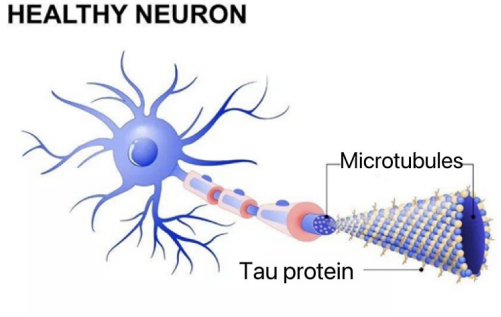
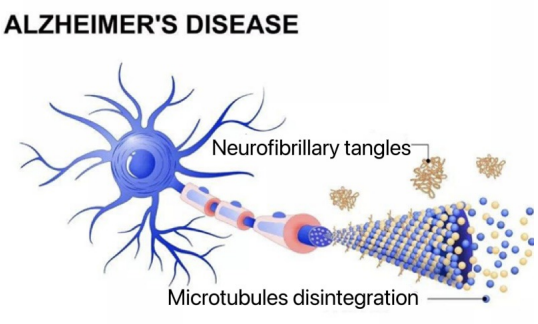
1. Status and Mechanisms

There are four common categories of senile dementia: 1. Cognitive decline due to neurodegenerative changes, such as Alzheimer's disease (AD). 2. Cognitive decline caused by non-degenerative diseases of the nervous system, such as vascular dementia. 3. Some treatable causes of cognitive decline, such as cognitive decline caused by nutritional deficiencies. Depression can also lead to reversible dementia. 4. Mixed or other dementia for which no cause can be found.

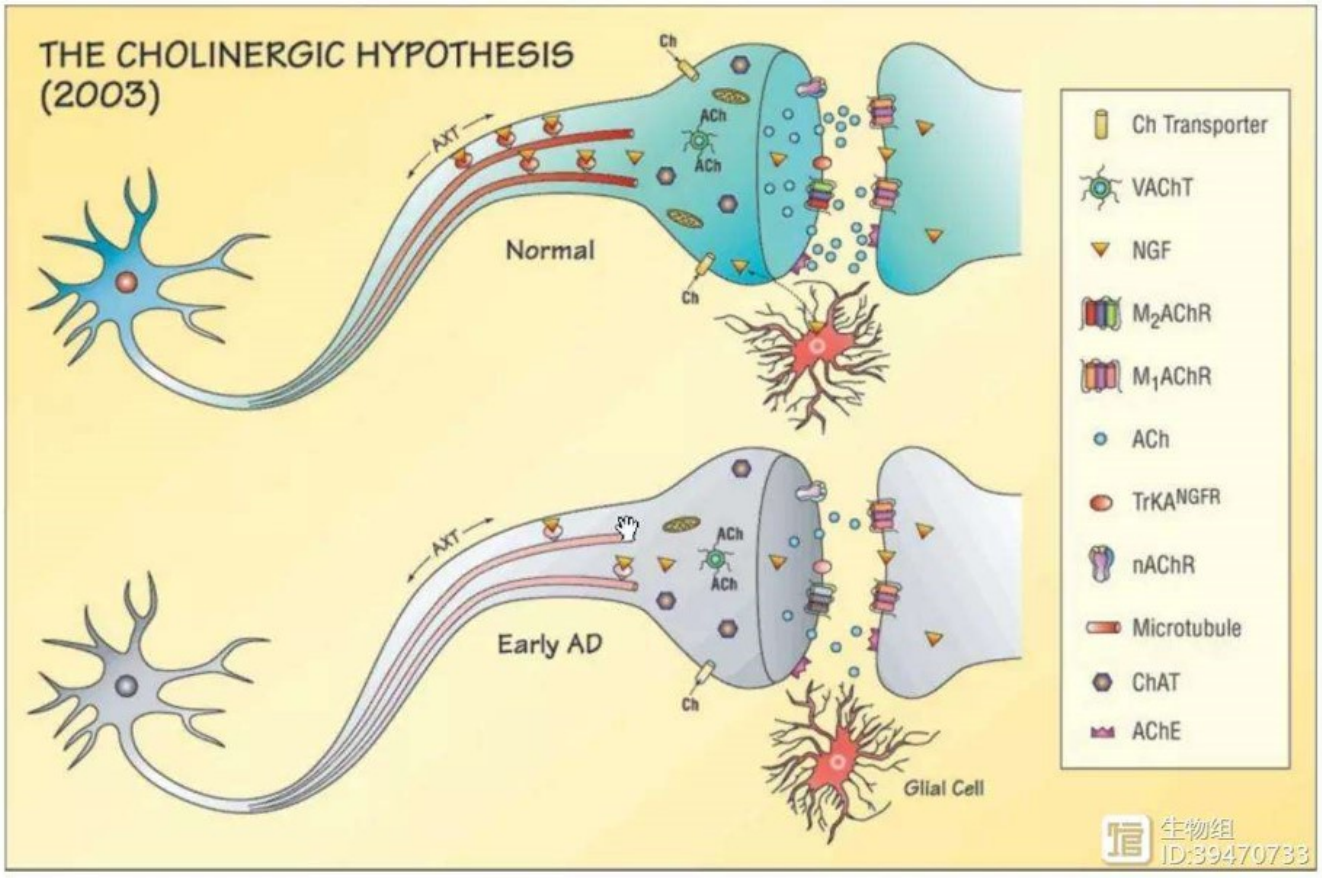
At present, there are 264 million elderly people over 60 years old in China. There are about 15.07 million senile dementia patients, of which 9.83 million are AD patients. On average, six out of every 100 elderly people suffer from senile dementia, and it is expected to reach 28.98 million in 2050. The number of senile dementia patients in China accounts for one quarter of the global total.

Aβ deposition is one of the most typical pathological features of senile dementia and plays a key role in the occurrence and development of senile dementia. Aβ is derived from amyloid precursor protein (APP). Normally, Aβ can be removed by mechanisms such as extracellular degradation, endocytosis or transport clearance. In AD patients, the above-mentioned Aβ clearance mechanism is hindered, resulting in excessive accumulation of Aβ in the brain. The neurotoxicity of Aβ is closely related to the degree of aggregation. Compared with monomeric and fibrotic Aβ, soluble and smaller Aβ Molecular oligomers are more neurotoxic. Aβ protein oligomerization activates glial cells and astrocytes, triggers an inflammatory response, induces abnormal phosphorylation of tau protein and neurofibrillary tangles, damages synapses and neurons, and eventually leads to neuronal apoptosis and dementia.





In addition, common explanation mechanisms include the theory of cholinergic deficiency, the theory of oxidative stress, and calcium homeostasis disorders. The theory of cholinergic deficiency believes that AD is caused by the lack of neurotransmitters such as acetylcholine (ACh) in the human brain, and the key reason for the lack of choline is the excessive content of acetylcholinesterase. The oxidative stress theory believes that because mitochondria are the target organelles of Aβ cytotoxicity, Aβ can specifically damage mitochondrial ETC through the action of transporters, causing irreversible damage, reducing the overall ATP level in cells, and promoting neuronal apoptosis. The calcium homeostasis dysregulation theory holds that persistent calcium disturbances in cells are the main cause of neurodegeneration including AD. In the nervous system, Ca2+ plays a fundamental role in regulating neuronal survival and plasticity. Due to excitotoxic stimulation and Aβ stimulation, intracellular calcium overload leads to apoptosis.



2. How to improve memory

The current common treatment methods include drug therapy and novel intervention. Drug therapy is mainly aimed at the above pathological mechanisms, and the pathological changes are improved through anti-Aβ drugs, cholinesterase inhibitors and other drugs. New approaches include targeted Aβ immunotherapy, Tau protein immunotherapy, metallomic therapy, etc.

3. Product recommendation

3.1 Ginseng Extract

Ginseng extract contains active components of ginsenosides, which affect calcium ion homeostasis, activate autophagy, protect nerve cells, and relieve apoptosis.

3.2 Wheat Germ Extract

Wheat germ contains a certain amount of spermidine, which can induce cytoprotective autophagy and maintain mitochondrial genome stability and mitochondrial membrane potential, inhibit the activation of apoptosis signals, improve mitochondrial function, and improve oxygen utilization.

3.3 Reishi Mushroom Extract

Ganoderma lucidum is rich in Ganoderma lucidum triterpenes and polysaccharides. Ganoderma lucidum triterpenes can inhibit AChE to maintain ACh levels and improve mitochondrial function. Ganoderma lucidum polysaccharide significantly reduces the production of malondialdehyde and reactive oxygen species by increasing the activity of neurons after hypoxia/reoxygenation treatment, and improves the activity of antioxidant enzymes.