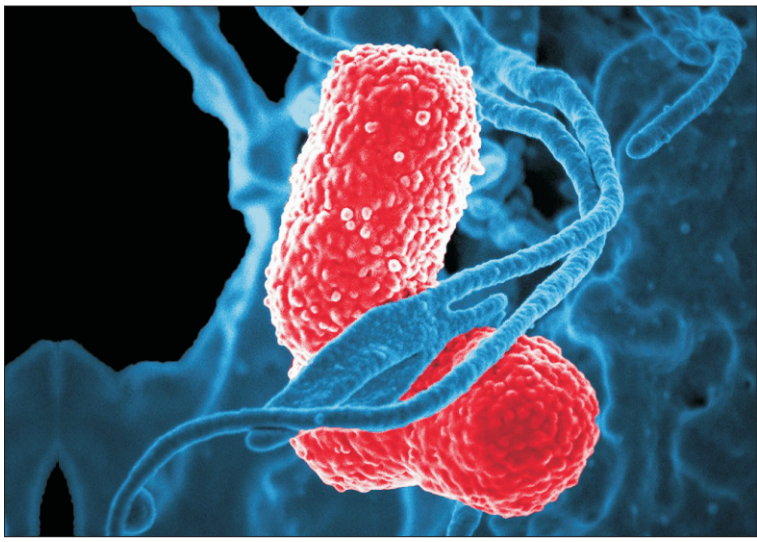
Global Warming Related

Endosymbiotic Archaea Induced Immunometabolonomic Syndrome

Ravikumar Kurup
Parameswara Achutha Kurup



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Ravikumar Kurup
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Chapter 1

Global Warming Related Immunometabolonomic Syndrome

Global warming leads to endosymbiotic actinidic archaeal growth which results in neanderthalisation of human species. The Neanderthal metabolic patterns were different from the homo sapien patterns. Stress of the ice age led to the induction of HO1. HO1 converts heme to bilirubin and carbon monoxide. This results in heme depletion. Heme depletion induces ALA synthase and results in porphyrinogenesis. Porphyrin photooxidation results in ROS generation and induction of HIF alpha resulting in the Warburg phenotype with increased glycolysis, PDH inhibition and mitochondrial dysfunction. Heme depletion leads to defective function of the TCA cycle heme enzyme aconitase and the mitochondrial enzyme cytochrome C oxidase. This results in the body generating energy by glycolysis as well as membrane sodium potassium ATPase inhibition induced ATP synthesis. Mitochondrial dysfunction leads to insulin resistance.

The PDH inhibition leads to less levels of the substrate acetyl CoA for the isoprenoid pathway for synthesis of cholesterol. Low level of cholesterol synthesis leads to low cortisol, testosterone, estrogen, vitamin D, coenzyme Q and bile acids. Bile acid deficiency can lead to defective modulation of LXR, FXR and PXR leading to metabolic syndrome x. CoQ deficiency leads to mitochondrial dysfunction. Vitamin D deficiency leads to immune activation. Low cortisol levels lead to defective stress response. Low testosterone and estrogen leads to an asexual state.

The archaea can induce biological transmutation metals. There is conversion of magnesium to calcium. This results in mitochondrial PT pore dysfunction and cell death, NFKB activation and immune stimulation, release of monoamine neurotransmitters resulting schizophrenia and autism, calcium induced oncogene activation and defective insulin secretion producing metabolic syndrome. There is also conversion of zinc to copper. Zinc is required for glutamate and GABA transmission. Zinc functions as a neurotransmitter in

the prefrontal cortex. The zinc containing neurons are called gluzinergic neurons. Copper is required for monoamine transmission and the function of cerebellum, brain stem and basal ganglia the primitive parts of the brain. Thus the conversion of zinc to copper results in prefrontal cortex atrophy and cerebellar dominance. Biological transmutation can also generate energy.

The archaea induced Warburg phenotype can produce PDH blockade and accumulation of pyruvate. The pyruvate enters the GABA shunt generating succinyl CoA and glycine resulting in porphyrin synthesis. Porphyrin can produce quantal perception of low level EMF resulting in prefrontal cortex atrophy and cerebellar dominance. Since glycine is used up for porphyrin synthesis serine is not formed. There is no cystathionine synthesis leading onto hyperhomocysteinemia. The PDB blockade results in accumulation of pyruvate which gets converted to glutamate and ammonia resulting in a hyperammonemic syndrome.

The BKCD enzyme has a structure similar to PDH. BKCD deficiency results in accumulation of branched chain amino acids. To counteract this, the Neanderthals developed defects in neutral amino acids renal transporter. This resulted in a Hartnup's disease mutation and nicotinic acid deficiency consequent to loss of tryptophan. The function of sirtuins a histone deacetylase inhibitor also involved in protein acetylation is affected. Nicotinic acids are sirtuin inhibitors. The PDH blockade results in defective generation of acetyl CoA and defective protein acetylation. There are nearly 3500 acetylated proteins. Thus PDH blockade can modulate proteonomics.

The archaea induced RNA viroids can block mRNA and regulate RNA function. The porphyrins can intercalate with DNA and RNA modulating their function. Sirtuin induced HDAC inhibition modulated by Hartnup's disease mutation and nicotinic acid deficiency can also modulate genomic function. The

RNA viroids generated by archaea can get integrated into DNA, function as jumping genes and modulate genomic function.

The archaea can catabolize cholesterol to digoxin which can produce membrane sodium potassium ATPase inhibition increasing intracellular calcium and reducing intracellular magnesium. There is decrease in magnesium and increase in calcium results in mitochondrial PT pore dysfunction and cell death, NFKB activation and immune stimulation, release of monoamine neurotransmitters resulting schizophrenia and autism, calcium induced oncogene activation and defective insulin secretion producing metabolic syndrome.

Global warming leads to increase in endosymbiotic actinidic archaeal growth. Archaea are extremophiles. The actinidic archaea survive by catabolizing cholesterol. The archaea and its antigens induce HIF alpha and activate the glycolytic pathway. The glycolytic pathway activation induces increased conversion of glucose to fructose by activation of the sorbitol pathway. Glucose is converted to sorbitol by the enzyme aldose reductase and sorbitol is converted to fructose by the action of sorbitol dehydrogenase. Fructose is phosphorylated by hexokinase or fructokinase to fructose phosphate. Hexokinase has a low km value for fructose and minimal amounts of fructose will be converted to fructose phosphate depleting the cellular ATP. ATP is converted to AMP and by the action of AMP deaminase is converted to uric acid. Thus there is resultant hyperuricemia and the depletion of ATP also produces membrane sodium potassium ATPase inhibition. Inhibition of membrane sodium potassium ATPase increases intracellular calcium and depletes magnesium. This produces cell death by opening up the mitochondrial PT pore, NFKB activation and immune activation, glutamate excitotoxicity and oncogene activation leading to systemic disorders. The depletion of ATP finally inhibits hexokinase as such and glucose phosphorylation stops blocking the glycolytic pathway and its coupling to the mitochondrial oxidative phosphorylation by the action of PT

pore hexokinase. The cell is depleted of energy by glycolysis and the oxidative phosphorylation scheme and dies. Thus global warming via induction of glycolysis and Warburg phenotype and the increased conversion of glucose to fructose and the resultant cellular depletion of ATP can produce systemic disorders and cell dysfunction as well as death. This can produce the global warming related systemic syndrome.

Global warming leads to neanderthalisation of the human species consequent to growth of actinidic archaea. The Neanderthals were accustomed to a ketogenic high fat, high protein diet. The ketone bodies were oxidised to generate ATP in the mitochondria. The neanderthalised humans due to actinidic archaeal growth due to consumption of a glucogenic diet leads to induction of glycolytic enzymes. The glycolytic enzymes are cytosolic. The glycolytic enzymes are antigenic in the neanderthalised humans. The glycolytic enzymes were suppressed in homo neanderthalis who ate ketogenic diet. This results in suppression of induced glycolytic enzymes by antibody formation in homo neoneanderthalis which arises due to archaeal growth consequent to global warming. The blockade of glycolysis results in blockade of cell energetics. This results in hyperglycemia and metabolic syndrome x. The glucose is converted to sorbitol by aldose reductase and sorbitol is converted to fructose by fructokinase. Fructokinase enzyme is native to Neanderthals as they consumed fruits along with fat and protein from meat. Fructose is phosphorylated to fructose phosphate which depletes the cell of ATP. This inhibits membrane sodium potassium ATPase leading onto increase in intracellular calcium and reduction in intracellular magnesium. This produces glutamate excitotoxicity and neurodegeneration, oncogene activation and malignancy, NFKB activation and autoimmune disease, release of mono amine neurotransmitters from presynaptic vesicles and schizophrenia and all systemic diseases. The increased glucose gets metabolised by archaeal glycolysis and citric acid cycle. The pyruvate generated

by archaeal glycolysis enters the GABA shunt scheme generating succinyl CoA and glycine which are substrates for porphyrin synthesis. The archaeal citric acid cycle can be reductive generating carbon dioxide fixation akin to the Calvin cycle of photosynthesis or oxidative generating acetyl CoA which is used for cholesterol synthesis by the archaeal mevalonate pathway. The archaeal glycolysis also generates fructose 1,6 diphosphate which enters the pentose phosphate pathway producing D xylulose phosphate which is a substrate for DXP pathway of archaeal cholesterol synthesis. The archaea synthesizes cholesterol by both the mevalonate pathway and DXB pathway. The archaea can use cholesterol for energetics by catabolizing it. The cholesterol ring is oxidised to pyruvate which enters the GABA shunt which provides substrates for the citric acid cycle. The pyruvate is converted to glutamate and ammonia. The archaea can oxidise ammonia for energy. The side chain of cholesterol is oxidised to butyrate and propionate which can also be further utilised for energy purposes. The archaeal energetics depends on glycolysis, citric acid cycle, ammonia oxidation and cholesterol catabolism. The antibodies against the glycolytic enzymes aldolase, enolase, GAPDH and pyruvic kinase contributes to metabolic syndrome x, schizophrenia, mood disorders, autism, multiple sclerosis, lupus, Alzheimer's disease and Parkinson's disease. The upregulation of glycolysis contributes to neoplastic state. The antibodies are produced against induced glycolytic enzymes as well as archaeal glycolytic enzymes. The blockade of glycolysis leads to a secondary mitochondrial dysfunction. The glycolytic scheme is coupled to mitochondrial oxidative phosphorylation by mitochondrial PT pore hexokinase. The antibodies against glycolysis blocks glycolysis and produce secondary mitochondrial dysfunction. The cell uses all its energetics. The depletion of ATP by phosphorylation of fructose produces membrane sodium potassium ATPase inhibition and cell hibernation as well as stem cell transformation. The human tissue systems come

to a halt and form a framework for archaeal colonies to thrive. The human body becomes a zombie for archaeal colonies which are eternal. This affects the function of organ systems like the liver producing cirrhosis, the lung producing interstitial lung disease, renal fibrosis and CRF, cardiomyopathy and Alzheimer's disease. This can be called as the zombie syndrome. The depletion of ATP by phosphorylation of fructose generates ADP and AMP which by action of AMP deaminase produces uric acid and hyperuricemia. The zombie syndrome converts the human body to a framework for an archaeal colony network. The archaea can secrete RNA and DNA viroids which can recombine with human endogenous retroviral sequences and human DNA sequences generating new RNA viruses, DNA viruses and bacteria. Thus the zombie syndrome results in the generation of new bacteria and viruses. The zombie syndrome can be treated by suppression of glycolysis. This can be done by giving a ketogenic diet derived from fibre short chain fatty acids - butyrate and acetate, polyunsaturated fatty acids and short chain fatty acids like lauric acid.

The global warming zombie syndrome results in conversion of glucose to fructose by the induction of aldose reductase consequent to dehydration. The glucose is first converted to sorbitol by aldose reductase and then by sorbitol dehydrogenase to fructose. Fructose has got a high value for ketokinase and is phosphorylated and enters the pentose phosphate pathway. Fructose is converted to glucosamine phosphate and galactosamine phosphate. There is increased synthesis of glycosaminoglycans. This produces GAG accumulation in the vessels producing mucoid angiopathy, kidney producing MEN, heart producing EMF, pancreas producing CCP and thyroid producing MNG. It can also result in fibrosis of the lung and liver producing cirrhosis and interstitial lung disease. These diseases are common in warm tropical countries, south of equator and can be called as the Lemurian syndrome. The increase km value of fructose for ketokinase results in increased phosphorylation of fructose over

glucose producing hyperglycemia and metabolic syndrome. The phosphorylation of fructose depletes the cell of ATP and converts ATP to AMP and ADP which is acted upon by ATP deaminase producing uric acid. The channelling of fructose into the pentose phosphate pathway results in increased production of ribose and nucleic acid synthesis and uric acid production consequent to purine degradation. There is hyperuricemia. The tubular defect of MEN produces hypokalemia and hyponatremia. The increase in fructose produces fructose glycation of proteins resulting in the formation of antigenic proteins and autoimmune disease. Fructose can produce inflammation and autoimmunity. This is called as fructositis. The increase in fructose which is channelled to the pentose phosphate pathway and ribose synthesis results in increased nucleic acid synthesis and cancer formation. The depletion of cellular ATP consequent to phosphorylation of fructose results in cell death and neuronal degeneration. Cell death in intestinal mucosa breaches the blood gut barrier producing leaky guts syndrome, acute phase response and metabolic syndrome x as well as autoimmunity. The phosphorylation of fructose and depletion of ATP results in membrane sodium potassium ATPase inhibition and increase in intracellular calcium and reduction in intracellular magnesium. This results in insulin resistance, glutamate excitotoxicity, oncogene activation, monoamine secretion from presynaptic vesicles and schizophrenia as well as NFKB activation and autoimmunity. The channelling of fructose to glucosamine synthesis and increased synthesis of GAG results in increased heparan sulphate synthesis which will combine with proteins forming amyloid. Amyloid formation is the basis of motor neuron disease where ribonucleoproteins form amyloid. In Parkinson’s disease alpha synuclein forms amyloid. In Alzheimer’s disease beta amyloid is formed. The tumour suppressor proteins forms amyloid and results in oncogenesis. The islet associated amyloid

polypeptide forms the basis of defective insulin secretion in metabolic syndrome x.

Global warming leads to aldose reductase induction. Aldose reductase converts glucose to sorbitol. Sorbitol is converted to fructose by sorbitol dehydrogenase. Fructose is phosphorylated by fructokinase and ketokinases have a higher km value for fructose than glucose. This results in rapid phosphorylation of fructose and depletion of cellular ATP. The depletion of cellular ATP has two consequences. ATP is converted to ADP and AMP. ADP and AMP are acted upon by deaminases generating uric acid. Hyperuricemia is a feature of global warming related metabolic phenomena. The depletion of ATP results in renal tubular dysfunction producing loss of electrolytes and amino acids. This results in non-specific aminoaciduria, hypokalemia and hyponatremia. There is no edema or hypertension. This produces a chronic tubulointerstitial disease called Mesoamerican nephropathy. The cellular depletion of ATP can produce cell death leading onto neuronal degeneration. The depletion of ATP produces membrane sodium potassium ATPase inhibition and increase in intracellular calcium and reduction in intracellular magnesium. This produces immune activation by NFKB induction, oncogene activation, glutamate excitotoxicity and neurodegeneration, release of monoamines into synaptic junction and schizophrenia. The depletion of ATP can also affect the gut blood barrier producing an acute phase response and leaky gut syndrome. The acute phase response can lead to metabolic syndrome x. The depletion of ATP owing to the affinity of ketokinases for fructose leads to lack of phosphorylation of glucose. This leads to hyperglycemia and metabolic syndrome x. The glucose that accumulates gets converted to fructose producing the syndrome of fructositis.

Fructose has got two metabolic fates. The fructose can get phosphorylated to fructose phosphate and enter the pentose phosphate pathway generating ribose

important in nucleic acid synthesis. The purine catabolism can generate uric acid. The synthesis of nucleic acid can lead to increased cell proliferation and oncogenesis. Thus global warming related fructositis can lead to oncogenesis. The fructose that gets accumulated can also enter the pathway for glycosaminoglycan and proteoglycan synthesis. The fructose is phosphorylated and converted to fructose phosphate which can be converted to glucosamine and galactosamine which are substrates for glycosaminoglycan synthesis. This results in accumulation of connective tissue mucopolysaccharides in the body and tissues leading onto disease states like endomyocardial fibrosis, chronic calcific pancreatitis, multinodular goitre and mucoid angiopathy which can be classified as a cardiovascular and endocrine syndrome of unknown origin related to global warming akin to MEN. The accumulation of mucopolysaccharides can occur in the liver producing cirrhosis of the liver and in the lung producing interstitial lung disease. The mucopolysaccharide, heparan sulphate can combine with prion proteins producing amyloid deposition leading onto conformational diseases. This include the prion proteins leading onto Creutzfeldt Jakob's disease, copper zinc dismutase and motor neuron disease, alpha synuclein and Parkinson's disease and tumour suppressor protein and cancer. The relation between islet associated amyloid polypeptide and diabetes mellitus is well known. Thus the conversion of fructose to GAG results in conformational disease and amyloid accumulation.

The accumulation of fructose results in fructosylation of proteins akin to glycation of proteins. Fructosylated proteins are antigenic. This results in increased frequency of autoimmune diseases like lupus and multiple sclerosis. The conversion to glucose to fructose and its phosphorylation depletes the cell of ATP and results in membrane sodium potassium ATPase inhibition. This increases the intracellular calcium which releases neurotransmitters from presynaptic vesicles. Thus there is an increase in glutamate and monoaminergic

transmission leading to schizophrenia and autism. The increase in intracellular calcium can open up the mitochondrial PT pore releasing cyto C which activates the caspase cascade and cell death. This produces neuronal degeneration.

The conversion of glucose to fructose and phosphorylation of fructose results in depletion of cellular ATP. This stops the phosphorylation of glucose by glucokinase and generation of glucose 6 phosphate stops. The glycolytic process, the TCA cycle and its coupling to mitochondrial oxidative phosphorylation is inhibited. The body depends upon fatty acids and amino acids for energetics. The body can survive only on a ketogenic diet. The accumulated glucose forms a substrate for utilisation by endosymbiotic archaea. The archaea have glycolytic pathway which can convert glucose to pyruvate. The pyruvate can then enter the GABA shunt pathway generating succinyl CoA and glycine, the substrates for porphyrin synthesis by archaea and humans. Porphyrin supramolecular arrays or porphyrions can transfer electrons synthesizing ATP. The archaea also has a partial citric acid cycle and the pyruvate generated by archaeal glycolysis can enter the partial citric acid cycle. The citrate can be used for lipid synthesis. The acetyl CoA generated from pyruvate by archaea can be used for cholesterol synthesis. The archaea can catabolize cholesterol to generate energy. The cholesterol ring is oxidised to pyruvate and the side chain oxidised to butyrate and propionate. This can be used for mitochondrial generation of ATP. The pyruvate generated by archaeal glycolysis can also undergo a reverse citric acid cycle for carbon dioxide fixation akin to the Calvin cycle. Thus the archaeal glycolysis, partial citric acid cycle, reverse citric acid cycle of carbon dioxide fixation, cholesterol synthesis by the mevalonate and DXP pathway and cholesterol catabolism dominates. The fructose generated by conversion to glucose can enter the pentose phosphate pathway generating D xylulose phosphate and can be used to synthesize cholesterol. The archaeal pyruvate generated by glycolysis can also

be converted to acetyl CoA which can enter the archaeal mevalonate pathway of cholesterol synthesis. Thus the archaea has got both pathway of cholesterol synthesis - the DXP pathway and mevalonate pathway. The fructose generated by conversion from glucose due to global warming enters the DXP pathway of cholesterol synthesis, the pentose phosphate pathway and nucleic acid synthesis and GAG synthesis. This can lead to multiple organ dysfunction with fibrosis and mucopolysaccharide accumulation which can be called as a Lemurian syndrome. The initial group of diseases EMF, CCP, MNG and mucoid angiopathy occur south of the equator in South India, South Africa and South America. The global warming related MEN is reported from Central America and South America. MEN belongs to the group of EMF, CCP, MNG and mucoid angiopathy. This can be called as the Lemurian syndrome as South Africa, South India, Australia and parts of South America were part of one single continental entity in the long past when Neanderthals exist. The global warming and resulting conversion of glucose to fructose results in depletion of ATP due to more efficient phosphorylation of fructose over glucose. This results in ineffective glucose phosphorylation and accumulation leading to archaeal growth. The accumulated fructose is converted to D xylulose phosphate and used for the DXP pathway of cholesterol synthesis by the archaea. The cholesterol is catabolized by the endosymbiotic actinidic archaea generating digoxin. The growth of actinidic archaea results in increased porphyrin synthesis and perception of low level electromagnetic fields by dipolar porphyrin mediated quantal systems. The digoxin induced membrane sodium potassium ATPase inhibition in the setting of dipolar porphyrins in the cell can produce a pumped phonon system of quantal perception. This results in atrophy of the prefrontal cortex and cerebellar dominance leading to neanderthalisation of the human brain. This results from endosymbiotic archaeal growth resulting from metabolonomics related to global warming. The same

metabolonomics related to global warming results in the genesis of the Lemurian syndrome described above.

This syndrome can also be called as fructosemia or fructositis. Oxidative stress can induce aldose reductase and convert glucose to fructose. The endosymbiotic archaea synthesizes digoxin by cholesterol catabolism. Digoxin inhibits membrane sodium potassium ATPase and increases intracellular calcium opening up the mitochondrial PT pore. This produces mitochondrial dysfunction and oxidative stress. Oxidative stress can induce aldose reductase and convert all glucose to fructose. Fructose has got a low km value for ketokinase compared to glucose and is preferentially phosphorylated. The glucose remains unphosphorylated and the glycolytic scheme and its coupled mitochondrial oxidative phosphorylation is slowed down or inhibited. The mitochondrial ATP and citrate can inhibit the phosphofructokinase and glycolysis, but cannot inhibit fructokinase or aldose reductase. Therefore the conversion of glucose to fructose continues. The fructose generated can inhibit mitochondrial function leading to more oxidative stress and still further induction of aldose reductase. The efficient phosphorylation of fructose depletes the cell of ATP producing oxidative stress and induction of NFKB resulting in chronic inflammation. Oxidative stress due to depletion of ATP can produce still further induction of aldose reductase and generation of fructose. The depletion of ATP makes the patient fatigue. The increase in fructose inhibits satiety and the patient feeding behaviour is altered leading onto obesity. This leads to a metabolic syndrome x. Metabolic syndrome x is a fat storage syndrome akin to hibernation. The fructose that is generated is converted to alpha glycerophosphate which is used for triglyceride synthesis. Fructose consumption can increase triglyceride synthesis and fatty liver. The depleted ATP due to fructose phosphorylation generates AMP and ADP which is converted to uric acid by deaminases. Uric acid can inhibit mitochondrial

function. Uric acid can contribute to insulin resistance and metabolic syndrome x. Uric acid can also produce endothelial dysfunction contributing to coronary artery disease and stroke. Uric acid inhibits aconitase which leads to accumulation of citrate. Uric acid can induce citrate lyase and fatty acyl CoA synthase leading onto fatty acid synthesis. The accumulated citrate can block the glycolytic pathway. The accumulated glucose gets converted to fructose by aldose reductase. Uric acid decreases reduced NADPH and oxidised NAD+. This affects the redox potential and leads to oxidative stress which further induces aldose reductase. Aldose reductase can be induced by hyperosmotic stress. This includes that created by hyperglycemia consequent to blockade of glucose phosphorylation as a result of selective fructose phosphorylation because of the low km value of fructose for ketokinase. The same mechanism operates in the dehydration induced by global warming. This leads to hyperosmolarity which induces aldose reductase. These mechanisms are similar to what happens in hibernation in animals. Hibernating animals develop a metabolic syndrome put on weight, store fat, increase triglycerides, develop fatty liver and develop insulin resistance. Hibernation and metabolic syndrome x are fat storage syndromes. The Neanderthals evolved in the cold Eurasian steppes and developed a hibernation syndrome similar to metabolic syndrome with fat storage. The stress of the ice age would have induced redox stress, induce aldose reductase and converted glucose to fructose. The fructose would have been selectively phosphorylated to fructose phosphate which would have entered the pentose phosphate pathway generating ribose for nucleic acid synthesis, the glucosamine pathway for glycosaminoglycan synthesis and/or converted to alpha glycerophosphate for fatty acid and triglyceride synthesis. Fructosamia can also affect brain function. Fructose can inhibit BDNF and inhibit cortical growth producing a cerebellar dominant brain and prefrontal cortex atrophy. This would have resulted in neanderthalisation of the brain. The

conversion of fructose phosphate to ribose via the pentose phosphate pathway increases nucleic acid synthesis and cell proliferation. This leads to oncogenesis. The cell proliferation can also contribute to the bulky phenotype of the Neanderthal population. The fructokinase enzyme acts as an obesity switch. The opening of the obesity switch also contributes to the bulky phenotype Neanderthal population. The oxidative stress and osmotic stress of the ice age and global warming can induce aldose reductase mediated conversion of glucose to fructose via the enzyme sorbitol dehydrogenase. This also leads to induction of fructokinase, generation of fructose, fructosemia and fructositis. The increased fructose can fructosylate proteins producing antigenic proteins and autoimmune disease. Thus fructosemia can contribute to oncogenesis, metabolic syndrome x, neurodegeneration, psychiatric disorders like schizophrenia and autism as well as autoimmune disease. Fructosemia can contribute to insulin resistance. The selective phosphorylation of fructose owing to the low km value of ketokinase for fructose results in nonphosphorylation of glucose and hyperglycemia. Insulin resistance leads to further induction of aldose reductase and fructokinase. Fructose can produce ATP depletion and oxidative stress. Oxidative stress can induce NFKB producing chronic inflammation and TNF alpha can produce insulin resistance acting at the level of insulin receptor. The insulin resistance activates the aldose reductase fructokinase system still further which is also further activated by the osmotic and oxidative stress of extremes of climate like global warming and ice age. This leads to the Lemurian systemic syndrome of fructosemia and fructositis.

The fructose metabolic pathway is called fructolysis. Oxidative stress and osmotic stress due to global warming and actinidic archaeal growth leads to induction of aldose reductase. This converts glucose to sorbitol and sorbitol is acted upon by sorbitol dehydrogenase produce fructose. Fructose normally undergoes fructolysis. Glycolysis is inhibited at the level of

phosphofructokinase by ATP and citrate. The fructolytic pathway and fructose metabolism is confined to the liver and certain tissues and is not under this regulatory control. Fructose is converted to fructose 1-phosphate by fructokinase. Fructose 1-phosphate is acted upon by aldolase B or fructose 1-phosphate aldolase converting it into dihydroxy acetone phosphate. Dihydroxy acetone phosphate has got two fates: It is acted upon by triose phosphate isomerase to glyceraldehyde 3-phosphate. Glyceraldehyde 3-phosphate is coverted to glucose 6-phosphate and then glucose 1-phosphate. Glucose 1-phosphate is used for glycogenesis. Dihydroxy acetone phosphate is acted upon by glycerol 3-phosphate dehydrogenase to glycerol 3-phosphate. Fructose 1-phosphate can be oxidised to pyruvate. Pyruvate can be decarboxylated to acetyl CoA. Acetyl CoA is used for fatty acid and cholesterol synthesis. Thus leads to triglyceride synthesis and VLDL formation. Fructose can thus be converted to storage glycogen, triglycerides and cholesterol. Global warming and ice age are extremophilic states. The human body goes into a state of hibernation and stores nutrients as glycogen and triglycerides. Fructose can increase lypogenic enzymes pyruvate kinase, malate dehydrogenase, citrate lyase, acetyl CoA carboxylase, pyruvate dehydrogenase and fatty acid synthase. Thus the metabolism is switched to the hibernatory mode from glucose catabolism. Glucose catabolism stops. The glucose that accumulates enters the archaeal primitive glycolytic and partial citric acid cycle as well as the GABA shunt pathway. The GABA shunt pathway of the archaea generates succinyl CoA and glycine the substrates for the porphyrin synthesis. Porphyrins can self organise to form supramolecular structures which can self replicate called porphyrions. Porphyrions are the ultimate self replicators. They can have a photoinduction induced electron transport chain and ATP synthesis. The porphyrions are dipolar and in the setting of porphyrin intercalating cell membrane producing sodium potassium ATPase inhibition can produce a

pumped phonon system. This is a superconductive state at room temperature and can produce quantal perception. The porphyrins are macromolecular structures with a wave particular existence. Porphyrions are the ultimate quantal observer and mediate the conversion of the quantal foam to the particulate world. This global warming induced fructosemia and accumulated free glucose get catabolized by partial citric acid cycle and GABA shunt of archaea to porphyrins generating porphyrions which can assume the quantal foam state and inhabit a multiverse universe with an eternal existence. The porphyrions can undergo photooxidation generating redox stress. Redox stress will further induce aldose reductase and increase fructosemia. The glucose remains unphosphorylated owing to the high km value of glucose for ketokinase as compared to fructose. The free glucose is catabolized by archaeal enzymes to porphyrins and porphyrions. The human body becomes a zombie for self replicating porphyrions. The porphyrions have quantal perception of low level of EMF. This produces prefrontal cortex atrophy and cerebellar dominance leading to neanderthalisation of the human brain. The human metabolic pathways of glycolysis and oxidative phosphorylation are blocked. The synthesis of glycogen, lipids, cholesterol and glycosaminoglycans dominates. The body switches into the anabolic hibernatory mode. The fructokinase induced by osmotic stress and oxidative stress of global warming is the switch for hibernatory mode leading onto metabolic syndrome or fat storage disease. The free glucose undergoes catabolism by archaeal glycolysis and GABA shunt to porphyrions. The porphyrins can act as a template formation of RNA viroids, DNA viroids, prions and they eventually symbiosed and live together as nanoarchaea. Thus the nanoarchaea can arise from porphyrin templates. The supramolecular porphyrin arrays can have an electron transport chain and ATP synthesis, a primitive form of mitochondria. The nanoarchaea can form as well as self replicate on porphyrin templates. The porphyrin supramolecular arrays or

porphyrions can also self replicate. The human body becomes a zombie for abiogenetic porphyrions and nanoarchaea. The porphyrions can have a macroscopic quantal existence and inhabit multiverse universes. Thus the human metabolism grinds to a halt and the world of nanoarchaea and porphyrions which are eternal steps in. The human race as we know of extincts owing to the metabolonomics of global warming. It is back to the work board of evolution once more.

The Neanderthals lived in the Eurasian Steppes which was cold. They evolved hibernatory metabolism and a fat storage syndrome to protect them from the cold. The Neanderthals evolved due to endosymbiotic archaeal growth. Endosymbiotic archaea are extremophiles and grow in extremes of climate - the ice age and global warming. The global warming results in increase in endosymbiotic archaeal growth and neanderthalisation of the homo sapien species. Neanderthalisation is a symbiotic phenomena. The global warming can lead to dehydration and osmotic stress. The archaea can catabolize cholesterol generating digoxin which can induce redox stress. Osmotic stress and redox stress leads to induction of the enzyme aldose reductase which converts glucose to sorbitol. Sorbitol is acted upon by sorbitol dehydrogenase and converted to fructose. Fructose can enter three metabolic schemes. The fructose is converted to alpha glycerophosphate and triglycerides. This results in storage of glucose and fructose as fat and a fat storage syndrome. The subcutaneous fat protects against variation in climatic temperature. The fructose can inhibit mitochondrial function and mitochondrial beta oxidation of fatty acids accentuating storage of fat. Fat can further fuel insulin resistance by fatty acids acting upon the insulin receptor. Insulin resistance leads to still further aldose reductase induction. The fructose can also enter the glucosamine pathway resulting in GAG synthesis and accumulation of mucopolysaccharides and proteoglycans. This can lead onto systemic connective tissue accumulation in visceral organs like liver producing

cirrhosis, lung producing interstitial lung disease, kidney producing the MEN syndrome, the pancreas producing pancreatic fibrosis and CCP, the heart producing cardiomyopathy and EMF and the vascular tree producing mucoid angiopathy. The fructose has got a low km value for ketokinases as compared to glucose and gets selectively phosphorylated. This results in cellular depletion of ATP and membrane sodium potassium ATPase inhibition resulting in increase of intracellular calcium. The calcium produces mitochondrial PT pore dysfunction and redox stress. Redox stress can induce aldose reductase. The depletion of ATP results in further inhibition of phosphorylation of glucose. This results in hyperglycemia. The archaea has got the glycolytic pathway, a partial citric acid cycle and a reverse citric acid cycle for carbon dioxide fixation. The archaea can induce the Warburg phenotype in the human tissues with increase glycolysis, inhibition of pyruvate dehydrogenase and mitochondrial oxidative phosphorylation inhibition. This results in accumulation of pyruvate which enters the GABA shunt scheme generating succinyl CoA and glycine for porphyrin synthesis. The accumulated glucose due to blockade of glucose phosphorylation is metabolized by archaea generating pyruvate. The pyruvate can enter the TCA cycle via pyruvate dehydrogenase generating acetyl CoA for fatty acid and cholesterol synthesis. The archaea can catabolize cholesterol and generate energy. The archaea can convert pyruvate via GABA shunt as said before to porphyrins. Thus there is increased porphyrin synthesis, cholesterol synthesis and catabolism, triglyceride synthesis, nucleic acid synthesis and GAG synthesis. The metabolic patterns change. The blockade of glucose phosphorylation by fructose results in hyperglycemia which can block or inhibit porphyrin accumulation. The porphyrins have a wave particle existence in the macroscopic state and contributes to quantal perception or extrasensory perception in homo neanderthalis. The cellular depletion of ATP consequent to fructose

phosphorylation results in generation of ADP and AMP which are acted upon by deaminases producing uric acid. Uric acid can block mitochondrial function generating redox stress and further induction of aldose reductase. Uric acid can inhibit the TCA cycle due to aconitase inhibition. This results in accumulation of citrate which blocks glycolysis still further. The citrate is acted upon by the induced citrate lyase and fatty acid synthase producing fatty acids. This can lead to triglyceride synthesis and accumulation and a fat storage syndrome. Fatty acids can block glucose metabolism and the glycolytic pathway. The mitochondrial function is blocked by uric acid, fructose and digoxin. The porphyrin arrays can function as a supramolecular organism called porphyrions and transfer electrons and photons. This functions as a primitive form of mitochondria generating ATP. The inhibition of membrane sodium potassium ATPase due to ATP depletion results in membrane sodium potassium ATPase mediated ATP synthesis. The porphyrin array and membrane sodium potassium ATPase mediated ATP synthesis can provide for body energetics. The fructosemia can lead to increased ribose synthesis via the pentose phosphate pathway inducing nucleic acid synthesis and cell proliferation. This can lead to oncogenesis. The fructosemia can lead to fructosylation of proteins producing antigenic proteins. The depletion of ATP consequent to fructose phosphorylation can produce redox stress, NFKB activation and immune activation. This leads to autoimmune disease. Fructose can inhibit brain derived neurotrophic growth factor and lead to genesis of schizophrenia and autism. The cellular depletion of ATP due to fructose phosphorylation can lead to cell death and neurodegeneration. The low km value of fructose for ketokinase can lead to selective phosphorylation of fructose at a rapid rate depleting the cell of ATP stores. This inhibits glucose phosphorylation producing inhibition of glucose metabolism resulting in hyperglycemia and metabolic syndrome. The uric acid generated by this pathway can result in endothelial dysfunction, coronary artery

disease and strokes. Thus the global warming related actinidic archaeal growth can lead onto fructositis, fructosemia and a Lemurian syndrome affecting multiple organ systems.

The oxidative stress due to global warming induces endosymbiotic archaeal growth resulting in oxidative stress and induction of NOX enzyme. This leads to activation of stress induced heme oxygenase which converts heme to bilirubin and biliverdin. The deficiency of heme leads to induction of ALA synthase and porphyrin synthesis. This leads onto porphyrogenesis and porphyrinuria. The porphyrins can form self replicating supramolecular aggregates called porphyrions. The porphyrions can have photon and electron transport resulting in ATP synthesis. The porphyrions are dipolar molecules and have a wave particle existence. Porphyrins intercalating the neuronal membrane produces sodium potassium ATPase inhibition and this results in a pumped phonon system of quantal perception involving dipolar porphyrions. Thus the porphyrions are capable of information storage and acts like a quantal computer. The porphyrions can act as a template for other porphyrions to form. They can therefore self replicate. The porphyrins can also act as a template for the formation of RNA viroids, DNA viroids and prions. They all symbiose together to generate nanoarchaea. The porphyrions can get photooxidised to generate free radicals. Free radical stress induces aldose reductase which converts glucose to fructose and fructose phosphate. The fructose phosphate can be directed to GAG synthesis via glucosamine, lipid synthesis via conversion to alpha glycerophosphate and nucleic acid synthesis via conversion to ribose. The depletion of glucose owing to conversion to fructose increases porphyrin synthesis and porphyrion formation still further. The deficiency of heme produces mitochondrial oxidative phosphorylation defects due to cytochrome C deficiency. The accumulated glucose, fructose and porphyrions can glycate, fructosylate and porphyrinate proteins. This leads onto development of new antigens and autoimmune disease.

The glycation, fructosylation and porphyrination results in development of antibodies against glycolytic enzymes and pyruvate kinase as well as antimitochondrial antibodies. They suppress the energy metabolism and produce and immune mediated metabolic syndrome with defective glycolysis, defective mitochondrial oxidative phosphorylation and eventual immune activation due to oxidative stress, oncogenesis initiated by fructose and cell death due to depletion of ATP by fructose phosphorylation. The starting process involves development of antibodies against porphyrinated, fructosylated and glycated proteins, enzymes and nucleic acid. This can be called as an immune mediated metabolic syndrome and leads to metabolic syndrome x, oncogenesis, autoimmunity, psychiatric disorders and neurodegenerations.

The Neanderthals are symbiotic life form due to archaeal endosymbiosis. The archaea induces the Warburg phenotype with increased glycolysis and the blockade of the TCA cycle and mitochondrial oxidative phosphorylation. The Warburg phenotype is seen in autoimmune disease, schizophrenia, autism, cancer, degeneration and metabolic syndrome x. The Neanderthals ate a ketogenic diet of fat and protein to suppress the glycolytic pathway. The Neanderthal hybrids formed by homo sapien mating had a high carbohydrate diet due to grain cultivation in settled colonies. This tends to increased glycolysis and accentuates the Warburg phenotype and associated disorders. The glycolytic pathway is upregulated and the mitochondrial oxidative phosphorylation is inhibited. To counteract this certain disease patterns developed in the hybrid population as a adaptive mechanism. These group of disorders develop autoantibodies against glycolytic enzymes. The cell envelope is of archaeal origin and the glycolytic enzymes are cytosolic. This is opposed to the mitochondrial oxidative phosphorylation scheme which is rickettsial in origin. The primitive parts of the brain the cerebellum functions as an archaeal colony network and promotes the Warburg phenotype and glycolysis. The

cerebellar brain is dominant in Neanderthals. The HLA genes are neanderthalic in origin and modulate lymphocytic function. The lymphocytes depend on glycolysis for its energy needs. The neocortex functions as a retroviral colony and promotes mitochondrial oxidative phosphorylation. The HERV genes functions as jumping genes and they can jump and insert themselves in between glycolytic enzyme genetic sequences producing mutations and mutated glycolytic enzymes. The glycolytic pathway becomes dysfunctional. Antibodies are formed against the mutated glycolytic proteins. Thus glycolysis and energy metabolism comes to a halt due to the inhibitory effect of the selfish HERV genes which needs mitochondrial function and ROS generation for its replicatory function and communicating with the cell. Disorders like autoimmune disease, schizophrenia, autism, cancer, degeneration and metabolic syndrome x are disorders of glycolysis and have an autoimmune component against glycolytic enzymes. Glycolytic inhibition and ketogenic diet is one way to treat autoimmune disease, schizophrenia, autism, cancer, degeneration and metabolic syndrome x. All autoimmune diseases develop to suppress the Warburg phenotype in Neanderthal hybrids. The increased glycolysis contributes to oncogenesis via the mitochondrial PT pore hexokinase. The increased glycolysis produces nuclear cell death via the GAPDH pathway. The phosphoglycerate gets converted to phosphoserine and glycine which can modulate NMDA. Fructose 1,6 diphosphate enters the pentose phosphate pathway generating NADPH which activates NOX modulating NMDA function. Thus the glycolytic pathway can modulate the NMDA pathway contributing to schizophrenia and autism due to dysfunction of consciousness. The PDH inhibition accumulates pyruvate which enters the GABA shunt generating succinyl CoA and glycine as well as GABA. Succinyl CoA and glycine are substrates for porphyrin synthesis and contributes to quantal perception important in schizophrenia and autism. The increased lymphocytic glycolysis

and glycolytic antigens contribute to autoimmune disease. Glycolytic antigens also contribute to neurodegeneration, neuropsychiatric disorders and metabolic syndrome x. GAD antibodies are involved in metabolic syndrome x. Autoimmunity is a part of antibody mediated attempt to inhibit glycolysis and Warburg phenotype in Neanderthal hybrids who consume a high carbohydrate diet. This as a by-product generates neurodegeneration, autoimmune disease, schizophrenia, autism, cancer and civilisational disease. All these can be controlled by glycolytic inhibitors and ketogenic diet.

Chapter 2

Endosymbiotic Archaeal Metabolonomics, Neoneanderthalisation and Human Disease - The Origins of Cancer, Autoimmune Disease, Neurodegeneration, Metabolic Syndrome X and Schizophrenia/Autism - Relation to Retroviral Resistance

Introduction

Actinidic archaea has been related to global warming and human diseases especially autoimmune disease, neurodegeneration, neuropsychiatric disorder, neoplasm and metabolic syndrome x. The growth of endosymbiotic actinidic archaea in relation to climate change and global warming leads to neanderthalisation of the human mind-body system. Neanderthal anthropometry and metabolonomics has been described in autoimmune disease, neurodegeneration, neuropsychiatric disorder, neoplasm and metabolic syndrome x especially the Warburg phenotype and hyperdigoxinemia. The human body is driven by archaeal metabolism which contributes to neanderthalisation of the homo sapien species. Digoxin produced by archaeal cholesterol catabolism produces neanderthalisation. Prefrontal cortical atrophy and cerebellar hyperplasia has been related to autoimmune disease, neurodegeneration, neuropsychiatric disorder, neoplasm and metabolic syndrome x in this communication. This leads on to dysautonomia with sympathetic hyperactivity and parasympathetic neuropathy in these disorders. Actinidic archaeal related cerebellar dominance leads to changes in brain function. The archaeal cholesterol catabolism leads to ring oxidase activity generated pyruvate. This enters the GABA shunt pathway producing succinyl CoA and glycine contributing to porphyrin synthesis. The porphyrins contribute to the pathology of these disorders. The archaeal generated digoxin and porphyrins are thus crucial to the evolution of these disorders. Retroviral resistance has been described in Neanderthal species. The increased incidence of archaeal mediated neanderthalisation contributes to retroviral resistance. Digoxin produces intracellular magnesium deficiency which inhibits reverse transcriptase activity and retroviral replication. The porphyrins by photoinduction can induce retroviral death. Thus the archaeal mediated

neanderthalisation can contribute to civilisational diseases - autoimmune disease, neurodegeneration, neuropsychiatric disorder, neoplasm and metabolic syndrome x and retroviral resistance.[1-16] The data is described in this paper.

Materials and Methods

Fifteen cases, each of autoimmune disease, neurodegeneration, neuropsychiatric disorder, neoplasm, metabolic syndrome x and internet addicts were selected for the study. Each case had an age and sex matched control. Neanderthal anthropometric and phenotypic measurements which included protruding supra-orbital ridges, dolichocephalic skull, small mandible, prominent mid face and nose, short upper and lower limbs, prominent trunk, low index finger-ring finger ratio and fair complexion were evaluated in the cases study. Autonomic function tests were done to assess the sympathetic and parasympathetic system in each case. CT scan of the head was done to have a volumetric assessment of the prefrontal cortex and cerebellum. Blood cytochrome F420 activity was assessed by spectrophotometric measurement.

Results

All the case groups studied had higher percentage of Neanderthal anthropometric and phenotypic measurements. There was low index finger-ring finger ratio suggestive of high testosterone levels in all the patient population studied. In all the case groups studied, there also was prefrontal cortex atrophy and cerebellar hyperplasia. Similarly in the all the case groups studied, there was dysautonomia with sympathetic overactivity and parasympathetic neuropathy. Cytochrome F420 was detected in the entire case group studied showing endosymbiotic archaeal overgrowth.

Table 1. *Neanderthal phenotype and systemic disease.*

Disease	Cyt F420 activity	Neanderthal phenotype	Low index finger-ring finger ratio
Schizophrenia	69%	75%	65%
Autism	80%	75%	72%
Alzheimer's disease	89%	65%	75%
Parkinson's disease	70%	71%	80%
Non-Hodgkin's lymphoma	72%	60%	69%
Multiple myeloma	70%	68%	74%
Diabetes mellitus with stroke and CAD	65%	72%	72%
SLE/Lupus	75%	85%	74%
Multiple sclerosis	80%	75%	75%
Internet users	65%	72%	69%

Table 2. *Neanderthal phenotype and brain dysfunction.*

Disease	Dysautonomia	Prefrontal cortex atrophy	Cerebellar hypertrophy
Schizophrenia	65%	60%	70%
Autism	72%	69%	72%
Alzheimer's disease	60%	72%	60%
Parkinson's disease	62%	71%	68%
Non-Hodgkin's lymphoma	79%	65%	75%
Multiple myeloma	69%	72%	80%
Diabetes mellitus with stroke and CAD	64%	84%	69%
SLE/Lupus	75%	73%	72%
Multiple sclerosis	69%	74%	76%
Internet users	74%	84%	82%

Discussion

Neanderthal metabolonomics contribute to the pathogenesis of these disorders. There were Neanderthal phenotypic features in all the case groups studied as well as low index finger-ring finger ratios suggestive of increased testosterone levels. Neanderthalisation of the mind-body system occurs due to increased growth of actinidic archaea as a consequence of global warming.

Neanderthalisation of the mind leads to cerebellar dominance and prefrontal cortex atrophy. This leads to dysautonomia with parasympathetic neuropathy and sympathetic hyperactivity. Digoxin produced by archaeal cholesterol catabolism produces neanderthalisation. Prefrontal cortical atrophy and cerebellar hyperplasia has been related to autoimmune disease, neurodegeneration, neuropsychiatric disorder, neoplasm and metabolic syndrome x in this communication. This leads on to dysautonomia with sympathetic hyperactivity and parasympathetic neuropathy in these disorders. Actinidic archaeal related cerebellar dominance leads to changes in brain function. The archaeal cholesterol catabolism leads to ring oxidase activity generated pyruvate. This enters the GABA shunt pathway producing succinyl CoA and glycine contributing to porphyrin synthesis. The porphyrins contribute to the pathology of these disorders. The archaeal generated digoxin and porphyrins are thus crucial to the evolution of these disorders. Retroviral resistance has been described in Neanderthal species. The increased incidence of archaeal mediated neanderthalisation contributes to retroviral resistance. Digoxin produces intracellular magnesium deficiency which inhibits reverse transcriptase activity and retroviral replication. The porphyrins by photoinduction can induce retroviral death. Thus the archaeal mediated neanderthalisation can contribute to civilisational diseases - autoimmune disease, neurodegeneration, neuropsychiatric disorder, neoplasm and metabolic syndrome x and retroviral resistance.

Global warming and the ice age produces increased growth of extremophiles. This leads to increased growth of actinidic archaeal endosymbiosis in humans. There is archaeal proliferation in the gut which enters the cerebellum and brain stem by reverse axonal transport via the vagus. The cerebellum and brain stem can be considered as an archaeal colony. The archaea are cholesterol catabolizing and use cholesterol as a carbon and energy source. The actinidic

archaea activates the toll receptor HIF alpha inducing the Warburg phenotype resulting in increased glycolysis with generation of glycine as well as pyruvate dehydrogenase suppression. The accumulated pyruvate enters the GABA shunt generating of succinyl CoA and glycine. The archaeal catabolism of cholesterol produces ring oxidation and generation of pyruvate which also enters the GABA shunt scheme producing glycine and succinyl CoA. This leads to increased synthesis of porphyrins. In the setting of digoxin induced sodium potassium ATPase inhibition the dipolar porphyrins produce a pumped phonon system resulting in the Frohlich model Bose-Einstein condensate and quantal perception of low level EMF. Low level EMF pollution is common with internet usage. Perception of low level of EMF leads to neanderthalisation of the brain with prefrontal cortex atrophy and cerebellar hyperplasia. The archaea which reaches the cerebellum from the gut via the vagus nerve proliferates and makes the cerebellum dominant with resultant suppression and atrophy of the prefrontal cortex. This leads to wide spread autistic and schizophrenic traits in population. The actinidic archaea induces the Warburg phenotype with increased glycolysis, PDH inhibition and mitochondrial suppression. This produces neanderthalisation of the mind-body system. The actinidic archaea secretes RNA viroids which block HERV expression by RNA interference. The HERV suppression contributes to the inhibition of prefrontal cortex development in Neanderthals and cerebellar dominance. Archaeal digoxin produces sodium potassium ATPase inhibition and magnesium depletion causing reverse transcriptase inhibition and decreased generation of HERV. The HERV contributes to the dynamicity of the genome and are required for the development of the prefrontal cortex. The HERV suppression contributes to retroviral resistance in Neanderthals. The actinidic archaea catabolizes cholesterol leading to cholesterol depleted state. Cholesterol depletion also leads to poor synaptic connectivity and decreased development of prefrontal cortex.

This is not genetic change but a form of symbiotic change with endosymbiotic actinidic archaeal growth in the body and brain.

Internet use and low level EMF pollution is common in this century. This results in increased low level EMF perception by the brain by the digoxin-porphyrin mediated pumped phonon system created Bose-Einstein condensates contributing to prefrontal cortex atrophy and cerebellar dominance. Cerebellar dominance leads to schizophrenia and autism. There is an epidemic of autism and schizophrenia in the present day community. The porphyrin mediated extrasensory perception can contribute to communication among Neanderthals. Neanderthals did not have a language and used extrasensory perception as a form of group communication. Because of dominant extrasensory quantal perception, the Neanderthals did not have individual identity but only group identity. Cerebellar dominance results in creativity consequent to quantal perception and group perception. The neanderthalic traits contribute to innovation and creativity. Cerebellar dominance results in development of a symbolic language. The Neanderthals used dance and music as a form of communication. Painting as a form of communication was also common in Neanderthals. Neanderthal behaviour was robotic. Robotic behaviour is characteristic of cerebellar dominance. Robotic, symbolic and ritualistic behaviour is common with cerebellar dominance and is seen in autistic traits. The cerebellar dominance in Neanderthals leads to intuitive intelligence and a hypnotic quality to communication. The increased extrasensory quantal perception leads to more communion with nature and a form of eco-spirituality. The increasing use of dance and music as a form of communication and eco-spirituality is common in the modern century along with increased incidence of autism. The cholesterol depletion leads to bile acid deficiency and generation of small social groups in Neanderthals. Bile acid

binds to olfactory receptors and contributes to group identity. This can also contributes to the generation of autistic features in Neanderthals.

The Neanderthal population was predominantly autistic and schizophrenic. The modern population is a hybrid of homo sapiens and homo neanderthalis. This contributes to 10 to 20 per cent dominant hybrids who tend to have schizophrenic and autistic qualities and contributes to creativity of civilisation. The Neanderthals tend to be innovative and chaotic. They tend to be creative in art, literature, dance, spirituality and science. Eighty per cent of less dominant hybrids are stable and contribute to a stabilizing influence leading to growth of civilisation. The homo sapiens were stable and non-creative over a long period of their existence. There was a burst of creativity with generation of music, dance, painting, ornaments, the creation of concept of God and compassionate group behaviour around 10,000 years ago in the homo sapiens community. This correlated with the generation of Neanderthal hybrids when the Eurasian Neanderthal male mated with homo sapiens African females. The extrasensory/quantal perception due to dipolar porphyrins and digoxin induced sodium potassium ATPase inhibition and the generated pumped phonon system mediated quantal perception leads to the globalisation phenomena and feeling of the world being a global village. The archaeal cholesterol catabolism leads to increased synthesis of digoxin. Digoxin promotes tryptophan transport over tyrosine. Tyrosine deficiency leads to dopamine deficiency and morphine deficiency. This leads to a morphine deficiency syndrome in Neanderthals. This contributes to addiction traits and creativity. The increased tryptophan levels produce increased alkaloids like LSD contributing to ecstasy and spirituality of Neanderthal population. Addictive, ADHD and autistic features are related to the morphine deficiency state. The ketogenic diet consumed by the meat eating Neanderthals leads on to increased generation of hydroxy butyric acid which produces ecstasy and a dissociative type of anaesthesia contributing to the

Neanderthal psychology. The dopamine deficiency leads to decreased melanin synthesis and fairness of the population. This was responsible for the fair colour of the Neanderthals.

The Neanderthals were essentially meat eaters taking a ketogenic diet. The acetoacetic acid is converted to acetyl CoA which enters the TCA cycle. When the Neanderthal hybrids consume a glucogenic diet owing to the spread of settled civilisation it produces pyruvate accumulation owing to PDH suppression in Neanderthals. The increased archaeal growth activates the toll receptor and induces HIF alpha resulting in increased glycolysis, PDH suppression and mitochondrial dysfunction - the Warburg phenotype. The pyruvate enters the GABA shunt pathway producing glutamate, ammonia and porphyrins resulting in neuropathology of autism and schizophrenia. Neanderthals consuming a ketogenic diet produces more of GABA an inhibitory neurotransmitter resulting in the docile quiet nature of the Neanderthals. There is less production of glutamate the predominant excitatory neurotransmitter of the prefrontal cortex and consciousness pathways. This leads onto dominance of cerebellar function. The Neanderthal hybrids have cerebellar dominance and less of conscious behaviour. Cerebellum is responsible for intuitive, unconscious behaviour as well as creativity and spirituality. The cerebellum is the site of extrasensory perception, magical acts and hypnosis. The predominant homo sapiens had prefrontal cortex dominance over the cerebellum resulting in more of conscious behaviour.

The Neanderthals consuming a glucogenic diet produces increased glycolysis in the setting of PDH inhibition. This produces the Warburg phenotype. There is increased lymphocytic glycolysis producing autoimmune diseases and immune activation. The increased levels of GAPD result in nuclear cell death and neurodegeneration. The predominance of glycolysis and suppression of mitochondrial function results in glycemia and metabolic syndrome x. The

increased mitochondrial PT pore hexokinase leads to cell proliferation and oncogenesis. The glycolytic intermediate 3-phosphoglycerate is converted to glycine resulting in NMDA excitotoxicity contributing to schizophrenia and autism. Cerebellar dominance is reported in schizophrenia and autism.

The cerebellar hyperplasia results in sympathetic hyperactivity and parasympathetic neuropathy. This contributes to cell proliferation and oncogenesis. Vagal neuropathy results in immune activation and autoimmune disease. Vagal neuropathy and sympathetic overactivity can contribute to glycogenolysis and lipolysis resulting in metabolic syndrome x. Cerebellar dominance and cerebellar cognitive affective dysfunction can contribute to schizophrenia and autism. The increased porphyrin synthesis resulting from succinyl CoA generated by GABA shunt and glycine generated by glycolysis contributes to increased extrasensory perception important in schizophrenia and autism. Sympathetic overactivity and parasympathetic neuropathy can contribute to neurodegeneration.

The archaeal cholesterol catabolism generates digoxin which produces sodium potassium ATPase inhibition and increase in intracellular calcium and decrease in intracellular magnesium. The increase in intracellular calcium produces oncogene activation and NFKB activation resulting in malignancies and autoimmune diseases. The increase in intracellular calcium opens the mitochondrial PT pore resulting in cell death and neurodegeneration. The increase in intracellular calcium can modulate the neurotransmitter release from presynaptic vesicles. This can modulate neurotransmission. Digoxin induced magnesium depletion can remove the magnesium block on the NMDA receptor resulting in NMDA excitotoxicity. Digoxin can modulate the glutamatergic thalamo-cortico-thalamic pathway and consciousness resulting in schizophrenia and autism. Digoxin induced magnesium depletion can inhibit reverse transcriptase activity and HERV generation modulating the dynamicity of the

genome. Digoxin induced intracellular calcium accumulation and magnesium depletion can modulate G-protein and protein tyrosine kinase dependent neurotransmitter and endocrine receptors. This can produce digoxin induced neuro-immuno-endocrine integration. Digoxin functions as a Neanderthal master hormone.

The actinidic archaea are cholesterol catabolising and leads to low levels of testosterone and estrogen. This leads on to asexual features and low reproductive rates of the Neanderthal population. The Neanderthals consume a low fibre diet with low lignan content. The actinidic archaea has cholesterol catabolizing enzymes generating more of testosterone than estrogens. This contributes to estrogen deficiency and testosterone overactivity. The Neanderthal population are hypermales with concommitant right hemispheric dominance and cerebellar dominance. Testosterone suppresses left hemispheric function. The high testosterone levels in Neanderthals contribute to a bigger brain. The Neanderthals males as well as females had a higher level of testosterone contributing to gender equality and gender neutral states. There was group identity and group motherhood with no differences between roles of both males and females. This also resulted in matrilinearity. The higher testosterone levels in males as well as females led to alternate type of sexuality and aberrant behaviour. Homo sapiens had higher reproductive rates and overtook the Neanderthal population resulting in its extinction. The homo sapien population was conservative with normal sexual mores, family values and patriarchal type of behaviour. The role of females the homo sapien community was inferior to males. The increasing generation of Neanderthal hybrids due to climate change mediated archaeal overgrowth leads to gender equality and equidominance of male and female in this century.

The cholesterol catabolism results in cholesterol depletion and bile acid deficiency. Bile acids bind to VDR and are immunomodulatory. Bile acid

deficiency leads to immune activation and autoimmune disease. Bile acids bind to FXR, LXR and PXR modulating lipid and carbohydrate metabolism. This leads to metabolic syndrome x in the presence of bile acid deficiency. Bile acid uncouples oxidative phosphorylation and its deficiency leads to obesity of metabolic syndrome x. Bile acids bind to olfactory receptors and are important in group identity. Bile acid deficiency leads to formation of small social groups in Neanderthals and genesis of autism. Cholesterol depletion also leads to vitamin D deficiency. Vitamin D binds to VDR and produces immunomodulation. Vitamin D deficiency leads to immune activation and autoimmune diseases. Vitamin D deficiency can also produce rickets and contribute to the phenotypic features of Neanderthals. Vitamin D deficiency can contribute to brain development resulting in macrocephaly. Vitamin D deficiency contributes to insulin resistance and truncal obesity of Neanderthals. Vitamin D deficiency contributes to the fairness of the Neanderthal skin as a phenotypic adaptation. The Neanderthal phenotypic features are due to vitamin D deficiency and insulin resistance.

Thus global warming and increased endosymbiotic actinidic archaeal growth leads to cholesterol catabolism and generation of the Warburg phenotype resulting in increased porphyrin synthesis, extrasensory low EMF perception, prefrontal cortex atrophy, insulin resistance and cerebellar dominance. This leads on to neanderthalisation of the body and brain.

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Chapter 3

Endosymbiotic Actinidic Archaeal Mediated Warburg Phenotype Mediates Human Disease State

Introduction

Endomyocardial fibrosis along with the root wilt disease of coconut is endemic to Kerala with its radioactive actinide beach sands. Actinides like rutile as well as organisms like phytoplasmas and viroids have been implicated in the etiology of these diseases.[1-4] The Warburg phenotype has been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration.[4] The possibility of Warburg phenotype induced by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism was considered in this paper.[5-8] An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described.[7, 9]

Materials and Methods

The following groups were included in the study: - endomyocardial fibrosis, Alzheimer's disease, multiple sclerosis, non-Hodgkin's lymphoma, metabolic syndrome x with cerebrovascular thrombosis and coronary artery disease, schizophrenia, autism, seizure disorder, Creutzfeldt Jakob's disease and acquired immunodeficiency syndrome. There were 10 patients in each group and each patient had an age and sex matched healthy control selected randomly from the general population. The blood samples were drawn in the fasting state before treatment was initiated. Plasma from fasting heparinised blood was used and the experimental protocol was as follows (I) Plasma+phosphate buffered saline, (II) same as I+cholesterol substrate, (III) same as II+rutile 0.1 mg/ml, (IV) same as II+ciprofloxacine and doxycycline each in a concentration of 1 mg/ml. Cholesterol substrate was prepared as described by Richmond.[10] Aliquots were withdrawn at zero time immediately after mixing and after incubation at 37 ℃ for 1 hour. The following estimations were carried out: - Cytochrome F420 and

hexokinase.[11-13] Cytochrome F420 was estimated flourimetrically (excitation wavelength 420 nm and emission wavelength 520 nm). Informed consent of the subjects and the approval of the ethics committee were obtained for the study. The statistical analysis was done by ANOVA.

Results

Plasma of control subjects showed increased levels of the above mentioned parameters with after incubation for 1 hour and addition of cholesterol substrate resulted in still further significant increase in these parameters. The plasma of patients showed similar results but the extent of increase was more. The addition of antibiotics to the control plasma caused a decrease in all the parameters while addition of rutile increased their levels. The addition of antibiotics to the patient's plasma caused a decrease in all the parameters while addition of rutile increased their levels but the extent of change was more in patient's sera as compared to controls. The results are expressed in tables 1-2 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time.

Table 1. *Effect of rutile and antibiotics on cytochrome F420.*

Group	CYT F420 % (Increase with Rutile)		CYT F420 % (Decrease with Doxy+Cipro)	
	Mean	**±SD**	**Mean**	**± SD**
Normal	4.48	0.15	18.24	0.66
Schizo	23.24	2.01	58.72	7.08
Seizure	23.46	1.87	59.27	8.86
AD	23.12	2.00	56.90	6.94
MS	22.12	1.81	61.33	9.82
NHL	22.79	2.13	55.90	7.29
DM	22.59	1.86	57.05	8.45
AIDS	22.29	1.66	59.02	7.50
CJD	22.06	1.61	57.81	6.04
Autism	21.68	1.90	57.93	9.64
EMF	22.70	1.87	60.46	8.06
F value	306.749		130.054	
P value	< 0.001		< 0.001	

Table 2. *Effect of rutile and antibiotics on hexokinase.*

Group	Hexokinase % change (Increase with Rutile)		Hexokinase % change (Decrease with Doxy+Cipro)	
	Mean	**± SD**	**Mean**	**± SD**
Normal	4.21	0.16	18.56	0.76
Schizo	23.01	2.61	65.87	5.27
Seizure	23.33	1.79	62.50	5.56
AD	22.96	2.12	65.11	5.91
MS	22.81	1.91	63.47	5.81
NHL	22.53	2.41	64.29	5.44
DM	23.23	1.88	65.11	5.14
AIDS	21.11	2.25	64.20	5.38
CJD	22.47	2.17	65.97	4.62
Autism	22.88	1.87	65.45	5.08
EMF	21.66	1.94	67.03	5.97
F value	292.065		317.966	
P value	< 0.001		< 0.001	

Discussion

There was increase in cytochrome F420 indicating archaeal growth. The archaea can synthesize and use cholesterol as a carbon and energy source.[6, 14] The archaeal origin of the enzyme activities was indicated by antibiotic induced suppression. The study indicates the presence of actinide based archaea with an alternate actinide based enzymes or metalloenzymes in the system as indicated by rutile induced increase in enzyme activities.[15, 16] The archaeal glycolytic hexokinase activity were increased. The part of the increased glycolytic hexokinase activity detected is human. The archaea can undergo magnetite and calcium carbonate mineralization and can exist as calcified nanoforms.[17]

Archaea can induce the host AKT PI3K, AMPK, HIF alpha and NFKB producing the Warburg metabolic phenotype.[18] The increased glycolytic hexokinase activity indicates the generation of the Warburg phenotype. The generation of the Warburg phenotype is due to activation of HIF alpha. This stimulates anaerobic glycolysis, inhibits pyruvate dehydrogenase, inhibits mitochondrial oxidative phosphorylation, stimulates heme oxygenase, stimulates VEGF and activates nitric oxide synthase. This can lead to increased cell proliferation and malignant transformation. The mitochondrial PT pore hexokinase is increased leading onto cell proliferation. There is induction of glycolysis, inhibition of PDH activity and mitochondrial dysfunction resulting in inefficient energetics and metabolic syndrome. The archaea and viroid generated cytokines can lead to TNF alpha induced insulin resistance and metabolic syndrome x. The increase in glycolysis can activate glyceraldehyde 3 phosphate dehydrogenase which gets translocated to the nucleus after polyadenylation. The PARP enzyme is activated by glycolysis mediated redox stress. This can produce nuclear cell death and neuronal degeneration. The increase in the glycolytic enzyme fructose 1,6 diphosphatase increases the pentose phosphate pathway. This generates NADPH which activates NOX.

NOX activation is related to NMDA activation and glutamate excitotoxicity. This leads onto neuronal degeneration.[18]

The increase in glycolysis activates the enzyme fructose 1,6 diphosphatase which activates the pentose phosphate pathway liberating NADPH. This increases NOX activity generating free radical stress and H_2O_2. Free radical stress is related to insulin resistance and metabolic syndrome x. Free radicals can activate NFKB producing immune activation and autoimmune disease. Free radicals can open the mitochondrial PT pore, produce release of cyto C and activate the caspase cascade. This produces cell death and neuronal degeneration. The free radicals can activate NMDA receptor and induce the enzyme GAD generating GABA. This activates the NMDA/GABA thalamo-cortico-thalamic pathway mediating conscious perception. Increased free radical generation can also initiate schizophrenia. Free radicals can also produce oncogene activation and malignant transformation. Free radicals can produce HDAC inhibition and HERV generation. The encapsulation of HERV particles in phospholipids vesicles can mediate the generation of the acquired immunodeficiency syndrome. Free radicals can also promote atherogenesis.[18]

The lymphocytes depend on glycolysis for its energy needs. The increase in glycolysis owing to the induction of Warburg phenotype can lead to immune activation. Immune activation can lead to autoimmune disease. TNF alpha can activate the NMDA receptor leading to glutamate excitotoxicity and neuronal degeneration. TNF alpha activating NMDA receptor can contribute to schizophrenia. TNF alpha can induce expression of HERV particles contributing to generation of acquired immunodeficiency syndrome. Immune activation has also been related to malignant transformation mediated by NFKB. TNF alpha can also act upon the insulin receptor producing insulin resistance. NOX activation consequent to the generation of the Warburg phenotype also

activates the insulin receptor. Thus there is a hyperinsulinemic state leading on to metabolic syndrome x.[18]

Thus the induction of the Warburg phenotype can lead to malignancy, autoimmune disease, metabolic syndrome x, neuropsychiatric disease and neuronal degeneration. The Warburg phenotype leads to inhibition of pyruvate dehydrogenase and accumulation of pyruvate. The accumulated pyruvate enters the GABA shunt pathway and is converted to citrate which is acted upon by citrate lyase and converted to acetyl CoA, used for cholesterol synthesis. The pyruvate can be converted to glutamate and ammonia which is oxidised by archaea for energy needs. The increased cholesterol substrate leads to increased archaeal growth and further induction of the Warburg phenotype.[18]

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Chapter 4

Endosymbiotic Actinidic Archaeal Cholesterol Catabolic Syndrome

Introduction

Cholesterol is converted to bile acids by cholesterol 7 aplha hydroxylase in archaea and human cells. Bile acids can regulate metabolism, neuronal transmission and immunity. Bile acids bind to VDR receptor and downregulate the innate immune response. Bile acid deficiency leads to autoimmune disease. Bile acids bind to FXR receptor increasing glycogenesis, inhibiting gluconeogenesis and inhibiting triglyceride and VLDL synthesis. Thus bile acids can prevent metabolic syndrome x. Bile acids bind to the PXR receptor increasing cholesterol degradation. Blocking the PXR receptor produces cholesterol toxicity. Bile acids bind to TGR 5 receptor converting T4 to T3 and uncouple oxidative phosphorylation. T3 functions as a neurotransmitter in the cerebellar cortex and leads to dominance of unconscious brain function and CCAS. Bile acids also bind to olfactory receptors and stimulate the limbic lobe. Bile acid deficiency can lead to autism and schizophrenia. Thus the archaeal cholesterol catabolic syndrome can lead to cholesterol deficiency and bile acid deficiency. CoQ deficiency can lead to an autonomic neuropathy also contributing to metabolic syndrome, psychiatric disorders and autoimmune disease as well as neurodegenerations and malignancy.

Actinidic archaea have been implicated in the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration.[1-9] Actinide based primitive organism like archaea have a mevalonate pathway and cholesterol catabolism. Cholesterol catabolism by actinidic archaea can lead to cholesterol depletion and a hypocholesterolemic state contributing to the pathogenesis of these disorders.[10-17]

Archaea can use cholesterol as a carbon and energy source. Archaeal cholesterol catabolism can lead to multiple systemic disease. Low cholesterol values in populations have been related to high mortality. The archaeal

cholesterol catabolizing enzymes were studied and the results in presented in this paper. This can be described as the endosymbiotic actinidic archaeal cholesterol catabolic syndrome.[10-17]

Materials and Methods

The following groups were included in the study: - meditation group, endomyocardial fibrosis, Alzheimer's disease, multiple sclerosis, non-Hodgkin's lymphoma, metabolic syndrome x with cerebrovascular thrombosis and coronary artery disease, schizophrenia, autism, seizure disorder, Creutzfeldt Jakob's disease and acquired immunodeficiency syndrome. There were 10 patients in each group and each patient had an age and sex matched healthy control selected randomly from the general population. The blood samples were drawn in the fasting state before treatment was initiated. Plasma from fasting heparinised blood was used and the experimental protocol was as follows (I) Plasma+phosphate buffered saline, (II) same as I+cholesterol substrate, (III) same as II+rutile 0.1 mg/ml, (IV) same as II+ciprofloxacine and doxycycline each in a concentration of 1 mg/ml. Cholesterol substrate was prepared as described by Richmond.[18] Aliquots were withdrawn at zero time immediately after mixing and after incubation at 37 ℃ for 1 hour. The following estimations were carried out: - Cytochrome F420, polycyclic aromatic hydrocarbon, digoxin, bile acid, cholesterol oxidase activity measured by hydrogen peroxide liberation, pyruvate, butyrate and propionate were estimated.[19-21] Cytochrome F420 was estimated flourimetrically (excitation wavelength 420 nm and emission wavelength 520 nm). Polycyclic aromatic hydrocarbon was estimated by measuring hydrogen peroxide liberated by using glucose reagent. Informed consent of the subjects and the approval of the ethics committee were obtained for the study. The statistical analysis was done by ANOVA.

Results

Plasma of control subjects showed increased levels of the above mentioned parameters with after incubation for 1 hour and addition of cholesterol substrate resulted in still further significant increase in these parameters. The plasma of patients showed similar results but the extent of increase was more. The addition of antibiotics to the control plasma caused a decrease in all the parameters while addition of rutile increased their levels. The addition of antibiotics to the patient's plasma caused a decrease in all the parameters while addition of rutile increased their levels but the extent of change was more in patient's sera as compared to controls. The results are expressed in tables 1-4 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time.

Table 1. *Effect of rutile and antibiotics on cytochrome F420 and PAH.*

Group	CYT F420 % (Increase with Rutile)		CYT F420 % (Decrease with Doxy+Cipro)		PAH % change (Increase with Rutile)		PAH % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.48	0.15	18.24	0.66	4.45	0.14	18.25	0.72
Schizo	23.24	2.01	58.72	7.08	23.01	1.69	59.49	4.30
Seizure	23.46	1.87	59.27	8.86	22.67	2.29	57.69	5.29
AD	23.12	2.00	56.90	6.94	23.26	1.53	60.91	7.59
MS	22.12	1.81	61.33	9.82	22.83	1.78	59.84	7.62
NHL	22.79	2.13	55.90	7.29	22.84	1.42	66.07	3.78
DM	22.59	1.86	57.05	8.45	23.40	1.55	65.77	5.27
Meditation	22.29	1.66	59.02	7.50	23.23	1.97	65.89	5.05
CJD	22.06	1.61	57.81	6.04	23.46	1.91	61.56	4.61
Autism	21.68	1.90	57.93	9.64	22.61	1.42	64.48	6.90
EMF	22.70	1.87	60.46	8.06	23.73	1.38	65.20	6.20
F value	306.749		130.054		391.318		257.996	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 2. *Effect of rutile and antibiotics on butyrate and propionate generation from cholesterol.*

Group	Butyrate % change (Increase with Rutile)		Butyrate % change (Decrease with Doxy+Cipro)		Propionate % change (Increase with Rutile)		Propionate % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.43	0.19	18.13	0.63	4.40	0.10	18.48	0.39
Schizo	22.50	1.66	60.21	7.42	22.52	1.90	66.39	4.20
Seizure	23.81	1.19	61.08	7.38	22.83	1.90	67.23	3.45
AD	22.65	2.48	60.19	6.98	23.67	1.68	66.50	3.58
MS	21.14	1.20	60.53	4.70	22.38	1.79	67.10	3.82
NHL	23.35	1.76	59.17	3.33	23.34	1.75	66.80	3.43
DM	23.27	1.53	58.91	6.09	22.87	1.84	66.31	3.68
Meditation	23.32	1.71	63.15	7.62	23.45	1.79	66.32	3.63
CJD	22.86	1.91	63.66	6.88	23.17	1.88	68.53	2.65
Autism	23.52	1.49	63.24	7.36	23.20	1.57	66.65	4.26
EMF	23.29	1.67	60.52	5.38	22.29	2.05	61.91	7.56
F value	380.721		171.228		372.716		556.411	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 3. *Effect of rutile and antibiotics on digoxin and bile acids.*

Group	Digoxin (ng/ml) (Increase with Rutile)		Digoxin (ng/ml) (Decrease with Doxy+Cipro)		Bile Acids % change (Increase with Rutile)		Bile Acids % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	0.11	0.00	0.054	0.003	4.29	0.18	18.15	0.58
Schizo	0.55	0.06	0.219	0.043	23.20	1.87	57.04	4.27
Seizure	0.51	0.05	0.199	0.027	22.61	2.22	66.62	4.99
AD	0.55	0.03	0.192	0.040	22.12	2.19	62.86	6.28
MS	0.52	0.03	0.214	0.032	21.95	2.11	65.46	5.79
NHL	0.54	0.04	0.210	0.042	22.98	2.19	64.96	5.64
DM	0.47	0.04	0.202	0.025	22.87	2.58	64.51	5.93
Meditation	0.56	0.05	0.220	0.052	22.29	1.47	64.35	5.58
CJD	0.53	0.06	0.212	0.045	23.30	1.88	62.49	7.26
Autism	0.53	0.08	0.205	0.041	22.21	2.04	63.84	6.16
EMF	0.51	0.05	0.213	0.033	23.41	1.41	58.70	7.34
F value	135.116		71.706		290.441		203.651	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 4. *Effect of rutile and antibiotics on pyruvate and hydrogen peroxide.*

Group	Pyruvate % change (Increase with Rutile)		Pyruvate % change (Decrease with Doxy+Cipro)		H_2O_2 % (Increase with Rutile)		H_2O_2 % (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.34	0.21	18.43	0.82	4.43	0.19	18.13	0.63
Schizo	20.99	1.46	61.23	9.73	22.50	1.66	60.21	7.42
Seizure	20.94	1.54	62.76	8.52	23.81	1.19	61.08	7.38
AD	22.63	0.88	56.40	8.59	22.65	2.48	60.19	6.98
MS	21.59	1.23	60.28	9.22	21.14	1.20	60.53	4.70
NHL	21.19	1.61	58.57	7.47	23.35	1.76	59.17	3.33
DM	20.67	1.38	58.75	8.12	23.27	1.53	58.91	6.09
Meditation	21.21	2.36	58.73	8.10	23.32	1.71	63.15	7.62
CJD	21.07	1.79	63.90	7.13	22.86	1.91	63.66	6.88
Autism	21.91	1.71	58.45	6.66	23.52	1.49	63.24	7.36
EMF	22.29	2.05	62.37	5.05	23.29	1.67	60.52	5.38
F value	321.255		115.242		380.721		171.228	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Discussion

There was increase in cytochrome F420 indicating archaeal growth. The archaea can synthesize and use cholesterol as a carbon and energy source.[22-24] The archaeal origin of the enzyme activities was indicated by antibiotic induced suppression. The study indicates the presence of actinide based archaea with an alternate actinide based enzymes or metalloenzymes in the system as indicated by rutile induced increase in enzyme activities.[22-24] The archaeal beta hydroxyl steroid dehydrogenase activity indicating digoxin synthesis and archaeal cholesterol hydroxylase activity indicating bile acid synthesis were increased.[22-24] The archaeal cholesterol oxidase activity was increased resulting in generation of pyruvate and hydrogen peroxide.[22-24] The pyruvate gets converted to glutamate and ammonia by the GABA shunt pathway. The archaeal aromatisation of cholesterol generating PAH was also detected.[22-24] This

indicates archaeal cholesterol aromatase activity. The archaeal cholesterol side chain oxidase activity generates butyrate and propionate. Thus archaeal cholesterol oxidase, cholesterol aromatase, cholesterol side chain oxidase, cholesterol hydroxylase and beta hydroxyl steroid dehydrogenase activity were detected in high levels in the patient population of endomyocardial fibrosis, Alzheimer's disease, multiple sclerosis, non-Hodgkin's lymphoma, metabolic syndrome x with cerebrovascular thrombosis and coronary artery disease, schizophrenia, autism, seizure disorder, Creutzfeldt Jakob's disease and acquired immunodeficiency syndrome. The archaeal cholesterol catabolizing enzymes were actinide dependent. The archaea can undergo magnetite and calcium carbonate mineralization and can exist as calcified nanoforms.[25] This leads to a cholesterol depleted state and hypocholesterolemic syndrome in patients with schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration.

Low cholesterol has been related to multiple systemic diseases. Low cholesterol is detected in patients with autism and schizophrenia. Low cholesterol is also associated with neuronal degenerations like Alzheimer's disease and Parkinson's disease. Cholesterol is required for the formation of synaptic connectivity in neuronal cultures. Depletion of cholesterol from the brain results in loss of synaptic connectivity in multiple neuronal circuits contributing to neuropsychiatric disorders and neuronal degeneration. Low cholesterol has also been related to malignancy. Cholesterol is required for contact inhibition. Absence of cholesterol results in loss of contact inhibition and uncontrolled cell proliferation. Low cholesterol has been related to autoimmune disease.[10-17]

The gut endotoxins and lipopolysaccharides are absorbed along with fat producing the syndrome of metabolic endotoxaemia. The endotoxins and lipopolysaccharides can combine with lipoproteins and are detoxified.

Metabolic endotoxaemia produces chronic immune activation and generation of superantigens. This has been related to the genesis of autoimmune disease. Metabolic endotoxaemia results in immune activation and generation of TNF alpha which modulates the insulin receptor producing insulin resistance. Insulin resistance is related to metabolic syndrome x and vascular thrombosis. Metabolic endotoxaemia has been related to neuronal degenerations like Alzheimer's disease and Parkinson's disease. Metabolic endotoxaemia related chronic immune activation drives the retroviral state. Metabolic endotoxaemia can induce NFKB which can drive malignant cell transformation. Thus hypocholesterolemia leads to non-detoxification of endotoxins and lipopolysaccharides resulting in metabolic syndrome x, neuronal degnerations and autoimmune disease.[10-17]

Infections have been related to schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. H. pylori infection and nocardiosis has been related to Parkinson's disease. Chlamydial infection and actinomycosis has been related to Alzheimer's disease. Clostridial infection has been related to motor neuron disease. Atypical mycobacterial infection had been related to malignancy like lymphoma. Staphylococal infections have been related to carcinoma of the breast. Gut bacterial infections had been related to rheumatoid disease. Toxoplasmosis has been related to schizophrenia. Gut bacteria with increase in gut firmicutes and decrease in bacteroides have been related to metabolic syndrome x. Chlamydial infections have been related to vascular disease. Low cholesterol leads to lack of lipoprotein binding to endotoxins.[10-17] The endotoxins and lipopolysaccharides are not detoxified.

Viral diseases have been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. The virus binds to lipid microdomains in the cell membrane. Cholesterol depletion leads to alteration in lipid microdomains and increased

entry of virus in the cell. Herpes virus infection and borna virus disease leads to schizophrenia. Enterovirus infection has been associated with motor neuron disease. Corona virus infection predisposes to Parkinson's disease. Herpes virus infection is implicated in Alzheimer's disease. Herpes virus infection and EBV infections predisposed to SLE. Retroviral infection - exogenous and endogenous have been related to schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. CMV infection and herpes infection has been related to atherogenesis. Prion disease has been related to alterations in cholesterol metabolism. Thus a cholesterol depleted state can lead to increased predilection to viral infection and systemic disease.[10-17]

The actinidic archaea uses cholesterol catabolism to generate energy. The cholesterol catabolizing enzymes of the archaea are dependent on actinides. The archaeal cholesterol catabolism leads to a cholesterol depleted state and systemic disease. Cholesterol depleted state have been related to high mortality. This can be described as the endosymbiotic actinidic archaeal cholesterol catabolic syndrome.[10-17]

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Chapter 5

The Porphyrions and Human Disease

Introduction

Actinidic archaea have been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. Actinidic archaea have a mevalonate pathway and are cholesterol catabolizing.[1-5] They can use cholesterol as a carbon and energy source. Archaeal cholesterol catabolism can generate porphyrins via the cholesterol ring oxidase generated pyruvate and GABA shunt pathway. Archaea can produce a secondary porphyria by inducing the enzyme heme oxygenase resulting in heme depletion and activation of the enzyme ALA synthase. Porphyrins have been related to schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. The role of archaeal porphyrins in regulation of cell functions and neuro-immuno-endocrine integration is discussed. Porphyrins are prebiotic molecules which are involved in abiogenesis and origin of life.[1-5] The porphyrions are self replicating supramolecular organisms which forms the precursor template on which the viroids, prions and nanoarchaea originate. Stress induced template directed abiogenesis of porphyrions, prions, viroids and archaea is a continuous process and can contribute to changes in brain structure and behavior as well as disease process.

Materials and Methods

The following groups were included in the study: - endomyocardial fibrosis, Alzheimer's disease, multiple sclerosis, non-Hodgkin's lymphoma, metabolic syndrome x with cerebrovascular thrombosis and coronary artery disease, schizophrenia, autism, seizure disorder, Creutzfeldt Jakob disease and acquired immunodeficiency syndrome. There were 10 patients in each group and each

patient had an age and sex matched healthy control selected randomly from the general population. The blood samples were drawn in the fasting state before treatment was initiated. Plasma from fasting heparinised blood was used and the experimental protocol was as follows (I) Plasma+phosphate buffered saline, (II) same as I+cholesterol substrate, (III) same as II+rutile 0.1 mg/ml, (IV) same as II+ciprofloxacine and doxycycline each in a concentration of 1 mg/ml. Cholesterol substrate was prepared as described by Richmond. Aliquots were withdrawn at zero time immediately after mixing and after incubation at 37 ℃ for 1 hour. The following estimations were carried out: - Cytochrome F420, free RNA, free DNA, polycyclic aromatic hydrocarbon, hydrogen peroxide, pyruvate, ammonia, glutamate, delta aminolevulinic acid, succinate, glycine and digoxin. Cytochrome F420 was estimated flourimetrically (excitation wavelength 420 nm and emission wavelength 520 nm). Polycyclic aromatic hydrocarbon was estimated by measuring hydrogen peroxide liberated by using glucose reagent. The study also involved estimating the following parameters in the patient population - digoxin, bile acid, hexokinase, porphyrins, pyruvate, glutamate, ammonia, acetyl CoA, acetyl choline, HMG CoA reductase, cytochrome C, blood ATP, ATP synthase, ERV RNA (endogenous retroviral RNA), H_2O_2 (hydrogen peroxide), NOX (NADPH oxidase), TNF alpha and heme oxygenase.[6-9] Informed consent of the subjects and the approval of the ethics committee were obtained for the study. The statistical analysis was done by ANOVA.

Results

Plasma of control subjects showed increased levels of the above mentioned parameters with after incubation for 1 hour and addition of cholesterol substrate resulted in still further significant increase in these parameters. The plasma of patients showed similar results but the extent of increase was more. The addition of antibiotics to the control plasma caused a decrease in all the

parameters while addition of rutile increased their levels. The addition of antibiotics to the patient's plasma caused a decrease in all the parameters while addition of rutile increased their levels but the extent of change was more in patient's sera as compared to controls. The results are expressed in section 1: tables 1-6 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time. There was upregulated archaeal porphyrin synthesis in the patient population which was archaeal in origin as indicated by actinide catalysis of the reactions. The cholesterol oxidase pathway generated pyruvate which entered the GABA shunt pathway. This resulted in synthesis of succinate and glycine which are substrates for ALA synthase.

The study showed the patient's blood and right hemispheric dominance had increased heme oxygenase activity and porphyrins. The hexokinase activity was high. The pyruvate, glutamate and ammonia levels were elevated indicating blockade of PDH activity, and operation of the GABA shunt pathway. The acetyl CoA levels were low and acetyl choline was decreased. The cyto C levels were increased in the serum indicating mitochondrial dysfunction suggested by low blood ATP levels. This was indicative of the Warburg's phenotype. There were increased NOX and TNF alpha levels indicating immune activation. The HMG CoA reductase activity was high indicating cholesterol synthesis. The bile acid levels were low indicating depletion of cytochrome P450. The normal population with right hemispheric dominance had values resembling the patient population with increased porphyrin synthesis. The normal population with left hemispheric dominance had low values with decreased porphyrin synthesis.

Section 1: Experimental Study

Table 1. *Effect of rutile and antibiotics on cytochrome F420 and PAH.*

Group	CYT F420 % (Increase with Rutile)		CYT F420 % (Decrease with Doxy+Cipro)		PAH % change (Increase with Rutile)		PAH % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.48	0.15	18.24	0.66	4.45	0.14	18.25	0.72
Schizo	23.24	2.01	58.72	7.08	23.01	1.69	59.49	4.30
Seizure	23.46	1.87	59.27	8.86	22.67	2.29	57.69	5.29
AD	23.12	2.00	56.90	6.94	23.26	1.53	60.91	7.59
MS	22.12	1.81	61.33	9.82	22.83	1.78	59.84	7.62
NHL	22.79	2.13	55.90	7.29	22.84	1.42	66.07	3.78
DM	22.59	1.86	57.05	8.45	23.40	1.55	65.77	5.27
AIDS	22.29	1.66	59.02	7.50	23.23	1.97	65.89	5.05
CJD	22.06	1.61	57.81	6.04	23.46	1.91	61.56	4.61
Autism	21.68	1.90	57.93	9.64	22.61	1.42	64.48	6.90
EMF	22.70	1.87	60.46	8.06	23.73	1.38	65.20	6.20
F value	306.749		130.054		391.318		257.996	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 2. *Effect of rutile and antibiotics on free RNA and DNA.*

Group	DNA % change (Increase with Rutile)		DNA % change (Decrease with Doxy+Cipro)		RNA % change (Increase with Rutile)		RNA % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.37	0.15	18.39	0.38	4.37	0.13	18.38	0.48
Schizo	23.28	1.70	61.41	3.36	23.59	1.83	65.69	3.94
Seizure	23.40	1.51	63.68	4.66	23.08	1.87	65.09	3.48
AD	23.52	1.65	64.15	4.60	23.29	1.92	65.39	3.95
MS	22.62	1.38	63.82	5.53	23.29	1.98	67.46	3.96
NHL	22.42	1.99	61.14	3.47	23.78	1.20	66.90	4.10
DM	23.01	1.67	65.35	3.56	23.33	1.86	66.46	3.65
AIDS	22.56	2.46	62.70	4.53	23.32	1.74	65.67	4.16
CJD	23.30	1.42	65.07	4.95	23.11	1.52	66.68	3.97
Autism	22.12	2.44	63.69	5.14	23.33	1.35	66.83	3.27
EMF	22.29	2.05	58.70	7.34	22.29	2.05	67.03	5.97
F value	337.577		356.621		427.828		654.453	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 3. *Effect of rutile and antibiotics on digoxin and delta aminolevulinic acid.*

Group	Digoxin (ng/ml) (Increase with Rutile)		Digoxin (ng/ml) (Decrease with Doxy+Cipro)		ALA % (Increase with Rutile)		ALA % (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	0.11	0.00	0.054	0.003	4.40	0.10	18.48	0.39
Schizo	0.55	0.06	0.219	0.043	22.52	1.90	66.39	4.20
Seizure	0.51	0.05	0.199	0.027	22.83	1.90	67.23	3.45
AD	0.55	0.03	0.192	0.040	23.67	1.68	66.50	3.58
MS	0.52	0.03	0.214	0.032	22.38	1.79	67.10	3.82
NHL	0.54	0.04	0.210	0.042	23.34	1.75	66.80	3.43
DM	0.47	0.04	0.202	0.025	22.87	1.84	66.31	3.68
AIDS	0.56	0.05	0.220	0.052	23.45	1.79	66.32	3.63
CJD	0.53	0.06	0.212	0.045	23.17	1.88	68.53	2.65
Autism	0.53	0.08	0.205	0.041	23.20	1.57	66.65	4.26
EMF	0.51	0.05	0.213	0.033	22.29	2.05	61.91	7.56
F value	135.116		71.706		372.716		556.411	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 4. *Effect of rutile and antibiotics on succinate and glycine.*

Group	Succinate % (Increase with Rutile)		Succinate % (Decrease with Doxy+Cipro)		Glycine % change (Increase with Rutile)		Glycine % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.41	0.15	18.63	0.12	4.34	0.15	18.24	0.37
Schizo	22.76	2.20	67.63	3.52	22.79	2.20	64.26	6.02
Seizure	22.28	1.52	64.05	2.79	22.82	1.56	64.61	4.95
AD	23.81	1.90	66.95	3.67	23.12	1.71	65.12	5.58
MS	24.10	1.61	65.78	4.43	22.73	2.46	65.87	4.35
NHL	23.43	1.57	66.30	3.57	22.98	1.50	65.13	4.87
DM	23.70	1.75	68.06	3.52	23.81	1.49	64.89	6.01
AIDS	23.66	1.67	65.97	3.36	23.09	1.81	65.86	4.27
CJD	22.92	2.14	67.54	3.65	21.93	2.29	63.70	5.63
Autism	21.88	1.19	66.28	3.60	23.02	1.65	67.61	2.77
EMF	22.29	1.33	65.38	3.62	22.13	2.14	66.26	3.93
F value	403.394		680.284		348.867		364.999	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 5. *Effect of rutile and antibiotics on pyruvate and glutamate.*

Group	Pyruvate % change (Increase with Rutile)		Pyruvate % change (Decrease with Doxy+Cipro)		Glutamate (Increase with Rutile)		Glutamate (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.34	0.21	18.43	0.82	4.21	0.16	18.56	0.76
Schizo	20.99	1.46	61.23	9.73	23.01	2.61	65.87	5.27
Seizure	20.94	1.54	62.76	8.52	23.33	1.79	62.50	5.56
AD	22.63	0.88	56.40	8.59	22.96	2.12	65.11	5.91
MS	21.59	1.23	60.28	9.22	22.81	1.91	63.47	5.81
NHL	21.19	1.61	58.57	7.47	22.53	2.41	64.29	5.44
DM	20.67	1.38	58.75	8.12	23.23	1.88	65.11	5.14
AIDS	21.21	2.36	58.73	8.10	21.11	2.25	64.20	5.38
CJD	21.07	1.79	63.90	7.13	22.47	2.17	65.97	4.62
Autism	21.91	1.71	58.45	6.66	22.88	1.87	65.45	5.08
EMF	22.29	2.05	62.37	5.05	21.66	1.94	67.03	5.97
F value	321.255		115.242		292.065		317.966	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 6. *Effect of rutile and antibiotics on hydrogen peroxide and ammonia.*

Group	H_2O_2 % (Increase with Rutile)		H_2O_2 % (Decrease with Doxy+Cipro)		Ammonia % (Increase with Rutile)		Ammonia % (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.43	0.19	18.13	0.63	4.40	0.10	18.48	0.39
Schizo	22.50	1.66	60.21	7.42	22.52	1.90	66.39	4.20
Seizure	23.81	1.19	61.08	7.38	22.83	1.90	67.23	3.45
AD	22.65	2.48	60.19	6.98	23.67	1.68	66.50	3.58
MS	21.14	1.20	60.53	4.70	22.38	1.79	67.10	3.82
NHL	23.35	1.76	59.17	3.33	23.34	1.75	66.80	3.43
DM	23.27	1.53	58.91	6.09	22.87	1.84	66.31	3.68
AIDS	23.32	1.71	63.15	7.62	23.45	1.79	66.32	3.63
CJD	22.86	1.91	63.66	6.88	23.17	1.88	68.53	2.65
Autism	23.52	1.49	63.24	7.36	23.20	1.57	66.65	4.26
EMF	23.29	1.67	60.52	5.38	22.29	2.05	61.91	7.56
F value	380.721		171.228		372.716		556.411	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Section 2: Patient Study

Table 1

Group	RBC Digoxin (ng/ml RBC Susp)		Cytochrome F420		HERV RNA (ug/ml)		H_2O_2 (umol/ml RBC)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
NO/BHCD	0.58	0.07	1.00	0.00	17.75	0.72	177.43	6.71
RHCD	1.41	0.23	4.00	0.00	55.17	5.85	278.29	7.74
LHCD	0.18	0.05	0.00	0.00	8.70	0.90	111.63	5.40
Schizo	1.38	0.26	4.00	0.00	51.17	3.65	274.88	8.73
Seizure	1.23	0.26	4.00	0.00	50.04	3.91	278.90	11.20
HD	1.34	0.31	4.00	0.00	51.16	7.78	295.37	3.78
AD	1.10	0.08	4.00	0.00	51.56	3.69	277.47	10.90
MS	1.21	0.21	4.00	0.00	47.90	6.99	280.89	11.25
SLE	1.50	0.33	4.00	0.00	48.20	5.53	278.59	11.51
NHL	1.26	0.23	4.00	0.00	51.08	5.24	283.39	10.67
Glio	1.27	0.24	4.00	0.00	51.57	2.66	278.19	12.80
DM	1.35	0.26	4.00	0.00	51.98	5.05	280.89	10.58
CAD	1.22	0.16	4.00	0.00	50.00	5.91	280.89	13.79
CVA	1.33	0.27	4.00	0.00	51.06	4.83	287.33	9.47
AIDS	1.31	0.24	4.00	0.00	50.15	6.96	278.58	12.72
CJD	1.48	0.27	4.00	0.00	49.85	6.40	286.16	10.90
Autism	1.19	0.24	4.00	0.00	52.87	7.04	274.52	9.29
DS	1.34	0.25	4.00	0.00	47.28	3.55	283.04	9.17
Cerebral Palsy	1.44	0.19	4.00	0.00	53.49	4.15	273.70	12.37
CRF	1.26	0.26	4.00	0.00	49.39	5.51	285.51	8.79
Cirr/Hep Fail	1.50	0.20	4.00	0.00	46.82	4.73	275.97	10.66
Muc Angio	1.40	0.32	4.00	0.00	46.37	4.87	290.37	9.10
EMF	1.51	0.29	4.00	0.00	47.47	4.34	287.49	9.81
CCP	1.35	0.22	4.00	0.00	48.54	5.97	277.50	7.51
Exposure to EMF	1.41	0.30	4.00	0.00	51.01	4.77	276.49	10.92
F value	60.288		0.001		194.418		713.569	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 2

Group	NOX (OD diff/hr/mgpro)		TNF ALP (pg/ml)		ALA (umol24)		PBG (umol24)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	0.012	0.001	17.94	0.59	15.44	0.50	20.82	1.19
RHCD	0.036	0.008	78.63	5.08	63.50	6.95	42.20	8.50
LHCD	0.007	0.001	9.29	0.81	3.86	0.26	12.11	1.34
Schizo	0.036	0.009	78.23	7.13	66.16	6.51	42.50	3.23
Seizure	0.038	0.007	79.28	4.55	68.28	6.02	46.54	4.55
HD	0.035	0.011	82.13	3.97	67.30	5.98	47.25	4.19
AD	0.036	0.007	79.65	5.57	67.32	5.40	49.83	3.45
MS	0.034	0.009	80.18	5.67	64.00	7.33	46.85	3.49
SLE	0.038	0.008	81.03	6.22	65.01	5.42	48.55	3.81
NHL	0.041	0.006	77.98	5.68	63.21	6.55	47.17	4.86
Glio	0.038	0.007	79.18	5.88	67.67	5.69	46.84	4.43
DM	0.041	0.005	78.36	6.68	64.72	6.81	48.15	3.36
CAD	0.038	0.009	78.15	3.72	66.66	7.77	47.00	3.81
CVA	0.037	0.007	77.59	5.24	69.02	4.86	46.33	4.01
AIDS	0.039	0.010	79.17	5.88	67.78	4.41	48.03	3.64
CJD	0.039	0.006	80.41	5.70	66.99	3.71	47.94	5.33
Autism	0.036	0.006	76.71	5.25	68.16	4.92	42.04	2.38
DS	0.035	0.009	80.30	6.65	64.99	6.72	45.69	4.18
Cerebral Palsy	0.038	0.008	80.02	6.82	65.56	6.28	44.58	4.52
CRF	0.039	0.008	81.36	5.37	67.61	5.55	46.81	4.62
Cirr/Hep Fail	0.037	0.010	77.61	4.42	66.28	6.55	48.23	2.36
Muc Angio	0.039	0.010	79.38	5.14	67.86	5.65	44.08	2.81
EMF	0.035	0.008	80.04	4.69	64.76	5.23	44.82	3.46
CCP	0.040	0.006	80.34	4.73	66.68	4.14	48.70	3.35
Exposure to EMF	0.038	0.007	76.41	5.96	68.41	5.53	47.27	3.42
F value	44.896		427.654		295.467		183.296	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 3

Group	Uroporphyrin (nmol24)		Coproporphyrin (nmol/24)		Protoporphyrin (Ab unit)		Heme (uM)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	50.18	3.54	137.94	4.75	10.35	0.38	30.27	0.81
RHCD	250.28	23.43	389.01	54.11	42.46	6.36	12.47	2.82
LHCD	9.51	1.19	64.33	13.09	2.64	0.42	50.55	1.07
Schizo	267.81	64.05	401.49	50.73	44.30	2.66	12.82	2.40
Seizure	290.44	57.65	436.71	52.95	49.59	1.70	13.03	0.70
HD	286.84	24.18	432.22	50.11	49.36	4.18	11.81	0.80
AD	259.61	33.18	433.17	45.61	49.68	3.30	12.09	1.12
MS	277.36	15.48	440.35	25.34	50.81	3.21	11.87	1.84
SLE	294.51	58.62	447.39	39.84	52.94	3.67	12.95	1.53
NHL	310.25	40.44	495.98	39.11	54.80	4.04	11.76	1.37
Glio	304.19	14.16	479.35	58.86	53.73	5.34	13.68	1.67
DM	285.46	29.46	422.27	33.86	49.80	4.01	12.83	2.07
CAD	314.01	17.82	426.14	24.28	49.51	2.27	11.39	1.10
CVA	320.85	24.73	402.16	33.80	46.74	4.28	11.26	0.95
AIDS	306.61	22.47	429.72	24.97	49.32	5.13	11.60	1.23
CJD	317.92	29.63	429.24	18.29	50.02	4.58	11.76	1.32
Autism	318.84	82.90	423.29	47.57	47.50	2.87	12.37	2.09
DS	258.33	37.85	421.52	36.57	50.97	7.07	11.81	1.14
Cerbral Palsy	280.16	26.14	431.39	28.88	49.23	3.91	11.61	1.36
CRF	301.78	48.22	427.57	33.55	49.66	4.41	12.03	1.40
Cirr/Hep Fail	276.51	16.66	436.44	25.65	50.56	1.63	11.92	1.33
Muc Angio	303.86	13.91	441.58	25.51	47.86	3.34	12.13	1.10
EMF	300.90	31.96	443.22	38.14	51.37	4.86	12.61	2.00
CCP	287.09	15.63	442.85	49.61	50.36	3.49	12.01	1.53
Exposure to EMF	288.21	26.17	444.94	38.89	50.59	1.71	12.36	1.26
F value	160.533		279.759		424.198		1472.05	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 4

Group	Bilirubin (mg/dl)		Biliverdin (Ab unit)		ATP Synthase (umol/gHb)		SE ATP (umol/dl)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	0.55	0.02	0.030	0.001	0.36	0.13	0.42	0.11
RHCD	1.70	0.20	0.067	0.011	2.73	0.94	2.24	0.44
LHCD	0.21	0.00	0.017	0.001	0.09	0.01	0.02	0.01
Schizo	1.74	0.08	0.073	0.013	2.66	0.58	1.26	0.19
Seizure	1.84	0.07	0.070	0.015	3.09	0.65	1.66	0.56
HD	1.83	0.09	0.071	0.014	3.34	0.84	1.27	0.26
AD	1.77	0.13	0.073	0.016	3.34	0.75	2.06	0.19
MS	1.81	0.10	0.079	0.007	3.05	0.52	1.63	0.26
SLE	1.82	0.08	0.061	0.006	2.85	0.34	1.59	0.22
NHL	1.84	0.08	0.077	0.011	3.01	0.55	1.73	0.26
Glio	1.76	0.11	0.073	0.012	2.70	0.62	1.48	0.32
DM	1.77	0.19	0.067	0.014	3.19	0.89	1.97	0.11
CAD	1.75	0.12	0.080	0.007	2.99	0.65	1.57	0.37
CVA	1.82	0.10	0.079	0.009	2.98	0.78	1.49	0.27
AIDS	1.79	0.08	0.072	0.013	3.29	0.63	1.59	0.38
CJD	1.82	0.09	0.066	0.009	3.21	0.95	1.69	0.43
Autism	1.83	0.16	0.072	0.014	2.67	0.80	2.03	0.12
DS	1.85	0.07	0.071	0.015	3.15	0.73	1.17	0.11
Cerebral Palsy	1.85	0.09	0.069	0.012	3.14	0.46	1.56	0.39
CRF	1.76	0.22	0.070	0.012	3.14	0.57	1.53	0.33
Cirr/Hep Fail	1.81	0.10	0.076	0.009	3.01	0.47	1.32	0.26
Muc Angio	1.78	0.24	0.067	0.014	2.92	0.55	1.35	0.29
EMF	1.79	0.07	0.074	0.009	3.12	0.60	1.56	0.48
CCP	1.84	0.07	0.073	0.011	3.15	0.46	1.51	0.38
Exposure to EMF	1.75	0.22	0.073	0.013	3.39	1.03	1.37	0.27
F value	370.517		59.963		54.754		67.588	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 5

Group	Cyto C (ng/ml)		Lactate (mg/dl)		Pyruvate (umol/l)		RBC Hexokinase (ug glu phos / hr/mgpro)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	2.79	0.28	7.38	0.31	40.51	1.42	1.66	0.45
RHCD	12.39	1.23	25.99	8.10	100.51	12.32	5.46	2.83
LHCD	1.21	0.38	2.75	0.41	23.79	2.51	0.68	0.23
Schizo	11.58	0.90	22.07	1.06	96.54	9.96	7.69	3.40
Seizure	12.06	1.09	21.78	0.58	90.46	8.30	6.29	1.73
HD	12.65	1.06	24.28	1.69	95.44	12.04	9.30	3.98
AD	11.94	0.86	22.04	0.64	97.26	8.26	8.46	3.63
MS	11.81	0.67	23.32	1.10	102.48	13.20	8.56	4.75
SLE	11.73	0.56	23.06	1.49	100.51	9.79	8.02	3.01
NHL	11.91	0.49	22.83	1.24	95.81	12.18	7.41	4.22
Glio	13.00	0.42	22.20	0.85	96.58	8.75	7.82	3.51
DM	12.95	0.56	25.56	7.93	96.30	10.33	7.05	1.86
CAD	11.51	0.47	22.83	0.82	97.29	12.45	8.88	3.09
CVA	12.74	0.80	23.03	1.26	103.25	9.49	7.87	2.72
AIDS	12.29	0.89	24.87	4.14	95.55	7.20	9.84	2.43
CJD	12.19	1.22	23.02	1.61	96.50	5.93	8.81	4.26
Autism	12.48	0.79	21.95	0.65	92.71	8.43	6.95	2.02
DS-50	12.79	1.15	23.69	2.19	91.81	4.12	8.68	2.60
Cerebral Palsy	12.14	1.30	23.12	1.81	95.33	11.78	7.92	3.32
CRF	12.66	1.01	23.42	1.20	97.38	10.76	7.75	3.08
Cirr/Hep Fail	12.81	0.90	26.20	5.29	97.77	13.24	8.99	3.27
Muc Angio	12.84	0.74	23.64	1.43	96.19	12.15	10.12	1.75
EMF	12.72	0.92	25.35	5.52	103.32	13.04	9.44	3.40
CCP	12.23	0.94	23.66	1.64	94.36	8.06	8.53	2.64
Exposure to EMF	12.26	1.00	23.31	1.46	103.28	11.47	7.58	3.09
F value	445.772		162.945		154.701		18.187	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 6

Group	ACOA (mg/dl)		ACH (ug/ml)		Glutamate (mg/dl)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	8.75	0.38	75.11	2.96	0.65	0.03
RHCD	2.51	0.36	38.57	7.03	3.19	0.32
LHCD	16.49	0.89	91.98	2.89	0.16	0.02
Schizo	2.51	0.57	48.52	6.28	3.41	0.41
Seizure	2.15	0.22	33.27	5.99	3.67	0.38
HD	1.95	0.06	35.02	5.85	3.14	0.32
AD	2.19	0.15	42.84	8.26	3.53	0.39
MS	2.03	0.09	39.99	12.61	3.58	0.36
SLE	2.54	0.38	49.30	7.26	3.37	0.38
NHL	2.30	0.26	50.58	3.82	3.48	0.46
Glio	2.34	0.43	42.51	11.58	3.28	0.39
DM	2.17	0.40	41.31	10.69	3.53	0.44
CAD	2.37	0.44	49.19	6.86	3.61	0.28
CVA	2.25	0.44	37.45	7.93	3.31	0.43
AIDS	2.11	0.19	38.40	7.74	3.45	0.49
CJD	2.10	0.27	34.97	4.24	3.94	0.22
Autism	2.42	0.41	50.61	6.32	3.30	0.32
DS-50	2.01	0.08	39.34	8.15	3.30	0.48
Cerebral Palsy	2.06	0.35	40.79	9.34	3.24	0.34
CRF	2.24	0.32	37.52	4.37	3.26	0.43
Cirr/Hep Fail	2.13	0.17	46.20	4.95	3.25	0.40
Muc Angio	2.51	0.42	45.51	7.56	3.11	0.36
EMF	2.19	0.19	42.48	8.62	3.27	0.39
CCP	2.04	0.10	37.95	8.82	3.33	0.25
Exposure to EMF	2.14	0.19	37.75	7.31	3.47	0.37
F value	1871.04		116.901		200.702	
P value	< 0.001		< 0.001		< 0.001	

Table 7

Group	Se. Ammonia (ug/dl)		HMG Co A (HMG CoA/MEV)		Bile Acid (mg/ml)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	50.60	1.42	1.70	0.07	79.99	3.36
RHCD	93.43	4.85	1.16	0.10	25.68	7.04
LHCD	23.92	3.38	2.21	0.39	140.40	10.32
Schizo	94.72	3.28	1.11	0.08	22.45	5.57
Seizure	95.61	7.88	1.14	0.07	22.98	5.19
HD	94.60	8.52	1.08	0.13	28.93	4.93
AD	95.37	4.66	1.10	0.07	26.26	7.34
MS	93.42	3.69	1.13	0.08	24.12	6.43
SLE	101.18	17.06	1.14	0.07	19.62	1.97
NHL	91.62	3.24	1.12	0.10	23.45	5.01
Glio	93.20	4.46	1.10	0.09	23.43	6.03
DM	93.38	7.76	1.09	0.12	22.77	4.94
CAD	93.93	4.86	1.07	0.12	24.55	6.26
CVA	103.18	27.27	1.05	0.09	22.39	3.35
AIDS	92.47	3.97	1.08	0.11	23.28	5.81
CJD	93.13	5.79	1.09	0.12	21.26	4.81
Autism	94.01	5.00	1.12	0.06	23.16	5.78
DS-50	98.81	15.65	1.09	0.11	21.31	4.49
Cerebral Palsy	92.09	3.21	1.07	0.09	22.80	5.02
CRF	98.76	11.12	1.03	0.10	26.47	5.30
Cirr/Hep Fail	94.77	2.86	1.04	0.10	24.91	5.06
Muc Angio	92.40	4.34	1.12	0.08	24.37	4.38
EMF	95.37	5.76	1.08	0.08	25.17	3.80
CCP	93.42	5.34	1.01	0.09	23.87	4.00
Exposure to EMF	102.62	26.54	1.00	0.07	22.58	5.07
F value	61.645		159.963		635.306	
P value	< 0.001		< 0.001		< 0.001	

Abbreviations

NO/BHCD: Normal/Bi-hemispheric chemical dominance

RHCD: Right hemispheric chemical dominance

LHCD: Left hemispheric chemical dominance

Schizo: Schizophrenia

HD: Huntington's disease

AD: Alzheimer's disease

MS: Multiple sclerosis

SLE: Systemic lupus erythematosis

NHL: Non-hodgkin's lymphoma

Glio - Glioma

DM: Diabetes mellitus

CAD: Coronary artery disease

CVA: Cerebrovascular accident

AIDS: Acquired immunodeficiency syndrome

CJD: Creutzfeldt Jakob disease

DS: Down syndrome

CRF: Chronic renal failure

Cirr/Hep Fail - Cirrhosis/Hepatic failure

EMF: Endomyocardial fibrosis

CCP: Chronic calcific pancreatitis

Discussion

There was increase in cytochrome F420 indicating archaeal growth. The archaea can synthesize and use cholesterol as a carbon and energy source.[2, 10] The archaeal origin of the enzyme activities was indicated by antibiotic induced suppression. The study indicates the presence of actinide based archaea with an alternate actinide based enzymes or metalloenzymes in the system as indicated by rutile induced increase in enzyme activities.[11] The archaeal beta hydroxyl steroid dehydrogenase activity indicating digoxin synthesis.[12] The archaeal cholesterol oxidase activity was increased resulting in generation of pyruvate and hydrogen peroxide.[10] The pyruvate gets converted to glutamate and ammonia by the GABA shunt pathway. The pyruvate is converted to glutamate by serum glutamate pyruvate transaminase. The glutamate gets acted upon by glutamate dehydrogenase to generate alpha ketoglutarate and ammonia. Alanine is most commonly produced by the reductive amination of pyruvate via alanine transaminase. This reversible reaction involves the interconversion of alanine and pyruvate, coupled to the interconversion of alpha-ketoglutarate (2-oxoglutarate) and glutamate. Alanine can contribute to glycine. Glutamate is acted upon by Glutamic acid decarboxylase to generate GABA. GABA is converted to succinic semialdehyde by GABA transaminase. Succinic semialdehyde is converted to succinic acid by succinic semialdehyde dehydrogenase. Glycine combines with succinyl CoA to generate delta aminolevulinic acid catalysed by the enzyme ALA synthase. There was upregulated archaeal porphyrin synthesis in the patient population which was archaeal in origin as indicated by actinide catalysis of the reactions. The cholesterol oxidase pathway generated pyruvate which entered the GABA shunt pathway. This resulted in synthesis of succinate and glycine which are substrates for ALA synthase. The archaea can undergo magnetite and calcium carbonate mineralization and can exist as calcified nanoforms.[13]

The possibility of Warburg phenotype induced by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism was considered in this paper. The Warburg phenotype results in inhibition of pyruvate dehydrogenase and the TCA cycle. The pyruvate enters the GABA shunt pathway where it is converted to succinyl CoA. The glycolytic pathway is upregulated and the glycolytic metabolite phosphoglycerate is converted to serine and glycine. Glycine and succinyl CoA are the substrates for ALA synthesis. The archaea induces the enzyme heme oxygenase. Heme oxygenase converts heme to bilirubin and biliverdin. This depletes heme from the system and results in upregulation of ALA synthase activity resulting in porphyria. Heme inhibits HIF alpha. The heme depletion results in upregulation of HIF alpha activity and further strengthening of the Warburg phenotype. The porphyrin self oxidation results in redox stress which activates HIF alpha and generates the Warburg phenotype. The Warburg phenotype results in channeling acetyl CoA for cholesterol synthesis as the TCA cycle and mitochondrial oxidative phosphorylation are blocked. The archaea uses cholesterol as an energy substrate. Porphyrin and ALA inhibits sodium potassium ATPase. This increases cholesterol synthesis by acting upon intracellular SREBP. The cholesterol is metabolized to pyruvate and then the GABA shunt pathway for ultimate use in porphyrin synthesis. The porphyrins can self organize and self replicate into macromolecular arrays. The porphyrin arrays behave like an autonomous organism and can have intramolecular electron transport generating ATP. The porphyrin macroarrays can store information and can have quantal perception. The porphyrin macroarrays serves the purpose of archaeal energetics and sensory perception. The Warburg phenotype is associated with malignancy, autoimmune disease and metabolic syndrome x.

The role of archaeal porphyrins in regulation of cell functions and neuro-immuno-endocrine integration is discussed. Protoporphyrin binds to the peripheral benzodiazepine receptor regulating steroid and digoxin synthesis. Increased porphyrin metabolites can contribute to hyperdigoxinemia. Digoxin can modulate the neuro-immuno-endocrine system. Porphyrins can combine with membranes modulating membrane function. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. Porphyrins can complex with DNA and RNA modulating their function. Porphyrin interpolating with DNA can alter transcription and generate HERV expression. Heme deficiency can also result in disease states. Heme deficiency results in deficiency of heme enzymes. There is deficiency of cytochrome C oxidase and mitochondrial dysfunction. The glutathione peroxidase is dysfunctional and the glutathione system of free radical scavenging does not function. The cytochrome P450 enzymes involved in steroid and bile acid synthesis have reduced activity leading to steroid - cortisol and sex hormones as well as bile acid deficiency states. The heme deficiency results in dysfunction of nitric oxide synthase, heme oxygenase and cystathione beta synthase resulting in lack of gasotransmitters regulating the vascular system and NMDA receptor - NO, CO and H_2S. Heme has got cytoprotective, neuroprotective, anti-inflammatory and antiproliferative effects. Heme is also involved in the stress response. Heme deficiency leads to metabolic syndrome, immune disease, degenerations and cancer.[3-5]

The porphyrins can undergo photo-oxidation and auto-oxidation generating free radicals. The archaeal porphyrins can produce free radical injury. Free radicals produce NFKB activation, open the mitochondrial PT pore resulting in cell death, produce oncogene activation, activate NMDA receptor and GAD enzyme regulating neurotransmission and generates the Warburg phenotypes

activating glycolysis and inhibiting TCA cycle/oxphos. Porphyrins have been related to schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. The porphyrins can complex and intercalate with the cell membrane producing sodium potassium ATPase inhibition adding on to digoxin mediated inhibition. Porphyrins can complex with proteins and nucleic acid producing biophoton emission. Porphyrins complexing with proteins can modulate protein structure and function. Porphyrins complexing with DNA and RNA can modulate transcription and translation. The porphyrin especially protoporphyrins can bind to peripheral benzodiazepine receptors in the mitochondria and modulate its function, mitochondrial cholesterol transport and steroidogenesis. Peripheral benzodiazepine receptor modulation by protoporphyrins can regulate cell death, cell proliferation, immunity and neural functions. The porphyrin photo-oxidation generates free radicals which can modulate enzyme function. Redox stress modulated enzymes include pyruvate dehydrogenase, nitric oxide synthase, cystathione beta synthase and heme oxygenase. Free radicals can modulate mitochondrial PT pore function. Free radicals can modulate cell membrane function and inhibit sodium potassium ATPase activity. Thus the porphyrins are key regulatory molecules modulating all aspects of cell function.[3-5] There was an increase in free RNA indicating self replicating RNA viroids and free DNA indicating generation of viroid complementary DNA strands by archaeal reverse transcriptase activity. The actinides and porphyrins modulate RNA folding and catalyse its ribozymal action. Digoxin can cut and paste the viroidal strands by modulating RNA splicing generating RNA viroidal diversity. The viroids are evolutionarily escaped archaeal group I introns which have retrotransposition and self splicing qualities. Archaeal pyruvate producing histone deacetylase inhibition and porphyrins intercalating with DNA can produce endogenous retroviral (HERV) reverse transcriptase and integrase expression. This can integrate the

RNA viroidal complementary DNA into the noncoding region of eukaryotic noncoding DNA using HERV integrase as has been described for borna and ebola viruses. The archaea and viroids can also induce cellular porphyrin synthesis. Bacterial and viral infections can precipitate porphyria. Thus porphyrins can regulate genomic function. The increased expression of HERV RNA can result in acquired immunodeficiency syndrome, autoimmune disease, neuronal degenerations, schizophrenia and malignancy.[14, 15]

The archaea and viroids can regulate the nervous system including the NMDA/GABA thalamocorticothalamic pathway mediating conscious perception. Porphyrin photo-oxidation can generate free radicals which can modulate NMDA transmission. Free radicals can increase NMDA transmission. Free radicals can induce GAD and increase GABA synthesis. ALA blocks GABA transmission and upregulates NMDA. Protoporphyrins bind to GABA receptor and promote GABA transmission. Thus porphyrins can modulate the thalamo-cortico-thalamic pathway of conscious perception. The dipolar porphyrins, PAH and archaeal magnetite in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated Frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macroscopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrin molecules have a wave particle existence and can bridge the dividing line between quantal state and particulate state. Thus the porphyrins can mediate conscious and quantal perception. Porphyrins binding to proteins, nucleic acids and cell membranes can produce biophoton emission. Porphyrins by autooxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Cellular porphyrins photooxidation are involved in sensing

of earth magnetic fields and low level biomagnetic fields. Thus prophyrins can mediate extrasensory perception. The porphyrins can modulate hemispheric dominance. There is increased porphyrin synthesis and right hemispherical chemical dominance and decreased porphyrin synthesis in left hemispherical chemical dominance. The increase in archaeal porphyrins can contribute to the pathogenesis of schizophrenia and autism. Porphyria can lead to psychiatric disorders and seizures. Altered porphyrin metabolism has been described in autism. Porphyrin by modulating conscious and quantal perception is involved in the pathogenesis of schizophrenia and autism. Protoporphyrins block acetyl choline transmission producing a vagal neuropathy with sympathetic overactivity. Vagal neuropathy results in immune activation, vasospasam and vascular disease. A vagal neuropathy underlines neoplastic and autoimmune processes as well as metabolic syndrome x. Porphyrin induced increased NMDA transmission and free radical injury can contribute to neuronal degeneration. Free radicals can produce mitochondrial PT pore dysfunction. This can lead to cyto C leak and activation of the caspase cascade leading to apoptosis and cell death. Altered porphyrin metabolism has been described in Alzheimer's disease. The increased porphyrin photooxidation generated free radicals mediated NMDA transmission can also contribute to epileptogenesis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate brain function and cell death.[3, 4, 16]

The porphyrin photooxidation can generate free radicals which can activate NFKB. This can produce immune activation and cytokine mediated injury. The increase in archaeal porphyrins can lead to autoimmune disease like SLE and MS. A hereditary form of MS and SLE related to altered porphyrin metabolism has been described. The protoporphyrins binding to mitochondrial benzodiazepine receptors can modulate immune function. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing

crosslinking and altering protein conformation and function. Porphyrins can complex with DNA and RNA modulating their structure. Porphyrin complexed with proteins and nucleic acids are antigenic and can lead onto autoimmune disease.[3, 4] The porphyrin photo-oxidation mediated free radical injury can lead to insulin resistance and atherogenesis. Thus archaeal porphyrins can contribute to metabolic syndrome x. Glucose has got a negative effect upon ALA synthase activity. Therefore hyperglycemia may be reactive protective mechanism to increased archaeal porphyrin synthesis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can modulate mitochondrial steroidogenesis and metabolism. Altered porphyrin metabolism has been described in the metabolic syndrome x. Porphyrias can lead onto vascular thrombosis.[3, 4] The porphyrin photo-oxidation can generate free radicals inducing HIF alpha and producing oncogene activation. Heme deficiency can lead to activation of HIF alpha and oncogenesis. This can lead to oncogenesis. Hepatic porphyrias induced hepatocellular carcinoma. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate cell proliferation.[3, 4] The porphyrin can combine with prion proteins modulating their conformation. This leads to abnormal prion protein conformation and degradation. Archaeal porphyrins can contribute to prion disease. The porphyrins can intercalate with DNA producing HERV expression. The HERV particles generated can contribute to the retroviral state. The porphyrins in the blood can combine with bacteria and viruses and the photo-oxidation generated free radicals can kill them. The archaeal porphyrins can modulate bacterial and viral infections. The archaeal porphyrins are regulatory molecules keeping other prokaryotes and viruses on check.[3, 4] Thus the archaeal porphyrins can contribute to the pathogenesis of metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis. Archaeal porphyrin synthesis is crucial in the pathogenesis of these disorders. Porphyrins

may serve as regulatory molecules modulating immune, neural, endocrine, metabolic and genetic systems. The porphyrins photooxidation generated free radicals can produce immune activation, produce cell death, activate cell proliferation, produce insulin resistance and modulate conscious / quantal perception. The archaeal porphyrins functions as key regulatory molecules with mitochondrial benzodiazepine receptors playing an important role.[3, 4]

The metal actinides provide radiolytic energy, catalysis for oligomer formation and provide a coordinating ion for metalloenzymes all important in abiogenesis.[6] The metal actinide surfaces would by surface metabolism generate porphyrins from simple compounds like succinic acid and glycine. Porphyrins can exist as wave forms and particulate forms and can bridge the dividing line between the quantal world and particulate world. Porphyrin molecules can self organize into organisms with energy transduction, ATP synthesis and information storage with replicating capacity. A self replicating porphyrin microorganism may have played a role in the origin of life. Porphyrins can form templates on which macromolecules like polysaccharides, protein and nucleic acids can form. The macromolecules generated on actinidic porphyrins templates would have contributed to the actinidic nanoarchaea and the original organisms on earth. The data supports the persistence of an actinidic archaeal shadow biosphere which throws light on the actinide based origin of life and porphyrins as the premier prebiotic molecule.[17, 18]

Porphyrins play an important role in the genesis of the biological universe. The porphyrin macroarrays can form in the interstellar space on its own as porphyrins can exist both as particles and waves. Porphyrins form the bridging connection between the quantal world and the particulate world. The self generated porphyrins from the quantal foam can self organize to form macroarrays, can store information and self replicate. This can be called as an abiotic porphyrin organism. The porphyrin template would have generated

nucleic acids, proteins, polysaccharides and isoprenoids. This would have generated actinidic nanoarchaea in the interstellar space. The porphyrins have magnetic properties and the interstellar porphyrin organism can contribute to the interstellar grains and interstellar magnetic fields.[37] The cosmic dust grains of porphyrin macroarrays/nanoarchaeal organism occupy the intergalactic space and are thought to be formed of magnetotactic bacteria identified according to their spectral signatures. According to the Hoyle's hypothesis, the cosmic dust magnetotactic porphyrin macroarrays/nanoarchaeal organism plays a role in the formation of the intergalactic magnetic field. A magnetic field equal in strength to about one millionth part of the magnetic field of earth exists throughout much of our galaxy. The magnetic files can be used to trace the spiral arms of the galaxy following a pattern of field lines that connect young stars and dust in which new stars are formed at a rapid rate. Studies have shown that a fraction of the dust particles have elongated shape similar to bacilli and they are systematically lined up in our galaxy. Moreover the direction of alignment is such that the long axes of the dust tend to be at right angles to the direction of the galactic magnetic field at every point. Magnetotactic porphyrin macroarrays/nanoarchaeal organisms have the property to affect the degree of alignment that is observed. The fact that the magnetotactic porphyrin macroarrays/nanoarchaeal organisms appear to be connected to the magnetic field lines that thread through the spiral arms of the galaxy connecting one region of star formation to another support a role for them in star formation and in the mass distribution and rotation of stars. The nutrient supply for a population of interstellar porphyrin macroarrays/nanoarchaeal organisms comes from mass flows out of supernovas populating the galaxy. Giants arising in the evolution of such stars experience a phenomenon in which material containing nitrogen, carbon monoxide, hydrogen, helium, water and trace elements essential for life flows continuously outward into space. The interstellar

organisms need liquid water. Water exists only as vapour or solid in the interstellar space and only through star formation leading to associated planets and cometary bodies can there be access to liquid water. To control conditions leading to star formation is of paramount importance in cosmic biology. The rate of star formation is controlled by two factors: Too high a rate of star formation produces a destructive effect of UV radiation and destroys cosmic biology. Star formation as stated before produces water crucial for organism growth. Cosmic biology of magnetotactic organisms and star formation are thus closely interlinked. Systems like solar systems do not arise in random condensation of blobs of interstellar gas. Only by a rigorous control of rotation of various parts of the system would galaxies and solar system evolved. The key to maintaining control over rotation seems to lie in the intergalactic magnetic field as indeed the whole phenomena of star formation. The intergalactic magnetic fields owes its origin to the lining up of magnetotactic porphyrin macroarrays/nanoarchaeal organisms and the cosmic biology of interstellar organisms can prosper only by maintaining a firm grip on the interstellar magnetic field and hence on the rate of star formation and type of star system produced. This point to a cosmic intelligence or brain capable of computation, analysis and exploration of the universe at large - of magnetotactic porphyrin macroarrays/nanoarchaeal organism networks. The origin of life on earth according to the Hoyle's hypothesis would be by seeding of porphyrin macroarrays/nanoarchaeal organism from the outer intergalactic space. The porphyrin organism can also be generated on actinidic surfaces in earth. Comets carrying porphyrin organisms would have interacted with the earth. A thin skin of graphitized material around a single porphyrin macroarrays/nanoarchaeal organism or clumps of organism can shield the interior from destruction by UV light. The sudden surge and diversification of species of plants and animals and their equally sudden extinction has seen from fossil records point to sporadic

evolution produced by induction of fresh cometary genes with the arrival of each major new crop of comets.[38, 39] The porphyrin macroarrays organism can have a wave particle existence and bridge the world of bosons and fermions. The porphyrin macroarrays/nanoarchaeal organism can form biofilms and the porphyrin organism can form a molecular quantum computing cloud in the biofilm which forms an interstellar intelligence regulating the formation of star systems and galaxies. The porphyrin macroarrays / nanoarchaeal organism quantal computing cloud can bridge the wave particle world functioning as the anthropic observer sensing gravity which orchestrates the reduction of the quantal world of possibilities in to the macroscopic world. The actinide based porphyrin macroarrays/nanoarchaeal organism regulates the human system and biological universe.[19-21]

Porphyrins also have evolutionary significance since porphyria is related to Scythian races and contributes to the behavioural and intellectual characteristics of this group of population. Porphyrins can intercalate into DNA and produce HERV expression. HERV RNA can get converted to DNA by reverse transcriptase which can get integrated into DNA by integrase. This tends to increase the length of the non coding region of the DNA. The increase in non coding region of the DNA is involved in primate and human evolution. Thus, increased rates of porphyrin synthesis would correlate with increase in non-coding DNA length. The alteration in the length of the non coding region of the DNA contributes to the dynamic nature of the genome. Thus genetic and acquired porphyrias can lead to alteration in the non coding region of the genome. The alteration of the length of the non coding region of the DNA contributes to the racial and individual differences in populations. An increased length of non-coding region as well as increased porphyrin synthesis leads to increased cognitive and creative neuronal function. Porphyrins are involved in quantal perception and regulation of the thalamo-cortico-thalamic pathway of

conscious perception. Thus genetic and acquired porphyrias contribute to higher cognitive and creative capacity of certain races. Porphyrias are common among Eurasian Scythian races who have assumed leadership roles in communities and groups. Porphyrins have contributed to human and primate evolution.[3, 4]

The dipolar porphyrins, PAH and archaeal magnetite in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated Frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macroscopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrins by auto-oxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Cellular porphyrins photo-oxidation are involved in sensing of earth magnetic fields and low level biomagnetic fields. Porphyrins can thus contribute to quantal perception. Low level electromagnetic fields and light can induce porphyrin synthesis. Low level EMF can produce ferrochelatase inhibition as well as heme oxygenase induction contributing to heme depletion, ALA synthase induction and increased porphyrin synthesis. Light also induces ALA synthase and porphyrin synthesis. The increased porphyrin synthesized can contribute to increased quantal perception and can modulate conscious perception. The porphyrin induced biophotons and quantal fields can modulate the source from which low level EMF and photic fields were generated. Thus the porphyrin generated by extraneous low level EMF and photic fields can interact with the source of low level EMF and photic fields modulating it. Thus porphyrins can serve as a bridge between the human brain and the source of low level EMF and photic fields. This serves as a mode of communication between the human brain and EMF storage devices like internet. The porphyrins can also serve as the source

of communication with the environment. Environmental EMF and chemicals produce heme oxygenase induction and heme depletion increasing porphyrin synthesis, quantal perception and two-way communication. Thus induction of porphyrin synthesis can serve as a mechanism of communication between human brain and the environment by extrasensory perception.

The porphyrions are self replicating supramolecular organisms which forms the precursor template on which the viroids, prions and nanoarchaea originate. Stress induced template directed abiogenesis of porphyrions, prions, viroids and archaea is a continuous process and can contribute to changes in brain structure and behavior as well as disease process.

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