Chapter 13

Internet Exposure, Porphyrions and Systemic Diseases-Porphyrions Induce Cirrhosis Liver, Chronic Renal Failure, Vascular Thrombosis, Chronic Obstructive Pulmonary Disease and Interstitial Lung Disease

Introduction

The internet exposure leads to low level EMF induced heme oxygenase induction in the brain. The brain heme is depleted leading to increase in ALA synthase and porphyrin synthesis. The porphyrins self aggregate to form supramolecular organisms called porphyrions. The porphyrin acts as a template for the formation of RNA viroids, DNA viroids, isoprenoid organisms and prions which symbiosed to form nanoarchaea. The nanoarchaea contain magnetite and are magnetotactic and can have quantal perception as well as low level EMF perception. This leads to more of brain endosymbiotic nanoarchaeal growth. The nanoarchaea are capable of methanogenesis which contributes to global warming. The global warming related to internet exposure can produce still further increase in endosymbiotic archaeal symbiosis.

Actinidic archaea have been related to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. 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.[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 cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The role of archaeal porphyrins in regulation of cell functions and neuro-immuno-endocrine integration is discussed.[1-5] They can function as self replicating supramolecular organisms which can be called as porphyrions.

Materials and Methods

The following groups of systemic disease were included in the study: - cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. 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 selected from the general population. The blood samples were drawn in the fasting state before treatment was initiated. Plasma from fasting heparinised blood was used and the experimental protocol was as follows: (I) Plasma+phosphate buffered saline, (II) same as I+cholesterol substrate, (III) same as II+rutile 0.1 mg/ml, and (IV) same as II+ciprofloxacine and doxycycline each in a concentration of 1 mg/ml. Cholesterol substrate was prepared as described by Richmond. Aliquots were withdrawn at zero time immediately after mixing and after incubation at 37 ℃ for 1 hour. The following estimations were carried out: - Cytochrome F420, free RNA, free DNA, polycyclic aromatic hydrocarbon, hydrogen peroxide, pyruvate, ammonia, glutamate, delta aminolevulinic acid, succinate, glycine and digoxin. Cytochrome F420 was estimated flourimetrically (excitation wavelength 420 nm and emission wavelength 520 nm). Polycyclic aromatic hydrocarbon was estimated by measuring hydrogen peroxide liberated by using glucose reagent. The study also involved estimating the following parameters in the patient population-digoxin, bile acid, hexokinase, porphyrins, pyruvate, glutamate, ammonia, acetyl CoA, acetyl choline, HMG CoA reductase, cytochrome C, blood ATP, ATP synthase, ERV RNA (endogenous retroviral RNA), H_2O_2 (hydrogen peroxide), NOX (NADPH oxidase), TNF alpha and heme oxygenase.[6-9] Informed consent of the subjects and the approval of the ethics committee were obtained for the study. The statistical analysis was done by ANOVA.

Results

Plasma of control subjects showed increased levels of the above mentioned parameters with after incubation for 1 hour and addition of cholesterol substrate resulted in still further significant increase in these parameters. The plasma of patients showed similar results but the extent of increase was more. The addition of antibiotics to the control plasma caused a decrease in all the parameters while addition of rutile increased their levels. The addition of antibiotics to the patient's plasma caused a decrease in all the parameters while addition of rutile increased their levels but the extent of change was more in patient's sera as compared to controls. The results are expressed in section 1: tables 1-6 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time. There was upregulated archaeal porphyrin synthesis in the patient population which was archaeal in origin as indicated by actinide catalysis of the reactions. The cholesterol oxidase pathway generated pyruvate which entered the GABA shunt pathway. This resulted in synthesis of succinate and glycine which are substrates for ALA synthase.

The study showed the patient's blood and right hemispheric dominance had increased heme oxygenase activity and porphyrins. The hexokinase activity was high. The pyruvate, glutamate and ammonia levels were elevated indicating blockade of PDH activity, and operation of the GABA shunt pathway. The acetyl CoA levels were low and acetyl choline was decreased. The 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 were increased NOX and TNF alpha levels indicating immune activation. The HMG CoA reductase activity was high indicating cholesterol synthesis. The bile acid levels were low indicating depletion of cytochrome P450. The normal population with right hemispheric dominance had values resembling the patient

population with increased porphyrin synthesis. The normal population with left hemispheric dominance had low values with decreased porphyrin synthesis.

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
COPD	23.46	1.87	59.27	8.86	22.67	2.29	57.69	5.29
ILD	22.79	2.13	55.90	7.29	22.84	1.42	66.07	3.78
CAD/CVA	22.59	1.86	57.05	8.45	23.40	1.55	65.77	5.27
CRF	22.06	1.61	57.81	6.04	23.46	1.91	61.56	4.61
CLD	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
COPD	23.40	1.51	63.68	4.66	23.08	1.87	65.09	3.48
ILD	22.42	1.99	61.14	3.47	23.78	1.20	66.90	4.10
CAD/CVA	23.01	1.67	65.35	3.56	23.33	1.86	66.46	3.65
CRF	23.30	1.42	65.07	4.95	23.11	1.52	66.68	3.97
CLD	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
COPD	0.51	0.05	0.199	0.027	22.83	1.90	67.23	3.45
ILD	0.54	0.04	0.210	0.042	23.34	1.75	66.80	3.43
CAD/CVA	0.47	0.04	0.202	0.025	22.87	1.84	66.31	3.68
CRF	0.53	0.06	0.212	0.045	23.17	1.88	68.53	2.65
CLD	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
COPD	22.28	1.52	64.05	2.79	22.82	1.56	64.61	4.95
ILD	23.43	1.57	66.30	3.57	22.98	1.50	65.13	4.87
CAD/CVA	23.70	1.75	68.06	3.52	23.81	1.49	64.89	6.01
CRF	22.92	2.14	67.54	3.65	21.93	2.29	63.70	5.63
CLD	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
COPD	20.94	1.54	62.76	8.52	23.33	1.79	62.50	5.56
ILD	21.19	1.61	58.57	7.47	22.53	2.41	64.29	5.44
CAD/CVA	20.67	1.38	58.75	8.12	23.23	1.88	65.11	5.14
CRF	21.07	1.79	63.90	7.13	22.47	2.17	65.97	4.62
CLD	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
COPD	23.81	1.19	61.08	7.38	22.83	1.90	67.23	3.45
ILD	23.35	1.76	59.17	3.33	23.34	1.75	66.80	3.43
CAD/CVA	23.27	1.53	58.91	6.09	22.87	1.84	66.31	3.68
CRF	22.86	1.91	63.66	6.88	23.17	1.88	68.53	2.65
CLD	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. *Archaeal metabolonomics.*

Group	RBC Digoxin (ng/ml RBC Susp)		Cytochrome F420		HERV RNA (ug/ml)		H_2O_2 (umol/ml RBC)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
NO/BHCD	0.58	0.07	1.00	0.00	17.75	0.72	177.43	6.71
RHCD	1.41	0.23	4.00	0.00	55.17	5.85	278.29	7.74
LHCD	0.18	0.05	0.00	0.00	8.70	0.90	111.63	5.40
CAD	1.22	0.16	4.00	0.00	50.00	5.91	280.89	13.79
CVA	1.33	0.27	4.00	0.00	51.06	4.83	287.33	9.47
CRF	1.26	0.26	4.00	0.00	49.39	5.51	285.51	8.79
CLD	1.50	0.20	4.00	0.00	46.82	4.73	275.97	10.66
COPD	1.40	0.32	4.00	0.00	46.37	4.87	290.37	9.10
ILD	1.51	0.29	4.00	0.00	47.47	4.34	287.49	9.81
F value	60.288		0.001		194.418		713.569	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 2. *Porphyrin synthesis.*

Group	NOX (OD diff/hr/mgpro)		TNF ALP (pg/ml)		ALA (umol24)		PBG (umol24)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
NO/BHCD	0.012	0.001	17.94	0.59	15.44	0.50	20.82	1.19
RHCD	0.036	0.008	78.63	5.08	63.50	6.95	42.20	8.50
LHCD	0.007	0.001	9.29	0.81	3.86	0.26	12.11	1.34
CAD	0.038	0.009	78.15	3.72	66.66	7.77	47.00	3.81
CVA	0.037	0.007	77.59	5.24	69.02	4.86	46.33	4.01
CRF	0.039	0.008	81.36	5.37	67.61	5.55	46.81	4.62
CLD	0.037	0.010	77.61	4.42	66.28	6.55	48.23	2.36
COPD	0.039	0.010	79.38	5.14	67.86	5.65	44.08	2.81
ILD	0.035	0.008	80.04	4.69	64.76	5.23	44.82	3.46
F value	44.896		427.654		295.467		183.296	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 3. *Porphyrin patterns.*

Group	Uroporphyrin (nmol24)		Coproporphyrin (nmol/24)		Protoporphyrin (Ab unit)		Heme (uM)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	50.18	3.54	137.94	4.75	10.35	0.38	30.27	0.81
RHCD	250.28	23.43	389.01	54.11	42.46	6.36	12.47	2.82
LHCD	9.51	1.19	64.33	13.09	2.64	0.42	50.55	1.07
CAD	314.01	17.82	426.14	24.28	49.51	2.27	11.39	1.10
CVA	320.85	24.73	402.16	33.80	46.74	4.28	11.26	0.95
CRF	301.78	48.22	427.57	33.55	49.66	4.41	12.03	1.40
CLD	276.51	16.66	436.44	25.65	50.56	1.63	11.92	1.33
COPD	303.86	13.91	441.58	25.51	47.86	3.34	12.13	1.10
ILD	300.90	31.96	443.22	38.14	51.37	4.86	12.61	2.00
F value	160.533		279.759		424.198		1472.05	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 4. *Heme metabolism.*

Group	Bilirubin (mg/dl)		Biliverdin (Ab unit)		ATP Synthase (umol/gHb)		SE ATP (umol/dl)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	0.55	0.02	0.030	0.001	0.36	0.13	0.42	0.11
RHCD	1.70	0.20	0.067	0.011	2.73	0.94	2.24	0.44
LHCD	0.21	0.00	0.017	0.001	0.09	0.01	0.02	0.01
CAD	1.75	0.12	0.080	0.007	2.99	0.65	1.57	0.37
CVA	1.82	0.10	0.079	0.009	2.98	0.78	1.49	0.27
CRF	1.76	0.22	0.070	0.012	3.14	0.57	1.53	0.33
CLD	1.81	0.10	0.076	0.009	3.01	0.47	1.32	0.26
COPD	1.78	0.24	0.067	0.014	2.92	0.55	1.35	0.29
ILD	1.79	0.07	0.074	0.009	3.12	0.60	1.56	0.48
F value	370.517		59.963		54.754		67.588	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 5. *Mitochondrial dysfunction.*

Group	Cyto C (ng/ml)		Lactate (mg/dl)		Pyruvate (umol/l)		RBC Hexokinase (ug glu phos / hr/mgpro)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	2.79	0.28	7.38	0.31	40.51	1.42	1.66	0.45
RHCD	12.39	1.23	25.99	8.10	100.51	12.32	5.46	2.83
LHCD	1.21	0.38	2.75	0.41	23.79	2.51	0.68	0.23
CAD	11.51	0.47	22.83	0.82	97.29	12.45	8.88	3.09
CVA	12.74	0.80	23.03	1.26	103.25	9.49	7.87	2.72
CRF	12.66	1.01	23.42	1.20	97.38	10.76	7.75	3.08
CLD	12.81	0.90	26.20	5.29	97.77	13.24	8.99	3.27
COPD	12.84	0.74	23.64	1.43	96.19	12.15	10.12	1.75
ILD	12.72	0.92	25.35	5.52	103.32	13.04	9.44	3.40
F value	445.772		162.945		154.701		18.187	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 6. *GABA shunt.*

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
CAD	2.37	0.44	49.19	6.86	3.61	0.28
CVA	2.25	0.44	37.45	7.93	3.31	0.43
CRF	2.24	0.32	37.52	4.37	3.26	0.43
CLD	2.13	0.17	46.20	4.95	3.25	0.40
COPD	2.51	0.42	45.51	7.56	3.11	0.36
ILD	2.19	0.19	42.48	8.62	3.27	0.39
F value	1871.04		116.901		200.702	
P value	< 0.001		< 0.001		< 0.001	

Table 7. *Cholesterol synthesis and catabolism.*

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
CAD	93.93	4.86	1.07	0.12	24.55	6.26
CVA	103.18	27.27	1.05	0.09	22.39	3.35
CRF	98.76	11.12	1.03	0.10	26.47	5.30
CLD	94.77	2.86	1.04	0.10	24.91	5.06
COPD	92.40	4.34	1.12	0.08	24.37	4.38
ILD	95.37	5.76	1.08	0.08	25.17	3.80
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

CAD-Coronary artery disease

CVA-Cerebrovascular accident

CRF-Chronic renal failure

CLD-Chronic liver disease

COPD-Chronic obstructive pulmonary disease

ILD-Interstitial lung disease

Discussion

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis and Abiogenesis

The internet exposure leads to low level EMF induced heme oxygenase induction in the brain. The brain heme is depleted leading to increase in ALA synthase and porphyrin synthesis. The porphyrins self aggregate to form supramolecular organisms called porphyrions. The porphyrin acts as a template for the formation of RNA viroids, DNA viroids, isoprenoid organisms and prions which symbiosed to form nanoarchaea. The nanoarchaea contain magnetite and are magnetotactic and can have quantal perception as well as low level EMF perception. This leads to more of brain endosymbiotic nanoarchaeal growth.

Internet Exposure, Endosymbiotic Archaea, Cholesterol Catabolism and Porphyrinogenesis

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]

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis, Genomic Modulation and Cardio-Renal-Hepatic-Pulmonary Syndrome

The porphyrins can contribute to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The porphyrins can undergo photooxidation 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 cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. 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 photooxidation generates free radicals which can modulate enzyme function. Redox stress modulated enzymes include pyruvate dehydrogenase, nitric oxide synthase, cystathione beta synthase and heme oxygenase. Free radicals can modulate mitochondrial PT pore function. Free radicals can modulate cell membrane function and inhibit sodium potassium ATPase activity. Thus the porphyrins are key regulatory molecules modulating all aspects of cell function.[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 noncoding 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. The viroids

can contribute to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Viroids can modulate cell function by RNA interference. Thus porphyrins can regulate genomic function. The increased expression of HERV RNA can also result in cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. HERV RNA can also modulate cell function by RNA interference.[14, 15]

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis, the Warburg Phenotype and Cardio-Renal-Hepatic-Pulmonary Syndrome

The possibility of Warburg phenotype induced by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism in cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis 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. Heme deficiency results in cytochrome C oxidase dysfunction and mitochondrial dysfunction. 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 cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Mitochondrial dysfunction can contribute to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The upregulated glycolysis results in channelling of glycolytic metabolite fructose 1,6-diphosphate to the pentose phosphate pathway generating NADPH. NADPH activates the NOX enzyme which generates H_2O_2 producing redox stress. Redox stress is related to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The channelling of fructose 1,6-diphosphate to the pentose phosphate pathway results in generation of amino sugars involved in mucopolysaccharide biosynthesis. This results in connective tissue deposition in organ systems contributing to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The upregulated glycolysis results in immune activation as lymphocytes depend on glycolysis for its energy needs. This results in cytokine related tissue injury contributing to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis.

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis and Cardio-Renal-Hepatic-Pulmonary Syndrome

The role of porphyrins in regulation of cell functions and neuro-immuno-endocrine integration and its dysfunction is discussed in the setting of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. 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. Digoxin can produce membrane sodium potassium ATPase inhibition resulting in increased intracellular calcium. Porphyrins can combine with membranes modulating membrane function and producing sodium potassium ATPase inhibition. The increased intracellular calcium can activate NFKB producing immune activation and produce mitochondrial dysfunction. This contributes to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Hyperdigoxinemia has been detected in cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. This can contribute to protein processing defects and impaired lysosomal digestion of the defectively processed proteins which accumulates in tissues. This can contribute to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Porphyrins can complex with DNA and RNA modulating their function. Porphyrin interpolating with DNA can alter transcription and generate HERV expression. HERV expression can contribute to the pathogenesis of these systemic diseases-cirrhosis liver, chronic renal failure,

interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. 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. Mitochondrial dysfunction can also contribute to the pathogenesis of these systemic diseases-cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The glutathione peroxidase is dysfunctional and the glutathione system of free radical scavenging does not function. Redox stress can contribute to the pathogenesis of these systemic diseases-cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The cytochrome P450 enzymes involved in steroid and bile acid synthesis have reduced activity leading to steroid-cortisol and sex hormones, activated vitamin D as well as bile acid deficiency states. Bile acids are tissue protective. Bile acids like lithocholic acid bind to VDR receptor. Bile acid and activated vitamin D deficiency can lead to immune activation and cytokine injury in systemic diseases-cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. 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. Deficiency of gasotransmitters can contribute to vasospasm and vascular thrombosis. NO, CO and H_2S can combine with cytochrome C oxidase producing mitochondrial hibernation and tissue protection. Loss of gasotransmitters related mitochondrial hibernation can contribute to the pathogenesis of these systemic diseases-cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Heme has got cytoprotective, neuroprotective, anti-inflammatory and anti-proliferative effects. Heme is also involved in the stress response.

Heme deficiency leads to cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis.[3-5]

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis, Autoimmunity and Cardio-Renal-Hepatic-Pulmonary Syndrome

The porphyrin photooxidation can generate free radicals which can activate NFKB. This can produce immune activation and cytokine mediated injury. The increase in porphyrins can lead to cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can modulate immune function. Immune activation and cytokine mediated tissue injury is related to the pathogenesis of systemic diseases-cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. 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] Autoantibodies are related to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The porphyrin photooxidation mediated free radical injury can lead to insulin resistance and atherogenesis. Thus archaeal porphyrins can contribute to metabolic syndrome X. Glucose has got a negative effect upon ALA synthase activity. Therefore hyperglycemia may be reactive protective mechanism to increased archaeal porphyrin synthesis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can modulate mitochondrial steroidogenesis and metabolism. Altered porphyrin metabolism has been described in the metabolic syndrome X. Porphyrias can lead onto vascular thrombosis.[3, 4] Metabolic

syndrome X is a risk factor for cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The porphyrin photooxidation can generate free radicals inducing HIF alpha and producing oncogene activation. Heme deficiency can lead to activation of HIF alpha and oncogenesis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate cell proliferation.[3, 4] Oncogene activation, connective tissue deposition and fibrosis is related to cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. The porphyrins in the blood can combine with bacteria and viruses and the photooxidation 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] Infections have been related to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. EBV, CMV, herpes and chlamydial infections have been related to vascular thrombosis. Corona virus infection and chlamydial infection have been related to interstitial lung disease and COPD. Thus the porphyrins can contribute to the pathogenesis of these systemic diseases.[3, 4]

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis, Synaptic Modulation and Cardio-Renal-Hepatic-Pulmonary Syndrome

The archaea and viroids can regulate the nervous system including the NMDA/GABA thalamo-cortico-thalamic pathway mediating conscious perception. Porphyrin photooxidation can generate free radicals which can modulate NMDA transmission. Free radicals can increase NMDA transmission. Free radicals can induce GAD and increase GABA synthesis. ALA blocks GABA transmission and upregulates NMDA. Protoporphyrins bind to GABA receptor and promote GABA transmission. Thus porphyrins can modulate the

thalamo-cortico-thalamic pathway of conscious perception. The dipolar porphyrins, PAH and archaeal magnetite in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated Frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macroscopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrin molecules have a wave particle existence and can bridge the dividing line between quantal state and particulate state. Thus the porphyrins can mediate conscious and quantal perception. Porphyrins binding to proteins, nucleic acids and cell membranes can produce biophoton emission. Porphyrins by autooxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Cellular porphyrins photooxidation are involved in sensing of earth magnetic fields and low level biomagnetic fields. Thus prophyrins can mediate extrasensory perception. The porphyrins can modulate hemispheric dominance. There is increased porphyrin synthesis and RHCD and decreased porphyrin synthesis in LHCD. Porphyria can lead to psychiatric disorders and seizures. Neuropsychiatric syndromes are described in chronic hepatic encephalopathy, chronic renal failure and respiratory failure. Right hemispheric chemical dominance has been related to cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Protoporphyrins block acetyl choline transmission producing a vagal neuropathy with sympathetic overactivity. Vagal neuropathy results in immune activation, vasospasm and vascular disease. A vagal neuropathy underlines cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Porphyrin induced increase free radical injury can contribute to cell death. 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 in systemic diseases. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate brain function and cell death. Cell death mediated by porphyrins can contribute to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis.[3, 4, 16]

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis, Quantal Perception and Cardio-Renal-Hepatic-Pulmonary Syndrome

The dipolar porphyrins, PAH and archaeal magnetite in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated Frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macroscopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrins by autooxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Cellular porphyrins photooxidation are involved in sensing of earth magnetic fields and low level biomagnetic fields. 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. Low level of EMF can contribute to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis.

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis, Species Evolution and Cardio-Renal-Hepatic-Pulmonary Syndrome

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 noncoding DNA length. The alteration in the length of the noncoding region of the DNA contributes to the dynamic nature of the genome. Thus genetic and acquired porphyrias can lead to alteration in the noncoding region of the genome. The alteration of the length of the noncoding region of the DNA contributes to the racial and individual differences in populations. An increased length of noncoding 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 thalamo-cortico-thalamic pathway of conscious perception. Thus genetic and acquired porphyrias contribute to higher cognitive and creative capacity of certain races. Porphyrias are common among Eurasian Scythian races who have assumed leadership roles in communities and groups. The systemic diseases-cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis are common in the Scythian races and most of our patient population belonged to this group.[3, 4]

Internet Exposure, Endosymbiotic Archaea, Porphyrinogenesis and Cardio-Renal-Hepatic-Pulmonary Syndrome

An actinide dependent shadow biosphere of archaea and viroids in cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis is described. The archaeal porphyrins can contribute to the pathogenesis of cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis. Porphyrin synthesis is crucial in the pathogenesis of these disorders. Porphyrins may serve as regulatory molecules modulating immune, neural, endocrine, metabolic and genetic systems. The porphyrins photooxidation generated free radicals can produce immune activation, produce cell death, activate cell proliferation, produce insulin resistance and modulate conscious/quantal perception. The porphyrins functions as key regulatory molecules with mitochondrial benzodiazepine receptors playing an important role. The porphyrins photooxidation 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 porphyrins functions as key regulatory molecules with mitochondrial benzodiazepine receptors playing an important role. A porphyrin metabolic dysfunction is the principal critical abnormality

underlying cirrhosis liver, chronic renal failure, interstitial lung disease, chronic obstructive pulmonary disease and vascular thrombosis.

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