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Glycolysis, Global Warming and Human Stem Cell Transformation

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 groups 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. The Warburg phenotype results in conversion of the somatic cell to a stem cell phenotype.

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.

The Warburg phenotype and increased glycolysis and stem cell transformation results in civilisational disease. The increase in glycolysis and mitochondrial PT pore hexokinase results in cell growth, proliferation and cancer. The increase in GAPDH and its polyribosylation by PARP enzymes results in nuclear cell death and neurodegeneration. The increase in the glycolytic intermediate phosphoglycerate results in increased serine and glycine contributing to NMDA excitotoxicity the basis of neurodegeneration and neuropsychiatric disorders. The suppression of mitochondrial oxidative phosphorylation as well as changes in pancreatic glutamatergic transmission can contribute to metabolic syndrome x.

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.