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Porphyrin and Polycyclic Aromatic Hydrocarbon Templates Mediate Endosymbiotic Archaeal, Viroidal and Prion Replication - Porphyrions and Abiogenesis

Introduction

Abiogenesis is a replicative mechanism for primitive life forms. Actinidic archaea and viroids have been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. 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, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. Porphyrins can serve as templates for macromolecules like RNA, DNA, proteins and isoprenoidal molecules to form. The actinidic archaea induces porphyrinogenesis and porphyrins form a template for formation of macromolecules mediating actinidic archaeal and viroidal replication. This is a form of abiogenesis on a porphyrin template. The archaeal synthesized polycyclic aromatic hydrocarbons can also serve as a template for macromolecular replication and viroidal/ actinidic archaeal abiogenesis. The role of archaeal porphyrins in regulation of cell functions and neuro-immuno-endocrine integration is discussed. Porphyrins are prebiotic molecules which are involved in abiogenesis and origin of life.[1-5] 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 groups were included in the study: - endomyocardial fibrosis, Alzheimer's disease, multiple sclerosis, non-Hodgkin's lymphoma, metabolic syndrome x with cerebrovascular thrombosis and coronary artery disease, schizophrenia, autism, seizure disorder, Creutzfeldt Jakob's disease and acquired immunodeficiency syndrome. 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 patients with right hemispheric dominance, left hemispheric dominance and bi-hemispheric dominance drawn 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). Polycyclic aromatic hydrocarbon was estimated by measuring hydrogen peroxide liberated by using glucose reagent. 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] 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's blood and right hemispheric dominance 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 population with right hemispheric dominance had values resembling the patient population with increased porphyrin synthesis. The normal population with left hemispheric dominance had low values with decreased porphyrin synthesis.

The increase in cytochrome F420 and self replicating RNA indicating RNA viroids paralleled the rise in porphyrin levels indicating that porphyrins could serve as a template for actinidic archaeal and RNA viroidal abiogenesis and replication. The increase in cytochrome F420 and self replicating RNA indicating RNA viroids paralleled the rise in PAH levels indicating that PAH could serve as a template for archaeal and RNA viroidal abiogenesis and replication. There was an increase in porphyrins and PAH in CJD indicating prions may also self replicate on porphyrin and PAH templates.

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**
Normal	4.48	0.15	18.24	0.66	4.45	0.14	18.25	0.72
Schizo	23.24	2.01	58.72	7.08	23.01	1.69	59.49	4.30
Seizure	23.46	1.87	59.27	8.86	22.67	2.29	57.69	5.29
AD	23.12	2.00	56.90	6.94	23.26	1.53	60.91	7.59
MS	22.12	1.81	61.33	9.82	22.83	1.78	59.84	7.62
NHL	22.79	2.13	55.90	7.29	22.84	1.42	66.07	3.78
DM	22.59	1.86	57.05	8.45	23.40	1.55	65.77	5.27
AIDS	22.29	1.66	59.02	7.50	23.23	1.97	65.89	5.05
CJD	22.06	1.61	57.81	6.04	23.46	1.91	61.56	4.61
Autism	21.68	1.90	57.93	9.64	22.61	1.42	64.48	6.90
EMF	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
Normal	4.37	0.15	18.39	0.38	4.37	0.13	18.38	0.48
Schizo	23.28	1.70	61.41	3.36	23.59	1.83	65.69	3.94
Seizure	23.40	1.51	63.68	4.66	23.08	1.87	65.09	3.48
AD	23.52	1.65	64.15	4.60	23.29	1.92	65.39	3.95
MS	22.62	1.38	63.82	5.53	23.29	1.98	67.46	3.96
NHL	22.42	1.99	61.14	3.47	23.78	1.20	66.90	4.10
DM	23.01	1.67	65.35	3.56	23.33	1.86	66.46	3.65
AIDS	22.56	2.46	62.70	4.53	23.32	1.74	65.67	4.16
CJD	23.30	1.42	65.07	4.95	23.11	1.52	66.68	3.97
Autism	22.12	2.44	63.69	5.14	23.33	1.35	66.83	3.27
EMF	22.29	2.05	58.70	7.34	22.29	2.05	67.03	5.97
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
Normal	0.11	0.00	0.054	0.003	4.40	0.10	18.48	0.39
Schizo	0.55	0.06	0.219	0.043	22.52	1.90	66.39	4.20
Seizure	0.51	0.05	0.199	0.027	22.83	1.90	67.23	3.45
AD	0.55	0.03	0.192	0.040	23.67	1.68	66.50	3.58
MS	0.52	0.03	0.214	0.032	22.38	1.79	67.10	3.82
NHL	0.54	0.04	0.210	0.042	23.34	1.75	66.80	3.43
DM	0.47	0.04	0.202	0.025	22.87	1.84	66.31	3.68
AIDS	0.56	0.05	0.220	0.052	23.45	1.79	66.32	3.63
CJD	0.53	0.06	0.212	0.045	23.17	1.88	68.53	2.65
Autism	0.53	0.08	0.205	0.041	23.20	1.57	66.65	4.26
EMF	0.51	0.05	0.213	0.033	22.29	2.05	61.91	7.56
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
Normal	4.41	0.15	18.63	0.12	4.34	0.15	18.24	0.37
Schizo	22.76	2.20	67.63	3.52	22.79	2.20	64.26	6.02
Seizure	22.28	1.52	64.05	2.79	22.82	1.56	64.61	4.95
AD	23.81	1.90	66.95	3.67	23.12	1.71	65.12	5.58
MS	24.10	1.61	65.78	4.43	22.73	2.46	65.87	4.35
NHL	23.43	1.57	66.30	3.57	22.98	1.50	65.13	4.87
DM	23.70	1.75	68.06	3.52	23.81	1.49	64.89	6.01
AIDS	23.66	1.67	65.97	3.36	23.09	1.81	65.86	4.27
CJD	22.92	2.14	67.54	3.65	21.93	2.29	63.70	5.63
Autism	21.88	1.19	66.28	3.60	23.02	1.65	67.61	2.77
EMF	22.29	1.33	65.38	3.62	22.13	2.14	66.26	3.93
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
Normal	4.34	0.21	18.43	0.82	4.21	0.16	18.56	0.76
Schizo	20.99	1.46	61.23	9.73	23.01	2.61	65.87	5.27
Seizure	20.94	1.54	62.76	8.52	23.33	1.79	62.50	5.56
AD	22.63	0.88	56.40	8.59	22.96	2.12	65.11	5.91
MS	21.59	1.23	60.28	9.22	22.81	1.91	63.47	5.81
NHL	21.19	1.61	58.57	7.47	22.53	2.41	64.29	5.44
DM	20.67	1.38	58.75	8.12	23.23	1.88	65.11	5.14
AIDS	21.21	2.36	58.73	8.10	21.11	2.25	64.20	5.38
CJD	21.07	1.79	63.90	7.13	22.47	2.17	65.97	4.62
Autism	21.91	1.71	58.45	6.66	22.88	1.87	65.45	5.08
EMF	22.29	2.05	62.37	5.05	21.66	1.94	67.03	5.97
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**
Normal	4.43	0.19	18.13	0.63	4.40	0.10	18.48	0.39
Schizo	22.50	1.66	60.21	7.42	22.52	1.90	66.39	4.20
Seizure	23.81	1.19	61.08	7.38	22.83	1.90	67.23	3.45
AD	22.65	2.48	60.19	6.98	23.67	1.68	66.50	3.58
MS	21.14	1.20	60.53	4.70	22.38	1.79	67.10	3.82
NHL	23.35	1.76	59.17	3.33	23.34	1.75	66.80	3.43
DM	23.27	1.53	58.91	6.09	22.87	1.84	66.31	3.68
AIDS	23.32	1.71	63.15	7.62	23.45	1.79	66.32	3.63
CJD	22.86	1.91	63.66	6.88	23.17	1.88	68.53	2.65
Autism	23.52	1.49	63.24	7.36	23.20	1.57	66.65	4.26
EMF	23.29	1.67	60.52	5.38	22.29	2.05	61.91	7.56
F value	380.721		171.228		372.716		556.411	
P value	< 0.001		P < 0.001		< 0.001		< 0.001	

Section 2: Patient Study

Table 1

Group	RBC Digoxin (ng/ml RBC Susp)		Cytochrome F420		RNA viroid (ug/ml)		H_2O_2 (umol/ml RBC)	
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD
NO/BHCD	0.58	0.07	1.00	0.00	17.75	0.72	177.43	6.71
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
Seizure	1.23	0.26	4.00	0.00	50.04	3.91	278.90	11.20
HD	1.34	0.31	4.00	0.00	51.16	7.78	295.37	3.78
AD	1.10	0.08	4.00	0.00	51.56	3.69	277.47	10.90
MS	1.21	0.21	4.00	0.00	47.90	6.99	280.89	11.25
SLE	1.50	0.33	4.00	0.00	48.20	5.53	278.59	11.51
NHL	1.26	0.23	4.00	0.00	51.08	5.24	283.39	10.67
Glio	1.27	0.24	4.00	0.00	51.57	2.66	278.19	12.80
DM	1.35	0.26	4.00	0.00	51.98	5.05	280.89	10.58
CAD	1.22	0.16	4.00	0.00	50.00	5.91	280.89	13.79
CVA	1.33	0.27	4.00	0.00	51.06	4.83	287.33	9.47
AIDS	1.31	0.24	4.00	0.00	50.15	6.96	278.58	12.72
CJD	1.48	0.27	4.00	0.00	49.85	6.40	286.16	10.90
Autism	1.19	0.24	4.00	0.00	52.87	7.04	274.52	9.29
DS	1.34	0.25	4.00	0.00	47.28	3.55	283.04	9.17
Cerebral Palsy	1.44	0.19	4.00	0.00	53.49	4.15	273.70	12.37
CRF	1.26	0.26	4.00	0.00	49.39	5.51	285.51	8.79
Cirr/Hep Fail	1.50	0.20	4.00	0.00	46.82	4.73	275.97	10.66
Muc Angio	1.40	0.32	4.00	0.00	46.37	4.87	290.37	9.10
EMF	1.51	0.29	4.00	0.00	47.47	4.34	287.49	9.81
CCP	1.35	0.22	4.00	0.00	48.54	5.97	277.50	7.51
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**
NO/BHCD	0.012	0.001	17.94	0.59	15.44	0.50	20.82	1.19
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
Seizure	0.038	0.007	79.28	4.55	68.28	6.02	46.54	4.55
HD	0.035	0.011	82.13	3.97	67.30	5.98	47.25	4.19
AD	0.036	0.007	79.65	5.57	67.32	5.40	49.83	3.45
MS	0.034	0.009	80.18	5.67	64.00	7.33	46.85	3.49
SLE	0.038	0.008	81.03	6.22	65.01	5.42	48.55	3.81
NHL	0.041	0.006	77.98	5.68	63.21	6.55	47.17	4.86
Glio	0.038	0.007	79.18	5.88	67.67	5.69	46.84	4.43
DM	0.041	0.005	78.36	6.68	64.72	6.81	48.15	3.36
CAD	0.038	0.009	78.15	3.72	66.66	7.77	47.00	3.81
CVA	0.037	0.007	77.59	5.24	69.02	4.86	46.33	4.01
AIDS	0.039	0.010	79.17	5.88	67.78	4.41	48.03	3.64
CJD	0.039	0.006	80.41	5.70	66.99	3.71	47.94	5.33
Autism	0.036	0.006	76.71	5.25	68.16	4.92	42.04	2.38
DS-50	0.035	0.009	80.30	6.65	64.99	6.72	45.69	4.18
Cerebral Palsy	0.038	0.008	80.02	6.82	65.56	6.28	44.58	4.52
CRF	0.039	0.008	81.36	5.37	67.61	5.55	46.81	4.62
Cirr/Hep Fail	0.037	0.010	77.61	4.42	66.28	6.55	48.23	2.36
Muc Angio	0.039	0.010	79.38	5.14	67.86	5.65	44.08	2.81
EMF	0.035	0.008	80.04	4.69	64.76	5.23	44.82	3.46
CCP	0.040	0.006	80.34	4.73	66.68	4.14	48.70	3.35
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 (nmol24)		Coproporphyrin (nmol/24)		Protoporphyrin (Ab unit)		Heme (uM)	
	Mean	**±SD**	**Mean**	**±SD**	**Mean**	**±SD**	**Mean**	**±SD**
NO/BHCD	50.18	3.54	137.94	4.75	10.35	0.38	30.27	0.81
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
Seizure	290.44	57.65	436.71	52.95	49.59	1.70	13.03	0.70
HD	286.84	24.18	432.22	50.11	49.36	4.18	11.81	0.80
AD	259.61	33.18	433.17	45.61	49.68	3.30	12.09	1.12
MS	277.36	15.48	440.35	25.34	50.81	3.21	11.87	1.84
SLE	294.51	58.62	447.39	39.84	52.94	3.67	12.95	1.53
NHL	310.25	40.44	495.98	39.11	54.80	4.04	11.76	1.37
Glio	304.19	14.16	479.35	58.86	53.73	5.34	13.68	1.67
DM	285.46	29.46	422.27	33.86	49.80	4.01	12.83	2.07
CAD	314.01	17.82	426.14	24.28	49.51	2.27	11.39	1.10
CVA	320.85	24.73	402.16	33.80	46.74	4.28	11.26	0.95
AIDS	306.61	22.47	429.72	24.97	49.32	5.13	11.60	1.23
CJD	317.92	29.63	429.24	18.29	50.02	4.58	11.76	1.32
Autism	318.84	82.90	423.29	47.57	47.50	2.87	12.37	2.09
DS-50	258.33	37.85	421.52	36.57	50.97	7.07	11.81	1.14
Cerbral Palsy	280.16	26.14	431.39	28.88	49.23	3.91	11.61	1.36
CRF	301.78	48.22	427.57	33.55	49.66	4.41	12.03	1.40
Cirr/Hep Fail	276.51	16.66	436.44	25.65	50.56	1.63	11.92	1.33
Muc Angio	303.86	13.91	441.58	25.51	47.86	3.34	12.13	1.10
EMF	300.90	31.96	443.22	38.14	51.37	4.86	12.61	2.00
CCP	287.09	15.63	442.85	49.61	50.36	3.49	12.01	1.53
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**
NO/BHCD	0.55	0.02	0.030	0.001	0.36	0.13	0.42	0.11
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
Seizure	1.84	0.07	0.070	0.015	3.09	0.65	1.66	0.56
HD	1.83	0.09	0.071	0.014	3.34	0.84	1.27	0.26
AD	1.77	0.13	0.073	0.016	3.34	0.75	2.06	0.19
MS	1.81	0.10	0.079	0.007	3.05	0.52	1.63	0.26
SLE	1.82	0.08	0.061	0.006	2.85	0.34	1.59	0.22
NHL	1.84	0.08	0.077	0.011	3.01	0.55	1.73	0.26
Glio	1.76	0.11	0.073	0.012	2.70	0.62	1.48	0.32
DM	1.77	0.19	0.067	0.014	3.19	0.89	1.97	0.11
CAD	1.75	0.12	0.080	0.007	2.99	0.65	1.57	0.37
CVA	1.82	0.10	0.079	0.009	2.98	0.78	1.49	0.27
AIDS	1.79	0.08	0.072	0.013	3.29	0.63	1.59	0.38
CJD	1.82	0.09	0.066	0.009	3.21	0.95	1.69	0.43
Autism	1.83	0.16	0.072	0.014	2.67	0.80	2.03	0.12
DS-50	1.85	0.07	0.071	0.015	3.15	0.73	1.17	0.11
Cerebral Palsy	1.85	0.09	0.069	0.012	3.14	0.46	1.56	0.39
CRF	1.76	0.22	0.070	0.012	3.14	0.57	1.53	0.33
Cirr/Hep Fail	1.81	0.10	0.076	0.009	3.01	0.47	1.32	0.26
Muc Angio	1.78	0.24	0.067	0.014	2.92	0.55	1.35	0.29
EMF	1.79	0.07	0.074	0.009	3.12	0.60	1.56	0.48
CCP	1.84	0.07	0.073	0.011	3.15	0.46	1.51	0.38
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**
NO/BHCD	2.79	0.28	7.38	0.31	40.51	1.42	1.66	0.45
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
Seizure	12.06	1.09	21.78	0.58	90.46	8.30	6.29	1.73
HD	12.65	1.06	24.28	1.69	95.44	12.04	9.30	3.98
AD	11.94	0.86	22.04	0.64	97.26	8.26	8.46	3.63
MS	11.81	0.67	23.32	1.10	102.48	13.20	8.56	4.75
SLE	11.73	0.56	23.06	1.49	100.51	9.79	8.02	3.01
NHL	11.91	0.49	22.83	1.24	95.81	12.18	7.41	4.22
Glio	13.00	0.42	22.20	0.85	96.58	8.75	7.82	3.51
DM	12.95	0.56	25.56	7.93	96.30	10.33	7.05	1.86
CAD	11.51	0.47	22.83	0.82	97.29	12.45	8.88	3.09
CVA	12.74	0.80	23.03	1.26	103.25	9.49	7.87	2.72
AIDS	12.29	0.89	24.87	4.14	95.55	7.20	9.84	2.43
CJD	12.19	1.22	23.02	1.61	96.50	5.93	8.81	4.26
Autism	12.48	0.79	21.95	0.65	92.71	8.43	6.95	2.02
DS-50	12.79	1.15	23.69	2.19	91.81	4.12	8.68	2.60
Cerebral Palsy	12.14	1.30	23.12	1.81	95.33	11.78	7.92	3.32
CRF	12.66	1.01	23.42	1.20	97.38	10.76	7.75	3.08
Cirr/Hep Fail	12.81	0.90	26.20	5.29	97.77	13.24	8.99	3.27
Muc Angio	12.84	0.74	23.64	1.43	96.19	12.15	10.12	1.75
EMF	12.72	0.92	25.35	5.52	103.32	13.04	9.44	3.40
CCP	12.23	0.94	23.66	1.64	94.36	8.06	8.53	2.64
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**
NO/BHCD	8.75	0.38	75.11	2.96	0.65	0.03
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
Seizure	2.15	0.22	33.27	5.99	3.67	0.38
HD	1.95	0.06	35.02	5.85	3.14	0.32
AD	2.19	0.15	42.84	8.26	3.53	0.39
MS	2.03	0.09	39.99	12.61	3.58	0.36
SLE	2.54	0.38	49.30	7.26	3.37	0.38
NHL	2.30	0.26	50.58	3.82	3.48	0.46
Glio	2.34	0.43	42.51	11.58	3.28	0.39
DM	2.17	0.40	41.31	10.69	3.53	0.44
CAD	2.37	0.44	49.19	6.86	3.61	0.28
CVA	2.25	0.44	37.45	7.93	3.31	0.43
AIDS	2.11	0.19	38.40	7.74	3.45	0.49
CJD	2.10	0.27	34.97	4.24	3.94	0.22
Autism	2.42	0.41	50.61	6.32	3.30	0.32
DS-50	2.01	0.08	39.34	8.15	3.30	0.48
Cerebral Palsy	2.06	0.35	40.79	9.34	3.24	0.34
CRF	2.24	0.32	37.52	4.37	3.26	0.43
Cirr/Hep Fail	2.13	0.17	46.20	4.95	3.25	0.40
Muc Angio	2.51	0.42	45.51	7.56	3.11	0.36
EMF	2.19	0.19	42.48	8.62	3.27	0.39
CCP	2.04	0.10	37.95	8.82	3.33	0.25
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**
NO/BHCD	50.60	1.42	1.70	0.07	79.99	3.36
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
Seizure	95.61	7.88	1.14	0.07	22.98	5.19
HD	94.60	8.52	1.08	0.13	28.93	4.93
AD	95.37	4.66	1.10	0.07	26.26	7.34
MS	93.42	3.69	1.13	0.08	24.12	6.43
SLE	101.18	17.06	1.14	0.07	19.62	1.97
NHL	91.62	3.24	1.12	0.10	23.45	5.01
Glio	93.20	4.46	1.10	0.09	23.43	6.03
DM	93.38	7.76	1.09	0.12	22.77	4.94
CAD	93.93	4.86	1.07	0.12	24.55	6.26
CVA	103.18	27.27	1.05	0.09	22.39	3.35
AIDS	92.47	3.97	1.08	0.11	23.28	5.81
CJD	93.13	5.79	1.09	0.12	21.26	4.81
Autism	94.01	5.00	1.12	0.06	23.16	5.78
DS-50	98.81	15.65	1.09	0.11	21.31	4.49
Cerebral Palsy	92.09	3.21	1.07	0.09	22.80	5.02
CRF	98.76	11.12	1.03	0.10	26.47	5.30
Cirr/Hep Fail	94.77	2.86	1.04	0.10	24.91	5.06
Muc Angio	92.40	4.34	1.12	0.08	24.37	4.38
EMF	95.37	5.76	1.08	0.08	25.17	3.80
CCP	93.42	5.34	1.01	0.09	23.87	4.00
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

NO/BHCD: Normal/Bi-hemispheric chemical dominance

RHCD: Right hemispheric chemical dominance

LHCD: Left hemispheric chemical dominance

Schizo: Schizophrenia

HD: Huntington's disease

AD: Alzheimer's disease

MS: Multiple sclerosis

SLE: Systemic lupus erythematosis

NHL: Non-Hodgkin's lymphoma

Glio - Glioma

DM: Diabetes mellitus

CAD: Coronary artery disease

CVA: Cerebrovascular accident

AIDS: Acquired immunodeficiency syndrome

CJD: Creutzfeldt Jakob's disease

DS: Down syndrome

CRF: Chronic renal failure

Cirr/Hep Fail - Cirrhosis/Hepatic failure

EMF: Endomyocardial fibrosis

CCP: Chronic calcific pancreatitis

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] There was also an increase in archaeal HMG CoA reductase activity indicating increased cholesterol synthesis by the archaeal mevalonate pathway. 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 archaeal aromatization of cholesterol generating PAH, serotonin and dopamine was also detected.[17] The archaeal glycolytic hexokinase activity and archaeal extracellular ATP synthase activity were increased. The archaea can undergo magnetite and calcium carbonate mineralization and can exist as calcified nanoforms.[13]

The actinidic archaea and viroids induce porphyrinogenesis. Porphyrins serve as a template for formation of nucleic acids, protein and isoprenoidal moieties which get organized to form nanoarchaea and viroids. The porphyrin templates mediate viroidal and archaeal abiogenesis and replication. The archaeal synthesized PAH can also serve as a template for self replication of macromolecules and nanoarchaeal/viroidal abiogenesis. Porphyrin and PAH templates mediate actinidic archaeal and viroidal abiogenesis and replication. They are indicative of primitive porphyrin organism and PAH organism as original life forms on earth. Abiogenesis is a replicative mechanism for primitive organisms. The nanoarchaea and viroids may replicate by abiogenesis on porphyrin and PAH templates.

The metal actinides provide radiolytic energy, catalysis for oligomer formation and provide a coordinating ion for metalloenzymes all important in abiogenesis.[6] The metal actinide surfaces would by surface metabolism generate porphyrins from simple compounds like succinic acid and glycine. Polycyclic aromatic hydrocarbons can also be generated by actinidic surface metabolism. Porphyrins and polycyclic aromatic hydrocarbons can exist as wave forms and particulate forms and can bridge the dividing line between the quantal world and particulate world. Porphyrin and polycyclic aromatic hydrocarbons molecules can self organize into organisms with capacities for energy transduction, ATP synthesis, information storage and replicating ability. A self

replicating porphyrin or polycyclic aromatic hydrocarbons microorganism may have played a role in the origin of life. Porphyrins and polycyclic aromatic hydrocarbons can form templates on which macromolecules like polysaccharides, protein and nucleic acids can form. The macromolecules generated on actinidic porphyrins and polycyclic aromatic hydrocarbons templates would have contributed to the actinidic nanoarchaea/viroids and the original organisms on earth. The data supports the persistence of an actinidic archaeal and viroidal shadow biosphere which throws light on the actinide based origin of life and porphyrins/polycyclic aromatic hydrocarbons as the premier prebiotic molecule.[17, 18]

Porphyrins and polycyclic aromatic hydrocarbons play an important role in the genesis of the biological universe. The porphyrin and polycyclic aromatic hydrocarbons macroarrays can form in the interstellar space on its own as porphyrins can exist both as particles and waves. Porphyrins and polycyclic aromatic hydrocarbons form the bridging connection between the quantal world and the particulate world. The self generated porphyrins and polycyclic aromatic hydrocarbons from the quantal foam can self organize to form macroarrays, can store information and self replicate. This can be called as an abiotic porphyrin and polycyclic aromatic hydrocarbons organism. The porphyrin and polycyclic aromatic hydrocarbons template would have generated nucleic acids, proteins, polysaccharides and isoprenoids. This would have generated actinidic nanoarchaea/viroids in the interstellar space. The porphyrins and polycyclic aromatic hydrocarbons have magnetic properties and the interstellar porphyrin/polycyclic aromatic hydrocarbons organism can contribute to the interstellar grains and interstellar magnetic fields.[37] The cosmic dust grains of porphyrin/ polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organism occupy the intergalactic space and are thought to be formed of magnetotactic bacteria identified according to their spectral

signatures. According to the Hoyle's hypothesis, the cosmic dust magnetotactic porphyrin/polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organism plays a role in the formation of the intergalactic magnetic field. A magnetic field equal in strength to about one millionth part of the magnetic field of earth exists throughout much of our galaxy. The magnetic files can be used to trace the spiral arms of the galaxy following a pattern of field lines that connect young stars and dust in which new stars are formed at a rapid rate. Studies have shown that a fraction of the dust particles have elongated shape similar to bacilli and they are systematically lined up in our galaxy. Moreover the direction of alignment is such that the long axes of the dust tend to be at right angles to the direction of the galactic magnetic field at every point. Magnetotactic porphyrin / polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organism have the property to affect the degree of alignment that is observed. The fact that the magnetotactic porphyrin / polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organisms appear to be connected to the magnetic field lines that thread through the spiral arms of the galaxy connecting one region of star formation to another support a role for them in star formation and in the mass distribution and rotation of stars. The nutrient supply for a population of interstellar porphyrin/polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organisms comes from mass flows out of supernovas populating the galaxy. Giants arising in the evolution of such stars experience a phenomenon in which material containing nitrogen, carbon monoxide, hydrogen, helium, water and trace elements essential for life flows continuously outward into space. The interstellar organisms need liquid water. Water exists only as vapour or solid in the interstellar space and only through star formation leading to associated planets and cometary bodies can there be access to liquid water. To control conditions leading to star formation is of paramount importance in cosmic biology. The rate of star formation is controlled by two

factors: Too high a rate of star formation produces a destructive effect of UV radiation and destroys cosmic biology. Star formation as stated before produces water crucial for organism growth. Cosmic biology of magnetotactic organisms and star formation are thus closely interlinked. Systems like solar systems do not arise in random condensation of blobs of interstellar gas. Only by a rigorous control of rotation of various parts of the system would galaxies and solar system evolved. The key to maintaining control over rotation seems to lie in the intergalactic magnetic field as indeed the whole phenomena of star formation. The intergalactic magnetic fields owes its origin to the lining up of magnetotactic porphyrin/polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organisms and the cosmic biology of interstellar organisms can prosper only by maintaining a firm grip on the interstellar magnetic field and hence on the rate of star formation and type of star system produced. This point to a cosmic intelligence or brain capable of computation, analysis and exploration of the universe at large - of magnetotactic porphyrin/polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organism networks. The origin of life on earth according to the Hoyle's hypothesis would be by seeding of porphyrin/polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organism from the outer intergalactic space. The porphyrin/polycyclic aromatic hydrocarbon organism can also be generated on actinidic surfaces in earth. Comets carrying porphyrin/ polycyclic aromatic hydrocarbons organisms would have interacted with the earth. A thin skin of graphitized material around a single porphyrin/polycyclic aromatic hydrocarbons macroarrays and nanoarchaeal organism or clumps of organism can shield the interior from destruction by UV light. The sudden surge and diversification of species of plants and animals and their equally sudden extinction has seen from fossil records point to sporadic evolution produced by induction of fresh cometary genes with the arrival of each major new crop of comets.[38, 39] The porphyrin /

polycyclic aromatic hydrocarbon macroarrays organism can have a wave particle existence and bridge the world of bosons and fermions. The porphyrin/polycyclic aromatic hydrocarbon macroarrays and nanoarchaeal organism can form biofilms and the porphyrin organism can form a molecular quantum computing cloud in the biofilm which forms an interstellar intelligence regulating the formation of star systems and galaxies. The porphyrin/polycyclic aromatic hydrocarbon macroarrays and nanoarchaeal organism quantal computing cloud can bridge the wave particle world functioning as the anthropic observer sensing gravity which orchestrates the reduction of the quantal world of possibilities in to the macroscopic world. The actinide based porphyrin/polycyclic aromatic hydrocarbon macroarrays and nanoarchaeal organism regulates the human system and biological universe.[19-21]

The porphyrins can modulate cell functions and produce neuro-immuno-endocrine integration. The porphyrin/PAH can undergo photo-oxidation and auto-oxidation generating free radicals. The archaeal porphyrin/PAH 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 porphyrin/PAH can complex and intercalate with the cell membrane producing sodium potassium ATPase inhibition adding on to digoxin mediated inhibition. Porphyrin/PAH can complex with proteins and nucleic acid producing biophoton emission. Porphyrin/PAH complexing with proteins can modulate protein structure and function. Porphyrin/PAH 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/PAH 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 porphyrin/PAH 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 viroids can self replicate on porphyrin/PAH templates. The actinides, PAH 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 noncoding DNA using HERV integrase as has been described for borna and ebola viruses. The archaea and viroids can also induce cellular porphyrin/PAH synthesis. Bacterial and viral infections can precipitate porphyria. Thus porphyrin/PAH can regulate genomic function. The increased expression of HERV RNA can result in acquired immunodeficiency syndrome, autoimmune disease, neuronal degenerations, schizophrenia and malignancy.[14, 15]

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/PAH 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. 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. Porphyrin/PAH can combine with membranes modulating membrane function. Porphyrin/PAH can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. Porphyrin/PAH can complex with DNA and RNA modulating their function. Porphyrin/PAH 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, antiinflammatory and antiproliferative effects. Heme is also involved in the stress response. Heme deficiency leads to metabolic syndrome, immune disease, degenerations and cancer.[3-5]

The archaea and viroids can regulate the nervous system including the NMDA/GABA thalamo-cortico-thalamic pathway mediating conscious perception. Porphyrin photooxidation 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, 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 photooxidation 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, vasospasm 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 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 photooxidation 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 porphyrin and PAH 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. Porphyrin/PAH 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/PAH complexed with proteins and nucleic acids are antigenic and can lead onto autoimmune disease.[3, 4] The porphyrin/PAH 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.[3, 4] The

porphyrin/PAH 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.[3, 4] The porphyrin/PAH can combine with prion proteins modulating their conformation. This leads to abnormal prion protein conformation and degradation. Archaeal porphyrin/PAH can contribute to prion disease. The porphyrin/PAH can intercalate with DNA producing HERV expression. The HERV particles generated can contribute to the retroviral state. The porphyrin/PAH in the blood can combine with bacteria and viruses and the photooxidation generated free radicals can kill them. The archaeal porphyrin/PAH can modulate bacterial and viral infections. The archaeal porphyrins are regulatory molecules keeping other prokaryotes and viruses on check.[3, 4] 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/PAH is crucial in the pathogenesis of these disorders. Porphyrins may serve as regulatory molecules modulating immune, neural, endocrine, metabolic and genetic systems. The porphyrin/PAH photooxidation 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]

Porphyrin and PAH have contributed to the primate and human evolution. 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 and PAH 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 noncoding region of the DNA. The increase in noncoding region of the DNA is involved in primate and human evolution. Thus, increased rates of porphyrin and PAH synthesis would correlate with increase in non coding DNA length. The alteration in the length of the noncoding 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 noncoding region as well as increased porphyrin/PAH synthesis leads to increased cognitive and creative neuronal function. Porphyrins/PAH are involved in quantal perception and regulation of the thalamo-cortico-thalamic 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.[3, 4]

An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. The actinidic archaea and viroids induce porphyrinogenesis. Porphyrins serve as a template for formation of nucleic acids, protein and isoprenoidal moieties which get organized to form nanoarchaea and viroids. The porphyrin templates mediate viroidal and archaeal abiogenesis and replication. The archaeal synthesized PAH can also serve as a template for self replication of macromolecules and nanoarchaeal/viroidal abiogenesis. 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 serve as regulatory

molecules modulating immune, neural, endocrine, metabolic and genetic systems. The porphyrins photooxidation generated free radicals can produce benzodiazepine receptor modulation, immune activation, produce cell death, activate cell proliferation, produce insulin resistance, modulate conscious/quantal perception and mediate hemispheric dominance. The role of porphyrins and PAH in abiogenesis and origin of life as well as biological universe is discussed. Porphyrin and PAH templates mediate actinidic archaeal and viroidal abiogenesis and replication. They are indicative of primitive porphyrin organism and PAH organism as original life forms on earth.

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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