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# Neanderthalic Actinidic Archaea Mediates Biological Transmutation in Human Systems - Experimental Evidence

## Introduction

Biological transmutation has been postulated by several groups of workers in microbial systems.[1, 2] Quantizing structures of optimal size and shape are necessary for non barrier nuclear interactions. The situation is realized in microbial cultures. During the growth process, the replication of DNA and other biomacromolecules takes place. In the region of growth, the interatomic potential holes with slowly changing sizes are constantly appearing and in this situation non barrier nuclear interactions can take place. Actinidic archaea has been described in human systems from our laboratory and function as cellular endosymbionts regulating multiple cellular functions. The actinidic archaea utilizes an alternate biochemistry depended on actinides for enzyme catalysis. The seashores of Kerala are rich in actinidic elements present as rutile, illmenite and monazite. The actinidic archaea is an endosymbiont of the human cell and it is possible that the organism can mediate biological transmutation. Transmutation of magnesium to calcium can serve as a mechanism of regulation of the neuro-immuno-endocrine system. Deficiency of magnesium is seen in degenerations, malignancy, metabolic syndrome x, psychiatric disorders and immune disease.[3] The actinidic archaea can exist as nanoarchaea which can undergo magnetite and calcium mineralization. It is possible that magnesium is being transmuted biologically to calcium to produce amounts sufficient for calcium mineralization. Calcified nanoarchaea can produce a systemic immune activation contributing to the diverse pathologies of degenerations, malignancy, metabolic syndrome x, psychiatric disorders and immune disease to study biological transmutation of magnesium to calcium and cerium. The results are presented in this paper.

## Materials and Methods

Informed consent was obtained from all patients included in the study. The permission of the Ethics Committee of the Institute was obtained. Fasting blood was drawn for the study from normal individuals without any systemic disease.

Experimental system was as follows: The basic system contained patient's serum 0.5 ml + normal serum 0.25 ml + physiological buffered saline + cerium chloride 0.1 mg/ml. To the basic system $MgSO_4$ 0.1 mg/ml was added.

The $Mg^{++}$ and $Ca^{++}$ were estimated at 0 hour. The remaining portion was incubated for 16 hours at 37 ℃ for 16 hours. The $Mg^{++}$ and $Ca^{++}$ were estimated at the end of 16 hours. The estimation of $Mg^{++}$ and $Ca^{++}$ were done by using commercial kits. Cytochrome F420 was estimated flourimetrically (excitation wavelength 420 nm and emission wavelength 520 nm).

## Results

The results showed that there was a decrease in magnesium and a concomitant increase in calcium in incubated serum samples from normal individuals. The percentage decrease in magnesium was 15.68 to 31.48 percent. The percentage increase in calcium was 10.43 to 9.79 percent. There was detection of cytochrome F420 in the system by fluorescence indicating archaeal growth dependent on actinidic cerium. This showed that the actinidic archaea was mediating the biological transmutation of magnesium to calcium.

***Table 1.*** *Experimental biological transmutation.*

| Case | Time | Mg (mEq/l) | % change in Mg | Ca (ng/dl) | % change in Ca |
|---|---|---|---|---|---|
| Case 1 | 0 hr | 1.415 | | 0.796 | |
| | 16 hrs | 1.193 | 15.68 ↓ | 8.310 | 10.43 ↑ |
| Case 2 | 0 hr | 2.290 | | 0.764 | |
| | 16 hrs | 1.569 | 31.48 ↓ | 7.480 | 9.79 ↑ |

## Discussion

The results showed that there is biological transmutation of magnesium to calcium in human systems mediated by actinidic archaea dependent on cerium for its growth. Regulation of calcium and magnesium levels in the cell by archaeal mediated biological transmutation can regulate multiple physiological systems. Calcium can modulate the mitochondrial PT pore and cell death. Cellular calcium levels are also involved in oncogene activation. Magnesium levels in the cell can regulate glycosylation and protein processing modulating golgi body and lysosomal function. Presynaptic calcium levels can regulate synaptic transmission as well as neurotransmitter release into the synapse. Cellular calcium levels can activate NFKB producing immune activation. Magnesium and calcium levels can modulate mitochondrial function and metabolism.[3]

There is magnesium depletion from the system and calcium accumulation which can predispose to malignancy, immune disease, degenerations, schizophrenia and metabolic syndrome x.[3] The increased intracellular calcium can open up the mitochondrial PT pore producing a mitochondrial dysfunction. Magnesium deficiency can produce a mitochondrial ATP synthase defect. The opening of the mitochondrial PT pore produces volume dysregulation of the mitochondria, hyperosmolarity and expansion of the mitochondrial matrix space producing outer membrane rupture. This leads to release of cytochrome C into the cytoplasm, activating the caspase cascade and cell death. Mitochondrial dysfunction and related apoptosis as well as free radical generation has been related to neuronal degeneration. Decreased intracellular magnesium can lead to altered glyconjugate synthesis and a protein processing dysfunction. Protein processing golgi body dysfunction as well as ER stress has been related to neuronal degeneration. Altered cell surface glyconjugates can lead to defective contact inhibition and oncogenesis. This can also produce disordered synaptic connectivity and functional

neuropsychiatric disorders. Altered glyconjugates can lead to defective MHC antigen presenting pathway and autoimmune diseases. A defective presentation of viral antigens can lead to immune evasion by the virus and viral persistence as in AIDS. Increased intracellular calcium can activate the RAS oncogene by producing GTPase inhibition and magnesium deficiency related phosphorylation defects can inactivate the tumour suppressor genes. Both of these can contribute to oncogenesis. Increased calcium within the presynaptic neuron can lead to increased glutamate release into the synapse and increased postsynaptic neuronal calcium can increase the NMDA signal transduction. NMDA signal transduction modulates the thalamo-cortico-thalamic reverberatory circuit important in conscious perception and schizophrenia. Increased NMDA signal transduction can contribute to epilepsy and degenerations of neuronal systems. An increase in presynaptic neuronal calcium can promote dopaminergic receptor actions contributing to the hyperdopaminergic state seen in schizophrenia. A decrease in intracellular magnesium can block the phosphorylation reaction involved in protein tyrosine kinase receptor activity leading to insulin resistance and syndrome x. An increase in intracellular calcium can activate the NFKB signal transduction producing immune activation and autoimmune disease. Immune activation has also been related to syndrome x, degenerations, malignant transformation and psychiatric disease.

A calcium excess related PT pore dysfunction of mitochondria can generate free radicals. Free radicals can produce apoptosis, immune activation, insulin resistance and NMDA activity. Free radicals can activate NFKB producing immune activation and autoimmune disease. Free radicals can activate the NMDA receptor modulating conscious perception and leading onto schizophrenia. Free radicals can produce mitochondrial dysfunction and cell death. Free radicals can activate HIF alpha and oncogene activation producing malignant transformation. Free radicals can produce insulin resistance and

metabolic syndrome x.

A shadow biosphere of actinidic archaea has been described in degenerations, malignancy, metabolic syndrome x, psychiatric disorders and immune disease. The archaea transmutates magnesium to calcium for the purpose of biological mineralization. The archaea can exist as nanoarchaea which can get calcified to form calcified nanoarchaeal forms. Calcified nanoarchaeal particles can induce NFKB. This can produce a state of systemic immune activation. This activates the AKT PI3 cascade inducing the Warburg phenotype with anaerobic glycolysis which is the basis of most human disease. The increase in mitochondrial PT pore hexokinase can produce cellular proliferation. The malignant cells depend on glycolysis for its energy needs. The Warburg phenotype can produce malignant transformation. The lymphocytes depend of glycolysis for its energy needs. Increased glycolysis can lead to immune activation. The glycolytic enzyme glyceraldehyde 3 phosphate dehydrogenase mediates nuclear cell death. The glycolysis generated NADPH activates the NOX enzyme important in insulin receptor function and NMDA activity. Thus the creation of Warburg phenotype can produce malignancy, immune disease, degenerations, schizophrenia and metabolic syndrome x.

Thus the transmutation related free radical generation and altered calcium-magnesium ratios in the cell can alter synaptic transmission, mitochondrial function, golgi body/ER function, lysosomal function, immune activation, cell proliferation, insulin resistance and cell death. The actinidic archaea related biological transmutation is an important regulatory mechanism of the cell whose dysfunction can produce altered neuro-immuno-endocrine regulation. This can lead to human disease. The biological transmutation gives the actinidic archaea energy to survive and generates calcium for its biological mineralization.

# References

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