Chapter 3

Copper Zinc Transmutation and Brain Function

The actinidic archaea can mediate biological transmutation, biological fission and fusion reactions and can be considered as the philosopher's stone and elixir of life.

Copper and zinc ions act as neurotransmitters in the brain and can be transmutated from one to the other. Copper and zinc play a role in RNA splicing and RNA splicing play an important role in brain size determination. RNA can function as neurotransmitters storing information and modulating other neurotransmission function. Divalent metal ions are involved in RNA splicing and contribute to the diversity of RNA world and prefrontal cortex.

Zinc can modulate NMDA transmission and can increase NMDA transmission. Zinc deficiency can lead to schizophrenia. Zinc is also involved in steroidal hormone and testosterone synthesis and its deficiency leads to hypogonadism. Zinc is involved in the formation of the prefrontal cortex. Zinc deficiency leads to prefrontal cortex dysfunction and human consciousness. Zinc containing neurons are seen in layers II, III, V and IV of cerebral cortex. They are also seen in pyramidal and inverted neurons of visual cortex. Zinc can modulated neuronal migration. Zinc containing neurons form a subset of glutamatergic neurons in the forebrain - the limbic cortex and cerebral cortex. Zinc containing neurons are seen in the hippocambal mossy fibres, perirhinal cortex, amygdale, presubiculum, cinculate cortex and claustrum. Zinc pumping into synaptic vesicles involves the golgi apparatus. Zinc vesicular stimulation releases zinc into the synapse which binds to post synaptic receptor site. Zinc is taken back by reuptake into the presynaptic neuron. Zinc containing neurons are glutamatergic neurons in the cerebral cortex and amygdale. Non zinc containing glutamatergic neurons are seen in spinal cord. The zinc containing forebrain cortex is called gluzinergic neuronal cortex. Zinc is involved in NMDA excitotoxicity and NMDA transmission. Zinc is involved in insulin secretion

and its deficiency leads to metabolic syndrome X. Zinc is involved in the pathophysiology of diabetes mellitus.

Copper is involved in diencephalon and cerebellar function. It is involved in function of the primitive part of the brain. It is involved in regulation of the human unconscious. Copper increases testosterone function and synthesis and leads to hypersexual states. Copper deficiency and aceruloplasminemia can lead to insulin resistance and metabolic syndrome.

Zinc deficiency can lead to decreased cholesterol and bile acid synthesis while copper excess can lead to decrease cholesterol and bile acid synthesis. Cholesterol deficiency and bile acid deficiency leads to autism and schizophrenia. Bile acids can bind to olfactory lobe and modulate limbic lobe function. Bile acids have got wide diversity and contribute to group identity.

Copper and zinc have got an inverse relationship. When copper is high zinc is low and when zinc is high copper is low. Zinc increases cholesterol and bile acid synthesis while copper inhibits it. There is an inverse relationship between copper and zinc. Copper is required for dopamine beta hydroxlyase function regulates monoamine function. It is also required for the function of flavin containing amine oxidases and tyrosinase.

Zinc is obtained from meat sources and non-vegetarians have high zinc levels and predominant cortical function. Copper is obtained from vegetarian sources and vegs are high cerebellar function and CCAS. Copper and zinc can get transmutated to each other by archaea mediated biological transmutation.

High copper and low zinc levels are involved in autism, schizophrenia and ADHD. Copper and zinc can thus act as metallic neurotransmitters.

Copper and zinc can activate the AKT pathway and glycolysis. Increased glycolysis is involved in systemic disorders. The increased glycolysis can activate the mitochondrial PT pore hexokinase leading onto cell proliferation.

This produces oncogenesis. The lymphocytes depend on glycolysis for its energy needs. The lymphocytic proliferation can lead onto autoimmune disease. The glycolytic enzyme glyceraldehyde 3-phosphate dehydrogenase can be acted upon by PARP enzyme and transferred to the nucleus producing cell death and neuronal degeneration. The glycolytic metabololite phosphoglycerate can be converted to phosphoserine, serine and glycine. Serine and glycine are NMDA modulators affecting the thalamo-cortico-thalamic pathway of conscious perception. This can lead onto schizophrenia and autism. The increase in glycolysis suggests a Warburg phenotype and mitochondrial dysfunction which can contribute to the evolution of metabolic syndrome X. Thus copper zinc transmutation can contribute to metabolic syndrome X, cancer, neurodegeneration, autoimmune disease, schizophrenia and autism.