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Neanderthal Hybrids: Climate Change
Mediated Actinidic Archaeal
Endosymbiosis Generates Neanderthal
Hybrids and Mind-Body Phenotypic
Change - The Origins of Schizophrenia,
Autism and Epilepsy

Introduction

Actinidic archaea has been related to global warming and human diseases especially schizophrenia, autism and epilepsy. 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 schizophrenia, autism and epilepsy especially the Warburg phenotype and hyperdigoxinemia. Digoxin produced by archaeal cholesterol catabolism produces Neanderthalisation. Prefrontal cortical atrophy and cerebellar hyperplasia has been related to schizophrenia, autism and epilepsy 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.¹⁻¹⁶ The data is described in this paper.

Global warming can lead to osmotic stress consequent to dehydration. The increase in actinidic archaeal growth leads to cholesterol catabolism and digoxin synthesis. Digoxin produces membrane sodium potassium ATPase inhibition and increase in intracellular calcium producing mitochondrial dysfunction. This results in oxidative stress. The oxidative stress and osmotic stress can induce the enzyme aldose reductase which converts glucose to fructose. Fructose has got a low K_m value for ketokinase as compared to glucose. Therefore fructose gets phosphorylated more to fructose phosphate and the cell is depleted of ATP. The cell depletion of ATP leads to oxidative stress and chronic inflammation consequent to induction of NF κ B. Oxidative stress can open the mitochondrial PT pore producing release of cyto C and activation of the caspase cascade of cell death. The fructose phosphate can enter the pentose phosphate pathway synthesizing ribose and nucleic acid. The depletion of cellular ATP results in generation of AMP and ADP which are acted upon by deaminases causing

hyperuricemia. Uric acid can produce endothelial dysfunction and vascular disease. Uric acid can also produce mitochondrial dysfunction. The fructose phosphate can enter the glucosamine pathway synthesizing GAG and producing mucopolysaccharide accumulation. Fructose can fructosylate proteins making them antigenic and producing an autoimmune response. This can lead to global warming related psychiatric disease.

Materials and Methods

Fifteen cases, each of schizophrenia, autism and epilepsy 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	Neanderthal phenotype	Low index finger-ring finger ratio
Schizophrenia	69%	75%	65%
Autism	80%	75%	72%
Epilepsy	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%
Epilepsy	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.

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 catabolising 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 catabolises 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 contribute to the generation of autistic and schizophrenic features in Neanderthals. This also contributes to epileptogenesis.

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 archaical 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. This leads onto the ontogenesis of schizophrenia, autism and epilepsy.

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, autism and epilepsy.

The cerebellar hyperplasia results in sympathetic hyperactivity and parasympathetic neuropathy. This contributes to oncogene activation. Vagal neuropathy results in immune activation and autoimmunity important in schizophrenia, autism and epilepsy. Vagal neuropathy and sympathetic overactivity can contribute to glycogenolysis and lipolysis resulting in insulin resistance. Insulin resistance leads to schizophrenia, autism and epilepsy. 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 schizophrenia, autism and epilepsy.

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 NF κ B activation resulting in schizophrenia, autism and epilepsy. The increase in intracellular calcium opens the mitochondrial PT pore resulting in cell death of schizophrenia, autism and epilepsy. 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, autism and epilepsy. Digoxin induced

magnesium depletion can inhibit reverse transcriptase activity and HERV generation modulating the dynamicity of the genome. HERV expression has been related to schizophrenia, autism and epilepsy. 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. The dysfunction of this integrative phenomenon can lead to schizophrenia, autism and epilepsy. 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 catabolising enzymes generating more of testosterone than estrogens. This contributes to estrogen deficiency and testosterone overactivity. The Neanderthal population is hypermales with concomitant 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. The homo sapiens eat a high fibre diet with low cholesterol and high lignan content contributing to estrogen dominance, left hemispheric dominance and cerebellar hypoplasia. 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. This gender phenomenon can lead onto the ontogenesis of schizophrenia, autism and epilepsy.

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 autoimmunity in schizophrenia, autism and epilepsy. Bile acids bind to FXR, LXR and PXR modulating lipid and carbohydrate metabolism. This leads to insulin resistance in the presence of bile acid deficiency. Bile acid uncouples oxidative phosphorylation and its deficiency leads to insulin resistance. Insulin resistance is important in schizophrenia, autism and epilepsy. Schizophrenia is called as an insulin resistance state of the brain. 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 autoimmunity in schizophrenia, autism and epilepsy. 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. All these lead to schizophrenia, autism and epilepsy.

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. This phenomenon leads to the ontogenesis of schizophrenia, autism and epilepsy.

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