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Porphyrins and Quantal Perception – Role of Porphyrins in Environmental Communication/Modulation of Digital Information Storage/Processing System – Low Level of Electromagnetic Fields and Pathogenesis of Neurodegenerations – Alzheimer's Disease, Parkinson's Disease and Motor Neuron Disease

Introduction

Actinidic archaea has been described as endosymbionts in humans. Actinidic archaea have a mevalonate pathway and are cholesterol catabolising. 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. The archaea can induce the enzyme heme oxygenase resulting in depletion of heme and induction of ALA synthase. This can lead to porphyrinogenesis. Low level of electromagnetic fields and geomagnetic fields can induce porphyrin synthesis by inhibiting the enzyme ferrochelatase which has got a ferromagnetic core. Inhibition of ferrochelatase produces deficiency of heme resulting in induction of ALA synthase. Low level of EMF can also induce heme oxygenase depleting heme and inducing ALA synthase. Porphyrins can undergo auto-oxidation generating biophotons and a quantal state. Porphyrin auto-oxidation is modulated by low level of electromagnetic fields and geomagnetic fields. Porphyrin microarrays can function as quantal computers storing information and can serve the purpose of extrasensory perception. Porphyrins can serve as a two way communicating bridge between digital information storage systems generating low level electromagnetic fields and human systems. The low level of EMF produced by digital system enhances porphyrin synthesis and serves the purpose of two way extrasensory perception and communication. The porphyrin quantal computers can in turn by biophoton emission modulate digital information storage system. 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. Porphyrins have been related to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Porphyrins can mediate the pathogenesis of low level electromagnetic fields inducing the above mentioned disease states. A hypothesis regarding the role of porphyrins and quantal perception as well as the role of porphyrins in environmental communication/modulation of digital information storage/processing system is presented. The relationship between low level of electromagnetic fields and neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease is highlighted[1-5].

Materials and Methods

The following groups were included in the study: – neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. There were 10 patients in each group and each patient had an age and sex matched healthy control selected randomly from the general population. There were also 10 normal people with right hemispheric dominance, left hemispheric dominance and bi-hemispheric dominance included in the study selected from the normal 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 and those with exposure to low level of EMF 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 and those with exposure to low level of EMF 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 section 1: 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 and those with exposure to low level of EMF 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, those with exposure to low level of EMF 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 cyto C 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 level 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.

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
PD	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
MND	22.06	1.61	57.81	6.04	23.46	1.91	61.56	4.61
Low level 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
PD	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
MND	23.30	1.42	65.07	4.95	23.11	1.52	66.68	3.97
Low level 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
PD	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
MND	0.53	0.06	0.212	0.045	23.17	1.88	68.53	2.65
Low level 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
PD	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
MND	22.92	2.14	67.54	3.65	21.93	2.29	63.70	5.63
Low level 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
PD	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
MND	21.07	1.79	63.90	7.13	22.47	2.17	65.97	4.62
Low level 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
PD	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
MND	22.86	1.91	63.66	6.88	23.17	1.88	68.53	2.65
Low level 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		< 0.001		< 0.001		< 0.001	

Section 2: Patient Study

Table 1

Group	RBC digoxin (ng/ml RBC Susp)		Cytochrome F 420		HERV RNA (ug/ml)		H_2O_2 (umol/ml RBC)		NOX (OD diff/hr/mgpro)	
	Mean	± SD	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	0.012	0.001
RHCD	1.41	0.23	4.00	0.00	55.17	5.85	278.29	7.74	0.036	0.008
LHCD	0.18	0.05	0.00	0.00	8.70	0.90	111.63	5.40	0.007	0.001
MND	1.23	0.26	4.00	0.00	50.04	3.91	278.90	11.20	0.038	0.007
PD	1.34	0.31	4.00	0.00	51.16	7.78	295.37	3.78	0.035	0.011
AD	1.10	0.08	4.00	0.00	51.56	3.69	277.47	10.90	0.036	0.007
Exposure to EMF	1.41	0.30	4.00	0.00	51.01	4.77	276.49	10.92	0.038	0.007
F value	60.288		0.001		194.418		713.569		44.896	
P value	< 0.001		< 0.001		< 0.001		< 0.001		< 0.001	

Table 2

Group	TNF ALP (pg/ml)		ALA (umol24)		PBG (umol24)		Uroporphyrin (nmol24)		Coproporphyrin (nmol/24)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
NO/BHCD	17.94	0.59	15.44	0.50	20.82	1.19	50.18	3.54	137.94	4.75
RHCD	78.63	5.08	63.50	6.95	42.20	8.50	250.28	23.43	389.01	54.11
LHCD	9.29	0.81	3.86	0.26	12.11	1.34	9.51	1.19	64.33	13.09
MND	79.28	4.55	68.28	6.02	46.54	4.55	290.44	57.65	436.71	52.95
PD	82.13	3.97	67.30	5.98	47.25	4.19	286.84	24.18	432.22	50.11
AD	79.65	5.57	67.32	5.40	49.83	3.45	259.61	33.18	433.17	45.61
Exposure to EMF	76.41	5.96	68.41	5.53	47.27	3.42	288.21	26.17	444.94	38.89
F value	427.654		295.467		183.296		160.533		279.759	
P value	< 0.001		< 0.001		< 0.001		< 0.001		< 0.001	

Table 3

Group	Protoporphyrin (Ab unit)		Heme (uM)		Bilirubin (mg/dl)		Biliverdin (Ab unit)		ATP synthase (umol/gHb)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
NO/BHCD	10.35	0.38	30.27	0.81	0.55	0.02	0.030	0.001	0.36	0.13
RHCD	42.46	6.36	12.47	2.82	1.70	0.20	0.067	0.011	2.73	0.94
LHCD	2.64	0.42	50.55	1.07	0.21	0.00	0.017	0.001	0.09	0.01
MND	49.59	1.70	13.03	0.70	1.84	0.07	0.070	0.015	3.09	0.65
PD	49.36	4.18	11.81	0.80	1.83	0.09	0.071	0.014	3.34	0.84
AD	49.68	3.30	12.09	1.12	1.77	0.13	0.073	0.016	3.34	0.75
Exposure to EMF	50.59	1.71	12.36	1.26	1.75	0.22	0.073	0.013	3.39	1.03
F value	424.198		1472.05		370.517		59.963		54.754	
P value	< 0.001		< 0.001		< 0.001		< 0.001		< 0.001	

Table 4

Group	SE ATP (umol/dl)		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	Mean	± SD
NO/BHCD	0.42	0.11	2.79	0.28	7.38	0.31	40.51	1.42	1.66	0.45
RHCD	2.24	0.44	12.39	1.23	25.99	8.10	100.51	12.32	5.46	2.83
LHCD	0.02	0.01	1.21	0.38	2.75	0.41	23.79	2.51	0.68	0.23
MND	1.66	0.56	12.06	1.09	21.78	0.58	90.46	8.30	6.29	1.73
PD	1.27	0.26	12.65	1.06	24.28	1.69	95.44	12.04	9.30	3.98
AD	2.06	0.19	11.94	0.86	22.04	0.64	97.26	8.26	8.46	3.63
Exposure to EMF	1.37	0.27	12.26	1.00	23.31	1.46	103.28	11.47	7.58	3.09
F value	67.588		445.772		162.945		154.701		18.187	
P value	< 0.001		< 0.001		< 0.001		< 0.001		< 0.001	

Table 5

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
MND	2.15	0.22	33.27	5.99	3.67	0.38
PD	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
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 6

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
MND	95.61	7.88	1.14	0.07	22.98	5.19
PD	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
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

AD: Alzheimer’s disease

MND: Motor neuron disease

PD: Parkinson’s disease

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 aminolevlunic 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]. This can lead on

to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

Low level electromagnetic fields and its porphyrin messengers can regulate the brain mediating quantal perception. Porphyrin microarrays serve the purpose of quantal perception. Porphyrin photo-oxidation can generate free radicals which can modulate NMDA transmission. Free radicals can increase NMDA transmission. ALA blocks GABA transmission and upregulates NMDA. Thus porphyrins can produce NMDA excitotoxicity contributing to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. The dipolar porphyrins in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated Frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macroscopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrin molecules have a wave particle existence and can bridge the dividing line between quantal state and particulate state. Thus the porphyrins can mediate quantal perception and perception of low level EMF contributing to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Porphyrins binding to proteins, nucleic acids and cell membranes can produce biophoton emission. Porphyrins by auto-oxidation 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 porphyrin microarrays can function as a quantal computer mediating extrasensory perception of low level EMF contributing to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. The porphyrins can modulate hemispheric dominance. There is

increased porphyrin synthesis and RHCD and decreased porphyrin synthesis in LHCD. RHCD can contribute to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Altered porphyrin metabolism has been described in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease[3, 4, 16]. Thus porphyrins microarrays can function as a quantal brain modulating extrasensory quantal perception. Porphyrin microarrays can function as a quantal brain in communication with digital world and geomagnetic fields. This can contribute to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

The dipolar porphyrins in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated Frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macroscopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrins by auto-oxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Porphyrin auto-oxidation is modulated by low level of electromagnetic fields and geomagnetic fields. Cellular porphyrins photo-oxidation are involved in sensing of earth magnetic fields and low level biomagnetic fields. Porphyrins can thus contribute to quantal perception. Low level electromagnetic fields and light can induce porphyrin synthesis. Low level EMF can produce ferrochelatase inhibition as well as heme oxygenase induction contributing to heme depletion, ALA synthase induction and increased porphyrin synthesis. Light also induces ALA synthase and porphyrin synthesis. The increased porphyrin synthesized can contribute to increased quantal perception and can modulate conscious perception. The human porphyrin microarrays induced biophotons and quantal fields can modulate the source from which low level EMF and photic fields were

generated. Thus the porphyrin generated by extraneous low level EMF and photic fields can interact with the source of low level EMF and photic fields modulating it. Thus porphyrins can serve as a bridge between the human brain and the source of low level EMF and photic fields. This serves as a mode of communication between the human brain and digital EMF storage devices like internet. The porphyrins can also serve as the source of communication with the environment. Environmental EMF and chemicals produce heme oxygenase induction and heme depletion increasing porphyrin synthesis, quantal perception and two-way communication. Thus induction of porphyrin synthesis can serve as a mechanism of communication between human brain and the environment by extrasensory perception. Porphyrin microarrays can function as quantal computers storing information and can serve the purpose of extrasensory perception. Porphyrins can serve as a two way communicating bridge between digital information storage systems generating low level electromagnetic fields and human systems. The low level of EMF produced by digital system enhances porphyrin synthesis and serves the purpose of two way extrasensory perception and communication. The human porphyrin quantal computers can in turn by biophoton emission modulate digital information storage system. The quantal perception of low level EMF can contribute to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

Low level of electromagnetic fields and its porphyrin messengers can induce the Warburg phenotype. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. The archaea can synthesize porphyrins and induce porphyrin synthesis. Porphyrins have been related to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Porphyrins can mediate the effect of low level electromagnetic fields inducing the Warburg phenotype leading to the above mentioned disease states. The Warburg phenotype results in inhibition of pyruvate dehydrogenase and

the TCA cycle. The pyruvate enters the GABA shunt pathway where it is converted to succinyl CoA. The glycolytic pathway is upregulated and the glycolytic metabolite phosphoglycerate is converted to serine and glycine. Glycine and succinyl CoA are the substrates for ALA synthesis. The archaea induces the enzyme heme oxygenase. Heme oxygenase converts heme to bilirubin and biliverdin. This depletes heme from the system and results in upregulation of ALA synthase activity resulting in porphyria. Heme inhibits HIF alpha. The heme depletion results in upregulation of HIF alpha activity and further strengthening of the Warburg phenotype. The porphyrin self oxidation results in redox stress which activates HIF alpha and generates the Warburg phenotype. The Warburg phenotype results in channelling acetyl CoA for cholesterol synthesis as the TCA cycle and mitochondrial oxidative phosphorylation are blocked. The archaea uses cholesterol as an energy substrate. Porphyrin and ALA inhibits sodium potassium ATPase. This increases cholesterol synthesis by acting upon intracellular SREBP. The cholesterol is metabolized to pyruvate and then the GABA shunt pathway for ultimate use in porphyrin synthesis. The porphyrins can self organize and self replicate into macromolecular arrays. The porphyrin arrays behave like an autonomous organism and can have intramolecular electron transport generating ATP. The porphyrin macroarrays can store information and can have quantal perception. The porphyrin macroarrays serves the purpose of archaeal energetics and sensory perception. The Warburg phenotype is associated with neurodegenerations-alzheimer’s disease, parkinson’s disease and motor neuron disease. Low level electromagnetic fields can induce the Warburg phenotype contributing to neurodegenerations-alzheimer’s disease, parkinson’s disease and motor neuron disease.

The role of porphyrins and low level electromagnetic fields in regulation of cell functions and neuro-immuno-endocrine integration is discussed. Low levels of EMF fields can induce digoxin synthesis. Protoporphyrin binds to the

peripheral benzodiazepine receptor regulating steroid and digoxin synthesis. Increased porphyrin metabolites can contribute to hyperdigoxinemia. Digoxin can modulate the neuro-immuno-endocrine system. Low level of EMF fields can modulate membrane, nucleic acid and protein structure and function via induction of porphyrin synthesis. Porphyrins can combine with membranes modulating membrane function. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. Porphyrins can complex with DNA and RNA modulating their function. Porphyrin interpolating with DNA can alter transcription and generate HERV expression. Low level of EMF fields through modulation of porphyrin metabolism can produce heme deficiency by inhibiting heme oxygenase and ferrochelatase. Heme deficiency can also result in disease states. Heme deficiency results in deficiency of heme enzymes. There is deficiency of cytochrome C oxidase and mitochondrial dysfunction. The glutathione peroxidase is dysfunctional and the glutathione system of free radical scavenging does not function. The cytochrome P450 enzymes involved in steroid and bile acid synthesis have reduced activity leading to steroid-cortisol and sex hormones as well as bile acid deficiency states. The heme deficiency results in dysfunction of nitric oxide synthase, heme oxygenase and cystathione beta synthase resulting in lack of gasotransmitters regulating the vascular system and NMDA receptor-NO, CO and H_2S. Heme has got cytoprotective, neuroprotective, anti-inflammatory and antiproliferative effects. Heme is also involved in the stress response. Heme deficiency leads to metabolic syndrome, immune disease, degenerations and cancer[3-5]. Low level electromagnetic fields can modulate cell functions and neuro-immuno-endocrine-genetic integration via induction of porphyrin synthesis. This can contribute to neurodegenerations-alzheimer’s disease, parkinson’s disease and motor neuron disease.

Low level electromagnetic fields via modulating porphyrin metabolism can produce an autonomic neuropathy. Protoporphyrins block acetyl choline transmission producing a vagal neuropathy with sympathetic over activity. Vagal neuropathy results in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. A vagal neuropathy underlines neoplastic and autoimmunity in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Low level electromagnetic fields by modulating porphyrin metabolism can induce cell death. Porphyrin induced increased NMDA transmission and free radical injury can contribute to neuronal degeneration. Free radicals can produce mitochondrial PT pore dysfunction. This can lead to cyto C leak and activation of the caspase cascade leading to apoptosis and cell death. Altered porphyrin metabolism has been described in Alzheimer's disease. The increased porphyrin photo-oxidation generated free radicals mediated NMDA transmission can also contribute to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate brain function and cell death[3, 4, 16].

Low level electromagnetic fields by modulating porphyrin metabolism can generate redox stress to regulate cell functions. The porphyrins can undergo photo-oxidation and auto-oxidation generating free radicals. The archaeal porphyrins can produce free radical injury. Free radicals produce NFKB activation, open the mitochondrial PT pore resulting in cell death, activate NMDA receptor and generates the Warburg phenotypes activating glycolysis and inhibiting TCA cycle/oxphos. Porphyrins have been related to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Low level electromagnetic fields by modulating porphyrin metabolism can regulate cell membrane sodium potassium ATPase. The porphyrins can complex and intercalate with the cell membrane producing sodium potassium ATPase inhibition adding on to digoxin mediated inhibition. Porphyrins can

complex with proteins and nucleic acid producing biophoton emission. Low level electromagnetic fields by modulating porphyrin metabolism can regulate DNA, RNA and protein structure and function. Porphyrins complexing with proteins can modulate protein structure and function. Porphyrins complexing with DNA and RNA can modulate transcription and translation. Low level electromagnetic fields by modulating porphyrin metabolism can regulate mitochondrial function, peripheral benzodiazepine receptor and steroidogenesis. The porphyrin especially protoporphyrins can bind to peripheral benzodiazepine receptors in the mitochondria and modulate its function, mitochondrial cholesterol transport and steroidogenesis. Peripheral benzodiazepine receptor modulation by protoporphyrins can regulate cell death, cell proliferation, immunity and neural functions. Low level electromagnetic fields by modulating porphyrin metabolism and inducing redox stress can regulate enzyme systems. The porphyrin photo-oxidation generates free radicals which can modulate enzyme function. Redox stress modulated enzymes include pyruvate dehydrogenase, nitric oxide synthase, cystathione beta synthase and heme oxygenase. Free radicals can modulate mitochondrial PT pore function. Free radicals can modulate cell membrane function and inhibit sodium potassium ATPase activity. Thus the porphyrins are key regulatory molecules modulating all aspects of cell function[3-5]. Low level of electromagnetic fields by modulating porphyrin metabolism can induce viroidal and HERV expression. There was an increase in free RNA indicating self replicating RNA viroids and free DNA indicating generation of viroid complementary DNA strands by archaeal reverse transcriptase activity. The actinides and porphyrins modulate RNA folding and catalyse its ribozymal action. Digoxin can cut and paste the viroidal strands by modulating RNA splicing generating RNA viroidal diversity. The viroids are evolutionarily escaped archaeal group I introns which have retrotransposition and self splicing qualities. Porphyrin photo-oxidation induced redox stress can produce HDAC

inhibition. Archaeal pyruvate producing histone deacetylase inhibition and porphyrins intercalating with DNA can produce endogenous retroviral (HERV) reverse transcriptase and integrase expression. This can integrate the RNA viroidal complementary DNA into the noncoding region of eukaryotic non coding DNA using HERV integrase as has been described for borna and ebola viruses. The archaea and viroids can also induce cellular porphyrin synthesis. Bacterial and viral infections can precipitate porphyria and neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Thus porphyrins can regulate genomic function. The increased expression of HERV RNA can result in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease[14, 15].

Low level electromagnetic fields by modulating porphyrin metabolism and generating redox stress can produce immune activation. 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 autoimmunity in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 autoimmunity in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease[3, 4]. Low level electromagnetic fields by modulating porphyrin metabolism and inducing redox stress can produce insulin resistance. The porphyrin photo-oxidation mediated free radical injury can lead to insulin resistance and neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. 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 neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease[3, 4]. Low level electromagnetic fields by modulating porphyrin metabolism and inducing redox stress/heme deficiency can activate HIF alpha. The porphyrin photo-oxidation can generate free radicals inducing HIF alpha and producing neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease[3, 4]. Low level electromagnetic fields by modulating porphyrin metabolism can regulate prion protein conformation. The porphyrin can combine with prion proteins modulating their conformation. This leads to abnormal prion protein conformation and degradation. Archaeal porphyrins can contribute to prion disease important in neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Low level electromagnetic fields by modulating porphyrin metabolism can produce redox stress and regulate HERV expression. The porphyrins can also intercalate with DNA producing HERV expression. The HERV particles generated can contribute to the retroviral state. HERV sequences can produce neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease[3, 4].

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 increased porphyrin synthesis in the Scythian races contributes to higher level of extrasensory quantal perception in this racial group. This contributes to higher level of cognitive and spiritual function of the brain in this racial group. This racial group has increased incidence of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

The porphyrins can contribute to the role of low level electromagnetic fields in the pathogenesis of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states-neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease is described. Archaeal porphyrin synthesis and induction of endogenous 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. Thus the porphyrins contribute to the inducing role of low level electromagnetic fields in the pathogenesis of neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease. Low level electromagnetic fields and its porphyrin messengers can regulate immune, neural, endocrine, metabolic and genetic systems[3, 4]. A hypothesis regarding the role of porphyrins and quantal perception as well as the role of porphyrins in environmental communication/modulation of digital information storage/processing system is presented. Thus porphyrin microarrays can function as a quantal computer mediating extrasensory perception. Porphyrin microarrays in human systems and brain owing to the wave particle nature of porphyrins can bridge the quantal world and particulate world. The relationship between low level of electromagnetic fields and human disease is highlighted. This can lead on to neurodegenerations-alzheimer's disease, parkinson's disease and motor neuron disease.

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