Plasticity Neural Network Based on Astrocytic effects at Critical Period, Synaptic Competition and Strength Rebalance by Current and Mnemonic Brain Plasticity and Synapse Formation

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In brief: The mechanism of our proposed Neural Network (NN) is very well in line with the results of the latest MIT brain plasticity study, in which researchers found that as a synapse strengthens, neighboring synapses automatically weaken themselves to compensate [14]. Nevertheless, our Neural Network treats synapses as a group of intelligent agents to achieve synaptic strength rebalance. And Dr. Luo’s team at Stanford University has put forward that competition regarding synapse formation for dendritic morphogenesis is crucial [15]. In tests, possible explanations of dendrite morphogenesis are derived, which demonstrate that dendrite generation, to a certain extent, is curbed by synapse formation, also reflected PNN simulates synapse formation [15]. The paper’s analysis suggests retrograde mechanism, was reflected in PNN through retrograde retrieval mnemonic gradient of synaptic effective range weight [16]. The decrease of neurogenesis was considered throughout aging. And PNN simulates synapse formation, decrease of neurons at the same time [17]. But controversy was claimed that human hippocampal neurogenesis persists throughout aging, PNN considered it may have a new and longer circuit in late iteration [18]. We try to conduct research on the mechanism of failure in brain plasticity by model at the closure of critical period in details by contrasting with studies before [19]. Current and mnemonic brain plasticity as well as synaptic effective range are also taken into account in the study. By simulations, the PNN gave the two cases with and without negative memories persistence. And the paper proposed α7-nAChR-dependent astrocytic responsiveness is an integral part of the cellular substrate underlying memory persistence by impairing fear memory[20]. Mnemonic brain plasticity involves the plus or minus disturbance-astrocytic brain plasticity phagocytic
synapses factor. The effect of astrocyte made local synaptic effective range remain in an appropriate length at critical period[21]. Both negative and positive memories can be reflected in brain cortex, and its simulations fits very well with experiments in the relationship of human intelligence and cortical thickness, individual differences in brain[22]. PNN also considered the memory engram cells that strengthened Synaptic strength, and memory engram cells are helpful to the memory retrieval process[23]. And paper found that 1064-nm Transcranial photobiomodulation (tPBM) applied to the right prefrontal cortex (PFC) improves visual working memory capacity[24], the effect of PNN (memory structure) may be the same with smaller losses in signals. The Dr. Christian Kerskens and David López Pérez suggests consciousness is non-classical [25], so PNN introduces mnemonic brain plasticity by quantum computing in retrograde mechanism, non-classical brain plasticity in extracted relatively good or inferior memories. Through the PNN tests, we found that the astrocytic memory persistence factor, like the astrocytic phagocytic synapses factor, produces the effect of reducing the local accumulation of synapses. Considering both negative and positive memories persistence help activate synapse length changes, and therefore yields better results than considering only positive memory persistence. In the calculation to update the synaptic effective range, the PNN in which only the synaptic phagocytosis effect is considered regardless of the gradient update, proves simple and effective. Cutting edge imaging and genetic tools are combined in their experimental studies, whereas our research lays more emphasis on the model, derivation and simulation of a new NN. Therefore, is it possible to reduce the number of animal experiments and their suffering by simulating and planning the factors of biological experiments through Deep Learning?

Summary: Beyond the consideration of the basic connection, this research focuses on a new Neutral Network (NN) model, referred as PNN (Plasticity Neural Network) in which the current and mnemonic effective range between synapses and neurons plastic changes with iterations, are both taken into account at the same time, specifically, the current and mnemonic synaptic effective range weights.

In addition, this mechanism of synaptic strength rebalance is consistent with the findings in the recent research on brain plasticity at the MIT Picower Institute. Regarding the importance of this mechanism [14], our Neural Network is, it treats synapses as a group of intelligent agents to achieve synaptic strength rebalance. Dr. Luo’s team at Stanford University has mentioned that the competition regarding synapse formation for dendritic morphogenesis is important, also reflected PNN simulates synapse formation[15]. We try to examine the mechanism of failure in brain plasticity by model at the closure of critical period in details by contrasting with studies before[19].

The PNN model is not just modified on the architecture of NN based on current gradient informational synapse formation and brain plasticity, but
also the mnemonic negative and positive gradient informational synapse formation and mnemonic brain plasticity at critical period. By simulations, the PNN gave the two cases with and without negative memories persistence. And the paper proposed α7-nAChR-dependent astrocytic responsiveness is an integral part of the cellular substrate underlying memory persistence by impairing fear memory[20]. In addition, mnemonic brain plasticity involves the plus or minus disturbance-astrocytic brain plasticity phagocytic synapses factor, through which the synaptic homeostasis is achieved[21]. The effect of astrocyte made local synaptic effective range remain in an appropriate length at critical period[21]. Both negative and positive memories can be reflected in brain cortex, and its simulations fits very well with experiments in the relationship of human intelligence and cortical thickness, individual differences in brain[22]. The PNN also considered the memory engram cells that strengthened Synaptic strength, and memory engram cells are helpful to the memory retrieval process[22]. And paper found that 1064-nm Transcranial photobiomodulation (tPBM) applied to the right prefrontal cortex (PFC) improves visual working memory capacity[24], the effect of PNN (memory structure) may be the same with smaller losses in signals.

For a given neuron, the synaptic plasticity of the connecting neurons from input to output units is enhanced and diminished with iterations. Considering the special Recurrent Neural Network (RNN) to obtain PNN, each input variable corresponds to neurons shared connection weights over a period of time interval; the weights are updated by the MSE loss of the activation function of output within this synaptic effective range. The synaptic effective range is reflected in the time interval, for which the real value of the simulation would lead to gradient-based change in the synaptic effective range by Back Propagation. The Back Propagation of PNN includes shared connection weights and synaptic effective range weights. A simulation of PNN was conducted on the cognitive processes in the brain from infancy to senile phase, which is interpreted by the observation of decreasing the synapse population and increasing the minimum of synaptic effective range in PNN evolution of synapse formation, hence the loss of diversity and plasticity. This explanation is similar to the Dr. Luo and his colleagues’ research, and on PNN which shows that the synapse formation in a certain extent may inhibit dendrite. And we also analyzed the different brain plasticity by Tables at critical or the closure of critical period. When ORPNN contains both astrocytic synapse formation memory persistence factor and astrocytic brain plasticity phagocytic synapses factor will have better results in correlation coefficients of Tables respectively at critical period.

The cosine filter is used in the tests for comparing the result of the three cases, namely gradient optimized synaptic effective range (ORPNN), random synaptic effective range (RRPNN), and constant synaptic effective range (CRPNN). The result indicates that the ORPNN runs the best. Through the PNN tests, we found that the astrocytic memory persistence factor, like the
phagocytic synapses factor, produces the effect of reducing the local accumulation of synapses. Considering both negative and positive memories persistence help activate synapse length changes, and therefore yields better results than considering only positive memory persistence. In the calculation to update the synaptic effective range, the PNN in which only the synaptic phagocytosis effect is considered regardless of the gradient update, proves simple and effective. Therefore, is it possible to reduce the number of animal experiments and their suffering by simulating and planning the factors of biological experiments through Deep Learning? And our question is can model construction, formula derivation and algorithm testing serve to promote the development of computational neuroscience and brain cognition?

1 Introduction

Researchers in the Deep Learning community have long regarded simulation of the human brain as an important means of advancing Neural Networks (NN). In machine learning and computational neuroscience, Artificial Neural Networks (ANN) are used as mathematical and computational models that mimic the structure and function of biological Neural Networks. Estimations and approximations conducted by the functions have witnessed great success over the recent decades. The more commonly used Neural Networks models today include Perceptron in 1957 [1], Hopfield network in 1982 [2], Boltzmann in 1983 [3], Back Propagation (BP) in 1986 [4], Convolutional Neural Networks (CNN) in 1989 (from Neocognitron [5] to CNN [6]), Spiking Neural Networks (SNN) in 1997 [7], Long Short-Term Memory (LSTM) in 1997 [8], Deep Belief Network (DBN) in 2006 [9], Deep Neural Networks (DNN) in 2012 [10], Deep Forest (DF) in 2019 [11] and Deep Residual Learning (ResNet) in 2016 [12].

Human brains can be very flexible due to their plasticity. Neural activity can induce the strengthening or weakening (potentiation or depression) of synapses to produce plasticity. The brain plasticity therefore refers to the connections of synapses and neurons, and to establish new connections as a result of learning and experiencing, thus exerting impacts on the behaviors of individuals. The synapses are the roles between neurons permitting the transmission of signals or stimulus. Therefore, our new NN model takes into account not only the synaptic connections, but also how the synaptic effective range at current and historical moments changes plastically with the iteration, particularly at critical period, namely, in addition to the shared weights of the current and mnemonic synaptic connections, it also considers the weights of the current and mnemonic synaptic effective ranges (brain plasticity) and gradient information (synapse formation) at critical period.

Last year, researchers presented the One Hundred Year Study on Artificial Intelligence (AI100) 2021 Study Panel Report, in which one question concerns how much progress we have made in UNDERSTANDING the PRINCIPLES of HUMAN INTELLIGENCE. First, a basic tenet of cognitive neuroscience
is that individual characteristics such as working memory and executive control are critical for domain-independent intelligence, which determines an individual’s performance on all cognitive tasks regardless of mode or subject. A second hypothesis gaining traction is that higher-ability persons enjoy more efficient patterns of brain connectivity—higher-level brain regions in the parietal-frontal cortex. The third concept is more revolutionary, which suggests that the neural correlates of intelligence are dispersed throughout the brain. According to this theory, human intelligence is fundamentally flexible, constantly updating past information and making new predictions [13]. The new NN proposed in this thesis is grounded on the first and third notion, taking into consideration the current and past brain plasticity and current gradient informational and mnemonic positive and negative gradient informational synapse formation at critical period and closure of critical period.

Scientists of MIT’s Picower Institute have unveiled for the first time how this balance is achieved in synapses: Professor Mriganka Sur likens this behavior to a massive school of fish in the sea. Immediately when the lead fish changes direction, other fish will follow suit, presenting a delicate marine ”dance”. “Collective behaviors of complex systems always have simple rules. When one synapse goes up, within 50 micrometers there is a decrease in the strength of other synapses using a well-defined molecular mechanism.” the scientists stated [14]. The lead behavior of the school of fish is well embodied in our PNN. However, our Neural Network is no longer confined to the simple physical concept of synaptic strength strength rebalance - it treats synapses as a group of intelligent agents to achieve synaptic strength rebalance.

Findings largely consistent with that of Dr. Luo’s team at Stanford University in PNN tests are obtained in our study, that is, the synapse formation will inhibit dendrites generation to a certain extent [15], with the reason shown in our simulation results being that synaptic growth leads to a reduction in changes in synaptic effective range, which disrupts diversity and plasticity of brain.

The Alcino J Silva’s team suggested that memory ensembles recruit presynaptic neurons during learning by a retrograde mechanism [16]. The retrograde mechanism reflected in memory retrieval process of PNN.

The Alvarez-Buylla’s lab proposed that recruitment of young neurons to the primate hippocampus decreases rapidly during the first years of life, and that neurogenesis in the dentate gyrus does not continue, or is greatly rare, in adult humans [17]. The PNN gave the simulation results about decrease of neurons.

But Boldrini et al suggested healthy older subjects display preserved neurogenesis. It is possible that ongoing hippocampal neurogenesis sustains human-specific cognitive function throughout life [18]. And PNN’s guess decline in neurogenesis may be repaired by a new and longer circuit in late iteration.

The finding has significant implications for the learning and further development of NN. Moreover, the mechanism of our NN is basically consistent with
that of the latest brain plasticity study conducted by MIT, in which researchers found that as a synapse strengthens, neighboring synapses automatically weaken themselves to compensate [14]. In tests, possible explanations of dendrite morphogenesis are derived, which demonstrate that dendrite generation, to a certain extent, is curbed by synapse formation [15]. Unconventional astrocyte connexin signaling hinders expression of extracellular matrix-degrading enzyme matrix metalloproteinase 9 (MMP9) through RhoA-guanosine triphosphatase activation for controlling critical period closure. The effect of astrocyte impacts on brain plasticity and synapse formation is an important mechanism of our Neural Network at critical period, and failures by the closure of critical period result in neurodevelopmental disorders [19]. In the model at critical period, the hypothesis is the best previous brain plasticity affects current brain plasticity and the best previous synapse formation, relatively good synapse formation and relatively inferior synapse formation affects current synapse formation. While their experimental research features a combination of cutting-edge imaging and genetic tools, our study lays more emphasis on the model, derivation and simulation of a new NN. At the same time, the current and mnemonic brain plasticity-the synaptic effective range are taken into consideration. Furthermore, the architecture of new NN is based on current gradient informational and mnemonic positive and negative gradient informational synapse formation for memory persistence. By simulations, the PNN gave the two cases with and without negative memories persistence. And the paper proposed $\alpha_7$-nAChR-dependent astrocytic responsiveness is an integral part of the cellular substrate underlying memory persistence by impairing fear memory [20]. In addition, mnemonic brain plasticity involves the plus or minus disturbance-astrocytic brain plasticity phagocytic synapses factor, through which the dynamic synaptic strength balance is achieved [21]. The effect of astrocyte made local synaptic effective range remain in an appropriate length at critical period [21]. Our new NN may give more inspirations to experiments in the relationship of human intelligence and cortical thickness, individual differences in brain [22]. By using learning-dependent cell labeling, Dr. Susumu Tonagawa and his team identified an increase of synaptic strength and dendritic spine density specifically in consolidated memory engram cells. For improving Synaptic strength in PNN, the memory retrieval process by memory engram cells includes mnemonic negative and positive gradient information [23]. In this study, researchers found that 1064-nm tPBM applied to the right prefrontal cortex (PFC) improves visual working memory capacity [24], and PNN has same effect for improving memory capacity by memory structure. The Dr. Christian Kerskens and David López Pérez of Trinity College Institute of Neuroscience’s findings suggest that they may have witnessed entanglement mediated by consciousness-related brain functions, because consciousness-related or electrophysiological signals are unknown in nuclear magnetic resonance (NMR). Those brain functions must then operate non-classically, which would mean that consciousness is non-classical [25],
so PNN introduces brain plasticity of extracted relatively good or inferior memories, which reflected consciousness by quantum computing.

Our newly proposed NN is named the Plasticity Neural Network (PNN). For a given neuron, the synaptic plasticity of the connecting neurons from input to output units is first enhanced and then diminished with iterations. Synaptic competition causes the enhancement of signal-stimulated synapses to be reflected in an increase in synaptic effective range, while the weakening of peripherally stimulated synapses is reflected in a shortening of synaptic effective range.

The following papers are academic frontiers in neuroscience and artificial neural networks which we endeavor to make connections with our PNN.

Their research shows that SynCAM 1 actively limits cortical plasticity in the mature brain. Plasticity tapers off when the brain matures and the conclusion is sufficiently substantiated by visual input in adult animal models [26]. As was stated by Dr. Biederer of Tufts University’s medical School, “Our research has identified a mechanism underlying the control of brain plasticity, and most excitingly, we can demonstrate that there is an active process of inhibiting plasticity in the adult brain.”

The Self-Back Propagation (SBP) phenomenon, first discovered in hippocampal neurons [27, 31], involves cross-layer Back Propagation (BP) of Long-Term Potentiation (LTP) and Long-Term Depression (LTD) from output to input synapses of a neuron to enable the strengthening or weakening of synaptic connections. Other forms of nonlocal spreads of LTP and LTD in the pre- and post-synaptic neurons have already been researched extensively [27, 28, 31]. The SBP phenomenon induces a form of nonlocal activity-dependent synaptic plasticity that may endow developing neural circuits with the capacity to modify the weights of input synapses on a neuron in accordance with the status of its output synapses [28]. The existence of SBP was demonstrated in developing retinotectal circuits in vivo [29, 30]. These papers endeavor to shed light on plasticity rules involving activity-dependent modification of synapses to obtain synaptic activity [27–31].

Bertens and Lee proposed an evolvable neural unit (ENU) that can evolve individual somatic and synaptic compartment models of neurons in a scalable manner and try to solve a T-maze environment task [32].

Wang and Sun stated that in a unidirectional RNN, the linking with the emotion regions and the somatic motor cortex includes three basic units: input units arriving from the emotion regions, only one hidden unit composed of self-feedback connected medial prefrontal cortex neurons, and output units located at the somatic motor cortex [33]. If they used PNN regardless of RNN may give more insights in their research.

In the simulations of PNN in brain plasticity at critical period, we had an idea to modify the synapses for repairing brain plasticity, and later we found this paper to build and remodel of synapses [34].

Instead of conducting research simply on the basis of Self-Back Propagation phenomenon, the PNN we propose in this thesis focuses more on plasticity
and strength rebalance as a result of its tapering off—the brain from infancy to senile phase during critical period and the closure of critical period. The model also delves into the competition of brain plasticity which introduces a novel form of current gradient informational and mnemonic positive and negative gradient informational synapse formation, as well as current and mnemonic brain plasticity at critical period. The strengthening and weakening of synaptic connections help to curb and boost current and historical synaptic effective ranges, resulting in an increased accuracy of PNN.

The research, based on Genetic Algorithm or Particle Swarm Optimization in search of the connection weights for each time interval of the dynamic problems, and on PNN to find the weights of the connection and the effective range, takes into account the specificity of the new coronavirus variant, whose viral strain is highly variable. The viral load of the Delta (or Omicron) strain is 1,260 times higher than the prevalent strain of last year, thus doubling the infectious rate of last year’s original strain. The major difference between PNN and RNN is that the former has a formula to estimate the parameters (shared connection weights) for their respective parameters’ ranges (synaptic effective range weights) at critical period. So PNN is based on the practical dynamic problem in an effort to obtain the new NN. As for the selection of the appropriate mechanism, we chose the hypotheses and possible explanations of synaptic strength rebalance, competition and effect of astrocyte [14–25]. In this way, not only the architecture of NN is transformed by current gradient information, but also mnemonic positive and negative gradient informational synapse formation and mnemonic brain plasticity are taken into account. The mnemonic gradient information needs to consider astrocytic synapse formation memory persistence factor (including both negative and positive memories). By simulations, the PNN gave the two cases with and without negative memories persistence. And the paper proposed α7-nAChR-dependent astrocytic responsiveness is an integral part of the cellular substrate underlying memory persistence by impairing fear memory[20]. In addition, mnemonic brain plasticity involves the astrocytic brain plasticity phagocytic synapses factor [21]. And also comparing with the simulations and experiments in the relationship of human intelligence and cortical thickness, individual differences in brain [22]. In PNN, the memory retrieval process includes mnemonic negative and positive gradient information, and used in formula (2) [23]. And PNN has same effect for improving memory capacity by memory structure of salient point and concave point [24].

After representing the synaptic plasticity competition in Formula (2), strength rebalance in Formula (3), current gradient informational and mnemonic positive and negative gradient informational synapse formation at critical period in Formula (2), current and mnemonic brain plasticity at critical period in Formula (4), cosine filter is used in tests to verify the PNN and compare the results of the following three situations: the case dubbed as CRPNN, in which the synaptic effective range for various neurons remains unchanged; the RRPNN case, in which synaptic effective range for each neuron generates
in a random manner; the last one is the ORPNN case, in which optimization of synaptic effective range connects neurons with iterations. The findings also show that synapse formation will, to some extent, inhabit the tests of PNN. And we also analyzed the different brain plasticity by Tables at critical period or the closure of critical period. The effect of astrocytic synapse formation memory persistence factor and astrocytic brain plasticity phagocytic synapses factor in Tables.

2 Method

We developed PNN under the premise that the sum of synaptic effective ranges of these neurons’ connections remains a constant value for the synaptic strength rebalance. For RNN, the size of training sets has been determined by sum of synaptic effective ranges \( l_{\text{max}} \). This hypothesis works for RNN as well as BP. However, synapse formation will lead to convergence and the loss of plasticity, therefore diminishing the cognitive abilities of PNN. For RNN, the input variables correspond to the different types of neurons’ within a time interval, and each time interval corresponds to a connection weight. and the weight is updated by the MSE loss of the activation function of output within this synaptic effective range. The synaptic effective range is reflected in the time interval, and alters with the gradient by simulating real value- training sets with iterations.

Formula (1) and (2) are both obtained through the reasoning of MSE loss function gradient. The activation function for the amount of change in the output result is processed through the tanh function.

Formula (1) updates the shared connection weight of the \( k \)th synapse of the \( m \)th input neuron, \( f() \) represents the output of different variables and synapses, \( m \) represents the label of the input variable, \( k \) represents label of synapses connecting two neurons, and the range of values of time interval \( t \) is \([n3(m, k) n3(m, k+1)]\). \( h(t) \) indicates actual output, and \( f(t) \) represents desired output. \( n3(m, k) \) and \( n3(m, k+1) \) depict the location of the \( m \)th input variable and \( k \)th synaptic initiation and termination points. \( \eta = 1/1E3 \times (j_{\text{max}} - j)/j_{\text{max}} \), namely, the learning rate is reflected in formulas (1) and (2).

\[
g_w(m, k) = [f(t) - h(t)] \times \Delta f(t-1)_{w(m, k)}/1E3 \\
\times (j_{\text{max}} - j)/j_{\text{max}} \times [1 - \Delta f(t-1)^2]
\]

\[
w(m, k) = w(m, k) - g_w(m, k)
\]

The synaptic range of effective weights of the \( m \)th input variable and the \( k \)th synapse is updated by Formula (2). The relationship between the synaptic location and the effective range satisfies the equation \( n3(m, k+1) - n3(m, k) = n1(m, k) \), in which \( n1(m, k) \) represents the synaptic effective range. When the synaptic position of the lead neurons is changed, these neurons, much like
the heads in a massive school of fish, also exerts an impact on the post-synaptic neurons. Formula (2) considers current gradient informational and mnemonic positive and negative gradient informational synapse formation updated by the gradient of the best previous synaptic effective range weight \( g_r(m,k)_{\text{best}} \), or gradient of relatively inferior synaptic effective range weight \( g_r(m,k)_{\text{worse}} \), or relatively good synaptic effective range weight \( g_r(m,k)_{\text{better}} \), so that architecture of PNN bears current and mnemonic gradient information at critical period. With reference to Formula (3), Formula (2) includes current and mnemonic gradient information, or \( r(m,k)_{r(m,k)} = \frac{-r(m,k)}{\text{sum}(r(m,:))} + \frac{1}{\text{sum}(r(m,:))} \times (l_{\text{max}} - k_{\text{max}} \times l_{\text{min}}) \). \( MW_{i,i=1,2,3} \) is mnemonic weight.

\[
g_r(m,k) = [f(t) - h(t)] \times \Delta f(t - 1)_{n1(m,k)} \times n_1(m,k)_{r(m,k)}' \\
\times \eta \times [1 - \Delta f(t - 1)^2] = [f(t) - h(t)] \\
\times \Delta f(t - 1)_{n1(m,k)}/1E3 \times (j_{\text{max}} - j)/j_{\text{max}} \times [1 - \Delta f(t - 1)^2] \tag{2} \\
\times \left[ \frac{-r(m,k)}{\text{sum}(r(m,:))^2} + \frac{1}{\text{sum}(r(m,:))} \right] \times (l_{\text{max}} - k_{\text{max}}l_{\text{min}}) \\
\]

\( r(m,k) = r(m,k) - g_r(m,k) \)

\( \text{or } r(m,k) = r(m,k) - g_r(m,k) + [g_r(m,k)_{\text{best}} - g_r(m,k)] \times M \)

\( \text{or } r(m,k) = r(m,k) - g_r(m,k) + [MW_1 \times g_r(m,k)_{\text{best}} + MW_2 \times g_r(m,k)_{\text{worse}} - g_r(m,k)] \times M \)

\( MW_1 = 0.6666, MW_2 = 0.3333 \)

\( \text{or } r(m,k) = r(m,k) - g_r(m,k) + [MW_1 \times g_r(m,k)_{\text{best}} + MW_2 \times g_r(m,k)_{\text{worse}} + MW_3 \times g_r(m,k)_{\text{better}} - g_r(m,k)] \times M \)

\( MW_1 = 0.5, MW_2 = 0.25, MW_3 = 0.25 \)

Formula (3) serves to update the range of connecting neurons at the \( k \)th synapse of the \( m \)th input variable, and normalization of result is achieved through \( r(m,k)/\text{sum}(r(m,:)) \) in Formula (3). The result of \( r(m,k) = r(m,k) - g_r(m,k) + [0.6666 \times g_r(m,k)_{\text{best}} + 0.3333 \times g_r(m,k)_{\text{worse}} - g_r(m,k)] \times M \) may be better than \( r(m,k) = r(m,k) - g_r(m,k) \) in simulation, because negative and positive memories reflecting the diversity. If Formula (2) just has current gradient information, \( r(m,k) = r(m,k) - g_r(m,k) \), else if Formula (2) includes current and mnemonic gradient information, \( r(m,k) = r(m,k) - g_r(m,k) + [0.6666 \times g_r(m,k)_{\text{best}} + 0.3333 \times g_r(m,k)_{\text{worse}} - g_r(m,k)] \times M \)
needs to consider forgotten memory in synapse formation, memory persistence of astrocytic function gradually lost.

The derivation of memory persistence factor is as follows: The update of \( r(m,k) = r(m,k) - g_r(m,k) \) is based on the gradient descent method. From \( r(m,k) = r(m,k) - g_r(m,k)_{\text{best}} \), we can obtain \( r(m,k)_{t+1} = r(m,k)_t - g_r(m,k)_{\text{best}} \). \( r(m,k)_{t+1} \) is the best previous synaptic effective range weight, and \( g_r(m,k)_{\text{best}} \) is the gradient of the best previous synaptic effective range weight. Time reversal from \( t+1 \) to \( t \) to return to in situ to maintain memory can also be written as \( r(m,k)_t = r(m,k)_{t+1} + g_r(m,k)_{\text{best}} \), after which, by adding \( r(m,k)_{t+1} = r(m,k)_t - g_r(m,k) \) and \( r(m,k)_t = r(m,k)_{t+1} + g_r(m,k)_{\text{best}} \), we get \( r(m,k) = r(m,k) - 0.5 \times g_r(m,k) + 0.5 \times g_r(m,k)_{\text{best}} \). Considering the flexibility of learning rate, it can also be written as \( r(m,k) = r(m,k) - g_r(m,k) + [g_r(m,k)_{\text{best}} - g_r(m,k)] \times M \). If we take both positive and negative memories into account, introducing the gradient of the best previous synaptic effective range weight \( g_r(m,k)_{\text{best}} \), gradient of relatively inferior synaptic effective range weight \( g_r(m,k)_{\text{worse}} \) and gradient of relatively good synaptic effective range weight \( g_r(m,k)_{\text{better}} \), therefore the equation can be modified as follows: 

\[
MW_1 \times g_r(m,k)_{\text{best}} + MW_2 \times g_r(m,k)_{\text{worse}} + MW_3 \times g_r(m,k)_{\text{better}} - g_r(m,k)
\]

of \( r(m,k) = r(m,k) - g_r(m,k) + [MW_1 \times g_r(m,k)_{\text{best}} + MW_2 \times g_r(m,k)_{\text{worse}} + MW_3 \times g_r(m,k)_{\text{better}} - g_r(m,k)] \times M \) illustrated that recruitment of presynaptic neurons is triggered by downstream neurons through a retrograde mechanism[16], so the mnemonic gradients \( MW_1 \times g_r(m,k)_{\text{best}}, MW_2 \times g_r(m,k)_{\text{worse}}, MW_3 \times g_r(m,k)_{\text{better}} \) are positive, and the current gradient \(-g_r(m,k)\) is negative.

The element \( g_r(m,k)_{\text{worse},i} \) or \( g_r(m,k)_{\text{better},i} \) of historic set \( WORSE = \{g_r(m,k)_{\text{worse},i} \in N^* \} \) or \( BETTER = \{g_r(m,k)_{\text{better},i} \in N^* \} \) also can be considered in the formula (2), and latest \( g_r(m,k)_{\text{worse}} \) or \( g_r(m,k)_{\text{better}} \) could be replaced by later \( g_r(m,k)_{\text{worse},i} \) or \( g_r(m,k)_{\text{better},i} \). The gradient information of later element \( g_r(m,k)_{\text{worse},i} \) or \( g_r(m,k)_{\text{better},i} \) is stored in memory engram cells of cortices, namely larger \( i \) of \( g_r(m,k)_{\text{worse},i} \) or \( g_r(m,k)_{\text{better},i} \) will be selected with upper probabilities.

Why formula (2) chooses the elements of historic set \( WORSE \) or \( BETTER \)?

Based on optimality principle, the relatively good gradient information can help to search optimal solution, and the relatively inferior gradient information
can help to improve diversity of searching optimal solution. The location of relatively inferior gradient information is salient point, the directions of search both are downward and become more efficient. The location of relatively good gradient information is concave point, the directions of search both are upward and improve the efficiency.

But memory-forgotten means that the signals from input to output units dissipate with resistances in brain and do not activate the relevant gradient information, and these resistances in brain increase by aging.

In the model at critical period, the hypothesis is the best previous synapse formation and relatively inferior synapse formation affects current synapse formation. Bad memory of synapse formation $M = \exp(-7 \times j/j_{\max}) \times \text{rand}$ or good memory $M = \exp(-5 \times j/j_{\max}) \times \text{rand}$ can be called the astrocytic synapse formation memory persistence factor. $j/j_{\max}$ illustrates the decreasing weights of factor we present at critical period in $M$.

$$n_1(m, k) = \frac{r(m, k)}{\text{sum}(r(m, :))} \times (l_{\max} - k_{\max} l_{\min}) + l_{\min}$$  \hspace{1cm} (3)
For a certain input variable, the neuron has a total of $k_{\text{max}}$ synapses. $l_{\text{min}}$ shows that the synaptic range change enjoys a certain degree of plasticity, and the minimum synaptic effective range is $l_{\text{min}}$. $g_r(m, k)$ controls $l_{\text{min}}$ with iterations and represents the elasticity or plasticity of synapses, and if the
value of \( l_{\text{min}} \) is overly small, the change of \( n_1(m,k) \) will be too elastic and may make the test results deteriorate. \( k_{\text{max}} \) indicates the synapse population involved in the neurons from input to output units, and \( l_{\text{max}} \) represents the sum of ranges of time series and is a constant. For the signal of a neuron (i.e., an input variable), when one synapse strengthens, there must be surrounding synapses that are weakened. In the case when \( l_{\text{max}} = k_{\text{max}} \times l_{\text{min}} \), i.e., the case in which the synaptic effective range is constant, \( n_1(m,k) = l_{\text{min}} \) is also a constant value. That is, for \( l_{\text{min}} = l_{\text{max}} / k_{\text{max}} \), constant-range synapses also satisfy Formula (2) and Formula (3). Formula (2) can be named the synaptic competitive formula, and Formula (3) can be dubbed as the synaptic strength rebalance formula.

\[
 r(m,k) = r(m,k) - [r(m,k)_{\text{best}} - r(m,k)] \times \text{rand} + P \\
\text{or } r(m,k) = r(m,k) + [MW_1 \times r(m,k)_{\text{best}} + MW_2 \times r(m,k)_{\text{worse}} + MW_3 \times r(m,k)_{\text{better}} - r(m,k)] \times \text{rand} + P, \text{ if } MW_1 = 0.5, MW_2 = 0.25, MW_3 = 0.25
\]

\[
 Q_P = (\text{rand} > 0.5) \times \text{sign}(\text{rand} - 0.5) \times \beta \times |r(m,k)_{\text{best}} + \text{rand} \times [r(m,k)_{\text{best}} - r(m,k)_{\text{better}}] - \sum_{i=j-L\text{EN}+1}^{j} \frac{r(m,k)_{i,\text{better}}}{L\text{EN}}| \times \ln(\frac{1}{\text{rand}})
\]

\[
 Q_N = (\text{rand} > 0.5) \times \text{sign}(\text{rand} - 0.5) \times \beta \times |r(m,k)_{\text{best}} + \text{rand} \times [r(m,k)_{\text{best}} - r(m,k)_{\text{better}}] - \sum_{i=j-L\text{EN}+1}^{j} \frac{r(m,k)_{i,\text{worse}}}{L\text{EN}}| \times \ln(\frac{1}{\text{rand}})
\]

\[
 P = \text{sign}(\text{rand} - 0.5) \times \text{rand} \times (j_{\text{max}} - j) / j_{\text{max}} \\
r(m,k) = r(m,k) + \text{rand} \times [R + Q_P + Q_N - r(m,k)] + P
\]

Formula (4) updates \( r(m,k) \) by current and mnemonic synaptic effective range weight, the best previous \( r(m,k)_{\text{best}} \). Formula (4) is named the brain plasticity formula. In the model featuring critical period, the hypothesis is best previous brain plasticity affects current brain plasticity. \( P = \text{sign}(\text{rand} - 0.5) \times \text{rand} \times (j_{\text{max}} - j) / j_{\text{max}} \) implements plus or minus disturbance of brain plasticity-astrocytic brain plasticity phagocytic synapses factor, and this also enables dynamic synaptic strength balance. The dynamic forgotten memory astrocytic synapse formation memory persistence factor and disturbance astrocytic brain plasticity phagocytic synapses factor shown in Fig. 2. \( (j_{\text{max}} - j) / j_{\text{max}} \) illustrates we give decreasing weights of factor at critical period in \( P \). \text{sign}(\text{rand} - 0.5) \) gives Formula \( P \) both positive and negative results. \( P \) can be negative because
of astrocytes phagocytose synapses, but considering the strength rebalance of synaptic effect, $P$ should be positive and negative. The $P$ decreases with the maturation response of astrocytes.

In our opinion, when you are considering one problem, whether positive or negative, the brain plasticity may turn to quantum and satisfies exponential decay by aging. It is important to maintain inner peace, for reducing these effects of exponential decay, the flatter exponential decay curve. The actions of neurons may like particles, have exponential decay at the $LEN$ points of relatively good or inferior memories, these extracted memories may be quantum, and distribution probability of $y = R - r(m, k)_{i, \text{better}}$ or $y = R - r(m, k)_{i, \text{worse}}$ satisfies $|\Psi(y)|^2 \propto \frac{\exp[-2|y| \times L(t)]}{L(t)}$. The wave function describes synaptic effective range weights or brain plasticity, The length of time is $LEN$ form ($j - LEN + 1$)th iteration to $j$th iteration, and by using Mont Carlo method to calculate $Q_N = (\text{rand} > 0.5) \times \text{sign(\text{rand} - 0.5) \times } \beta \times |r(m, k)_{\text{best}} + \text{rand} \times [r(m, k)_{\text{best}} - r(m, k)_{\text{better}}]| - \sum_{i=j-LEN+1}^{j} \frac{r(m, k)_{i, \text{worse}}}{LEN} \times \ln(\frac{1}{\text{rand}}), L(t) = 2 \times \beta \times |r(m, k)_{\text{best}} + \text{rand} \times [r(m, k)_{\text{best}} - r(m, k)_{\text{better}}]| - \sum_{i=j-LEN+1}^{j} \frac{r(m, k)_{i, \text{better}}}{LEN} \times \ln(\frac{1}{\text{rand}}), Q_P = (\text{rand} > 0.5) \times \text{sign(\text{rand} - 0.5) \times } \beta \times |r(m, k)_{\text{best}} + \text{rand} \times [r(m, k)_{\text{best}} - r(m, k)_{\text{better}}]| - \sum_{i=j-LEN+1}^{j} \frac{r(m, k)_{i, \text{better}}}{LEN} \times \ln(\frac{1}{\text{rand}}), i \beta = 1$, and $\beta$ is shrinkage-expansion coefficient, a superposition of wave function of time. The length of time $LEN$, it may be long-term memory or short-term memory in quantum computing. $j$ is current iteration. $i$ is $i$th iteration. $Q_N + Q_P$ means a linear superposition of two wave functions. $r(m, k)_{i, \text{better}}$ is relatively good synaptic effective range weight in $i$th iteration, $r(m, k)_{i, \text{worse}}$ is relatively inferior synaptic effective range weight in $i$th iteration, $r(m, k)_{i, \text{best}}$ is best previous synaptic effective range weight in $i$th iteration. And ($\text{rand} > 1/2$) means it has $2^2 = 4$ scenarios of the relatively inferior and good two-qubit: $\{1,1\}, \{1,0\}, \{0,1\}$ or $\{0,0\}$. The quantum uncertainty of relatively inferior and good range weights, because of scan resolution of the cortical folds. The effects of astrocytic phagocytic synapses $P$ and quantum computing $Q$ in calculations are very similar in minus or plus, the process of phagocytic synapses may a normal computer by random or gradient, but also a quantum computer. It may be that the process of phagocytic synapses is driven by positive and negative memory of brain plasticity, by excitatory/inhibitory interactions.

The pseudocode of PNN is as follows, which encompasses Forward and Back Propagation.
Fig. 1 Positive (the best previous gradient information, relatively good gradient information) and Negative (relatively inferior gradient information) synapse formation: (a) Biological experiment results [22] (b) Normal IQ (c) High IQ (d) Low IQ (e) LEN points of relatively good and inferior memories for quantum computing
Fig. 2  Astrocytic synapse formation memory persistence factor and astrocytic brain plasticity phagocytic synapses factor: (a) Astrocytic synapse formation memory persistence factor (b) Astrocytic brain plasticity phagocytic synapses factor
Require: initialization parameter: generation of randomly connection weight \( w(m,k) \), neuron \( m \), synapse \( k \), and effective range weight \( r(m,k) \).

#1 Do \( j=1:j_{\text{max}} \) (\( j_{\text{max}} \) is maximum iterations)

#2 While the condition of loss function is satisfied, it stops iterating

#3 Do \( g=1:g_{\text{max}} \) (\( g_{\text{max}} \) is number of neurons)

Using mnemonic the best previous solution to update the \( w(g,k) \) and \( g.r \) (m,k) While it satisfies the conditions Update \( r(m,k) \) at formula (4) by current and mnemonic synaptic effective range weight for brain plasticity and implements plus or minus disturbance of brain plasticity–P, astrocytic brain plasticity phagocytic synapses factor at critical period, it also considers quantum computing \( R+Q.N+Q.P \) to update \( r(m, k) \)

#4 Do \( m=1:m_{\text{max}} \)

#5 Do \( k=1:k_{\text{max}} \) (\( k_{\text{max}} \) is synapse population)

#6 Do \( t=n_{3}(m,k):n_{3}(m,k+1) \) (\( t \) is time point)

When Back Propagation position moves from \( n_{3}(m,k) \) to \( n_{3}(m,k+1) \), update synapse connection weight at Formula (1) \( w(g,k) \), synaptic effective range weight at Formula (2) \( r(m,k) \) for competition by the current gradient informational and mnemonic positive and negative gradient informational synapse formation and consider the forgotten memory–M, astrocytic synapse formation memory persistence factor at critical period.

#6 End do

#5 End do

Update weights of synaptic connections and effective ranges, then Forward Propagation is performed to obtain loss

Then result of Back Propagation updates the neuron's synaptic position by Formula (3) \( n_{1}(m,k) \) for strength rebalance

#3 End do

#2 End while

#1 End do

*As for updating \( w(g,k) \) and \( r(m,k) \), the global synaptic effective range weights \( r(m,k) \) need to be changed by overall optimization, while the same type of connection weight \( w(g,k) \) just needs to be changed by local optimization. I.e., research on disease dynamics, when infection rates (or cure rates) change, the time interval of the infection rate and cure rates both need to be updated at the same time.
3 Discussion

3.1 Comparison of PNNs in some scenarios

Parameters regarding the abovementioned test is as follows: testing environment is WIN11 and MATLAB2020a, the parameters for updating the weights remain the same for the three tests, and the number of iterations is $j_{\text{max}} = 40000$. The tests employ cosine filter, in which input are filter from point $t + 1$ to $t + 4$, and prediction of output is filter at $t + 5$ point. $l_{\text{min}} = 2$, $k_{\text{max}} = 9$, $l_{\text{max}} = 44$. Three methods were all considered mnemonic the best previous solution, so RRPNN and ORPNN both take into consideration the current and mnemonic connection weight -brain plasticity and synaptic effective range- synapse formation. But CRPNN only pays heed to the current and mnemonic connection weight, as the synaptic effective range remains constant in synapse formation. Only ORPNN takes into account the current gradient informational and mnemonic positive and negative gradient informational synapse formation, and it also considers current and mnemonic brain plasticity. In the Fig.4, the ORPNN model includes two scenarios, in addition to phagocytic synapses factor, one only considering the best previous memory of synapse formation ORPNN-MF(P)-PF, and the other considering both the best previous memory and the relatively inferior memory of synapse formation ORPNN-MF(P&N)-PF. MF(P) represents positive memory persistence, and MF(P&N) represents positive and negative memories persistence.

Fig.3 shows respectively the flow charts of RNN, CRPNN, RRPNN and ORPNN.

A total of 3 tests were conducted, with the results shown in Fig.4, in which the results of the variation of the logarithm of the loss of PNN with iterations are also provided.

Result of the first test: the correlation coefficient between CRPNN simulation data and actual data is 0.8057, the correlation coefficient between RRPNN simulation data and real data is 0.8706, and the correlation coefficient between M(P)ORPNN and M(P&N) ORPNN simulation data and real data are 0.9709 and 0.9687. Result of the second test: the correlation coefficients are 0.8292, 0.8921, 0.9668 and 0.9646 respectively. Result of the third test: the correlation coefficients are 0.8031, 0.8728, 0.9622 and 0.9588 respectively.

The results of the RRPNN test turned out to be satisfactory, and though the connection weights are updated by equation (1) and mnemonic the best previous solution, more satisfactory optimization results could be found for random synaptic effective range locations.

The ORPNN showed strong fluctuation in the iterations, whereas the final convergence turned out better and faster than the RRPNN and the CRPNN. Iteration of CRPNN is not convergent. RRPNN has slow convergence.

The number of iterations is $j_{\text{max}} = 40000$. In order to study the effect of the astrocytic synapse formation memory persistence factor and astrocytic brain plasticity phagocytic synapses factor, Fig.7 is obtained. In the first scenario of ORPNN-PF, only the astrocytic brain plasticity phagocytic synapses factor is
RNN flow chart

(a) RNN $U$ is the weight from the input layer to the hidden layer, $V$ is the weight from the hidden layer to the output layer, $S$ is the vector of the hidden layer, $X$ is the vector of the input layer, and $O$ is the vector of the output layer. $W$ is the connection weight.

CRPNN and RRPNN flow charts

(b) CRPNN and RRPNN CRPNN is very similar to RNN, except that the iteration time is changed from $+1$ to $+n_1$. The $n_1$ in RRPNN is a variable, and the length of $n_1$ is randomly generated within a certain range.

taken into account with the best previous $r(m,k)_{best}$ and a correlation coefficient of 0.9515. The second scenario ORPNN-MF(P)-PF takes into account both the astrocytic synapse formation positive memory persistence factor in formula (2) and astrocytic brain plasticity phagocytic synapses factor in formula (4), memory persistence factor considers positive memory. It may have better results and a correlation coefficient of 0.9573 at critical period. The third scenario ORPNN excludes the astrocytic synapse formation memory persistence factor and astrocytic brain plasticity phagocytic synapses factor at the
3.2 Synapse formation affects brain function - inhibiting dendrites

We then modified the ORPNN model accordingly for analysis. By modifying $l_{\text{min}}$, we managed to simulate the process of brain cognition from infancy to the senile phase. $l_{\text{min}}$ exhibits elasticity at the beginning as a decimal value, then
first test results belong to CRPNN, RRPNN, and ORPNN respectively.

gradually decreases in plasticity. In this way the increase in minimum synaptic effective range from $l_{\text{min}} = 2$ to $l_{\text{max}}/k_{\text{max}} \approx 5$ made diversity of synaptic effective range progressively smaller. CRPNN corresponds to the senile phase period of brain cognition as it satisfies $l_{\text{min}} = l_{\text{max}}/k_{\text{max}} = 44/9 \approx 5$, and the funny thing is that the iteration is not convergent at CRPNN.

We obtained a possible explanation to the research findings of Dr. Luo’s team through PNN. We drew the relationship of different $l_{\text{min}}$ (minimum synaptic effective range), different $k_{\text{max}}$ (synapse population) and correlation coefficients, and the results are shown in Fig. 8 below. We also conducted another test, in which the cosine filter and cycle gradually diminish by time -variable cycle cosine filter, and the number of iterations is $j_{\text{max}} = 80000$. The functions of dendrites are to receive and process signals from other neurons. Our simulation results reveal that synapse formation will inhibit dendrites to a certain extent, furthering the findings of Dr. Luo’s team at the Stanford University. An explanation to this is that synaptic growth will lead to a reduction in the changes in synaptic effective range, and an overgrowth will inevitably disrupt the diversity and plasticity of human brain. $l_{\text{min}} = 2 - 5$, $k_{\text{max}} = 7 - 9$.

Fig. 8 concerns the relationship between synapse formation and correlation
coefficients in ORPNN. The synapse formation could involve a decrease in $k_{\text{max}}$ and an increase in $l_{\text{min}}$. The PNN situation can be thought as the Finite Element Method, in which neurons of the brain can be regarded as nodes of FEM. As increased in the number of nodes and finer meshes can lead to more accurate computational results of FEM, more neurons and smaller minimum synaptic effective range can also help us obtain more desirable and accurate PNN results. When synapse population $k_{\text{max}}$ decreases and sum of synaptic effective ranges $l_{\text{max}}$ is a constant, the diversity of local synaptic effective range may be improved as the synaptic effective range enlarges from $l_{\text{min}}$ to $l_{\text{max}}/k_{\text{max}}$, so $k_{\text{max}}$ decreases may enhance correlation coefficients by accident, as you can observe from Fig. 9 (a) $l_{\text{min}} = 3 - 4$, $k_{\text{max}} = 7 - 8$ and Fig. 9 (b) $l_{\text{min}} = 2 - 3$, $k_{\text{max}} = 7 - 9$. When the minimum synapse length is too small, the probability of local synaptic accumulation increases, which also affects the optimization results, It can be seen from Table 2.
3.3 Effects of astrocytes are reflected in phagocytic synapses factor and memory persistence factor during critical and closure critical period

As for the ORPNN-MF(P)-PF situation (ORPNN both containing astrocytic synapse formation positive memory persistence factor and astrocytic brain plasticity phagocytic synapses factor at critical period), synaptic effective ranges with different $l_{\text{min}}$ and different input variables are provided in Table 1. When $l_{\text{min}} = 5$, the synapse grows to a certain extent, and synaptic effective range remains almost the same, thus leading to a worsened simulation result. When the synapse grows, standard deviation of synaptic effective range at the end of the iteration diminishes, and the correlation coefficients decreases to a certain extent when $l_{\text{min}} = 5$. From $l_{\text{min}} = 2$ to $l_{\text{min}} = 5$, synaptic effective range gradually loses its diversity and plasticity. The number of iterations is $j_{\text{max}} = 80000$. And the results of synaptic effective range and correlation coefficient when iteration equals 20,000, 40,000, 60,000 and 80,000 were also observed, as 4444 in Table represents the synaptic effective ranges are all 4, and 0.9641, 0.9683, 0.9683 and 0.9645 are the correlation coefficients when iteration equals 20,000, 40,000, 60,000 and 80,000, respectively.
As for the ORPNN-PF situation (ORPNN only containing astrocytic brain plasticity phagocytic synapses factor), synaptic effective ranges with different $l_{\text{min}}$ and different input variables are provided in Table 2.

Comparing with the other Tables, the effect of astrocyte made local synaptic effective range remain in an appropriate length at critical period, as one can observe in Tables 1, 3, 4 and 5. Else some synapses have longer length and disorder, resulting in the alignment of other synaptic effective ranges, as one can observe in Table 2. The simulations in Tables similar to that failure to the closure of critical period results in neurodevelopmental disorders, and then astrocytes become mature [19]. The calculation of the correlation coefficient was better than that of Tables 2, 4 and 5 but worse than Table 3 at critical period, as model of Deep Learning contains both astrocytic synapse formation memory persistence factor and astrocytic brain plasticity phagocytic synapses factor in Table 1 and Fig.10(b).

Table 2 shows that the local accumulation of synapse length is accompanied by Vanishing Gradients by Fig.9 and Tables. It can be seen from Table 2 that activity of synapse formation gradually diminishes with iterations and growth.
of the \( l_{\text{min}} \), the calculation of the correlation coefficient in Table 2 also turns out worse than in other Tables.

For Table 3 which considers only phagocytic synapses factor, synapse formation showed good activity with iterations and \( l_{\text{min}} \) growth. We can find better results of correlation coefficients in Tables respectively. As the probability of Vanishing Gradients is small when considering phagocytic synapses factor by Fig.11(c) and Tables, the result of Table 3 converges even at \( l_{\text{min}} = 5 \), while that of other Tables converge poorly at \( l_{\text{min}} = 5 \).

Table 4 only deals with the positive memory persistence factor, which maintains the slow change in synapse length, and the synapse formation shows a worse activity with iterations and minimum synaptic effective range growth than in Tables 1-3 and 5. The Vanishing Gradients of the positive memory persistence factor is worse than that of Tables 1, 3 and 5, and better than that of Table 2 by Fig.12(d) and Tables.

Table 5 deals with the positive and negative memories persistence factor, which maintains the active change of synapse length, and the synapse formation shows a better activity with iterations and minimum synaptic effective range growth than in Tables 1 and 3. The Vanishing Gradients of the positive
Fig. 9 ORPNN test results are $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively (a) ORPNN

and negative memories persistence factor is worse than that of Tables 1 and 3, and better than that of Table 2 and 4 by Fig. 13(e) and Tables.

When $l_{\text{min}} \approx l_{\text{max}}/k_{\text{max}} = 44/9 \approx 5$, similar to the case of CRPNN, it can be seen from Tables 1-5 that the correlation coefficient effect is not so good at this time, and even Tables 1, 2, 5 is less ideal.

ORPNN contains both astrocytic synapse formation positive memory persistence factor and astrocytic brain plasticity phagocytic synapses factor-ORPNN-MF(P)-PF. We propose the simulation of $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively, and the correlation coefficients are 0.9645, 0.9464, 0.9242 and 0.8734 respectively. ORPNN contains neither astrocytic synapse formation memory persistence factor nor astrocytic brain plasticity phagocytic synapses factor at the closure of critical period-ORPNN, and the correlation coefficients are 0.8730, 0.9282, 0.8846 and 0.8579 respectively. ORPNN contains both astrocytic synapse formation positive memory persistence factor with positive memory - ORPNN-MF(P), and the correlation coefficients are 0.9382, 0.9265, 0.9115 and 0.8663 respectively. ORPNN only
Fig. 10 ORPNN test results are \( l_{\text{min}} = 2 - 5 \) and \( k_{\text{max}} = 9 \) respectively (b) ORPNN-MF(P)-PF contains astrocytic synapse formation memory persistence factor with negative and positive memories ORPNN-MF(P&N), and the correlation coefficients are 0.9592, 0.9423, 0.9228 and 0.8878 respectively. Different \( l_{\text{min}} \) affects simulations can be observed in Fig.9-13. The number of iterations is \( j_{\text{max}} = 80000 \).
Fig. 11 ORPNN test results are $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively (c) ORPNN-PF

Table 1 Synaptic effective range with different $l_{\text{min}}$ and different variables - brain plasticity ORPNN-MF(P)-PF, test results are $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively

| S.L. | S.1 | S.2 | S.3 | S.4 | S.5 | S.6 | S.7 | S.8 | S.9 | Std | correlation coefficients |
|------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|--------------------------|
| $l_{\text{min}} = 2$ |
| V.1 | 5555 | 4444 | 5555 | 6666 | 6666 | 4444 | 5555 | 6666 | 4444 | 1.2693 | 0.9641 |
| V.2 | 5555 | 5555 | 5555 | 4444 | 5555 | 5555 | 6666 | 5555 | 4444 | 0.6009 | 0.9683 |
| V.3 | 4555 | 6666 | 4444 | 5555 | 5555 | 4444 | 5555 | 5555 | 5555 | 1.2693 | 0.9683 |
| V.4 | 4444 | 4444 | 5555 | 5555 | 5555 | 6666 | 4444 | 5555 | 4444 | 0.6009 | 0.9683 |
| $l_{\text{min}} = 3$ |
| V.1 | 5555 | 5555 | 4444 | 5555 | 6666 | 5555 | 5555 | 4444 | 5555 | 5555 | 0.7817 | 0.9409 |
| V.2 | 5555 | 6666 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.7817 | 0.9456 |
| V.3 | 6555 | 5555 | 5555 | 4444 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.7817 | 0.9456 |
| V.4 | 5444 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.9464 |
| $l_{\text{min}} = 4$ |
| V.1 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.9464 |
| V.2 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.9464 |
| V.3 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.9464 |
| V.4 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.9464 |
| $l_{\text{min}} = 5$ |
| V.1 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.8759 |
| V.2 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.8759 |
| V.3 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.8759 |
| V.4 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.8759 |

When $l_{\text{min}} = 5$, $S.9 = 4 < (l_{\text{min}} = 5)$ as a result of condition of constraint: sum of synaptic effective ranges is constant $= 44$ and reflects synaptic strength rebalance.
Fig. 12 ORPNN test results are \( l_{\text{min}} = 2 - 5 \) and \( k_{\text{max}} = 9 \) respectively. (d) ORPNN-MF(P)

Table 2 Synaptic effective range with different \( l_{\text{min}} \) and different variables-brain plasticity ORPNN, test results are \( l_{\text{min}} = 2 - 5 \) and \( k_{\text{max}} = 9 \) respectively

| \( l_{\text{min}} \) | S.1 | S.2 | S.3 | S.4 | S.5 | S.6 | S.7 | S.8 | S.9 | Std | correlation coefficients |
|-----------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|--------------------------|
| \( l_{\text{min}} = 2 \) | \* | \* | \* | \* | \* | \* | \* | \* | \* | \* | 0.3333 | 0.8579 |
| \( l_{\text{min}} = 3 \) | \* | \* | \* | \* | \* | \* | \* | \* | \* | \* | 0.3333 | 0.8579 |
| \( l_{\text{min}} = 4 \) | \* | \* | \* | \* | \* | \* | \* | \* | \* | \* | 0.3333 | 0.8579 |
| \( l_{\text{min}} = 5 \) | \* | \* | \* | \* | \* | \* | \* | \* | \* | \* | 0.3333 | 0.8579 |

2 We discovered the synaptic effective ranges are larger at local, resulting in the alignment of other synaptic effective ranges.

3 Vanishing Gradients
Fig. 13 ORPNN test results are $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively. (e)ORPNN-MF(P&N)

Table 3 Synaptic effective range with different $l_{\text{min}}$ and different variables-brain plasticity ORPNN-PF, test results are $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively

| V.L. | S.1 | S.2 | S.3 | S.4 | S.5 | S.6 | S.7 | S.8 | S.9 | Std | correlation coefficients |
|------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|--------------------------|
| $l_{\text{min}} = 2$ |      |     |     |     |     |     |     |     |     |     |                          |
| V.1  | 3444 | 4444 | 7444 | 6666 | 7777 | 6666 | 4444 | 6666 | 4433 | 1.4340 | 0.9472                   |
| V.2  | 7677 | 4444 | 7777 | 4444 | 6556 | 5444 | 4444 | 4544 | 5554 | 1.3642 | 0.9497                   |
| V.3  | 6666 | 6666 | 5555 | 7777 | 6666 | 5555 | 4444 | 4444 | 4444 | 1.4530 | 0.9527                   |
| V.4  | 3333 | 4444 | 7777 | 5555 | 5444 | 5555 | 7777 | 5555 | 4433 | 1.5366 | 0.9537                   |
| $l_{\text{min}} = 3$ |      |     |     |     |     |     |     |     |     |     |                          |
| V.1  | 4444 | 4444 | 6666 | 6677 | 5555 | 6666 | 5555 | 6666 | 4433 | 1.2693 | 0.9672                   |
| V.2  | 4444 | 4544 | 5555 | 6555 | 5555 | 5555 | 6666 | 5555 | 5555 | 0.6009 | 0.9686                   |
| V.3  | 6666 | 4444 | 4444 | 5555 | 6666 | 5555 | 5555 | 5555 | 5555 | 0.7817 | 0.9681                   |
| V.4  | 4444 | 4544 | 4444 | 6366 | 6666 | 5555 | 5655 | 5555 | 5555 | 0.7817 | 0.9700                   |
| $l_{\text{min}} = 4$ |      |     |     |     |     |     |     |     |     |     |                          |
| V.1  | 5555 | 4444 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.7817 | 0.9527                   |
| V.2  | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.9218                   |
| V.3  | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.9218                   |
| V.4  | 4444 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.9326                   |
| $l_{\text{min}} = 5$ |      |     |     |     |     |     |     |     |     |     |                          |
| V.1  | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.8992                   |
| V.2  | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.8992                   |
| V.3  | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.8992                   |
| V.4  | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 5555 | 0.3333 | 0.8992                   |
Table 4  Synaptic effective range with different \(l_{\text{min}}\) and different variables-brain plasticity ORPNN-MF(P), test results are \(l_{\text{min}} = 2 - 5\) and \(k_{\text{max}} = 9\) respectively

| L.V. | S.1 | S.2 | S.3 | S.4 | S.5 | S.6 | S.7 | S.8 | Std | correlation coefficients |
|------|-----|-----|-----|-----|-----|-----|-----|-----|-----|---------------------------|
| \(l_{\text{min}} = 2\) | V.1 | 555 | 444 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9414                    |
|       | V.2 | 555 | 444 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9350                    |
|       | V.3 | 555 | 444 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9302                    |
|       | V.4 | 555 | 444 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9382                    |
| \(l_{\text{min}} = 3\) | V.1 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9417                    |
|       | V.2 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9352                    |
|       | V.3 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9335                    |
|       | V.4 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9265                    |
| \(l_{\text{min}} = 4\) | V.1 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9444                    |
|       | V.2 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9433                    |
|       | V.3 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9333                    |
|       | V.4 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9115                    |
| \(l_{\text{min}} = 5\) | V.1 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9434                    |
|       | V.2 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.8333                    |
|       | V.3 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.8738                    |
|       | V.4 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.8663                    |

Table 5  Synaptic effective range with different \(l_{\text{min}}\) and different variables-brain plasticity ORPNN-MF(P&N), test results are \(l_{\text{min}} = 2 - 5\) and \(k_{\text{max}} = 9\) respectively

| L.V. | S.L. | S.1 | S.2 | S.3 | S.4 | S.5 | S.6 | S.7 | S.8 | S.9 | Std | correlation coefficients |
|------|------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|---------------------------|
| \(l_{\text{min}} = 2\) | V.1 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9650 | 0.9417                  |
|       | V.2 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9650 | 0.9650                  |
|       | V.3 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9650 | 0.9650                  |
|       | V.4 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9650 | 0.9650                  |
| \(l_{\text{min}} = 3\) | V.1 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9417 | 0.9359                  |
|       | V.2 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9415 | 0.9415                  |
|       | V.3 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9415 | 0.9415                  |
|       | V.4 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9415 | 0.9415                  |
| \(l_{\text{min}} = 4\) | V.1 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9433 | 0.9068                  |
|       | V.2 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9333 | 0.9068                  |
|       | V.3 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9333 | 0.9068                  |
|       | V.4 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9333 | 0.9228                  |
| \(l_{\text{min}} = 5\) | V.1 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.9433 | 0.8894                  |
|       | V.2 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.8894 | 0.8894                  |
|       | V.3 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.8894 | 0.8894                  |
|       | V.4 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 555 | 0.8894 | 0.8894                  |
4 A simple PNN

A simple PNN—in terms of plasticity, only correlation coefficient and synaptic phagocytosis are considered rather than gradients update. Here, the PNN is simplified to only consider the synaptic phagocytosis formula (4) with updated synaptic effective range weight and formula (1) with updated shared connection weight.

Two approaches are proposed: one approach is to evaluate the \( f(t) \) and \( h(t) \) correlation coefficient of \( r(m, k) \) (synaptic effective range weight) corresponding to the time range, each variable and synaptic position corresponding to one time range \( t \) and \( t \in [n_3(m, k), n_3(m, k + 1)] \), and then considering whether or not to cancel the update of the previous-generation \( r(m, k) \) depending on whether current correlation coefficient is improved compared to the previous generation; the other approach favors not processing \( r(m, k) \) at all.

By comparing Table.6-7 and Fig.14-15, the author found that first approach garners better simulation results, and the processing helps activate the synaptic effective range that varies with iteration.

![Graph](image)

**Fig. 14** simple ORPNN-PF without correlation coefficient correction, test results are \( l_{\text{min}} = 2 - 5 \) and \( k_{\text{max}} = 9 \) respectively.
Fig. 15 simple ORPNN-PF with correlation coefficient correction, test results are $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively.
Table 6  Synaptic effective range with different $l_{\text{min}}$ and different variables-brain plasticity simple ORPN-PF without correlation coefficient correction, test results are $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively

| L.V. | S.1 | S.2 | S.3 | S.4 | S.5 | S.6 | S.7 | S.8 | S.9 | Std | correlation coefficients |
|------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|--------------------------|
| l_{\text{min}} = 2 | V.1 | 555 | 444 | 333 | 444 | 777 | 444 | 666 | 444 | 577 | 1.2693 | 0.9404 |
|     | V.2 | 555 | 444 | 333 | 444 | 666 | 999 | 444 | 666 | 444 | 1.9003 | 0.9473 |
|     | V.3 | 544 | 333 | 333 | 444 | 788 | 655 | 555 | 555 | 588 | 1.7038 | 0.9421 |
|     | V.4 | 333 | 544 | 444 | 555 | 655 | 555 | 555 | 555 | 588 | 1.3642 | 0.9525 |

Table 7  Synaptic effective range with different $l_{\text{min}}$ and different variables-brain plasticity simple ORPN-PF with correlation coefficient correction, test results are $l_{\text{min}} = 2 - 5$ and $k_{\text{max}} = 9$ respectively

| L.V. | S.1 | S.2 | S.3 | S.4 | S.5 | S.6 | S.7 | S.8 | S.9 | Std | correlation coefficients |
|------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|--------------------------|
| l_{\text{min}} = 2 | V.1 | 555 | 444 | 333 | 444 | 777 | 444 | 666 | 444 | 577 | 1.7638 | 0.9427 |
|     | V.2 | 555 | 444 | 333 | 444 | 666 | 999 | 444 | 666 | 444 | 1.6159 | 0.9454 |
|     | V.3 | 444 | 544 | 444 | 788 | 655 | 555 | 555 | 555 | 588 | 0.9473 | 0.9084 |
|     | V.4 | 333 | 544 | 444 | 555 | 655 | 555 | 555 | 555 | 588 | 0.9421 | 0.9525 |

5 Mechanism, Interpretability and findings

In addition to the shared weights of synaptic connections, we proposed a new Neural Network that includes weights of synaptic ranges for Forward propagation and Back propagation. And it gave a unified model of brain plasticity and synapse formation. And using more simulations to compare with the experiments which RNN cannot be achieved [14–25].

The mechanism of PNN enables the appropriate synapse to change the effective range for dendrite morphogenesis at critical period. When one synapse transfers a signal from one neuron to other with a better stimulation signal, PNN will change the current synaptic effective range based on Mean Squared Error (MSE) of loss function, with reference to current and mnemonic brain plasticity, current gradient informational and mnemonic positive and negative gradient informational synapse formation, and vice versa for the same reason.

In terms of the infectious disease dynamics, the infection rates of primitive strains and Delta strains varies in different time ranges, and the time interval corresponding to the cure rate will not be in line with the time interval corresponding to the infection rate, nor will the cure rate correspond exactly to the
synaptic effective range effect of the infection rate, due to its high dependence on the medical condition.

Explaining in terms of synaptic competition, PNN is well-positioned to calculate the time ranges corresponding to the cure and infection rates, i.e., the synaptic effective ranges, through data processing, and then obtain the appropriate cure and infection rates, i.e., the weights of synaptic connections. During the emergency and non-emergency period, i.e., critical period and closure of critical period, astrocytic effect is similar to government policies such as lockdown and mask rules, thereby affecting infection rate, cure rate and time ranges corresponding to them.

The optimization of synaptic effective range is also similar to the Residual Neural Network (ResNet)[12], as more layers don’t necessarily perform better in training than fewer layers, and the number of layers is determined by training the synaptic effective range by gradient update with iterations.

By previous discussion’s comparisons, we can summarize as follows:

PNN considers synaptic strength balance in dynamic of phagocytosing of synapses and static of constant sum of synapses length, it implements rebalancing strength between synapses[14]. From input to output units, neurons go through hidden layers, and when the lead synaptic effective range changes at critical period, these neurons, like a school of fish, also affect the postsynaptic neurons.

Synapse formation will inhibit dendrites generation to a certain extent in experiments, by simulations synapse formation will inhibit the function of dendrites such as receiving signals then affecting cognition [15]. The effects of phagocytosing of synapses also help to synaptic growth. Because of rebalancing of synaptic strength, the phagocytosing of synapses makes neighboring synapses easily to grow. High IQ will have more neurons and their actions process fast, but synapse formation will inhibit those. Because of the rebalancing strength from input to output units, synapse formation leads to less neurons, and longer distance between neurons causes processing slower.

The Alcino J Silva’s team suggested that memory ensembles by a retrograde mechanism [16]. Our research gave retrograde formula in preprint arXiv:2203.11740 version 1, it used mnemonic gradient of the best previous synaptic effective range weight to update current gradient of synaptic effective range weight, it employs the retrograde mechanism, the mnemonic best previous gradient is positive, and current gradient is negative. Mnemonic gradient of the best previous synaptic effective range weight may be stored in the memory engram cells. And preprint version 2, it makes more sense the best previous memory retrieval process employs a retrograde mechanism through derivation of formula. The preprint version 3 considered the mnemonic relatively inferior gradient of synaptic effective range weight by a retrograde mechanism.

The Alvarez-Buylla’s lab proposed that human hippocampal neurogenesis drops sharply in children to undetectable levels in adults [17]. While PNN’s simulations consider synapse formation, synapse formation leads to the decrease of neurogenesis, through the process from infancy to the senile phase.
But another study claimed human hippocampal neurogenesis persists throughout aging [18]. We guess if it may activate a new and longer circuit (a larger $l_{\text{max}}$) in late iteration, even if synapse formation happens then decrease of neurons in early iteration, so a new circuit may lead to neurogenesis persists and may increase neurons in senile phase.

Closing the critical period (Such as astrocytic memory persistence or astrocytic phagocytosis at critical period) will cause neurological disorder in experiments, but worse results in PNN simulations [19].

The positive and negative mnemonic synapse formation formula which considers gradient information of retrograde circuit, although it has same effects like brain plasticity phagocytosis in simulation comparing with experiments. The memory persistence gradient information of retrograde circuit similar to the Enforcing Resilience in a Spring Boot, in addition to considering the mnemonic brain plasticity of architecture. The relatively good and inferior gradient information in synapse formation of retrograde circuit like the folds of the brain. Only considering positive (best previous gradient information) memory persistence in simulation will inhibit synapse length changes with iterations. The memory of fear learning (or negative memory) can activate Synapses and observed obviously. By simulations, the PNN gave the two cases with and without negative memories. The brain plasticity was much easier to restore without negative memories, and the paper proposed $\alpha$7-nAChR-dependent astrocytic responsiveness is an integral part of the cellular substrate underlying memory persistence by impairing fear memory [20].

Astrocytic phagocytosis will avoid the local accumulation of synapses by simulation (Lack of astrocytic phagocytosis causes excitatory synapses and functionally impaired synapses accumulate in experiments and lead to destruction of cognition, but local longer synapses and worse results in PNN simulations) [21].

The brain cortex may be the space-time accumulation of the loss function. And the thicker cortex and more diverse individuals in brain may have high IQ in simulations and experiments [22].

The weights of synaptic ranges’ mnemonic negative and positive gradient information (or mnemonic relatively good or inferior gradient information) is not mnemonic brain plasticity, it used stimulus information to update brain plasticity. The stimulus information may be stored in the memory engram cells for inhibiting and inducing synapses and updating synaptic strength [23].

Based on principles of Optics, the relatively good or inferior gradient information’s location is near the core of lens, and signals from input to output units with smaller losses go through core of lens. So different cortexes’ relatively good and inferior gradient information is stimulated by stronger signals. And the relatively good or inferior gradient information may reflect improved memory retrieval process. And paper found that 1064-nm Transcranial photobiomodulation (tPBM) applied to the right prefrontal cortex (PFC) improves visual working memory capacity [24]. The effects of PNN (memory structure)
and tPBM (longer wavelengths) may be the same, powerful penetrability of signals for improving memory capacity.

The Trinity College Institute of Neuroscience’s findings may have witnessed entanglement mediated by consciousness-related brain functions, the consciousness-related or electrophysiological signals are unknown in NMR. Those brain functions must then operate non-classically, which would mean that consciousness is non-classical [25]. So PNN introduces mnemonic brain plasticity by quantum computing in global brain, but normal computing in details by Deep Learning, Gradient Method and Statistics in gradient of synaptic connections, gradient of effective ranges, astrocytic memory persistence by memory engram cells, astrocytic phagocytosis, different types neurons and synapses.

The 3 findings were as follows:
1. Memory persistence factor also inhibits local synaptic accumulation, and the model inspires experiments.
2. But the thickest cortex and the most diverse individuals in brain may have low IQ in simulation, so the PNN can guide the experiments, and experiments are special situations of models. And our guess is that more advanced cortexes near the core of brain.
3. It may be the process of phagocytic synapses is driven by positive and negative memories of brain plasticity, based on $r(m,k) = r(m,k) + \text{rand} \times [R + Q_N + Q_P - r(m,k)] + P$ in formula (4).

6 Conclusion

1 Based on the RNN architecture, we accomplished the model construction, formula derivation and algorithm testing for PNN. We elucidated the mechanism of PNN based on the latest MIT research on synaptic strength rebalance, and also grounded our study on the basis of findings of the Stanford research, which suggested that synapse formation is important for competition in dendrite morphogenesis. The effects of astrocyte impact on brain plasticity and synapse formation is an important mechanism of our Neural Network at critical period or the closure of critical period. In addition to formula (1) and (2), which is derived from Back Propagation and features the synaptic shared connection weight and the effective range weight respectively. We also managed to figure out synaptic competition by current gradient informational and mnemonic positive and negative gradient informational synapse formation at critical period through formula (2), synaptic strength rebalance through formula (3) and (4), and current and mnemonic brain plasticity at critical period through formula (4). However, our Neural Network is no longer confined to the simple physical concept of synaptic strength rebalance - it treats synapses as a group of intelligent agents to achieve synaptic strength rebalance.

2 The mnemonic positive and negative gradient informational synapse formation needs to consider the forgotten memory-astrocytic synapse formation
memory persistence factor. Mnemonic brain plasticity involves plus or minus disturbance-astrocytic brain plasticity phagocytic synapses factor, thus enabling dynamic synaptic strength balance. The effect of astrocyte made local synaptic effective range remain in an appropriate length at critical period. We give decreasing weights of factors at critical period. We try to deduce the mechanism of failure in brain plasticity by model at the closure of critical period in details by contrasting with previous studies. The simulation in Tables similar to that failure to the closure of critical period results in neurodevelopmental disorders[19]. The findings of our study proved meaningful, which revealed that much like astrocytic phagocytic synapses factor, memory persistence factor also inhibit local synaptic accumulation [21]. The memory persistence factor obtained by the simulation of the mathematical model also inhibits the local accumulation of synapses.

3 Test results of the 3 scenarios all concern mnemonic the best previous solution. All the PNNs with gradient-optimized synaptic effective range fared better than those with random synaptic effective range, which in turn outperformed PNNs with constant synaptic effective range. Constant synaptic effective range is achieved when the minimum synaptic effective range grows to coincide with the maximum synaptic effective range, which usually takes place in the cognition of the senile phase. The iteration of CRPNN is difficult to convergence due to a lack of plasticity and diversity, and RRPNN demonstrates slow convergence in testing.

4 We believe our work is of profound significance for the development of NN research. Furthermore, PNN takes into account the mnemonic positive and negative gradient informational synapse formation, and brain plasticity and synapse formation change architecture of NN is a new method of Deep Learning. We can obtain better results in Tables of correlation coefficients respectively, when ORPNN contains both astrocytic synapse formation memory persistence factor and astrocytic brain plasticity phagocytic synapses factor at critical period. Both mnemonic the best previous gradient information, the relatively good gradient information and the relatively inferior gradient information are considered, which increases the activity of the synaptic effective range with iteration changes and thereby improves the simulation results. The relatively good and inferior gradient information in synapse formation of retrograde circuit like the folds of the brain[16]. And we discussed the relationship of human intelligence and cortical thickness, individual differences in brain by simulations and experiments, and PNN can guide the experiments, and experiments are special situations of models[22]. Our model PNN fits very well with the memory engram cells that strengthened Synaptic strength [23]. The effects of PNN’s memory structure and longer wavelengths of tPBM may be the same, powerful penetrability of signals for improving memory capacity [24].

5 In addition to the shared weights of synaptic connections, Forward propagation and Back propagation also include weights of synaptic ranges. We
developed the concept of weights for NN and divided the weights into categories, namely weights of synaptic shared connection and effective range. The latter reflects synaptic competition, strength rebalance, current and mnemonic brain plasticity and current gradient informational and mnemonic negative and positive gradient informational synapse formation at critical period. From input to output units, they go through hidden layers, and when the lead synaptic effective range changes at critical period, these neurons, like a school of fish, also affect postsynaptic neurons.

6 We also attempted to simulate PNN cognition process from infancy to the senile phase, and by analyzing relationship between different synapse formation and correlation coefficients. We may reasonably assume that the minimum synaptic effective range increases and synapse population decreases for synapse formation of senile phase.

7 In the next step of our study, we took the real pandemic data into consideration, and PNN may well be used to process the pandemic data of the original strain and the variant Delta strain, in an effort to optimize the time interval of the synaptic effective range, and then different connection weights will be applied for cure and infection rates. We will calculate the connection weights by PNN, and the synaptic effective range, based on which we then formulate contingency plans for different period.

8 By testing, we made a similar observation to that of Dr. Luo’s team through PNN: synapse formation to a certain extent is detrimental to dendrites, and synaptic formation leads to a reduction in changes of the synaptic effective range, therefore disrupting diversity and plasticity, including results considering memory persistence factor and phagocytic synapses factor separately.

9 The synaptic competition mechanism NN is suitable for RNN architecture as well as BP, and our next step is to conduct research on the implementation of synaptic competition, strength rebalance and critical period by BP.

10 In simulation, if a same type of shared connection weight changes, all the weights of effective ranges may be consequently updated for better results.

11 To be honest, the effects of astrocyte affect on brain plasticity and synapse formation was inspired by Particle Swarm Optimization (PSO) [35] and consolidation of concrete, and critical period of brain plasticity really depends on inertia weight of PSO [36]. Only synapse formation affects on brain cognition refers biological tests of neuroscience [15], but also is relevant to the FEM. Synaptic strength rebalance is actually inspired by the conservation of energy. The question we proposed is whether the promotion of computational neuroscience and brain cognition was achieved by model construction, formula derivation or algorithm testing, and whether we can reduce the number of animal experiments and their suffering through the guidance of model simulation planning. We resorted to the Artificial Neural Network (ANN), Evolutionary Computation and other numerical methods for hypotheses, possible explanations and rules, rather than only biological experiments.
12 In formula (2), the PNN gradient information considering both positive and negative memories outperforms the case where only positive memory is considered, and the synaptic effective range of the former is more active with iteration changes, yielding better optimization results.

13 We simplified the PNN to only consider the update of synaptic phagocytosis rather than the gradient update, and decided whether to cancel the update of synaptic phagocytosis based on the correlation coefficient corresponding to the time range between the current and previous generation, each variable and synaptic position corresponding to one time range. The model considering whether or not to cancel the phagocytic update which yields better simulation results than model not processing, and the synaptic effective range is more active with iterations.

14 Particle Swarm Optimization (PSO) considers the global optimal solution and the best previous solution to update the velocity of particles-2 parameters, and refers PNN can also introduce the relatively good and the relatively inferior solution to update the velocity of particle-4 parameters.

15 In formula (2) and (4), we suggest the PNN is not only a classical normal computer in synapse formation and brain plasticity, but also a non-classical quantum computer in brain plasticity in extracted relatively good or inferior memories. And relatively good or inferior memories both have exponential decay. When you are considering one problem, whether positive or negative, the brain plasticity turns to quantum and satisfies exponential decay.

16 The process of phagocytic synapses may both have quantum computing and traditional computing, and may be the process of phagocytic synapses is driven by positive and negative memories of brain plasticity, through interactions of excitatory and inhibitory transmitters.

### Author contributions

Conceptualization, J.B.T., B.Q.S., Y.X., J.Q.L and C.W., Formal analysis, J.B.T., B.Q.S., W.D.Z., S.Y.Q., L.K.C., and Y.X., Funding acquisition, C.W., and B.Q.S.; Investigation, J.B.T., C.W., B.Q.S., L.K.C., Y.X., J.Q.L and S.Y.Q.; Methodology and Coding, J.B.T., Supervision, B.Q.S., Y.X., J.Q.L, C.W., S.Y.Q., and W.D.Z.; Writing-original draft, J.B.T., J.X.Z., and L.K.C.; Writing-review & editing, W.D.Z. B.Q.S., J.Q.L, L.K.C., and Y.X.; All authors have read and agreed to the published version of the manuscript.

### Competing interests

The authors declare no competing interest

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