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The Homo Neanderthalis and the Dravidians: A Common Origin and Relation to Harappan Civilisation and Vedas

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Abstract

INTRODUCTION: The postulated Lemurian part of the Indian sub-continent in South India is inhabited by the dominant Nair community. The dominant Nair community also has a high incidence of autism. Neanderthal anthropometric features have been described in autism. Neanderthal metabolonomics have also been described in autism. It is possible that homo neanderthalis would have originated in the super continent which occupied the southern ocean. The island of Sumatra is home to another human species homo floresiensis which lived along with homo neanderthalis. This suggests an oceanic origin of homo neanderthalis in the supercontinent in the southern ocean. Recurrent Tsunamis would have forced the migration of homo neanderthalis to the Eurasian land mass especially to Harappa, Sumeria, Etruscia, Egypt and Basque country. There is a high incidence of Neanderthal genes in the Basque population. The language spoken in Harappa, Sumeria, Etruscia, Egypt and Basque country had a Dravidian sub-stratum. The population in these areas are matrilineal and female dominant.

MATERIALS AND METHODS: Neanderthal anthropometric features were evaluated in the Nair community and in autism. The parameters checked include dolichocephalic skull, prominent supraorbital ridge and mid face large flat nose and ring finger index finger ratios.

RESULTS: The Nair community had a high prevalence of Neanderthal anthropometric features. Neanderthal anthropometric features were also dominant in autism.

CONCLUSION: This suggests an out of oceania hypothesis for the origin of homo neanderthalis.

Key words: Archaea; Lemuria; Rigveda; Dravidians; Neanderthals

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INTRODUCTION

The postulated Lemurian part of the Indian sub-continent in South India is inhabited by the dominant Nair community. The dominant Nair community also has a high incidence of autism. Neanderthal anthropometric features have been described in autism. Neanderthal metabolonomics have also been described in autism. It is possible that homo neanderthalis would have originated in the super continent which occupied the southern ocean. The island of Sumatra is home to another human species homo floresiensis which lived along with homo neanderthalis. This suggests an oceanic origin of homo neanderthalis in the supercontinent in the southern ocean. Recurrent Tsunamis would have forced the migration of homo neanderthalis to the Eurasian land mass especially to Harappa, Sumeria, Etruscia, Egypt and Basque country. There is a high incidence of Neanderthal genes in the Basque population. The language spoken in Harappa, Sumeria, Etruscia, Egypt and Basque country had a Dravidian sub-stratum. The population in these areas are matrilineal and female dominant. This suggests an out of oceanic hypothesis for the origin of homo neanderthalis.

MATERIALS AND METHODS

Neanderthal anthropometric features were evaluated in the Nair community and in autism. The parameters checked include dolichocephalic skull, prominent supraorbital ridge and mid face large flat nose and ring finger index finger ratios.

RESULT ANALYSIS

The Nair community had a high prevalence of Neanderthal anthropometric features. Neanderthal anthropometric features were also dominant in autism.

Table 1
Incidence of Autism in Nair, Autistic and Non Nair Population

Groups	Autism	Percentage
Nair	68 cases	68%
Non-nair	32 cases	32%
Total	100	

Table 2
Anthropometric Features in Nair, Autistic and Non Nair Population

Groups	Neanderthal anthropometric	Total cases	Percentage
Nair	72 cases	100	72%
Non-nair	21 cases	100	21%
Autism	81 cases	100	81%

DISCUSSION

Neanderthal anthropometric features were seen in autism and Nair community dominating the part of the Indian subcontinent derived from Lemuria. This suggests a Lemurian supercontinent origin of the homo neanderthalis. The homo neanderthalis shared the Lemurian super continent with another human species called homo floresiensis. Homo floresiensis has been detected in the island of Sumatra in Indonesia. The Nair community dominates the Kerala coast of South India. The Nair community is matrilineal and Dravidian. There are other civilisations speaking the Dravidian language important in human evolution like Harappa, Sumeria, Etruscia, Egypt and Basque country. These civilisations may have a Neanderthal substratum. They would have migrated to the Eurasian land mass from the Lemurian supercontinent when it was destroyed by Tsunamis in the Indian ocean. The Tsunamis would have evolved due to archaeal overgrowth in the southern ocean during the ice age. The archaea are extremophiles. The archaeal overgrowth in the Indian ocean bed in the ice age would have released methane. This would have triggered movement of the earth crust, earthquakes and Tsunamis. The same endosymbioticarchaeal growth would have led to evolution of homo neanderthalis. The endosymbioticarchaeal metabolism in primates would have generated the species homo neanderthalis. The homo neanderthalis contributed to the civilisations of Harappa, Sumeria, Etruscia, Egypt, basque and celts. They were all matrilineal with gender equality. They had a symbolic language predominantly non-vocal. Music, dance and painting as a form of communication were prevalent in these societies. This is exemplified by the Harappan language dominated by Harappan seals and the Egyptian hieroglyphics. The concept of spirituality is evolved in these societies including the worship of the mother goddess.

The increased prevalence of autism in the Dravidian Nair community has been documented. Neanderthal anthropometric features are documented in autistic children and Nairs. Nairs and Dravidians have evolved from the Indian ocean supercontinent called Lemuria. Lemuria was flooded and broken up in a Indian ocean tsunami according to Sangam literature. The archaeal growth due to climate change in the ocean bed would have led to tsunamis and earth quake. The data supports the persistence of an actinide and cholesterol based shadow biosphere which throws light on the actinide based origin of life and cholesterol as the premier prebiotic molecule. The presence of placer deposits and mineral sands containing monazite, illmenite, rutile and thorium in the Lemurian supercontinent would have made it the ideal place for the primitive cell, nanoarchaea, eukaryote, multicellular eukaryote, primates and humans to evolve. Anthropological studies have provided evidence for the evolution of primates and homosapiens in the rift valley of Kenya part of the prehistoric Lemurian continent. The actinidic nanoarcheal growth would have led to methane burps in the ocean bed contributing to earthquakes and Tsunamis producing extinction of the Lemurian supercontinent. It also supports the abiogenesis on radioactive actinidic beach sands through the process of surface metabolism. This gives support to the role of actinidicarchaea as the third element that controls life and its role in the evolution of the multicellular eukaryote, primates and humans. Civilization and humans would have evolved in the placer deposits and actinidic sand rich pre-historic Lemurian supercontinent in the Indian and Southern ocean.

The Dravidian population that migrated out settled in the Eurasian land mass. This later evolved into Harappan civilization in India. The other coterminus civilization of Sumeria, Egypt, Celts and Basque were of similar Lemurian origin. All the civilizations have the Akkadian-Dravidian script. The homo neanderthalis would have evolved due to increased archaeal growth in the ice age. The same extremes of climate during global warming can result in growth of archaea and neanderthalisation. The archaea catabolises cholesterol. This leads to vitamin D deficiency and bile acid deficiency leading to metabolic syndrome x and rickets producing the Neanderthal phenotype. The cholesterol catabolism generates porphyrins which are dipolar and in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system and Bose-

Einstein condensate producing quantal perception. This would have led to extra sensory perception and the intuitive spiritual Neanderthal phenotype. The increased EMF perception by porphyrins would have produced prefrontal cortex atrophy and cerebellar dominance resulting in the Neanderthal brain. The digoxin induced magnesium deficiency can produce reverse transcriptase inhibition and blocking of HERV expression resulting in blunting of synaptic connectivity and prefrontal cortex atrophy. The homo sapiens have less of digoxin and a prominent prefrontal cortex and smaller cerebellum.

The Indus valley civilization has a Neanderthal origin. The Rigveda is of Harappan origin. The Varuna- the principal God of Vedas is just an oceanic God pointing to the Lemurian and Dravidian origin of Vedas. The other Gods of the Vedas were also of Dravidian or asuric origin. The Aryans were primitive homo sapien invaders from Eurasia who stole the civilization knowledge of the Dravidian asuric Harappans. Thus all primitive civilizations were of Neanderthal origin. The asuric Dravidians matriarchal and gender equal. They had a socialized society which operated as a primitive type of communism. The initial global language in Harappa. Sumeria, Egypt, Semitic and celtic countries were Akkadian Dravidian. The homo sapiens got their civilisational knowledge from the Neanderthals by hybridization. The Sanskrit is a modified Akkadian Dravidian language. The global warming leads to extremes of climate and growth of archaea leading to neanderthalisation of human species.

The increased prevalence of the Neanderthal anthropometric features in the Nair community and autism suggests a Lemurian origin for homo neanderthalis. This suggests an out of oceania hypothesis for homo neanderthalis with later migration to the Eurasian land mass consequent to destruction of the supercontinent by Tsunamis. The Tsunamis would have been precipitated by increased archaeal growth in the oceanic beds and movements in the earth crust produced by released methane. The homo neanderthalis also originated due to increased endosymbioticactinidicarchaeal growth.

The extinction of homo neanderthalis could be attributed to archaeal synthesised compounds including digoxin and porphyrins. The cholesterol catabolism generated 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 with 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. 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. Protoporphyrine binds to the peripheral benzodiazepine receptor in regulating steroid and digoxin synthesis. Increased porphyrin metabolites can contribute to hyperdigoxinemia. Digoxin can modulate

the neuroimmunoendocrine 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 cysthathione 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. The porphyrins can undergo photo-oxidation and autooxidation 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.

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 non coding 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.

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 thalamocorticothalamic 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 macrosopic 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 photo-oxidation 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 cytoC 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 photo-oxidation 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. The porphyrin photo-oxidation 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. The porphyrin photo-oxidation mediated free radical injury can result in 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 increase 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. 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. The porphyrin can combine with prion proteins modulating their conformation. This results in 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.

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 be used as regulatory molecules modulating immune, neural, endocrine, metabolic and genetic systems. The porphyrins photo-oxidation 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. Thus the archaeal synthesised compounds would have led to neanderthalisation of homo sapiens and their eventual extinction and death of these great civilisations.

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