**ROLE OF METAL IONS IN PATHOGENISIS OF ALZHEIMERS DISEASE**

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Alzheimer disease is a neuro-degenerative disease of the brain. It usually affects people over the age of 65 years, with a progressive decline in memory, thinking, language and learning capacity.

The pathophysiology of AD is related to the injury and death of neurons, especially in the areas of the brain that are involved with memory and learning. Alzheimer disease is the most common dementia, accounting for 50%-75% of the total, with a greater proportion in the higher age ranges. There are nearly 18 million people with dementia in the world today. The number of people with dementia is expected to increase steadily over the next 25 years. By 2025, there will be about 34 million people with dementia in the world.

There is accumulating evidence that interactions between β-amyloid and copper, iron, and zinc are associated with the pathophysiology of Alzheimer’s disease (AD). A significant dyshomeostasis of copper, iron, and zinc has been detected, and the mismanagement of these metals induces β-amyloid precipitation and neurotoxicity.

1) Copper mediates the aggregation of β-amyloid and facilitating the generation of oxidative stress.

2) Iron precipitates β-amyloid to a lesser degree than does copper and catalyzes the Fenton reaction at a significantly slower rate.

3) Zinc has tendency to precipitate β-amyloid at physiological pH inhibits β-amyloid plaque clearance and degradation.

Experimental studies provide strong evidence that chelation strategies targeting these metals may have the potential to dissolve β-amyloid aggregations and inhibit oxidative stress.