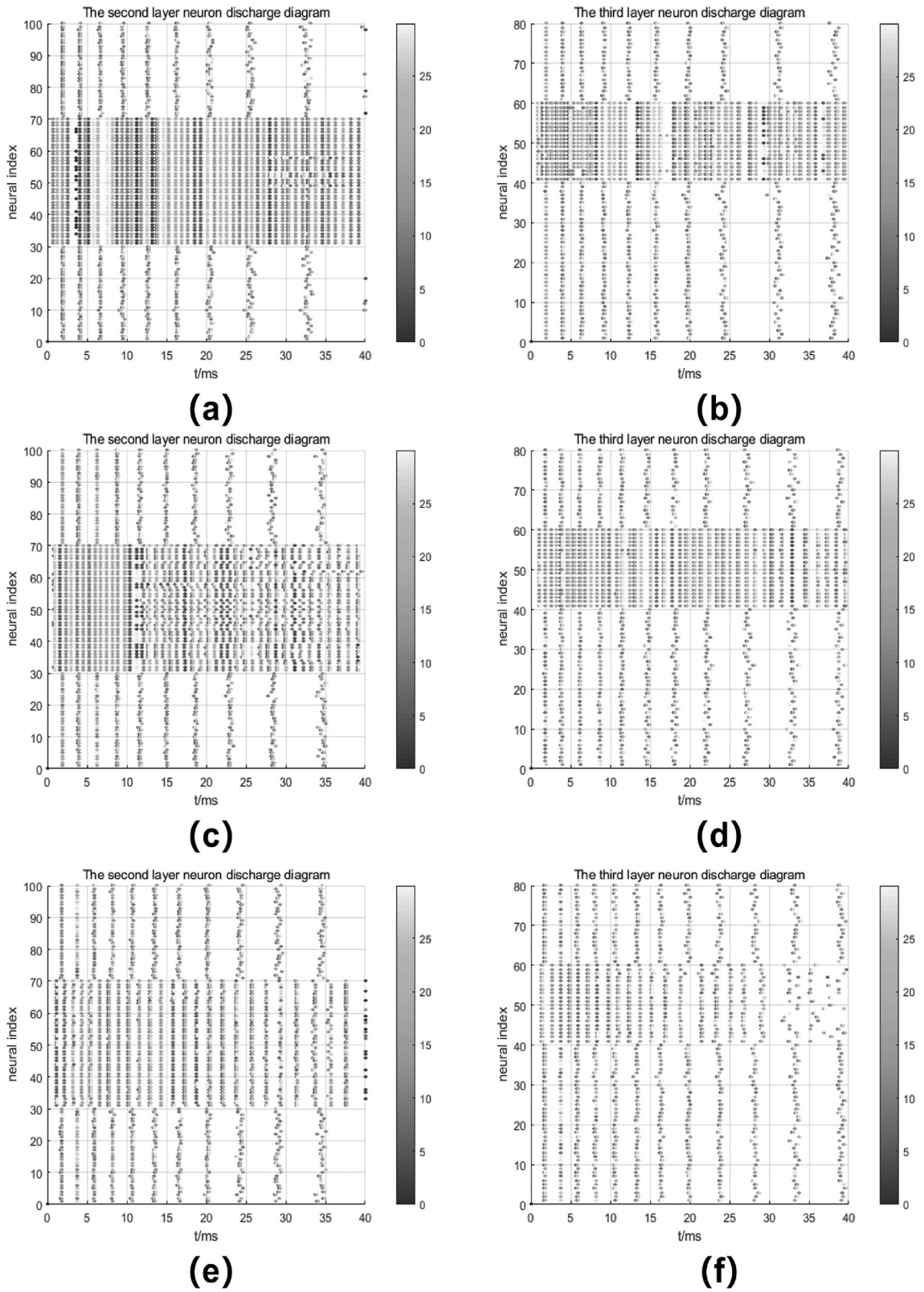


Fig. 10. The membrane voltage scatter plots of neurons in the first layer and the third layer under different degrees of synaptic compensation when the degree of synaptic loss is 0.8. (a)(c)(e) is the voltage scatter diagram of the first layer of the neuron membrane at the compensation degree of $k = 0.3, 0.5, 0.8$; (b)(d)(f) is the voltage scatter diagram of the third layer of the neuron membrane at the compensation degree of $k = 0.3, 0.5$ and 0.8.

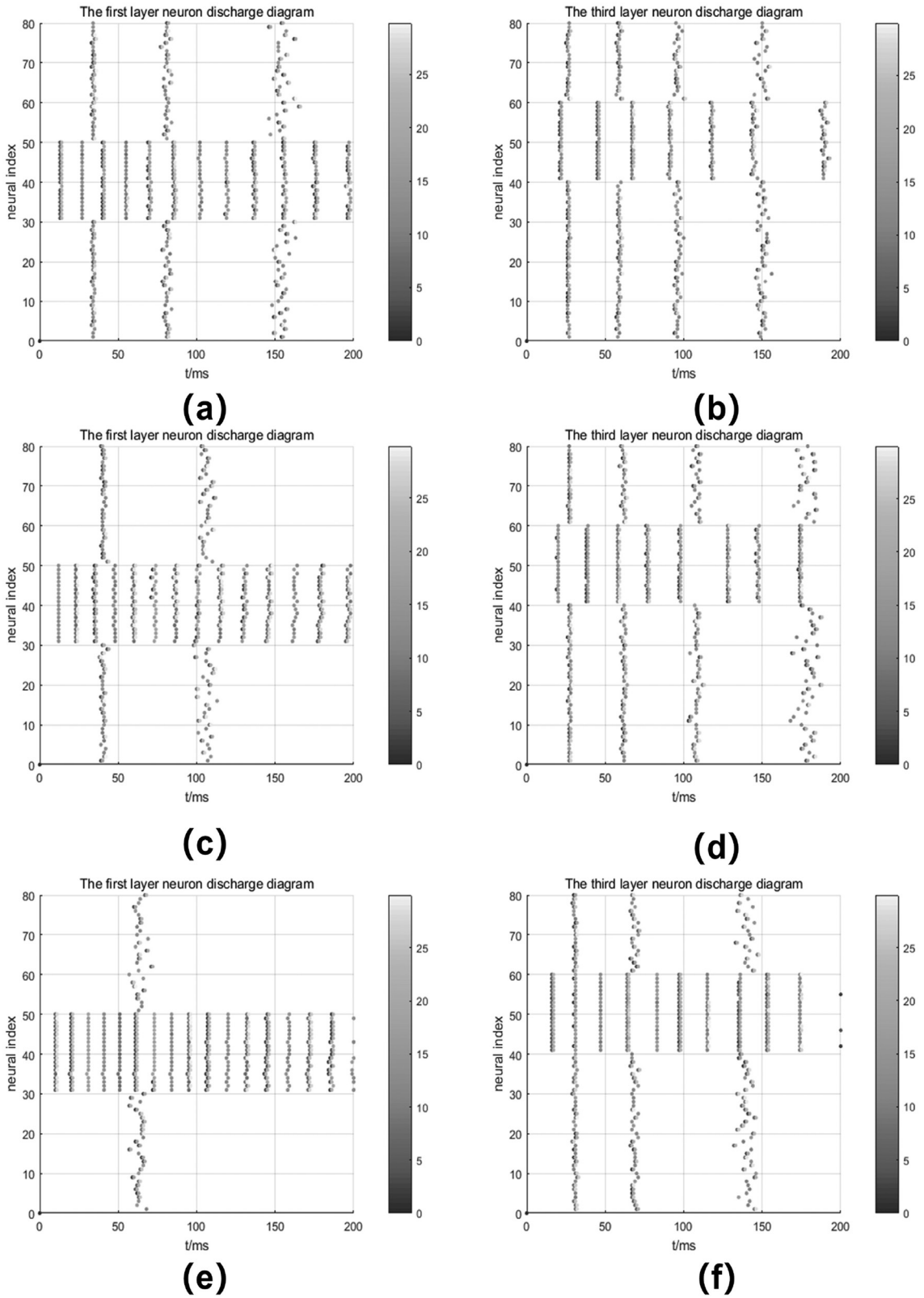
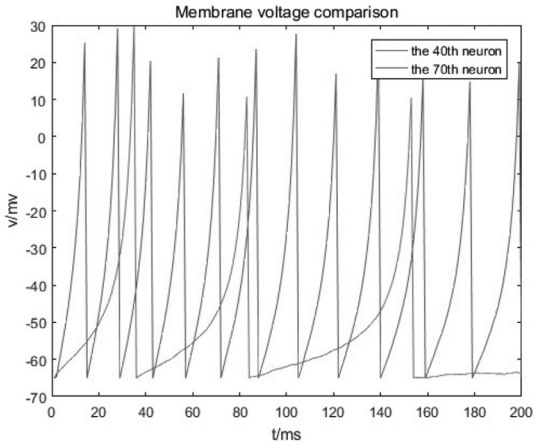
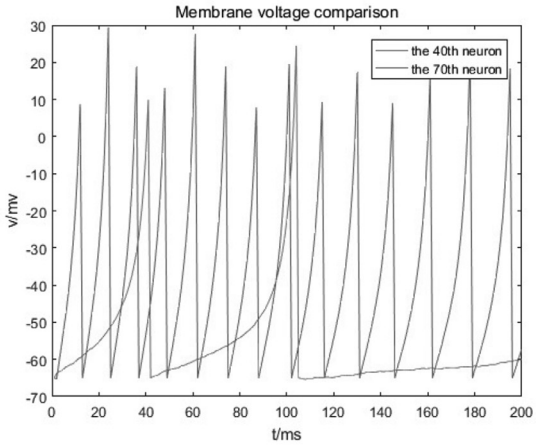


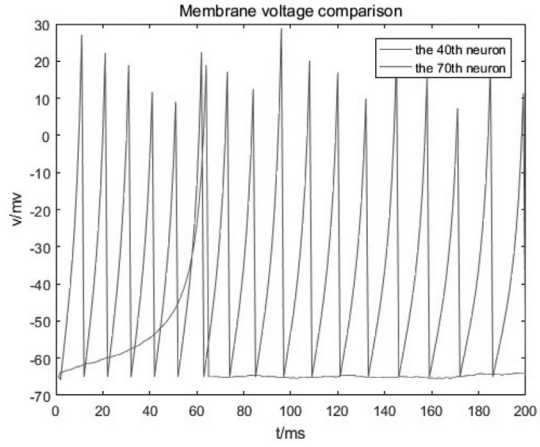
Fig. 11. The membrane voltage scatter plots of neurons in the first layer and the third layer under different degrees of synaptic compensation when the degree of synaptic loss is 0.8. (a)(c)(e) is the voltage scatter diagram of the first layer of the neuron membrane at the compensation degree of $k = 0.3, 0.5, 0.8$; (b)(d)(f) is the voltage scatter diagram of the third layer of the neuron membrane at the compensation degree of $k = 0.3, 0.5, 0.8$.



(a) $\Delta\sigma = 0.8\ k = 0.3$



(b) $\Delta\sigma = 0.8\ k = 0.5$



(c) $\Delta\sigma = 0.8\ k = 0.8$

Fig. 12. Changes of membrane voltage of neurons 40th and 70th in the first layer with $\Delta\sigma = 0.8\ k = 0.3, 0.5, 0.8$.

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