Chapter 5

The Ardhanareswara - Neanderthal Metabolonomics and Androgynous Behavioural Patterns

Introduction

Neanderthal genes have been described in the homo sapien population. The Neanderthal brain has a prominent cerebellar cortex and small prefrontal cortex. This results in defective vocalization, symbolic speech, impulsive behaviour, obsessive traits, intuition and extrasensory perception. The Neanderthal brain structure results in female dominance and matriarchal social patterns. It was considered plausible that Neanderthal genomics and metabolonomics could also contribute to androgynous behaviour. Autistic patients tend to have Neanderthal metabolonomics and phenotype. It has been demonstrated that Neanderthal phenotype is due to symbiosis by actinidic archaea using cholesterol as an energy substrate. The actinidic archaea catabolizes cholesterol with ring A being oxidized to pyruvate which gets channelled to the GABA shunt pathway resulting in the formation of glycine and succinyl CoA. This results in porphyrin synthesis. The side chain oxidation results in generation of short chain fatty acids. Cholesterol is also converted to steroidogenic estrogens and testosterone. The increasing growth of actinidic archaea converts the body metabolites the cholesterol which is subsequently oxidized and depleted. Cholesterol is also converted by actinidic archaea to endogenous digoxin which helps to integrate the neuro-immuno-endocrine system. Digoxin produces sodium potassium ATPase inhibition and increased in intracellular calcium inducing nitric oxide synthase and heme oxygenase generating gasotransmitters nitric oxide and carbon monoxide important in smooth muscle contraction and autonomic function.[1-16] The study deals with assessment of Neanderthal metabolonomics in androgynous individuals.

Materials and Methods

Fifty healthy individuals with androgynous behaviour and free of any disease were chosen for the study. Each individual had a normal age and sex matched control. The estimations done in the blood samples collected include cytochrome F420 activity, cholesterol oxidase activity - cholesterol ring oxidase activity, cholesterol side chain oxidase activity, digoxin, lactate, pyruvate, ALA levels and hexokinase activity. Neanderthal anthropometry was studied in the androgynous population. The statistical analysis was done by ANOVA. Informed consent and permission of the Ethics Committee was obtained.

Results

The results of the study were as follows. The androgynous individuals had increased cytochrome F420 activity, cholesterol oxidase activity, ring oxidase activity and digoxin synthesis. The androgynous had decreased PDH activity as indicated by increased pyruvate and lactate levels. The androgynous group had increased GABA shunt pathway as indicated by increased pyruvate. The androgynous group had increased porphyrin synthesis as indicated by increased ALA levels. They had increased hexokinase activity indicating a Warburg phenotype in this group. The androgynous group had features of Neanderthal metabolism as indicated by pyruvate dehydrogenase suppression. The androgynous group has the Neanderthal anthropometric phenotype with slanting forehead, large face, stubby nose, prominent mandibles, low 2D:4D ratio, large coarse trunk, macrocephaly and longer second toe as compared to big toe.

Table 1. *Anthropometric features in androgynous population*.

Groups	Neanderthal anthropometric	Total	Percentage
Normal	0 cases	50	0
Androgyny	40 cases	50	40

Table 2. *Effect of cerium and antibiotics on cytochrome F420.*

Group	CYT F420 % (Increase with Cerium)		CYT F420 % (Decrease with Doxy+Cipro)	
	Mean	**± SD**	**Mean**	**± SD**
Normal	4.48	0.15	18.24	0.66
Androgyny	22.79	2.13	55.90	7.29
F value	306.749		130.054	
P value	< 0.001		< 0.001	

Table 3. *Effect of cerium and antibiotics on digoxin.*

Group	Digoxin (ng/ml) (Increase with Cerium)		Digoxin (ng/ml) (Decrease with Doxy+Cipro)	
	Mean	**± SD**	**Mean**	**± SD**
Normal	0.11	0.00	0.054	0.003
Androgyny	0.55	0.06	0.219	0.043
F value	135.116		71.706	
P value	< 0.001		< 0.001	

Table 4. *Effect of cerium and antibiotics on pyruvate.*

Group	Pyruvate % change (Increase with Cerium)		Pyruvate % change (Decrease with Doxy+Cipro)	
	Mean	**± SD**	**Mean**	**± SD**
Normal	4.34	0.21	18.43	0.82
Androgyny	20.99	1.46	61.23	9.73
F value	321.255		115.242	
P value	< 0.001		< 0.001	

Table 5. *Effect of cerium and antibiotics on delta amino levulinic acid.*

Group	ALA % (Increase with Cerium)		ALA % (Decrease with Doxy+Cipro)	
	Mean	**± SD**	**Mean**	**± SD**
Normal	4.40	0.10	18.48	0.39
Androgyny	23.20	1.57	66.65	4.26
F value	372.716		556.411	
P value	< 0.001		< 0.001	

Table 6

Group	RBC digoxin (ng/ml RBC Susp)		Cytochrome F420		ALA (umol24)		Pyruvate (umol/l)		RBC hexokinase (ug glu phos / hr/mgpro)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Normal	0.18	0.05	0.00	0.00	3.86	0.26	23.79	2.51	0.68	0.23
Androgyny	1.38	0.26	4.00	0.00	68.16	4.92	102.48	13.20	8.46	3.63
F value	60.288		0.001		295.467		154.701		18.187	
P value	< 0.001		< 0.001		< 0.001		< 0.001		< 0.001	

Discussion

The study indicates that androgynous individuals tend to have the Neanderthal phenotype with skeletal characteristics. The androgynous individuals may have more of Neanderthal genotype. The metabolonomics in androgyny is suggestive of Neanderthal phenotype. There is increased actinidic archaeal symbiosis as indicated by increase in cytochrome F420 activity. The actinidic archaea uses cholesterol as a metabolic substrate. There is ring oxidation of cholesterol generating pyruvate. The pyruvate enters the GABA shunt pathway producing glycine and succinyl CoA. This results in porphyrin synthesis. The cholesterol is also converted to steroidal glycoside digoxin. Digoxin and porphyrin intercalation in the cell membrane produces sodium potassium ATPase inhibition and accumulation of intracellular calcium. The increase in intracellular calcium induces nitric oxide synthase, heme oxygenase and cystathione synthase generating nitric oxide, carbon monoxide and hydrogen sulphide. This results in vasodilation of the blood spaces in the corpora cavernosa and increasing autonomic function of the genitourinary system resulting in obsessive traits. The increasing cholesterol catabolism by actinidic archaea results in depletion of cholesterol from the body. This produces inhibition of estrogen and testosterone synthesis. This results in an asexual state and androgynous behaviour. The brain function depends on

testosterone and estrogens. The sex hormones modulate hemispheric dominance. The estrogens produce left hemispheric dominance and testosterones produce right hemispheric dominance. The lack of estrogens and testosterones in androgyny results in equidominance. This leads to equal function of the right hemisphere and left hemisphere and a state of creativity mixed with practicality. The right hemisphere is concerned with creative behaviour and the left hemisphere is concerned with practical behaviour. Equidominance results in the generation of a new phenotype with dominance of both creativity and practicality. Equidominance and lack of estrogens and testosterones can contribute to the social state of matriarchy. There is female dominance in society. The behavioural patterns between the male and female section of the population becomes homogenized. This results in generation of matrilineal societies and the demise of patriarchy.

Porphyrinuria and porphyria are the hallmarks of androgyny. This contributes to neuro-immuno-endocrine regulation and disease states associated with androgyny. The cholesterol is catabolized to porphyrins. Porphyrins are dipolar molecules and can contribute to quantal perception which is more in androgyny contributing to creativity, spirituality and extrasensory perceptive modes of this phenotype. Low level electromagnetic fields and its porphyrin messengers can regulate the brain mediating conscious and quantal perception. Porphyrin microarrays serve the purpose of quantal and conscious perception. The archaea and viroids via porphyrin synthesis can regulate the nervous system including the NMDA/GABA thalamo-cortico-thalamic 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 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 porphyrin microarrays can function as a quantal computer mediating extrasensory perception. Porphyrin microarrays in human systems and brain owing to the wave particle nature of porphyrins can bridge the quantal world and particulate world. The porphyrins can modulate hemispheric dominance. There is increased porphyrin synthesis and RHCD and decreased porphyrin synthesis in LHCD. 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. Thus porphyrins microarrays can function as a quantal brain modulating extrasensory quantal perception. Porphyrin microarrays can function as a quantal brain in communication with digital world and geomagnetic fields. The dipolar porphyrins 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 autooxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Porphyrin autooxidation is modulated by low level of electromagnetic fields and geomagnetic fields. Cellular porphyrins photooxidation 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 human porphyrin microarrays 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 digital 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. Porphyrin microarrays can function as quantal computers storing information and can serve the purpose of extrasensory perception. Porphyrins can serve as a two way communicating bridge between digital information storage systems generating low level electromagnetic fields and human systems. The low level of EMF produced by digital system enhances porphyrin synthesis and serves the purpose of two way extrasensory perception and communication. The human porphyrin quantal computers can in turn by biophoton emission modulate digital information storage system.

Low level of electromagnetic fields and its porphyrin messengers can induce the Warburg phenotype. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. The archaea can synthesize porphyrins and induce porphyrin synthesis. Porphyrins have been related to schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. Porphyrins can mediate the effect of low level electromagnetic fields inducing the Warburg phenotype leading to the above mentioned disease states. 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 channelling 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. Low level electromagnetic fields can induce the Warburg phenotype contributing to human disease.

The role of porphyrins and low level electromagnetic fields in regulation of cell functions and neuro-immuno-endocrine integration is discussed. Low levels of EMF fields can induce digoxin synthesis. 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. Low level of EMF fields can modulate membrane, nucleic acid and protein structure and function via induction of porphyrin synthesis. 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. Low level of EMF fields through modulation of porphyrin metabolism can

produce heme deficiency by inhibiting heme oxygenase and ferrochelatase. 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. Low level electromagnetic fields can modulate cell functions and neuro-immuno-endocrine-genetic integration via induction of porphyrin synthesis. Low level electromagnetic fields via modulating porphyrin metabolism can produce an autonomic neuropathy. Protoporphyrins block acetyl choline transmission producing a vagal neuropathy with sympathetic over activity. Vagal neuropathy results in immune activation, vasospasm and vascular disease. A vagal neuropathy underlines neoplastic and autoimmune processes as well as metabolic syndrome x. Low level electromagnetic fields by modulating porphyrin metabolism can induce cell death. 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 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. Low level electromagnetic fields by modulating porphyrin metabolism can generate redox stress to regulate cell functions. 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. Low level electromagnetic fields by modulating porphyrin metabolism can regulate cell membrane sodium potassium ATPase. 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. Low level electromagnetic fields by modulating porphyrin metabolism can regulate DNA, RNA and protein structure and function. Porphyrins complexing with proteins can modulate protein structure and function. Porphyrins complexing with DNA and RNA can modulate transcription and translation. Low level electromagnetic fields by modulating porphyrin metabolism can regulate mitochondrial function, peripheral benzodiazepine receptor and steroidogenesis. 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. Low level electromagnetic fields by modulating porphyrin metabolism and inducing redox stress can regulate enzyme systems. The

porphyrin photooxidation 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. Low level of electromagnetic fields by modulating porphyrin metabolism can induce viroidal and HERV expression. 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. Porphyrin photooxidation induced redox stress can produce HDAC inhibition. 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. Low level electromagnetic fields by modulating porphyrin metabolism and generating redox stress can produce immune activation. 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. Low level electromagnetic fields by modulating porphyrin metabolism and inducing redox stress can produce insulin resistance. The porphyrin photooxidation 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. Low level electromagnetic fields by modulating porphyrin metabolism and inducing redox stress/heme deficiency can activate HIF alpha. The porphyrin photooxidation 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. Low level electromagnetic fields by modulating porphyrin metabolism can regulate prion protein conformation. 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. Low level electromagnetic fields by modulating porphyrin metabolism can produce redox stress and regulate HERV expression. The porphyrins can also intercalate with DNA producing HERV expression. The HERV particles generated can contribute to the retroviral state associated with androgyny. The porphyrins in the blood can combine with bacteria and viruses and the photooxidation generated free radicals can kill them. Low level electromagnetic fields by modulating porphyrin metabolism can lead to increase predilection for viral and bacterial infections. The archaeal porphyrins can modulate bacterial and viral infections. The archaeal porphyrins are regulatory molecules keeping other prokaryotes and viruses on check.

Thus the actinidic archaeal symbiosis results in neanderthalisation of the population and generation of androgyny. The actinidic archaeal overgrowth and symbiosis is a consequence of global warming. Archaea are extremophiles and increase in density during periods of climate change. The actinidic archaeal catabolism of cholesterol generates digoxin and increased intracellular calcium resulting in formation of excess of gasotransmitters important in autonomic function of structures like the corpora cavernosa. The cholesterol catabolism results in depletion of cholesterol and to a state of lack of sex hormone synthesis. This produces an asexual state resulting in a social system of matriarchy related to androgyny. The actinidic archaeal cholesterol catabolism generates porphyrins producing the extrasensory quantal perceptive state associated with androgyny. This contributes to the creativity of the androgynous state. The porphyrin synthesis associated with androgyny also contributes to the disease states associated with it. This includes autoimmune disease, cancer, degenerations, acquired immunodeficiency syndrome, metabolic syndrome x and all civilisational disease.

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