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The Porphyrins, Origin of Life in Biological Universe and Evolution/Regulation of the Human System

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Abstract

Objectives: Actinidic archaea 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. The role of archaeal porphyrins in regulation of cell functions and neuroimmunoendocrine integration is discussed. Porphyrins are prebiotic molecules which are involved in abiogenesis and origin of life.

Methodology: 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. The following estimations were carried out: Cytochrome F420, free RNA, free DNA, polycyclic aromatic hydrocarbon, hydrogen peroxide, pyruvate, ammonia, glutamate, succinate, glycine, delta aminolevulinic acid and digoxin. The study also involved estimating the following parameters in the patient population- Hexokinase, porphyrins, pyruvate, glutamate, ammonia, succinic acid, serine, glycine, HMG CoA reductase, cytochrome C, blood ATP and heme oxygenase.

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 and rutile to the patient's plasma produced the same changes but the extent of change was more in patient's sera as compared to controls. 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 had increased heme oxygenase activity, increased serine, glycine, succinic acid and porphyrins. The hexokinase activity was high. The pyruvate, glutamate, ammonia, GABA and succinic acid levels were elevated indicating blockade of PDH activity, and operation of the GABA shunt pathway. 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. The HMG CoA reductase activity was high indicating cholesterol synthesis.The RHCD population had values similar to the patient population. The LHCD population had opposite values.

Conclusion: An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. The archaeal porphyrins can contribute to the pathgenesis 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 photo-oxidation generated free radicals can produce immune activation, produce cell death, activate cell proliferation, produce insulin resistance and modulate conscious/quantal perception. Porphyrins can regulate hemispheric dominance. The archaeal porphyrins functions as key regulatory molecules with mitochondrial benzodiazepine receptors playing an important role. The role of porphyrins in abiogenesis and origin of life as well as biological universe is discussed.

Key words: Actinide; Archaea; Porphyrins; GABA shunt; Peripheral Benzodiazepine receptor; Delta aminolevulinic acid

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INTRODUCTION

Actinidic archaea have been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, auto-immune disease and neuronal degeneration. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states are described. Actinidic archaea have a mevalonate pathway and are cholesterol catabolizing[1-5]. 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. The role of archaeal porphyrins in regulation of cell functions and neuroimmunoendocrine integration is discussed. Porphyrins are prebiotic molecules which are involved in abiogenesis and origin of life[1-5].

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 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. 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^{o}C 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), H2O2 (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 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 was 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.

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 P value < 0.001		F value 130.054 P value < 0.001		F value 391.318 P value < 0.001		F value 257.996 P value < 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 P value < 0.001		F value 356.621 P value < 0.001		F value 427.828 P value < 0.001		F value 654.453 P value < 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 P value < 0.001		F value 71.706 P value < 0.001		F value 372.716 P value < 0.001		F value 556.411 P value < 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 P value < 0.001		F value 680.284 P value < 0.001		F value 348.867 P value < 0.001		F value 364.999 P value < 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 P value < 0.001		F value 115.242 P value < 0.001		F value 292.065 P value < 0.001		F value 317.966 P value < 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 P value < 0.001		F value 171.228 P value < 0.001		F value 372.716 P value < 0.001		F value 556.411 P value < 0.001	

Section 2: Patient Study

Table 1
RBC Dig (ng/ml RBC Susp)

Group	Mean	± SD
NO/BHCD	0.58	0.07
RHCD	1.41	0.23
LHCD	0.18	0.05
Schizo	1.38	0.26
Seizure	1.23	0.26
HD	1.34	0.31
AD	1.10	0.08
MS	1.21	0.21
SLE	1.50	0.33
NHL	1.26	0.23
Glio	1.27	0.24
DM	1.35	0.26
CAD	1.22	0.16
CVA	1.33	0.27
AIDS	1.31	0.24
CJD	1.48	0.27
Autism	1.19	0.24
DS	1.34	0.25
Cerbral Palsy	1.44	0.19
CRF	1.26	0.26
Cirr/Hep Fail	1.50	0.20
Muc Angio	1.40	0.32
EMF	1.51	0.29
CCP	1.35	0.22
Exposure to EMF	1.41	0.30
F value	60.288	
P value	< 0.001	

Table 2
Cytochrome F 420

Group	Mean	± SD
NO/BHCD	1.00	0.00
RHCD	4.00	0.00
LHCD	0.00	0.00
Schizo	4.00	0.00
Seizure	4.00	0.00
HD	4.00	0.00
AD	4.00	0.00
MS	4.00	0.00
SLE	4.00	0.00
NHL	4.00	0.00
Glio	4.00	0.00
DM	4.00	0.00

To be continued

Continued

Group	Mean	± SD
CAD	4.00	0.00
CVA	4.00	0.00
AIDS	4.00	0.00
CJD	4.00	0.00
Autism	4.00	0.00
DS	4.00	0.00
Cerbral Palsy	4.00	0.00
CRF	4.00	0.00
Cirr/Hep Fail	4.00	0.00
Muc Angio	4.00	0.00
EMF	4.00	0.00
CCP	4.00	0.00
Exposure to EMF	4.00	0.00
F value	0.001	
P value	< 0.001	

Table 3
ERV RNA (ug/ml)

Group	Mean	± SD
NO/BHCD	17.75	0.72
RHCD	55.17	5.85
LHCD	8.70	0.90
Schizo	51.17	3.65
Seizure	50.04	3.91
HD	51.16	7.78
AD	51.56	3.69
MS	47.90	6.99
SLE	48.20	5.53
NHL	51.08	5.24
Glio	51.57	2.66
DM	51.98	5.05
CAD	50.00	5.91
CVA	51.06	4.83
AIDS	50.15	6.96
CJD	49.85	6.40
Autism	52.87	7.04
DS-50	47.28	3.55
Cerbral Palsy	53.49	4.15
CRF	49.39	5.51
Cirr/Hep Fail	46.82	4.73
Muc Angio	46.37	4.87
EMF	47.47	4.34
CCP	48.54	5.97
Exposure to EMF	51.01	4.77
F value	194.418	
P value	< 0.001	

Table 4
H2O2 (umol/ml RBC)

Group	Mean	± SD
NO/BHCD	177.43	6.71
RHCD	278.29	7.74
LHCD	111.63	5.40
Schizo	274.88	8.73
Seizure	278.90	11.20
HD	295.37	3.78
AD	277.47	10.90
MS	280.89	11.25
SLE	278.59	11.51
NHL	283.39	10.67
Glio	278.19	12.80
DM	280.89	10.58
CAD	280.89	13.79
CVA	287.33	9.47
AIDS	278.58	12.72
CJD	286.16	10.90
Autism	274.52	9.29
DS-50	283.04	9.17
Cerbral Palsy	273.70	12.37
CRF	285.51	8.79
Cirr/Hep Fail	275.97	10.66
Muc Angio	290.37	9.10
EMF	287.49	9.81
CCP	277.50	7.51
Exposure to EMF	276.49	10.92
F value	713.569	
P value	< 0.001	

Table 5
NOx (OD diff/hr/mgpro)

Group	Mean	± SD
NO/BHCD	0.012	0.001
RHCD	0.036	0.008
LHCD	0.007	0.001
Schizo	0.036	0.009
Seizure	0.038	0.007
HD	0.035	0.011
AD	0.036	0.007
MS	0.034	0.009
SLE	0.038	0.008
NHL	0.041	0.006
Glio	0.038	0.007
DM	0.041	0.005
CAD	0.038	0.009

To be continued

Continued

Group	Mean	± SD
CVA	0.037	0.007
AIDS	0.039	0.010
CJD	0.039	0.006
Autism	0.036	0.006
DS-50	0.035	0.009
Cerbral Palsy	0.038	0.008
CRF	0.039	0.008
Cirr/Hep Fail	0.037	0.010
Muc Angio	0.039	0.010
EMF	0.035	0.008
CCP	0.040	0.006
Exposure to EMF	0.038	0.007
F value	44.896	
P value	< 0.001	

Table 6
TNF ALP (pg/ml)

Group	Mean	± SD
NO/BHCD	17.94	0.59
RHCD	78.63	5.08
LHCD	9.29	0.81
Schizo	78.23	7.13
Seizure	79.28	4.55
HD	82.13	3.97
AD	79.65	5.57
MS	80.18	5.67
SLE	81.03	6.22
NHL	77.98	5.68
Glio	79.18	5.88
DM	78.36	6.68
CAD	78.15	3.72
CVA	77.59	5.24
AIDS	79.17	5.88
CJD	80.41	5.70
Autism	76.71	5.25
DS-50	80.30	6.65
Cerbral Palsy	80.02	6.82
CRF	81.36	5.37
Cirr/Hep Fail	77.61	4.42
Muc Angio	79.38	5.14
EMF	80.04	4.69
CCP	80.34	4.73
Exposure to EMF	76.41	5.96
F value	427.654	
P value	< 0.001	

Table 7
ALA (umol24)

Group	Mean	± SD
NO/BHCD	15.44	0.50
RHCD	63.50	6.95
LHCD	3.86	0.26
Schizo	66.16	6.51
Seizure	68.28	6.02
HD	67.30	5.98
AD	67.32	5.40
MS	64.00	7.33
SLE	65.01	5.42
NHL	63.21	6.55
Glio	67.67	5.69
DM	64.72	6.81
CAD	66.66	7.77
CVA	69.02	4.86
AIDS	67.78	4.41
CJD	66.99	3.71
Autism	68.16	4.92
DS-50	64.99	6.72
Cerbral Palsy	65.56	6.28
CRF	67.61	5.55
Cirr/Hep Fail	66.28	6.55
Muc Angio	67.86	5.65
EMF	64.76	5.23
CCP	66.68	4.14
Exposure to EMF	68.41	5.53
F value	295.467	
P value	< 0.001	

Table 8
PBG (umol24)

Group	Mean	± SD
NO/BHCD	20.82	1.19
RHCD	42.20	8.50
LHCD	12.11	1.34
Schizo	42.50	3.23
Seizure	46.54	4.55
HD	47.25	4.19
AD	49.83	3.45
MS	46.85	3.49
SLE	48.55	3.81
NHL	47.17	4.86
Glio	46.84	4.43
DM	48.15	3.36
CAD	47.00	3.81

To be continued

Continued

Group	Mean	± SD
CVA	46.33	4.01
AIDS	48.03	3.64
CJD	47.94	5.33
Autism	42.04	2.38
DS-50	45.69	4.18
Cerbral Palsy	44.58	4.52
CRF	46.81	4.62
Cirr/Hep Fail	48.23	2.36
Muc Angio	44.08	2.81
EMF	44.82	3.46
CCP	48.70	3.35
Exposure to EMF	47.27	3.42
F value	183.296	
P value	< 0.001	

Table 9
URO (nmol24)

Group	Mean	± SD
NO/BHCD	50.18	3.54
RHCD	250.28	23.43
LHCD	9.51	1.19
Schizo	267.81	64.05
Seizure	290.44	57.65
HD	286.84	24.18
AD	259.61	33.18
MS	277.36	15.48
SLE	294.51	58.62
NHL	310.25	40.44
Glio	304.19	14.16
DM	285.46	29.46
CAD	314.01	17.82
CVA	320.85	24.73
AIDS	306.61	22.47
CJD	317.92	29.63
Autism	318.84	82.90
DS-50	258.33	37.85
Cerbral Palsy	280.16	26.14
CRF	301.78	48.22
Cirr/Hep Fail	276.51	16.66
Muc Angio	303.86	13.91
EMF	300.90	31.96
CCP	287.09	15.63
Exposure to EMF	288.21	26.17
F value	160.533	
P value	< 0.001	

Table 10
COPRO (nmol/24)

Group	Mean	± SD
NO/BHCD	137.94	4.75
RHCD	389.01	54.11
LHCD	64.33	13.09
Schizo	401.49	50.73
Seizure	436.71	52.95
HD	432.22	50.11
AD	433.17	45.61
MS	440.35	25.34
SLE	447.39	39.84
NHL	495.98	39.11
Glio	479.35	58.86
DM	422.27	33.86
CAD	426.14	24.28
CVA	402.16	33.80
AIDS	429.72	24.97
CJD	429.24	18.29
Autism	423.29	47.57
DS-50	421.52	36.57
Cerbral Palsy	431.39	28.88
CRF	427.57	33.55
Cirr/Hep Fail	436.44	25.65
Muc Angio	441.58	25.51
EMF	443.22	38.14
CCP	442.85	49.61
Exposure to EMF	444.94	38.89
F value	279.759	
P value	< 0.001	

Table 11
PROTO (Ab unit)

Group	Mean	± SD
NO/BHCD	10.35	0.38
RHCD	42.46	6.36
LHCD	2.64	0.42
Schizo	44.30	2.66
Seizure	49.59	1.70
HD	49.36	4.18
AD	49.68	3.30
MS	50.81	3.21
SLE	52.94	3.67
NHL	54.80	4.04
Glio	53.73	5.34
DM	49.80	4.01
CAD	49.51	2.27

To be continued

Continued

Group	Mean	± SD
CVA	46.74	4.28
AIDS	49.32	5.13
CJD	50.02	4.58
Autism	47.50	2.87
DS-50	50.97	7.07
Cerbral Palsy	49.23	3.91
CRF	49.66	4.41
Cirr/Hep Fail	50.56	1.63
Muc Angio	47.86	3.34
EMF	51.37	4.86
CCP	50.36	3.49
Exposure to EMF	50.59	1.71
F value	424.198	
P value	< 0.001	

Table 12
HEME (uM)

Group	Mean	± SD
NO/BHCD	30.27	0.81
RHCD	12.47	2.82
LHCD	50.55	1.07
Schizo	12.82	2.40
Seizure	13.03	0.70
HD	11.81	0.80
AD	12.09	1.12
MS	11.87	1.84
SLE	12.95	1.53
NHL	11.76	1.37
Glio	13.68	1.67
DM	12.83	2.07
CAD	11.39	1.10
CVA	11.26	0.95
AIDS	11.60	1.23
CJD	11.76	1.32
Autism	12.37	2.09
DS-50	11.81	1.14
Cerbral Palsy	11.61	1.36
CRF	12.03	1.40
Cirr/Hep Fail	11.92	1.33
Muc Angio	12.13	1.10
EMF	12.61	2.00
CCP	12.01	1.53
Exposure to EMF	12.36	1.26
F value	1472.05	
P value	< 0.001	

Table 13
Bilirubin (mg/dl)

Group	Mean	± SD
NO/BHCD	0.55	0.02
RHCD	1.70	0.20
LHCD	0.21	0.00
Schizo	1.74	0.08
Seizure	1.84	0.07
HD	1.83	0.09
AD	1.77	0.13
MS	1.81	0.10
SLE	1.82	0.08
NHL	1.84	0.08
Glio	1.76	0.11
DM	1.77	0.19
CAD	1.75	0.12
CVA	1.82	0.10
AIDS	1.79	0.08
CJD	1.82	0.09
Autism	1.83	0.16
DS-50	1.85	0.07
Cerbral Palsy	1.85	0.09
CRF	1.76	0.22
Cirr/Hep Fail	1.81	0.10
Muc Angio	1.78	0.24
EMF	1.79	0.07
CCP	1.84	0.07
Exposure to EMF	1.75	0.22
F value	370.517	
P value	< 0.001	

Table 14
Biliverdin (Ab unit)

Group	Mean	± SD
NO/BHCD	0.030	0.001
RHCD	0.067	0.011
LHCD	0.017	0.001
Schizo	0.073	0.013
Seizure	0.070	0.015
HD	0.071	0.014
AD	0.073	0.016
MS	0.079	0.007
SLE	0.061	0.006
NHL	0.077	0.011
Glio	0.073	0.012
DM	0.067	0.014
CAD	0.080	0.007

To be continued

Continued

Group	Mean	± SD
CVA	0.079	0.009
AIDS	0.072	0.013
CJD	0.066	0.009
Autism	0.072	0.014
DS-50	0.071	0.015
Cerbral Palsy	0.069	0.012
CRF	0.070	0.012
Cirr/Hep Fail	0.076	0.009
Muc Angio	0.067	0.014
EMF	0.074	0.009
CCP	0.073	0.011
Exposure to EMF	0.073	0.013
F value	59.963	
P value	< 0.001	

Table 15
ATP Synth (umol/gHb)

Group	Mean	± SD
NO/BHCD	0.36	0.13
RHCD	2.73	0.94
LHCD	0.09	0.01
Schizo	2.66	0.58
Seizure	3.09	0.65
HD	3.34	0.84
AD	3.34	0.75
MS	3.05	0.52
SLE	2.85	0.34
NHL	3.01	0.55
Glio	2.70	0.62
DM	3.19	0.89
CAD	2.99	0.65
CVA	2.98	0.78
AIDS	3.29	0.63
CJD	3.21	0.95
Autism	2.67	0.80
DS-50	3.15	0.73
Cerbral Palsy	3.14	0.46
CRF	3.14	0.57
Cirr/Hep Fail	3.01	0.47
Muc Angio	2.92	0.55
EMF	3.12	0.60
CCP	3.15	0.46
Exposure to EMF	3.39	1.03
F value	54.754	
P value	< 0.001	

Table 16
SE ATP (umol/dl)

Group	Mean	± SD
NO/BHCD	0.42	0.11
RHCD	2.24	0.44
LHCD	0.02	0.01
Schizo	1.26	0.19
Seizure	1.66	0.56
HD	1.27	0.26
AD	2.06	0.19
MS	1.63	0.26
SLE	1.59	0.22
NHL	1.73	0.26
Glio	1.48	0.32
DM	1.97	0.11
CAD	1.57	0.37
CVA	1.49	0.27
AIDS	1.59	0.38
CJD	1.69	0.43
Autism	2.03	0.12
DS-50	1.17	0.11
Cerbral Palsy	1.56	0.39
CRF	1.53	0.33
Cirr/Hep Fail	1.32	0.26
Muc Angio	1.35	0.29
EMF	1.56	0.48
CCP	1.51	0.38
Exposure to EMF	1.37	0.27
F value	67.588	
P value	< 0.001	

Table 17
Cyto C (ng/ml)

Group	Mean	± SD
NO/BHCD	2.79	0.28
RHCD	12.39	1.23
LHCD	1.21	0.38
Schizo	11.58	0.90
Seizure	12.06	1.09
HD	12.65	1.06
AD	11.94	0.86
MS	11.81	0.67
SLE	11.73	0.56
NHL	11.91	0.49
Glio	13.00	0.42
DM	12.95	0.56
CAD	11.51	0.47

To be continued

Continued

Group	Mean	± SD
CVA	12.74	0.80
AIDS	12.29	0.89
CJD	12.19	1.22
Autism	12.48	0.79
DS-50	12.79	1.15
Cerbral Palsy	12.14	1.30
CRF	12.66	1.01
Cirr/Hep Fail	12.81	0.90
Muc Angio	12.84	0.74
EMF	12.72	0.92
CCP	12.23	0.94
Exposure to EMF	12.26	1.00
F value	445.772	
P value	< 0.001	

Table 18
Lact (mg/dl)

Group	Mean	± SD
NO/BHCD	7.38	0.31
RHCD	25.99	8.10
LHCD	2.75	0.41
Schizo	22.07	1.06
Seizure	21.78	0.58
HD	24.28	1.69
AD	22.04	0.64
MS	23.32	1.10
SLE	23.06	1.49
NHL	22.83	1.24
Glio	22.20	0.85
DM	25.56	7.93
CAD	22.83	0.82
CVA	23.03	1.26
AIDS	24.87	4.14
CJD	23.02	1.61
Autism	21.95	0.65
DS-50	23.69	2.19
Cerbral Palsy	23.12	1.81
CRF	23.42	1.20
Cirr/Hep Fail	26.20	5.29
Muc Angio	23.64	1.43
EMF	25.35	5.52
CCP	23.66	1.64
Exposure to EMF	23.31	1.46
F value	162.945	
P value	< 0.001	

Table 19
Pyru (umol/l)

Group	Mean	± SD
NO/BHCD	40.51	1.42
RHCD	100.51	12.32
LHCD	23.79	2.51
Schizo	96.54	9.96
Seizure	90.46	8.30
HD	95.44	12.04
AD	97.26	8.26
MS	102.48	13.20
SLE	100.51	9.79
NHL	95.81	12.18
Glio	96.58	8.75
DM	96.30	10.33
CAD	97.29	12.45
CVA	103.25	9.49
AIDS	95.55	7.20
CJD	96.50	5.93
Autism	92.71	8.43
DS-50	91.81	4.12
Cerbral Palsy	95.33	11.78
CRF	97.38	10.76
Cirr/Hep Fail	97.77	13.24
Muc Angio	96.19	12.15
EMF	103.32	13.04
CCP	94.36	8.06
Exposure to EMF	103.28	11.47
F value	154.701	
P value	< 0.001	

Table 20
RBC Hexokinase (ug glu phos/hr/mgpro)

Group	Mean	± SD
NO/BHCD	1.66	0.45
RHCD	5.46	2.83
LHCD	0.68	0.23
Schizo	7.69	3.40
Seizure	6.29	1.73
HD	9.30	3.98
AD	8.46	3.63
MS	8.56	4.75
SLE	8.02	3.01
NHL	7.41	4.22
Glio	7.82	3.51
DM	7.05	1.86
CAD	8.88	3.09

To be continued

Continued

Group	Mean	± SD
CVA	7.87	2.72
AIDS	9.84	2.43
CJD	8.81	4.26
Autism	6.95	2.02
DS-50	8.68	2.60
Cerbral Palsy	7.92	3.32
CRF	7.75	3.08
Cirr/Hep Fail	8.99	3.27
Muc Angio	10.12	1.75
EMF	9.44	3.40
CCP	8.53	2.64
Exposure to EMF	7.58	3.09
F value	18.187	
P value	< 0.001	

Table 21
ACOA (mg/dl)

Group	Mean	± SD
NO/BHCD	8.75	0.38
RHCD	2.51	0.36
LHCD	16.49	0.89
Schizo	2.51	0.57
Seizure	2.15	0.22
HD	1.95	0.06
AD	2.19	0.15
MS	2.03	0.09
SLE	2.54	0.38
NHL	2.30	0.26
Glio	2.34	0.43
DM	2.17	0.40
CAD	2.37	0.44
CVA	2.25	0.44
AIDS	2.11	0.19
CJD	2.10	0.27
Autism	2.42	0.41
DS-50	2.01	0.08
Cerbral Palsy	2.06	0.35
CRF	2.24	0.32
Cirr/Hep Fail	2.13	0.17
Muc Angio	2.51	0.42
EMF	2.19	0.19
CCP	2.04	0.10
Exposure to EMF	2.14	0.19
F value	1871.04	
P value	< 0.001	

Table 22
ACH (ug/ml)

Group	Mean	± SD
NO/BHCD	75.11	2.96
RHCD	38.57	7.03
LHCD	91.98	2.89
Schizo	48.52	6.28
Seizure	33.27	5.99
HD	35.02	5.85
AD	42.84	8.26
MS	39.99	12.61
SLE	49.30	7.26
NHL	50.58	3.82
Glio	42.51	11.58
DM	41.31	10.69
CAD	49.19	6.86
CVA	37.45	7.93
AIDS	38.40	7.74
CJD	34.97	4.24
Autism	50.61	6.32
DS-50	39.34	8.15
Cerbral Palsy	40.79	9.34
CRF	37.52	4.37
Cirr/Hep Fail	46.20	4.95
Muc Angio	45.51	7.56
EMF	42.48	8.62
CCP	37.95	8.82
Exposure to EMF	37.75	7.31
F value	116.901	
P value	< 0.001	

Table 23
Glut (mg/dl)

Group	Mean	± SD
NO/BHCD	0.65	0.03
RHCD	3.19	0.32
LHCD	0.16	0.02
Schizo	3.41	0.41
Seizure	3.67	0.38
HD	3.14	0.32
AD	3.53	0.39
MS	3.58	0.36
SLE	3.37	0.38
NHL	3.48	0.46
Glio	3.28	0.39
DM	3.53	0.44
CAD	3.61	0.28

To be continued

Continued

Group	Mean	± SD
CVA	3.31	0.43
AIDS	3.45	0.49
CJD	3.94	0.22
Autism	3.30	0.32
DS-50	3.30	0.48
Cerbral Palsy	3.24	0.34
CRF	3.26	0.43
Cirr/Hep Fail	3.25	0.40
Muc Angio	3.11	0.36
EMF	3.27	0.39
CCP	3.33	0.25
Exposure to EMF	3.47	0.37
F value	200.702	
P value	< 0.001	

Table 24
Se. Amm.(ug/dl)

Group	Mean	± SD
NO/BHCD	50.60	1.42
RHCD	93.43	4.85
LHCD	23.92	3.38
Schizo	94.72	3.28
Seizure	95.61	7.88
HD	94.60	8.52
AD	95.37	4.66
MS	93.42	3.69
SLE	101.18	17.06
NHL	91.62	3.24
Glio	93.20	4.46
DM	93.38	7.76
CAD	93.93	4.86
CVA	103.18	27.27
AIDS	92.47	3.97
CJD	93.13	5.79
Autism	94.01	5.00
DS-50	98.81	15.65
Cerbral Palsy	92.09	3.21
CRF	98.76	11.12
Cirr/Hep Fail	94.77	2.86
Muc Angio	92.40	4.34
EMF	95.37	5.76
CCP	93.42	5.34
Exposure to EMF	102.62	26.54
F value	61.645	
P value	< 0.001	

Table 25
HMG Co A (HMG CoA/MEV)

Group	Mean	± SD
NO/BHCD	1.70	0.07
RHCD	1.16	0.10
LHCD	2.21	0.39
Schizo	1.11	0.08
Seizure	1.14	0.07
HD	1.08	0.13
AD	1.10	0.07
MS	1.13	0.08
SLE	1.14	0.07
NHL	1.12	0.10
Glio	1.10	0.09
DM	1.09	0.12
CAD	1.07	0.12
CVA	1.05	0.09
AIDS	1.08	0.11
CJD	1.09	0.12
Autism	1.12	0.06
DS-50	1.09	0.11
Cerbral Palsy	1.07	0.09
CRF	1.03	0.10
Cirr/Hep Fail	1.04	0.10
Muc Angio	1.12	0.08
EMF	1.08	0.08
CCP	1.01	0.09
Exposure to EMF	1.00	0.07
F value	159.963	
P value	< 0.001	

Table 26
Bile Acid (mg/ml)

Group	Mean	± SD
NO/BHCD	79.99	3.36
RHCD	25.68	7.04
LHCD	140.40	10.32
Schizo	22.45	5.57
Seizure	22.98	5.19
HD	28.93	4.93
AD	26.26	7.34
MS	24.12	6.43
SLE	19.62	1.97
NHL	23.45	5.01
Glio	23.43	6.03
DM	22.77	4.94
CAD	24.55	6.26

To be continued

Continued

Group	Mean	± SD
CVA	22.39	3.35
AIDS	23.28	5.81
CJD	21.26	4.81
Autism	23.16	5.78
DS-50	21.31	4.49
Cerbral Palsy	22.80	5.02
CRF	26.47	5.30
Cirr/Hep Fail	24.91	5.06
Muc Angio	24.37	4.38
EMF	25.17	3.80
CCP	23.87	4.00
Exposure to EMF	22.58	5.07
F value	635.306	
P value	< 0.001	

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 possibility of Warburg phenotype induced by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism was considered in this paper. The Warburg phenotype results in inhibition of pyruvate dehydrogenase and the TCA cycle. The pyruvate enters the GABA shunt pathway where it is converted to succinyl CoA. The glycolytic pathway is upregulated and the glycolytic metabolite phosphoglycerate is converted to serine and glycine. Glycine and succinyl CoA are the substrates for ALA synthesis. The archaea induces the enzyme heme oxygenase. Heme oxygenase converts heme to bilirubin and biliverdin. This depletes heme from the system and results in upregulation of ALA synthase activity resulting in porphyria. Heme inhibits HIF alpha. The heme depletion results in upregulation of HIF alpha activity and further strengthening of the Warburg phenotype. The porphyrin self oxidation results in redox stress which activates HIF alpha and generates the Warburg phenotype. The Warburg phenotype results in channeling acetyl CoA for cholesterol synthesis as the TCA cycle and mitochondrial oxidative phosphorylation are blocked. The archaea uses cholesterol as an energy substrate. Porphyrin and ALA inhibits sodium potassium ATPase. This increases cholesterol synthesis by acting upon intracellular SREBP. The cholesterol is metabolized to pyruvate and then the GABA shunt pathway for ultimate use in porphyrin synthesis. The porphyrins can self organize and self replicate into macromolecular arrays. The porphyrin arrays behave like an autonomous organism and can have intramolecular electron transport generating ATP. The porphyrin macroarrays can store information and can have quantal perception. The porphyrin macroarrays serves the purpose of archaeal energetics and sensory perception. The Warburg phenotype is associated with malignancy, autoimmune disease and metabolic syndrome x.

The role of archaeal porphyrins in regulation of cell functions and neuroimmunoendocrine 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 neuroimmunoendocrine system. Porphyrins can combine with membranes modulating membrane function. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. Porphyrins can complex with DNA and RNA modulating their function. Porphyrin interpolating with DNA can alter transcription and generate HERV expression. Heme deficiency can also result in disease states. Heme deficiency results in deficiency of heme enzymes. There is deficiency of cytochrome C oxidase and mitochondrial dysfunction. The glutathione peroxidase is dysfunctional and the glutathione system of free radical scavenging does not function. The cytochrome P450 enzymes involved in steroid and bile acid synthesis have reduced activity leading to steroid- cortisol and sex hormones as well as bile acid deficiency states. The heme deficiency results in dysfunction of nitric oxide synthase, heme oxygenase and cysthathione beta synthase resulting in lack of gasotransmitters regulating the vascular system and NMDA receptor- NO, CO and H_2S. Heme has got cytoprotective, neuroprotective, anti-inflammatory and antiproliferative effects. Heme is also involved in the stress response. Heme deficiency leads to metabolic syndrome, immune disease, degenerations and cancer.[3-5]

The porphyrins can undergo photo-oxidation and autooxidation generating free radicals. The archaeal porphyrins can produce free radical injury. Free radicals produce NFKB activation, open the mitochondrial PT pore resulting in cell death, produce oncogene activation, activate NMDA receptor and GAD enzyme regulating neurotransmission and generates the Warburg phenotypes activating glycolysis and inhibiting TCA cycle/oxphos. Porphyrins have been related to schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. The porphyrins can complex and intercalate with the cell membrane producing sodium potassium ATPase inhibition adding on to digoxin mediated inhibition. Porphyrins can complex with proteins and nucleic acid producing biophoton emission. Porphyrins complexing with proteins can modulate protein structure and function. Porphyrins complexing with DNA and RNA can modulate transcription and translation. The porphyrin especially protoporphyrins can bind to peripheral benzodiazepine receptors in the mitochondria and modulate its function, mitochondrial cholesterol transport and steroidogenesis. Peripheral benzodiazepine receptor modulation by protoporphyrins can regulate cell death, cell proliferation, immunity and neural functions. The porphyrin photo-oxidation generates free radicals which can modulate enzyme function. Redox stress modulated enzymes include pyruvate dehydrogenase, nitric oxide synthase, cystathione beta synthase and hemeoxygenase. 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. The increased expression of HERV RNA can result in acquired immunodeficiency syndrome, autoimmune disease, neuronal degenerations, schizophrenia and malignancy[14,15].

The archaea and viroids can regulate the nervous system including the NMDA/GABA thalamocorticothalamic pathway mediating conscious perception. Porphyrin photo-oxidation can generate free radicals which can modulate NMDA transmission. Free radicals can increase NMDA transmission. Free radicals can induce GAD and increase GABA synthesis. ALA blocks GABA transmission and upregulates NMDA. Protoporphyrins bind to GABA receptor and promote GABA transmission. Thus porphyrins can modulate the thalamocorticothalamic pathway of conscious perception. The dipolar porphyrins, PAH and archaeal magnetite in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macrosopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrin molecules have a wave particle existence and can bridge the dividing line between quantal state and particulate state. Thus the porphyrins can mediate conscious and quantal perception. Porphyrins binding to proteins, nucleic acids and cell membranes can produce biophoton emission. Porphyrins by autooxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Cellular porphyrins photo-oxidation are involved in sensing of earth magnetic fields and low level biomagnetic fields. Thus prophyrins can mediate extrasensory perception. The porphyrins can modulate hemispheric dominance. There is increased porphyrin synthesis and 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. Porphyrins by modulating conscious and quantal perception is involved in the pathogenesis of schizophrenia and autism. Protoporphyrins block acetyl choline transmission producing a vagal neuropathy with sympathetic overactivity. Vagal neuropathy results in immune activation, vasospasam and vascular disease. A vagal neuropathy underlines neoplastic and autoimmune processes as well as metabolic syndrome x. Porphyrin induced increased NMDA transmission and free radical injury can contribute to neuronal degeneration. Free radicals can produce mitochondrial PT pore dysfunction. This can lead to cytoC leak and activation of the caspase cascade leading to apoptosis and cell death. Altered porphyrin metabolism has been described in Alzheimer's disease. The increased porphyrin photo-oxidation generated free radicals mediated NMDA transmission can also contribute to epileptogenesis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate brain function and cell death[3,4,16].

The porphyrin photo-oxidation can generate free radicals which can activate NFKB. This can produce immune activation and cytokine mediated injury. The increase in archaeal porphyrins can lead to autoimmune disease like SLE and MS. A hereditary form of MS and SLE related to altered porphyrin metabolism has been described. The protoporphyrins binding to mitochondrial benzodiazepine receptors can modulate immune function. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. Porphyrins can complex with DNA and RNA modulating their structure. Porphyrin complexed with proteins and nucleic acids are antigenic and can lead onto autoimmune disease[3,4]. The porphyrin photo-oxidation 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 photo-oxidation can generate free radicals inducing HIF alpha and producing oncogene activation. Heme deficiency can lead to activation of HIF alpha and oncogenesis. This can lead to oncogenesis. Hepatic porphyrias induced hepatocellular carcinoma. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate cell proliferation[3,4]. 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. The porphyrins can intercalate with DNA producing HERV

expression. The HERV particles generated can contribute to the retroviral state. The porphyrins in the blood can combine with bacteria and viruses and the photo-oxidation generated free radicals can kill them. The archaeal porphyrins can modulate bacterial and viral infections. The archaeal porphyrins are regulatory molecules keeping other prokaryotes and viruses on check[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 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 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. Porphyrins can exist as wave forms and particulate forms and can bridge the dividing line between the quantal world and particulate world. Porphyrin molecules can self organize into organisms with energy transduction, ATP synthesis and information storage with replicating capacity. A self replicating porphyrin microorganism may have played a role in the origin of life. Porphyrins can form templates on which macromolecules like polysaccharides, protein and nucleic acids can form. The macromolecules generated on actinidic porphyrins templates would have contributed to the actinidic nanoarchaea and the original organisms on earth. The data supports the persistence of an actinidic archaeal shadow biosphere which throws light on the actinide based origin of life and porphyrins as the premier prebiotic molecule[17,18].

Porphyrins play an important role in the genesis of the biological universe. The porphyrin macroarrays can form in the interstellar space on its own as porphyrins can exist both as particles and waves. Porphyrins form the bridging connection between the quantal world and the particulate world. The self generated porphyrins from the quantal foam can self organize to form macroarrays, can store information and self replicate. This can be called as an abiotic porphyrin organism. The porphyrin template would have generated nucleic acids, proteins, polysaccarides and isoprenoids. This would have generated actinidic nanoarchaea in the interstellar space. The porphyrins have magnetic properties and the interstellar porphyrin organism can contribute to the interstellar grains and interstellar magnetic fields. The cosmic dust grains of porphyrin macroarrays/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 macroarrays/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 macroarrays/nanoarchaeal organism have the property to affect the degree of alignment that is observed. The fact that the magnetotactic porphyrin macroarrays/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 macroarrays/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 macroarrays/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 macroarrays/nanoarchaeal organism networks. The origin of life on earth according to the Hoyle's hypothesis would be by seeding of porphyrin macroarrays/nanoarchaeal organism from the outer intergalactic space. The porphyrin organism can also be generated on actinidic surfaces in earth. Comets carrying porphyrin organisms would have interacted with the earth. A thin skin of graphitized material around a single porphyrin macroarrays/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. The porphyrin macroarrays organism can have a wave particle existence and bridge the world of bosons and fermions. The porphyrin macroarrays/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 macroarrays/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 macroarrays/nanoarchaeal organism regulates the human system and biological universe[19-21].

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[3,4].

The dipolar porphyrins, PAH and archaeal magnetite in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macrosopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. 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. 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 porphyrin 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 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.

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