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Experimental Study on the Modeling of Alzheimer's Disease in Mice with Aluminum Maltol and the Treatment of Tremella Fuciformis and Its Compound Polysaccharides

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Abstract: *In this study, the pathological changes of human AD patients were simulated by using aluminum maltol to model Alzheimer's disease (AD) in mice. This study established an Alzheimer's disease (AD) mouse model using aluminum maltol to mimic pathological changes in human AD patients. Fifteen healthy mice were randomized into four groups: Normal, Model, Treatment 1 (tremella preparation), and Treatment 2 (good sober preparation). The Model and treatment groups received daily intraperitoneal aluminum maltol injections for 57 days, with oral administration of test agents initiated on day 39. Cognitive function was evaluated via the Y-shaped water maze test[1], while serum biomarkers (triglycerides, total cholesterol, total protein, urea nitrogen) and brain homogenate enzymes (acetylcholinesterase, β1/a secretase) were measured. Revealed significant cognitive impairment and biomarker alterations in the Model group compared to the Normal group (P<0.05 for all). Both treatment groups showed improved cognitive performance and reduced neuronal damage markers (P<0.05 vs. Model), characterized by lower β1 secretase and higher a secretase levels. Aluminum maltol successfully induced AD-like symptoms in mice. Tremella fuciformis and its compound polysaccharides exhibited neuroprotective effects by enhancing cognitive function and alleviating neuronal injury, providing experimental evidence for their potential application in AD therapy.*

Keywords: *Alzheimer's Disease; Aluminum Poisoning; B-Amyloid Lyase; Traditional Chinese Medicine Treatment*

1. Introduction

Alzheimer's disease (AD) occurs in middle-aged, elderly, and pre-elderly individuals. It is the result of the combined effects of genes, lifestyles, and environmental factors. It is a neurodegenerative disease of the central nervous system characterized by progressive cognitive impairment and behavioral impairment. The typical histopathological changes are amyloid protein deposition and neuronal fiber tangles in the brain. Clinically, it manifests as cognitive impairments such as memory, language, calculation, perception, and behavior. Aluminum is a chronic metallic neurotoxin, and the brain is the accumulation site and main target organ of aluminum. Al can enter the brain tissue through the blood-brain barrier and accumulate to produce neurotoxic effects. Nerve cells are very sensitive to the toxicity of Al, and Al has a high affinity with brain proteins. Binding with brain proteins leads to protein degeneration, which in turn causes brain tissue atrophy. [2]. At present, the toxic effects of Al on the

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central nervous system have been confirmed by a large number of experimental studies and clinical diagnostic observations. Among them, the relationship between Al and AD is closer than that with other neurological diseases. The main active ingredient in Tremella fuciformis is Tremella polysaccharide, which can increase the activity of superoxide dismutase (SOD), scavenge superoxide anion free radicals, and at the same time, SOD reduces the sensitivity of the body to the adverse environment by scavenging free radicals, and reduces oxidative stress damage.

This study aims to prepare an animal model of nervous system injury by intraperitoneal injection of subchronic aluminum maltol at different doses in mice, and then treat them with Tremella fuciformis and its compound polysaccharides to explore the therapeutic effect of Tremella fuciformis and its compound polysaccharides in the treatment of AD, and further explore the treatment methods of AD.

2. Materials and Methods

2.1 Materials

2.1.1 Experimental Animals

Fifty-seven KM mice (8 weeks old, weighing about 30g) were provided by Changsha Tianqin Biotechnology Co., Ltd., with the license number: SCXK(Xiang)2022-0011, including 37 male mice and 20 female mice, with similar activity ability and a body mass of about 30 grams. They were randomly divided into 4 groups according to their body mass: the normal group (15 mice), the model group (Al3+ 0.3ml/mouse, 15 mice), the treatment group 1 (Al3+ 0.3ml/mouse, Tremella preparation 0.3ml/mouse, 15 mice), and the treatment group 2 (Al3+ 0.3ml/mouse, Tremella compound polysaccharide preparation (Hao Qingxing) 0.3ml/mouse, 15 mice) [3].

2.1.2 Main Reagents

The reagents and kits used in the study included anhydrous aluminum chloride ($AlCl_3$), maltol, 0.9% sodium chloride solution; Mouse α-Secretase ELISA Kit, Mouse β-site Amyloid Precursor Protein Cleaving Enzyme 1 (BACE1, β-Secretase) ELISA Kit, Acetylcholinesterase (AChE) Assay Kit; and the indicators to be detected, such as blood urea nitrogen (BUN), triglyceride (TG), total cholesterol (TCH), and total protein (TP) quantification.

2.1.3 Major Instruments

The SpectraMax M5 microplate reader, micropipettes, high-precision pipettors, thermostatic water bath, centrifuge, and 4°C refrigerator.

2.2 Methods

2.2.1 Toxicant Administration Protocols

Before exposure to the toxin, equal volumes of maltol solution and aluminum trichloride ($AlCl_3$) solution were mixed to prepare aluminum maltol solution, with a pH of 7.1 - 7.4. It was then filtered under reduced pressure and set aside for later use [4]. All the mice were exposed to the toxin for a total of 57 days, and were administered intraperitoneally at a dose of 0.3 ml per mouse. The injection was given continuously for 6 days, followed by a 1-day interval. All the mice had free access to water and food, and were fed with ordinary feed. All the utensils, such as the mouse cages and water bottles, did not contain aluminum products.

2.2.2 Observation of the Basic State of the Animals and Sample Collection

Daily observations of mice for physical signs, abnormal behaviors, fecal characteristics, alopecia, food intake, weight loss, and mortality were performed before drug administration. Before, during, and after the experiment, the Y-maze water maze assay was performed to measure and record swim latency and error frequency; after the experiment, take the eyeball blood, separate the serum, and measure the serum BUN, TC, TG, TP, etc.; sacrifice the mice, take the brain for grinding and homogenization, and measure the acetylcholinesterase (AchE) in the brain homogenate, the total protein (TP) in the brain homogenate, the brain β1 secretase, the brain α secretase, and the brain γ secretase.

2.2.3 Neurobehavioral Experiment

The learning and memory ability of mice was measured by the Y-shaped water maze experiment. One round of the Y-shaped water maze was conducted before, during, and after the exposure, three times a

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day for 5 consecutive days. In the experiment, the mice were gently placed in the water at the end of the long arm, with their tails facing the Y-shaped fork. If the mice reached the platform directly within 40 seconds, it was recorded as a success, and the time used was the escape latency. If the mice failed to find the platform within 40 seconds, it was recorded as a failure, and the escape latency was recorded as 40 seconds. Each time the mice reached the platform, they stayed for 5 seconds and then underwent repeated training. The average escape latency and average error rate of each group were calculated [5].

2.2.4 Preparation of Mouse Brain Tissue Homogenate

Respectively take the brains of the mice, store them in the refrigerator for frozen preservation, and take them out one by one when needed. Rinse the surface blood of the brain tissue with PBS phosphate buffer (0.01M, PH = 7.4), absorb the moisture with filter paper, weigh the brain, and then grind it with a glass homogenizer in an ice bath for 10 minutes. Use PBS phosphate buffer (0.01M, PH = 7.4) to make up the volume to obtain 10% brain homogenate, centrifuge, take the supernatant layer and store it in the refrigerator for later use. Measure the protein content, acetylcholinesterase AchE, α-secretase, β-secretase, and γ-secretase indicators in the homogenate.

2.2.5 Determination methods of main observation indicators

Use various kits and operate according to the instructions to determine the indicators involved in the experiment. α-Secretase, β-secretase, and γ-secretase activities were quantified using enzyme-linked immunosorbent assay (ELISA) with a microplate reader, and enzyme activities of individual samples were automatically calculated by the instrument.

2.3 Statistical methods

Data were processed by SPSS13.0 software and expressed as x ± S. A two-sided $P<0.05$ or two-sided $P<0.01$ was considered statistically significant.

3. Results

After the injection and detection experiment, the number of surviving mice in each group was as follows: 15 in the normal group, 13 in the model group, 14 in treatment group 1, and 15 in treatment group 2.

3.1 Body weight comparisons across toxicant-exposed mouse groups at early, middle, and late stages (see Table 1)

Table 1 Body weight comparisons across different mouse groups (x ± S) (g)

Group	Number of animals	Early stage (g)	Middle stage (g)	Late stage (g)
Normal group	15	31.13 ± 1.14b	41.67 ± 2.69a	46.19 ± 3.51ab
Model group	13	31.16 ± 1.40	37.58 ± 4.94▲a	36.92 ± 6.80▲▲a
Treatment group 1	14	30.77 ± 2.06	39.40 ± 4.05c	40.63 ± 3.74▲c
Treatment group 2	15	31.32 ± 2.31	38.84 ± 3.81a	38.79 ± 6.10▲▲a

Note: Comparison within groups: Comparison of before, during and after: Normal group: The body weight increased continuously; Model group: It increased significantly from the early stage to the middle stage and decreased from the middle stage to the late stage; Treatment group 1: It increased significantly from the early stage to the middle stage and increased to a certain extent from the middle stage to the late stage; Treatment group 2: It increased from the early stage to the middle stage and did not increase from the middle stage to the late stage.

3.2 The test time (s) of the water maze for each group of mice before poisoning, during modeling and in the later stage of modeling. Determination results

The water maze test time in the later stage of modeling for Treatment group 1 and Treatment group 2 was less than that of the Model group (see Table 2).

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Table 2 Comparison of the water maze test time (s) (±S) of each group of mice before poisoning, during poisoning and in the later stage of poisoning

Group	Number of animals	Early stage of poisoning (s)	Middle stage of poisoning (s)	Late stage of poisoning (s)
Normal group	15	4.22±0.62b	3.79±0.78a	4.91±1.14
Model group	13	3.96±0.55▲	5.68±2.01▲▲c	5.45±1.18c
Treatment group 1	14	3.88±0.59▲	5.54±1.56▲▲d	4.85±1.61
Treatment group 2	15	4.53±0.91f	6.65±1.79▲▲	5.16±1.21f

Note: Analysis of variance: Comparison between groups: Compared with Treatment group 2: $P < 0.05$, the difference is statistically significant; Comparison within the group for the early, middle and late stages: Model group: $F = 7.599$, $P = 0.008$, compared with before poisoning: $C < 0.01$, the difference is statistically significant; Treatment group 1: $F = 5.131$, $P = 0.010$, compared with before poisoning: $d < 0.01$, the difference is statistically significant; Treatment group 2, $F = 10.727$, $P = 0.001$, compared with during poisoning: $P < 0.01$, the difference is statistically significant.

3.3 Comparison of the memory overtime rate (%) of each group of mice before, during and after poisoning (see Table 3)

Table 3 Comparison of the memory overtime rate (%) of each group of mice before, during and after poisoning

Group	Number of animals	Before poisoning (%)	During poisoning (%)	After treatment (%)
Normal group	15	0	0	0
Model group	13	0	1.664%	2.879%
Treatment group 1	14	0	2.303%	0.308%
Treatment group 2	15	0	1.640%	0

Note: The overtime rate (%) of the model group increased after treatment, while that of the other groups decreased.

3.4 Brain β-secretase, α-secretase, and γ-secretase activities (U/L) across different mouse groups (see Table 4)

Table 4 Brain β-secretase, α-secretase, and γ-secretase activities (U/L) across different mouse groups (±S)

Group	Number of animals	Brain β-secretase 1	Brain α-secretase	Brain γ-secretase
Normal group	15	11.34±1.24▲	8.48±1.43	10.88±1.98
Model group	13	12.41±0.95	8.32±1.42	10.94±1.17
Treatment group 1	14	11.33±0.50▲▲	8.58±0.92▲	10.85±1.03
Treatment group 2	15	11.26±0.79▲▲	7.61±0.69	9.94±1.04

Note: Analysis of variance: Comparison between groups: For brain β-secretase: $F = 4.148$, $P = 0.012$. Compared with the model group, ▲$P < 0.05$, ▲▲$P < 0.01$, and the difference is statistically significant; For brain α-secretase, $F = 1.849$, $P = 0.155$. Compared with treatment group 2, ▲$P < 0.05$, and the difference is statistically significant; For γ-secretase: $F = 1.827$, $P = 0.154$. There is no statistically significant difference in the comparison between groups.

3.5 Determination results of AchE in the mouse brain, serum urea nitrogen and total protein (see Table 5)

Table 5 Comparison of AchE in the brain, serum urea nitrogen and total protein in each group of mice (±S)

Group	Number of animals	Brain AchE (u/mg.prot)	Urea nitrogen (mmol/L)	Total protein TP (g/L)
Normal group	15	0.45±0.22	7.96±0.03	150.24±9.78▲▲
Model group	13	0.50±0.24	6.72±0.85	145.10±11.31▲
Treatment group 1	14	0.49±0.22	6.33±1.02▲	129.57±20.88
Treatment group 2	15	0.43±0.20	7.54±2.16	144.08±13.46▲

Note: Analysis of variance: Comparison between groups: For brain AchE, $F = 0.196$, $P = 0.898$, and the

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difference is not statistically significant; For serum urea nitrogen, F = 2.623, P = 0.067. Compared with the normal group, ▲P < 0.05, and the difference is statistically significant; For serum total protein: F = 3.380, P = 0.030. Compared with treatment group 1, ▲P < 0.05, ▲▲P < 0.01, and the difference is statistically significant.

3.6 Determination results of serum total cholesterol (TC) and triglyceride (TG) in each group of mice (see Table 6)

The results showed that compared with the serum total cholesterol (TC) of the model group, the serum total cholesterol (TC) of treatment group 1 and treatment group 2 had a statistically significant difference (P < 0.05); compared with the serum triglyceride (TG) of the model group, the serum triglyceride (TG) of treatment group 1 and treatment group 2 had a statistically significant difference.

Table 6 Comparison of serum TC and TG in each group of mice (±S)

Group	Number of animals	TC (mmol/L)	TG (mmol/L)
Normal group	15	2.86±2.13▲▲	1.00±0.50
Model group	13	5.63±3.33	1.32±0.49
Treatment group 1	14	2.67±0.87▲▲	0.83±0.32▲
Treatment group 2	15	2.85±0.79▲▲	1.09±0.33

Note: Compared with the model group, ▲▲P < 0.01, and the difference is statistically significant; For serum TG: F = 2.113, P = 0.117.

3.7 Comparison of brain aluminum Al3+ content (μg/ml) in each group of mice (see Table 7)

Table 7 Comparison of brain aluminum Al3+ content (μg/ml) in each group of mice (±S)

Group	Number of animals	Al3+ content (μg/ml)
Normal group	15	0.22±0.01★
Model group	13	0.30±0.07
Treatment group 1	14	0.24±0.02★
Treatment group 2	15	0.23±0.02★

Note: Analysis of variance: Comparison of brain aluminum Al3+ among groups. Group 1: F = 3.408, p = 0.061. Compared with the model group: ★P < 0.05, and the difference is statistically significant.

3.8 Comparison of free radical () scavenging rate % in brain homogenates of each group of mice (see Table 8)

Table 8 Comparison of free radical () scavenging rate % in brain homogenates of each group of mice (±S) (g)

Group	Number of animals	( )clearance rate (%)
Normal group	15	21.41±3.14★
Model group	13	15.69±3.72
Treatment group 1	14	9.97±5.67▲▲★
Treatment group 2	15	6.53±2.37 ▲▲★★

Note: Analysis of variance: For () scavenging rate %: In comparison between groups, F = 16.719, P = 0.000. Compared with the normal group, ▲▲a < 0.01, and the difference is statistically significant; compared with the model group, ★P < 0.05, ★★a < 0.01, and the difference is statistically significant.

4. Discussion

Aluminum (aluminium, Al) and its chemical products are widely used in daily life. They can enter the human body through various channels such as diet, environmental contact, living habits, and occupational interference. There is obvious accumulation in the body and toxicity is produced. Al has a chronic neurotoxic effect on poultry in the natural environment. In recent years, due to environmental pollution, air pollution, and industrial pollution, the dissolution of Al has increased, increasing the risk of human enrichment of aluminum [6]. The brain is the accumulation site and main organ of action of Al. Al can enter the brain tissue through the blood-brain circulation barrier and accumulate and produce

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neurotoxic effects. Brain nerve cells are very sensitive to Al toxicity. Al has a high affinity with brain proteins. Binding with brain proteins leads to protein denaturation and then brain tissue atrophy. Al interferes with the memory and cognitive functions of the brain in the body. At present, experimental studies have shown that long-term aluminum exposure can lead to a decline in human cognitive ability and intelligence, and is closely related to the occurrence of neurological diseases such as Alzheimer's disease, neurodegenerative diseases, and dialysis encephalopathy. The Y-maze water maze assay is a validated behavioral paradigm for evaluating spatial learning and memory in animals. Variations in swimming distance, escape latency, and error counts directly reflect the spatial orientation learning ability of experimental subjects. [7]. This study demonstrated that subchronic intraperitoneal administration of aluminum maltol for AD model establishment prolonged the escape latency in the Y-maze water maze task, indicating that subchronic aluminum maltol exposure impaired spatial memory and learning capabilities in mice and confirming the successful development of an Alzheimer's disease model.

Tremella compound polysaccharide is composed of various Chinese herbal medicines such as Tremella, Poria cocos, Licorice, Smilax glabra, Coix seed, and Mung bean coat. Tremella polysaccharide, the main active ingredient in Tremella, can enhance the activity of superoxide dismutase (SOD), accelerate the removal of free radicals. At the same time, through the removal of free radicals by SOD, it reduces the sensitivity of the body to adverse environments and reduces oxidative stress damage to the body, and has the effects of preventing tumors and anti-aging [8]. In addition, Tremella polysaccharide can promote cell proliferation and division, reduce the number of senescent cells, and play an antioxidant and anti-aging role. In addition, Tremella polysaccharide can significantly enhance the phagocytic activity of immune cells and promote the ability to generate immune antibodies, and has the effect of improving immunity [9]. Pachymic acid in Poria cocos has effects such as anti-tumor, anti-inflammatory, antioxidant, hypoglycemic, and sedative and hypnotic [10]. Glycyrrhizic acid in licorice can improve the energy metabolism of cells in brain tissue, reduce cerebral edema, promote the recovery of brain function, and improve the memory of the body, which is helpful for alleviating Alzheimer's disease [11]. Smilax glabra is rich in various active ingredients and has physiological activities such as anti-oxidation and anti-aging, detoxification and dehumidification, protecting the liver, and dredging joints [12]. Coix seed has the functions of strengthening the spleen and removing dampness, clearing heat and discharging pus, and beautifying and nourishing the face. The extract of Coix seed has the effects of anti-tumor and anti-aging[13]. Mung bean coat contains a variety of amino acids, minerals and vitamins, which can effectively remove toxins and free radicals in the body. The diuretic and detumescence effect can promote the excretion of waste in the body. SOD is an active and potent substance in the body and can remove harmful substances produced by the body during metabolism. As an efficient free radical scavenger, SOD can quickly and effectively remove excessive free radicals produced by the body, block the production of elastase in time, and inhibit its activity. Free radicals attack cells and destroy cell membranes, leading to cell death and changes in cell membranes. Tremella polysaccharide can increase the activity of SOD in the brain tissue of the body and enhance the ability to remove free radicals, so as to achieve the effects of anti-oxidation and anti-aging.

In this experiment, maltol aluminum was used to model Alzheimer's disease in mice, and Tremella and its compound preparations were used to intervene in the modeled mice. The results showed that in treatment group 1 and treatment group 2 of Tremella and its compound preparations, the time length of the water maze test and the number of memory errors were both less than those in the model group. The levels of serum TC, TG and β-secretase 1 in brain tissue, free radical scavenging rate, and Al^{3+} content were significantly lower than those in the model group. The degree of cortical lesions and decay in the brain was lighter than that in the model group. The content of free radicals in the Tremella compound polysaccharide (treatment group 2) was lower. Whether it is that the Tremella compound preparation has better antioxidant capacity and aluminum excretion ability or is affected by other potential factors requires further experimental research and clinical diagnostic observation and exploration. This experiment confirmed that the aluminum excretion effect, free radical scavenging ability and anti-aging effect of the Tremella compound polysaccharide preparation are better than those of the single preparation of Tremella. However, the intervention site of the Tremella compound polysaccharide traditional Chinese medicine preparation still requires more further research.

5. Conclusions

Collectively, Aluminum and its compounds infiltrate the human body through multiple exposure routes and accumulate, exhibiting neurotoxicity preferentially in the brain, which triggers

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neurodegenerative disorders such as Alzheimer's disease. This study successfully developed a murine Alzheimer's disease model via subchronic intraperitoneal administration of aluminum maltolate, providing evidence that aluminum exposure impairs spatial learning and memory functions. Treatment with Tremella complex polysaccharides (comprising Tremella, Poria, and other medicinal components) significantly shortened the water maze completion time, reduced memory errors, decreased serum TC and TG levels, and lowered brain β-secretase 1, Al^{3+}, and free radical concentrations, thereby alleviating cerebral cortical lesions in modeled mice. Notably, the complex preparation (treatment group 2) demonstrated superior antioxidant and aluminum-chelating effects, significantly contributing to Alzheimer's treatment and paving the way for the integration of traditional Chinese medicine into neurodegenerative disease therapy.

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