Chapter 16

Archaeaon and Vitamin C Synthesis - The Vitaminocyte Organelle

Introduction

Ascorbic acid is not synthesized by primates and humans. Vitamin C is synthesized from monosaccharides especially mannose, galactose or glucose. Primates and humans have the mutated form of the enzyme L-gulonolactone oxidase and are therefore not able to synthesize vitamin C. Archaea are endosymbionts in the human cell and function as cellular organelle. Archaea have the vitamin C synthetic pathway. Therefore the human cell could be able to synthesize vitamin C using endosymbiotic archaea functioning as organelle.

Fructolysis in the archaeaon vitaminocyte can contribute to vitamin C and vitamin E synthesis. The Neanderthals have a higher density of archaeal symbiosis resulting in increasing number of vitaminocyte organelle. This results in increased synthesis endogenous ascorbic acid and tocopherol in Neanderthals which function as free radical scavengers. Free radicals are important in neuronal function and NMDA activity. Free radicals increase NMDA activity. Free radicals are also important as messengers of human endogenous retroviruses. Free radicals mediate the expression and reintegration into the genome where it functions as jumping genes contributing to genomic plasticity and dynamicity. Genomic dynamicity is consequently absent in Neanderthals due to higher synthesis of ascorbic acid and tocopherol by the vitaminocyte and free radical deficiency. Genomic dynamicity and HERV sequences contribute to development of synaptic connectivity, formation of cerebral cortex and brain size. This leads onto defective NMDA transmission, cerebral cortical dysfunction and cerebellar dominance in Neanderthals. The brain size in Neanderthals is bigger than the newer species of homo sapiens. The homo sapiens on the other hand has less of archaeal symbiotic density and fewer archaeal vitaminocyte organelle. The gene for vitamin C synthesis is already mutated in all human species and in the presence of decreased

density of archaeal vitaminocyte organelle in homo sapiens there is deficiency of ascorbic acid and tocopherols in homo sapiens. This results in reduced free radical scavenging, increased free radicals in the system, increased expression and reintegration of HERV sequences in to the genome. There is increased genomic dynamicity and plasticity and a dominant cerebral cortical function in homo sapien population and a smaller brain size. Thus the archaeal symbiosis and the resultant vitaminocyte organelle decides the human species type, brain size, cerebral cortical versus cerebellar dominance and the human consciousness.

Materials and Methods

10 normal individuals were drawn for the study. 10 ml of plasma from heparinised blood was taken for the study. The experimental protocols was as follows: (1) Plasma+buffered saline containing glucose 1 mg/ml with vitamin C concentration measured at 0 time and 2 hour time, and (2) Plasma+doxy 1 mg/ml+buffered saline containing glucose 1 mg/ml with vitamin C concentration measured at 0 time and 2 hr. time. Cytochrome F420 activity was also assessed.

Results

The vitamin C level were found to increase spontaneously from 9 mg/l at 0 time to 14 mg/l at 2 hr. in experimental protocol (1) containing plasma+buffered saline with glucose at 1 mg/ml. The solution also showed cytochrome F420 activity. The protocol (2) containing plasma+doxy+buffered saline containing glucose at 1 mg/ml had no vitamin C activity detected or cytochrome F420 activity detected. The archaeal endosymbiont or archaeaon could thus synthesize vitamin C.

Discussion

The study demonstrates that vitamin C is synthesized by endosymbiotic archaeaon. It functions as a vitaminocyte. The primates and humans lost the capacity to synthesize vitamin C. L-gulonolactone oxidase is deficient in humans. Vitamin C deficiency is a genetic disease. Vitamin C deficiency played an important role in human evolution. Vitamin C is an anti-oxidant. Its deficiency leads to free radical generation and modulation of monoaminergic and glutamatenergic neurotransmission and evolution of the cerebral cortex. The generation of free radicals may have played the role in conscious perception and the bigger size of the primate cerebral cortex as seen in homo sapiens. The capacity to generate vitamin C synthesis by endosymbiotic archaea may shrink the cerebral cortex and increase the cerebellar size leading onto the dominance of the unconscious brain as seen in homo neanderthalis. Vitamin C deficiency is implicated in disorders of consciousness like schizophrenia and autism.

Vitamin C deficiency leads to defective collagen synthesis and breaks in the vessel wall producing damage which is healed by adhesion of lipoprotein a to the vessel wall producing atherosclerosis. Atherosclerosis is a genetic vitamin C deficiency disease. This hypothesis was put forward by Linus Pauling. The capacity of endosymbiotic archaea to synthesize vitamin C may protect against it. Vitamin C is required for insulin secretion and its deficiency leads to diabetes mellitus and metabolic syndrome. Vitamin C deficiency leads to oncogenesis.

Vitamin C deficiency generates free radicals which can activate oncogenes producing cell proliferation. The defective collagen matrix that is formed can lead to metastasis. Oncogenesis can be considered as a vitamin C deficiency syndrome. Vitamin C is seen in high levels in lymphocytes. Vitamin C deficiency leads to immunosuppression and viral infections. Vitamin C is anti-viral agent. Vitamin C is required for lymphocyte function and its

deficiency leads to autoimmune disease. Vitamin C deficiency leads to free radical generation and cell death and neurodegeneration.

All the civilizational disorders of schizophrenia, autism, autoimmune disease, neurodegeneration, metabolic syndrome X, cancer and atherosclerosis. The archaeaon is the cellular organelle concerned with ascorbic acid synthesis and cyto protection. It can be considered as a vitaminocyte.[1-3]

The homo sapiens ate more of fruits, cereals and vegetables having evolved in the African Savannah. The diet was rich in vitamin C leading to eventual mutation and loss of the GULO gene. Hypoascorbemia is a genetic disease. The GULO gene is mutated in adult homo sapiens. The homo sapiens lack the GULO gene which gets mutated due to insertion of a HERV sequence in it. This leads onto the genetic disorder of hypoascorbemia in homo sapiens. The GULO gene is absent in the new world monkeys from which the homo sapiens originated. The old world monkeys have the GULO gene and they evolved into homo neanderthalis. The homo neanderthalis evolved in the Eurasian Steppes in the ice age and ate a carnivorous non-vegetarian diet which was deficient in vitamin C. The GULO gene was therefore evolutionarily preserved in homo neanderthalis to synthesize vitamin C which was deficient in their diet. The divergence of the old world and new world monkeys and the evolution of homo sapiens and homo neanderthalis coincided with the mutation of GULO gene. Vitamin C deficiency in homo sapiens leads to increased free radical generation. Free radicals are messengers for retroviral replication. This leads to increase in HERV sequences in homo sapien genome. The increase in HERV sequences in the homo sapien genome leads to genomic dynamicity and increased cortical synaptic connectivity contributing to the evolution of the homo sapien cerebral cortex. The homo neanderthalis have vitamin C synthesis and an active GULO gene contributing to more of vitamin C synthesis and reduced free radical

generation. This leads to less of HERV replication and reduction in HERV sequences in the homo neanderthalis genome. This contributes to the dominant cerebellum in the homo neanderthalis genome and a cerebellar cognitive affective disorder in homo neanderthalis. Vitamin C inhibits excessive activation of the immune system and tissue destruction. It converts Th0 cells to Th1 and increases the production of gamma interferons. Vitamin C inhibits the synthesis of proinflammatory cytokines. This contributes to the immune escape and archaeal endosymbiosis in homo neanderthalis. The GULO positive mice have increased HDL which is anti-inflammatory and contributes to immune escape and endosymbiosis by archaea in homo neanderthalis. Thus vitamin C contributes to the evolution of homo neanderthalis by archaeal endosymbiosis. Vitamin C is a co-factor for 4-hydroxy phenyl pyruvate dioxygenase. The HPPD enzyme is required for conversion of tyrosine to homogentisic acid and eventual synthesis of tocopherols and plastoquinones. The plastoquinones subserve archaeal energetics and contributes to archaeal endosymbiosis resulting in evolution of homo neanderthalis. The sperm activation of the oocyte requires redox stress. The vitamin C synthesis in homo neanderthalis contributes to inhibition of the sperm activation of the oocyte. The oxidative stress is required for meiosis and generation of germ cells. The decrease in redox stress consequent to vitamin C synthesis in homo neanderthalis leads to inhibition of meiosis. This contributes to parthenogenesis in homo neanderthalis. Thus vitamin C synthesis is one important reason for parthenogenesis in homo neanderthalis. Vitamin C synthesis in homo neanderthalis contributes to decreased HERV replication and HERV sequences in the genome. HERV sequences are required for the development of the placenta. This contributes to defective placentation in homo neanderthalis and parthenogenesis.

Vitamin C is co-factor for the synthesis of catecholamines - epinephrine, norepinephrine and dopamine. Vitamin C is also a co-factor for hydroxylation

of tryptophan and serotonin synthesis. The increased vitamin C synthesis in homo neanderthalis contributes to increased catecholaminergic, serotoninergic and dopaminergic transmission contributing to schizophreniform psychosis and fear flight fight response type of impulsive personality in homo neanderthalis. The increased catecholaminergic activity contributes to impulsivity and cerebellar cognitive affective disorder in homo neanderthalis. The increased vitamin C levels in homo neanderthalis blocks the glucose transporter GLUT contributing to a dependence on ketogenesis for energetics. The homo neanderthalis ate a carnivorous ketogenic diet. Vitamin C is required for tyrosine catabolism and generation of melanin.

Vitamin C is a co-factor for the enzyme N-trimethyl L-Lysine hydroxylase and gamma butyrobetaine hydroxylase required for carnitine synthesis. Vitamin C is required for mitochondrial function. Vitamin C is also a co-factor for peptidyl glycine alpha amidating monooxygenase which is required for removing the glyoxylate residues from the C terminal glycine of peptide hormones. Vitamin C is required for activation of peptide hormones like insulin and growth hormone. Vitamin C deficiency leads to increased free radical generation which is required for insulin activation. ROS species are required for insulin signaling. Vitamin C is transported by the GLUT and SVCT transporter and competes with glucose for transport into the cell. Therefore vitamin C deficiency can lead to hyperglycemia and insulin resistance in homo neanderthalis. Vitamin C deficiency leads to dysfunction of the enzymes prolyl 4-hydroxylase and prolyl 3-hydroxylase as well as lysyl hydroxylase. The prolyl hydroxylases are required for the activation of HIF alpha producing increased glycolysis and mitochondrial dysfunction. This contributes to the Warburg phenotype in homo neanderthalis. Thus vitamin C excess in homo neanderthalis leads to insulin resistance and diabetes mellitus.

Vitamin C synthesis and the archaeaon vitaminocyte thus played an important role in the evolution of homo neanderthalis and its parthenogenetic asexual reproduction. Vitamin C synthesis also contributed to the cerebellar dominance Neanderthal brain. Vitamin C deficiency and GULO mutation led to the evolution of homo sapien cerebral cortex by increasing HERV sequences in the genome.

The homo neanderthalis had vitamin C toxicity due to the presence of the archaeaon vitaminocyte. Vitamin C is a free radical scavenger but when it combines with reactive oxygen species can lead to the formation of pro-oxidants. The pro-oxidants generated by binding of ascorbic acid with ROS leads to tissue destruction and genesis of cancer, metabolic syndrome, neurodegeneration, autoimmunity and neuropsychiatric disease. Pro-oxidants can produce immune activation, insulin resistance, NMDA exitotoxicity, cell proliferation and increasing dopaminergic transmission leading to schizophreniform and autistic brain.

References

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