Chapter 12

The Porphyrions Modulates Conscious/Quantal Perception, Hemispheric Dominance and Human Personality - The Genesis of Right Hemispheric Dominant Global Porphyric Personality

Introduction

Actinidic archaea have been related to the pathogenesis of disorders of consciousness - schizophrenia and autism and modulates conscious/quantal perception. Actinidic archaea also determines hemispheric dominance. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. Actinidic archaea have a mevalonate pathway and are cholesterol catabolizing. They can use cholesterol as a carbon and energy source. Archaeal cholesterol catabolism can generate porphyrins via the cholesterol ring oxidase generated pyruvate and GABA shunt pathway. Archaea can produce a secondary porphyria by inducing the enzyme heme oxygenase resulting in heme depletion and activation of the enzyme ALA synthase. Porphyrins have been related to schizophrenia and autism. Porphyrins microarrays determine conscious/quantal perception and hemispheric dominance. Actinidic archaea and porphyrins can modulate personality profiles. The Scythian populations have an increased incidence of porphyrias and study aimed to elucidate this aspect. The role of actinidic archaea and porphyrins in modulating personality and behavioural traits is discussed in this report.[1-5] Porphyrins can function as self replicating supramolecular organisms which can be called as porphyrions. The porphyrions are self replicating supramolecular organisms which forms the precursor template on which the viroids, prions and nanoarchaea originate. Stress induced template directed abiogenesis of porphyrions, prions, viroids and archaea is a continuous process and can contribute to changes in brain structure and behavior as well as disease process.

Materials and Methods

The following disorders of consciousness were included in the study: - schizophrenia and autism. There were 10 patients in each group and each patient had an age and sex matched healthy control selected randomly from the general population. There were also 10 normal people belonging to the right hemispheric dominant/scythian group and left hemispheric dominant/non-scythian group selected from the general population. The blood samples were drawn in the fasting state before treatment was initiated. Plasma from fasting heparinised blood was used and the experimental protocol was as follows (I) Plasma+phosphate buffered saline, (II) same as I+cholesterol substrate, (III) same as II+rutile 0.1 mg/ml, (IV) same as II+ciprofloxacine and doxycycline each in a concentration of 1 mg/ml. Cholesterol substrate was prepared as described by Richmond. Aliquots were withdrawn at zero time immediately after mixing and after incubation at 37 ℃ for 1 hour. The following estimations were carried out: - Cytochrome F420, free RNA, free DNA, polycyclic aromatic hydrocarbon, hydrogen peroxide, pyruvate, ammonia, glutamate, delta aminolevulinic acid, succinate, glycine and digoxin. Cytochrome F420 was estimated flourimetrically (excitation wavelength 420 nm and emission wavelength 520 nm). The study also involved estimating the following parameters in the patient population - digoxin, bile acid, hexokinase, porphyrins, pyruvate, glutamate, ammonia, acetyl CoA, acetyl choline, HMG CoA reductase, cytochrome C, blood ATP, ATP synthase, ERV RNA (endogenous retroviral RNA), H_2O_2 (hydrogen peroxide), NOX (NADPH oxidase), TNF alpha and heme oxygenase.[6-9] The personality profiles of the normal population with right hemispheric, left hemispheric and bi-hemispheric dominance was assessed and related to actinidic archaeal density and porphyrin synthesis. Informed consent of the subjects and the approval of the

ethics committee were obtained for the study. The statistical analysis was done by ANOVA.

Results

Plasma of control subjects showed increased levels of the above mentioned parameters with after incubation for 1 hour and addition of cholesterol substrate resulted in still further significant increase in these parameters. The plasma of patients showed similar results but the extent of increase was more. The addition of antibiotics to the control plasma caused a decrease in all the parameters while addition of rutile increased their levels. The addition of antibiotics to the patient's plasma caused a decrease in all the parameters while addition of rutile increased their levels but the extent of change was more in patient's sera as compared to controls. The results are expressed in section 1: tables 1-6 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time. 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 study showed the patient population and right hemispheric dominant/scythian group had increased heme oxygenase activity and porphyrins. The hexokinase activity was high. The pyruvate, glutamate and ammonia levels were elevated indicating blockade of PDH activity, and operation of the GABA shunt pathway. The acetyl CoA levels were low and acetyl choline was decreased. The cytoC levels were increased in the serum indicating mitochondrial dysfunction suggested by low blood ATP levels. This was

indicative of the Warburg's phenotype. There were increased NOX and TNF alpha levels indicating immune activation. The HMG CoA reductase activity was high indicating cholesterol synthesis. The bile acid levels were low indicating depletion of cytochrome P450. The normal scythian/right hemispheric dominant population had values resembling the patient population with increased porphyrin synthesis. The normal non-scythian left hemispheric dominant population had low values with decreased porphyrin synthesis.

The right hemispheric chemical dominant/scythian population had higher archaeal density and porphyrin synthesis as compared to left hemispheric chemical dominance population. The right hemispheric chemical dominance group had a higher IQ, increased creativity, artistic talents, mathematical skills, intuitive memory, decreased language skills, asexual traits and aberrant sexual behavior, increased community bonding, spirituality, altruism and paganistic tendencies.

Section 1: Experimental Study

Table 1. *Effect of rutile and antibiotics on cytochrome F420 and PAH.*

Group	CYT F420 % (Increase with Rutile)		CYT F420 % (Decrease with Doxy+Cipro)		PAH % change (Increase with Rutile)		PAH % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Non-scyth/ LHCD	4.48	0.15	18.24	0.66	4.45	0.14	18.25	0.72
Scythians/RHCD	23.12	2.00	56.90	6.94	23.26	1.53	60.91	7.59
Schizophrenia	23.24	2.01	58.72	7.08	23.01	1.69	59.49	4.30
Autism	21.68	1.90	57.93	9.64	22.61	1.42	64.48	6.90
EMF exposure	22.70	1.87	60.46	8.06	23.73	1.38	65.20	6.20
F value	306.749		130.054		391.318		257.996	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 2. *Effect of rutile and antibiotics on free RNA and DNA.*

Group	DNA % change (Increase with Rutile)		DNA % change (Decrease with Doxy+Cipro)		RNA % change (Increase with Rutile)		RNA % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Non-scyth/ LHCD	4.37	0.15	18.39	0.38	4.37	0.13	18.38	0.48
Scythians/RHCD	23.52	1.65	64.15	4.60	23.29	1.92	65.39	3.95
Schizophrenia	23.28	1.70	61.41	3.36	23.59	1.83	65.69	3.94
Autism	22.12	2.44	63.69	5.14	23.33	1.35	66.83	3.27
EMF exposure	22.62	1.38	63.82	5.53	23.29	1.98	67.46	3.96
F value	337.577		356.621		427.828		654.453	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 3. *Effect of rutile and antibiotics on digoxin and delta aminolevulinic acid.*

Group	Digoxin (ng/ml) (Increase with Rutile)		Digoxin (ng/ml) (Decrease with Doxy+Cipro)		ALA % (Increase with Rutile)		ALA % (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Non-scyth/ LHCD	0.11	0.00	0.054	0.003	4.40	0.10	18.48	0.39
Scythians/RHCD	0.55	0.03	0.192	0.040	23.67	1.68	66.50	3.58
Schizophrenia	0.55	0.06	0.219	0.043	22.52	1.90	66.39	4.20
Autism	0.53	0.08	0.205	0.041	23.20	1.57	66.65	4.26
EMF exposure	0.51	0.05	0.199	0.027	22.83	1.90	67.23	3.45
F value	135.116		71.706		372.716		556.411	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 4. *Effect of rutile and antibiotics on succinate and glycine.*

Group	Succinate % (Increase with Rutile)		Succinate % (Decrease with Doxy+Cipro)		Glycine % change (Increase with Rutile)		Glycine % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Non-scyth/ LHCD	4.41	0.15	18.63	0.12	4.34	0.15	18.24	0.37
Scythians/RHCD	23.81	1.90	66.95	3.67	23.12	1.71	65.12	5.58
Schizophrenia	22.76	2.20	67.63	3.52	22.79	2.20	64.26	6.02
Autism	21.88	1.19	66.28	3.60	23.02	1.65	67.61	2.77
EMF exposure	22.28	1.52	64.05	2.79	22.82	1.56	64.61	4.95
F value	403.394		680.284		348.867		364.999	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 5. *Effect of rutile and antibiotics on pyruvate and glutamate.*

Group	Pyruvate % change (Increase with Rutile)		Pyruvate % change (Decrease with Doxy+Cipro)		Glutamate (Increase with Rutile)		Glutamate (Decrease with Doxy+Cipro)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Non-scyth/ LHCD	4.34	0.21	18.43	0.82	4.21	0.16	18.56	0.76
Scythians/RHCD	22.63	0.88	56.40	8.59	22.96	2.12	65.11	5.91
Schizophrenia	20.99	1.46	61.23	9.73	23.01	2.61	65.87	5.27
Autism	21.91	1.71	58.45	6.66	22.88	1.87	65.45	5.08
EMF exposure	20.94	1.54	62.76	8.52	23.33	1.79	62.50	5.56
F value	321.255		115.242		292.065		317.966	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 6. *Effect of rutile and antibiotics on hydrogen peroxide and ammonia.*

Group	H_2O_2 % (Increase with Rutile)		H_2O_2 % (Decrease with Doxy+Cipro)		Ammonia % (Increase with Rutile)		Ammonia % (Decrease with Doxy+Cipro)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Non-scyth/ LHCD	4.43	0.19	18.13	0.63	4.40	0.10	18.48	0.39
Scythians/RHCD	22.65	2.48	60.19	6.98	23.67	1.68	66.50	3.58
Schizophrenia	22.50	1.66	60.21	7.42	22.52	1.90	66.39	4.20
Autism	23.52	1.49	63.24	7.36	23.20	1.57	66.65	4.26
EMF exposure	23.81	1.19	61.08	7.38	22.83	1.90	67.23	3.45
F value	380.721		171.228		372.716		556.411	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Section 2: Patient Study

Table 1

Group	RBC Digoxin (ng/ml RBC Susp)		Cytochrome F420		HERV RNA (ug/ml)		H_2O_2 (umol/ml RBC)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Non-scythians	0.58	0.07	1.00	0.00	17.75	0.72	177.43	6.71
Scythians	1.34	0.31	4.00	0.00	51.16	7.78	295.37	3.78
RHCD	1.41	0.23	4.00	0.00	55.17	5.85	278.29	7.74
LHCD	0.18	0.05	0.00	0.00	8.70	0.90	111.63	5.40
Schizo	1.38	0.26	4.00	0.00	51.17	3.65	274.88	8.73
Autism	1.19	0.24	4.00	0.00	52.87	7.04	274.52	9.29
Exposure to EMF	1.41	0.30	4.00	0.00	51.01	4.77	276.49	10.92
F value	60.288		0.001		194.418		713.569	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 2

Group	NOX (OD diff/hr/mgpro)		TNF ALP (pg/ml)		ALA (umol24)		PBG (umol24)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Non-scythians	0.012	0.001	17.94	0.59	15.44	0.50	20.82	1.19
Scythians	0.035	0.011	82.13	3.97	67.30	5.98	47.25	4.19
RHCD	0.036	0.008	78.63	5.08	63.50	6.95	42.20	8.50
LHCD	0.007	0.001	9.29	0.81	3.86	0.26	12.11	1.34
Schizo	0.036	0.009	78.23	7.13	66.16	6.51	42.50	3.23
Autism	0.036	0.006	76.71	5.25	68.16	4.92	42.04	2.38
Exposure to EMF	0.038	0.007	76.41	5.96	68.41	5.53	47.27	3.42
F value	44.896		427.654		295.467		183.296	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 3

Group	Uroporphyrin (nmol/24)		Coproporphyrin (nmol/24)		Protoporphyrin (Ab unit)		Heme (uM)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Non-scythians	50.18	3.54	137.94	4.75	10.35	0.38	30.27	0.81
Scythians	286.84	24.18	432.22	50.11	49.36	4.18	11.81	0.80
RHCD	250.28	23.43	389.01	54.11	42.46	6.36	12.47	2.82
LHCD	9.51	1.19	64.33	13.09	2.64	0.42	50.55	1.07
Schizo	267.81	64.05	401.49	50.73	44.30	2.66	12.82	2.40
Autism	318.84	82.90	423.29	47.57	47.50	2.87	12.37	2.09
Exposure to EMF	288.21	26.17	444.94	38.89	50.59	1.71	12.36	1.26
F value	160.533		279.759		424.198		1472.05	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 4

Group	Bilirubin (mg/dl)		Biliverdin (Ab unit)		ATP Synthase (umol/gHb)		SE ATP (umol/dl)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Non-scythians	0.55	0.02	0.030	0.001	0.36	0.13	0.42	0.11
Scythians	1.83	0.09	0.071	0.014	3.34	0.84	1.27	0.26
RHCD	1.70	0.20	0.067	0.011	2.73	0.94	2.24	0.44
LHCD	0.21	0.00	0.017	0.001	0.09	0.01	0.02	0.01
Schizo	1.74	0.08	0.073	0.013	2.66	0.58	1.26	0.19
Autism	1.83	0.16	0.072	0.014	2.67	0.80	2.03	0.12
Exposure to EMF	1.75	0.22	0.073	0.013	3.39	1.03	1.37	0.27
F value	370.517		59.963		54.754		67.588	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 5

Group	Cyto C (ng/ml)		Lactate (mg/dl)		Pyruvate (umol/l)		RBC Hexokinase (ug glu phos / hr/mgpro)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Non-scythians	2.79	0.28	7.38	0.31	40.51	1.42	1.66	0.45
Scythians	12.65	1.06	24.28	1.69	95.44	12.04	9.30	3.98
RHCD	12.39	1.23	25.99	8.10	100.51	12.32	5.46	2.83
LHCD	1.21	0.38	2.75	0.41	23.79	2.51	0.68	0.23
Schizo	11.58	0.90	22.07	1.06	96.54	9.96	7.69	3.40
Autism	12.48	0.79	21.95	0.65	92.71	8.43	6.95	2.02
Exposure to EMF	12.26	1.00	23.31	1.46	103.28	11.47	7.58	3.09
F value	445.772		162.945		154.701		18.187	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 6

Group	ACOA (mg/dl)		ACH (ug/ml)		Glutamate (mg/dl)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Non-scythians	8.75	0.38	75.11	2.96	0.65	0.03
Scythians	1.95	0.06	35.02	5.85	3.14	0.32
RHCD	2.51	0.36	38.57	7.03	3.19	0.32
LHCD	16.49	0.89	91.98	2.89	0.16	0.02
Schizo	2.51	0.57	48.52	6.28	3.41	0.41
Autism	2.42	0.41	50.61	6.32	3.30	0.32
Exposure to EMF	2.14	0.19	37.75	7.31	3.47	0.37
F value	1871.04		116.901		200.702	
P value	< 0.001		< 0.001		< 0.001	

Table 7

Group	Se. Ammonia (ug/dl)		HMG Co A (HMG CoA/MEV)		Bile Acid (mg/ml)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Non-scythians	50.60	1.42	1.70	0.07	79.99	3.36
Scythians	94.60	8.52	1.08	0.13	28.93	4.93
RHCD	93.43	4.85	1.16	0.10	25.68	7.04
LHCD	23.92	3.38	2.21	0.39	140.40	10.32
Schizo	94.72	3.28	1.11	0.08	22.45	5.57
Autism	94.01	5.00	1.12	0.06	23.16	5.78
Exposure to EMF	102.62	26.54	1.00	0.07	22.58	5.07
F value	61.645		159.963		635.306	
P value	< 0.001		< 0.001		< 0.001	

Abbreviations

RHCD: Right hemispheric chemical dominance

LHCD: Left hemispheric chemical dominance

Schizo: Schizophrenia

Discussion

There was increase in cytochrome F420 indicating archaeal growth. The archaea can synthesize and use cholesterol as a carbon and energy source.[2, 10] The archaeal origin of the enzyme activities was indicated by antibiotic induced suppression. The study indicates the presence of actinide based archaea with an alternate actinide based enzymes or metalloenzymes in the system as indicated by rutile induced increase in enzyme activities.[11] The archaeal beta hydroxyl steroid dehydrogenase activity indicating digoxin synthesis.[12] The archaeal cholesterol oxidase activity was increased resulting in generation of pyruvate and hydrogen peroxide.[10] The 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 to 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.[13] The role of archaeal porphyrins in regulation of cell functions and neuro-immuno-endocrine integration is discussed. Protoporphyrine 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. 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 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 autism, seizure disorder, schizophrenia and chronic fatigue syndrome. Bile acids can bind to olfactory receptors and modulate limbic lobe function. Bile acids are involved in group and social cognition. Bile acid deficiency can lead to autism and schizophrenia. Bile acids are neuroprotective and its deficiency can contribute to autism, seizure disorder, schizophrenia and chronic fatigue syndrome. Mitochondrial dysfunction has been related to autism, seizure disorder, schizophrenia and chronic fatigue syndrome. NO, CO and H_2S deficiency can contribute to hypoglutamatergic state in schizophrenia and autism. As serine and glycine are utilized for porphyrin synthesis, they are not available for positive modulation of the NMDA receptor. This can contribute to NMDA receptor dysfunction in schizophrenia and autism.[3-5]

The archaea/viroids and porphyrins 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 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 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 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. Protoporphyrins block acetyl choline transmission producing a vagal neuropathy with sympathetic overactivity. Vagal neuropathy results in immune activation, vasospasm and vascular disease. A vagal neuropathy underlines functional neuropsychiatric disorders. 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 autism, seizure disorder, schizophrenia and chronic fatigue syndrome. 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.[3, 4, 16]

The right hemispheric chemical dominant hyperdigoxinemic population had higher archaeal density and porphyrin synthesis as compared to left hemispheric chemical dominant population. The right hemispheric chemical dominant group had a higher IQ, increased creativity, artistic talents, mathematical skills, intuitive memory, decreased language skills, asexual traits and aberrant sexual behavior, increased community bonding, spirituality, altruism and paganistic tendencies. Porphyrins mediate quantal perception. This creates a feeling of oneness with the world around you and contributes to community bonding, family bonding, global bonding and altruistic behavior. The quantal perception gives rise to pagan tendencies and a feeling of oneness with nature and a tendency for environmental ecospirituality. The porphyrin mediated quantal perception gives a sense of dissolution of the self and gives a spiritual perspective to the right hemispheric dominant personality. The porphyrin induced increased NMDA transmission and conscious/quantal perception can give rise to increased cognitive and intellectual abilities and a higher IQ to the right hemispheric dominant group. This gives rise to political and spiritual leadership traits in this group. The porphyrin mediated quantal perception in the right hemispheric dominant group gives rise to telescopic memory and intuitive memory traits. As quantal perception is dominant in the right hemispheric dominant group language skills deteriorate and the right hemispheric

personality is more prone to abstract thinking. The right hemispheric personality is more creative, generates more scientific ideas and has more mathematical skills. Right hemisphere is dominant for gestural traits and this contributes to increased artistic, dancing and acting skills in the right hemispheric dominant population. The heme depletion leads to defects in cytochrome P450 enzyme systems mediating sex hormone synthesis. The heme depletion also leads to defects in nitric oxide synthase activity and reduced nitric oxide synthesis. This leads to an asexual personality with also a higher incidence of aberrant sexual behaviour of the homosexual/lesbian. The right hemispheric porphyric personality has right parietal lobe dominance and quantal perception. The right parietal lobe of the brain is concerned with environmental interactions and quantal perception modulates this aspect of brain function. The porphyric personality has a higher amount of quantal perception and right parietal overactivity. They tend to be guided by environmental cues and this tends to produce uniformity in cultural and ethical values in the right hemispheric dominant porphyric personality. There is a suppression of frontal activity and the quality of introspection. This contributes to the homogeneity of global culture dominated by an increased in right hemispheric personality profiles due to environmental low level EMF pollution. The right hemispheric personality can be described as the porphyric personality or global personality.

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 auto-oxidation 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 photo-oxidation 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. Thus induction of porphyrin synthesis can serve as a mechanism of communication between human brain and the environment by extrasensory perception. Thus porphyrin mediated extrasensory perception can contribute to the pathogenesis of autism and schizophrenia. Porphyrin mediated extrasensory perception can modulate hemispheric dominance. Low level of EMF can regulate conscious/quantal perception and modulate hemispheric dominance. Low level of EMF can also modulate personality traits. There is environmental pollution with increased amount of low level EMF radiation in the environment. This leads to increased porphyrin synthesis and a rise in porphyrin mediated right hemispheric dominant personality traits in epidemic proportions. This contributes to the genesis of a personality profile in consonance with the globalization trends.

Porphyrins also have evolutionary significance since porphyria is related to Scythian races and contributes to the behavioural and intellectual characteristics of this group of population. Porphyrins can intercalate into DNA and produce HERV expression. HERV RNA can get converted to DNA by reverse transcriptase which can get integrated into DNA by integrase. This tends to increase the length of the non coding region of the DNA. The increase in non coding region of the DNA is involved in primate and human evolution. Thus, increased rates of porphyrin synthesis would correlate with increase in non coding DNA length. The alteration in the length of the non coding region of the DNA contributes to the dynamic nature of the genome. Thus genetic and acquired porphyrias can lead to alteration in the non coding region of the genome. The alteration of the length of the non coding region of the DNA contributes to the racial and individual differences in populations. An increased length of non coding region as well as increased porphyrin synthesis leads to increased cognitive and creative neuronal function. Porphyrins are involved in

quantal perception and regulation of the thalamocorticothalamic pathway of conscious perception. Thus genetic and acquired porphyrias contribute to higher cognitive and creative capacity of certain races. Porphyrias are common among Eurasian Scythian races who have assumed leadership roles in communities and groups. Porphyrins have contributed to human and primate evolution. There is high incidence of autism and schizophrenia in Scythian races and most of our patient population belonged to this group.[3, 4] The right hemispheric porphyrin mediated global personality traits are more prominent in Scythian races with increased porphyrin synthesis. The Scythians tend to have right hemispheric dominant traits and have assumed political and spiritual leadership of population groups. The increased porphyrin synthesis leads to increased HERV expression and its reintegration into the non-coding region of the genome. This increased porphyrin synthesis leads to increase in length and structure of the non-coding region of the genome. This leads on to the genomic evolution of a porphyrin personality which is right hemispheric dominant. The increased porphyrin synthesis consequent to increased HERV expression leads to further increase in length of the non-coding region of the genome. This can lead to the evolution of a new human phenotype and genotype with a right hemispheric dominance global personality profile.

The actinidic archaea mediated Warburg phenotype and the increased porphyrin synthesis contributes to a neuropsychiatric personality with autism and schizophrenia. 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 can contribute to neuropsychiatric disorders. The Warburg phenotype results in increased generation of fructose 1, 6, diphosphate which enters the pentose phosphate pathway generating NADPH. This activates NOX and generates H_2O_2. Redox stress and NOX activity can increase NMDA transmission modulating conscious/quantal perception. This can lead to schizophrenia and autism. The increase in glycolysis leads to immune activation as lymphocytes depends on glycolysis for energy needs. This leads to increased cytokine secretion and neuronal injury important in schizophrenia and autism. 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. Redox stress can modulate the NMDA/GABA thalamocorticothalamic pathway important in conscious perception. This contributes to the genesis of autism and schizophrenia. 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.[3-5] 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. HERV expression and viroids has been related to autism and schizophrenia. HERV RNA and viroidal RNA can modulate brain function by RNA interference. Redox stress can contribute to autism and schizophrenia.[14, 15] The porphyrin photo-oxidation can generate free radicals which can activate NFKB. This can produce immune activation and cytokine mediated injury. Immune activation has been related to autism and schizophrenia. The increase in archaeal porphyrins can lead to immune activation crucial in the pathogenesis of functional neuropsychiatric disorders. 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 pathology documented in autism, seizure disorder, schizophrenia and chronic fatigue syndrome.[3, 4] The protoporphyrins binding to mitochondrial benzodiazepine receptors can modulate mitochondrial steroidogenesis and metabolism. Sex steroids can modulate brain neurotransmission and contribute to the pathogenesis of autism, seizure disorder, schizophrenia and chronic fatigue syndrome.[3, 4] 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. Borna and herpes viruses has been related to autism, seizure disorder, schizophrenia and chronic fatigue syndrome.[3, 4] Thus the archaeal porphyrins can contribute to the pathogenesis of autism, seizure disorder, schizophrenia and chronic fatigue syndrome. Archaeal porphyrin synthesis is crucial in the pathogenesis of these disorders. Porphyrins may serve 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.[3, 4] The actinidic archaea mediated Warburg phenotype, immune activation, mitochondrial dysfunction, redox stress, HERV expression, viroidal expression, protein processing defect and digoxin synthesis can contribute to the genesis of tumors, autoimmune disease, degenerations and metabolic syndrome x. This contributes to a porphyric-right hemispheric dominant pathological personality which coincides with the personality profile described earlier.

An actinide dependent shadow biosphere of archaea and viroids is described in autism and schizophrenia. Porphyrin synthesis is crucial in the pathogenesis of these disorders. Porphyrins can modulate conscious and quantal perception and regulate hemispheric dominance. Right hemispheric dominant normal population with increased porphyrin synthesis had a higher IQ, increased creativity, artistic talents, mathematical skills, intuitive memory, decreased language skills, asexual traits and aberrant sexual behavior, increased community bonding, spirituality, altruism and paganistic tendencies. This can be described as the porphyric personality. The porphyric right hemispheric

dominant personality has a pathological component with increased incidence of autism, schizophrenia, tumors, autoimmune disease, degenerations and metabolic syndrome x. Porphyrins can modulate personality traits and human behavior. This contributes to the genesis of a human genotype/phenotype - the global right hemispheric dominant hyperdigoxinemic porphyric personality.

The porphyrions are self replicating supramolecular organisms which forms the precursor template on which the viroids, prions and nanoarchaea originate. Stress induced template directed abiogenesis of porphyrions, prions, viroids and archaea is a continuous process and can contribute to changes in brain structure and behavior as well as disease process.

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