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Neo-Neanderthalisation and Human Disease – The Origins of in Neurodegenerations – Alzheimer's Disease, Parkinson's Disease and Motor Neuron Disease

Introduction

Actinidic archaea has been related to global warming and human diseases especially neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease especially the Warburg phenotype and hyperdigoxinemia. Digoxin produced by archaeal cholesterol catabolism produces Neanderthalisation. Prefrontal cortical atrophy and cerebellar hyperplasia has been related to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease 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.

Materials and Methods

Fifteen cases, each of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease 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 over activity 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
Alzheimer's disease	89%	65%	75%
Parkinson's disease	70%	71%	80%
MND	80%	75%	75%
Internet users	65%	72%	69%

Table 2 *Neanderthal phenotype and brain dysfunction.*

Disease	Dysautonomia	Prefrontal cortex atrophy	Cerebellar hypertrophy
Alzheimer's disease	60%	72%	60%
Parkinson's disease	62%	71%	68%
MND	69%	74%	76%
Internet users	74%	84%	82%

Discussion

Neanderthal metabolonomics contribute to the pathogenesis of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 incidence of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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. This leads onto the pathogenesis of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

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 neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. There is an epidemic of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease in the present day community. The

porphyrin mediated extrasensory perception can contributes to low level EMF related neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

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 increase incidence of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 low level EMF related neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 leads to increased incidence of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

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 neurodegenerations-alzheimer's disease, parkinson's

disease and motor neuron disease. There is dominance of cerebellar function. The Neanderthal hybrids have cerebellar dominance and less of conscious behaviour. The predominant homo sapiens had prefrontal cortex dominance over the cerebellum resulting in more of conscious behaviour. Neanderthalisation leads to increased incidence of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

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 and immune activation producing autoimmunity in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 insulin resistance. The glycolytic intermediate 3-phosphoglycerate is converted to glycine resulting in NMDA excitotoxicity contributing to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Cerebellar dominance is seen in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

The cerebellar hyperplasia results in sympathetic hyperactivity and parasympathetic neuropathy. Vagal neuropathy results in immune activation and autoimmunity in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Vagal neuropathy and sympathetic over activity can contribute to glycogenolysis and lipolysis resulting in insulin resistance. Insulin resistance leads to increased incidence of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Cerebellar dominance and cerebellar cognitive affective dysfunction can contribute to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron

disease. The increased porphyrin synthesis resulting from succinyl CoA generated by GABA shunt and glycine generated by glycolysis contributes to increased extrasensory perception of low level EMF important in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Sympathetic over activity 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 autoimmunity important in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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. This can lead to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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. This defective neuro-immuno-endocrine integration can lead to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

The actinidic archaea are cholesterol catabolising and leads to low levels of testosterone and estrogen. 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 over activity. 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 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 change in endocrine status leads to increased incidence of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

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 important in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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. Vitamin D deficiency leads on to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

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 leads on to an epidemic of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

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