Chapter 7

The Globalisation of the Mind – Neanderthalic Actinidic Archaeal Porphyrins and Perception/Two-Way Communication with Internet Digital Fields, Low Level EMF, Background Radiation Perception – How the Unconscious Regulates the Internet

Introduction

Neanderthalic actinidic archaea and its synthesized porphyrins can perceive low level of EMF fields as well as internet digital electromagnetic fields. This is evidenced by the effect of low level of radioactivity is described from the beach sands of Kerala. The incidence of schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis, Alzheimer's diseases, Parkinson's disease, endomyocardial fibrosis, chronic calcific pancreatitis, multinodular goitre, mucoid angiopathy and recurrent viral epidemics is high in the Kerala population exposed to low level of background irradiation. Actinidic archaea has been described as endosymbiots 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 terrestrial background radiation 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 background radiation can also induce heme oxygenase depleting heme and inducing ALA synthase. Porphyrins can undergo autooxidation generating biophotons and a quantal state. Porphyrin autooxidation is modulated by low level background radiation. Porphyrin microarrays can function as quantal computers storing information and can serve the purpose of extrasensory perception. 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 schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. Porphyrins can mediate the pathogenesis of low level background radiation inducing the above mentioned disease states. The paper attempts to explore the relationship between low level radioactivity, porphyrin synthesis and disease pathology[1-5]. The above mentioned objectives are studied in the report.

Materials and Methods

The following groups were included in the study: – low level background irradiation group, schizophrenia, autism, seizure disorder, metabolic syndrome x, cerebrovascular thrombosis, coronary artery disease, malignancy, systemic lupus erythematosis, multiple sclerosis, Alzheimer's diseases, Parkinson's disease, endomyocardial fibrosis, chronic calcific pancreatitis, multinodular goitre, mucoid angiopathy and recurrent viral epidemics, 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. There were also 10 normal people with right hemispheric dominance, left hemispheric dominance and bihemispheric 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. 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. 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 radiation 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 and those with exposure to low level of radiation 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 and those with exposure to low level of radiation 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 background irradiation 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
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
Low level radiation	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
Low level radiation	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
Low level radiation	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
Low level radiation	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
Low level radiation	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
Low level radiation	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	

Abbreviations

AD: Alzheimer's disease

MS: Multiple sclerosis

NHL: Non-hodgkin's lymphoma

DM: Diabetes mellitus

AIDS: Acquired immunodeficiency syndrome

CJD: Creutzfeldt's-jakob disease

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
Schizo	1.38	0.26	4.00	0.00	51.17	3.65	274.88	8.73	0.036	0.009
Seizure	1.23	0.26	4.00	0.00	50.04	3.91	278.90	11.20	0.038	0.007
HD	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
MS	1.21	0.21	4.00	0.00	47.90	6.99	280.89	11.25	0.034	0.009
SLE	1.50	0.33	4.00	0.00	48.20	5.53	278.59	11.51	0.038	0.008
NHL	1.26	0.23	4.00	0.00	51.08	5.24	283.39	10.67	0.041	0.006
Glio	1.27	0.24	4.00	0.00	51.57	2.66	278.19	12.80	0.038	0.007
DM	1.35	0.26	4.00	0.00	51.98	5.05	280.89	10.58	0.041	0.005
CAD	1.22	0.16	4.00	0.00	50.00	5.91	280.89	13.79	0.038	0.009
CVA	1.33	0.27	4.00	0.00	51.06	4.83	287.33	9.47	0.037	0.007
AIDS	1.31	0.24	4.00	0.00	50.15	6.96	278.58	12.72	0.039	0.010
CJD	1.48	0.27	4.00	0.00	49.85	6.40	286.16	10.90	0.039	0.006
Autism	1.19	0.24	4.00	0.00	52.87	7.04	274.52	9.29	0.036	0.006
DS	1.34	0.25	4.00	0.00	47.28	3.55	283.04	9.17	0.035	0.009
Cerebral Palsy	1.44	0.19	4.00	0.00	53.49	4.15	273.70	12.37	0.038	0.008
CRF	1.26	0.26	4.00	0.00	49.39	5.51	285.51	8.79	0.039	0.008
Cirr/Hep Fail	1.50	0.20	4.00	0.00	46.82	4.73	275.97	10.66	0.037	0.010
Muc Angio	1.40	0.32	4.00	0.00	46.37	4.87	290.37	9.10	0.039	0.010
EMF	1.51	0.29	4.00	0.00	47.47	4.34	287.49	9.81	0.035	0.008
CCP/MNG	1.35	0.22	4.00	0.00	48.54	5.97	277.50	7.51	0.040	0.006
Low level background radiation	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
Schizo	78.23	7.13	66.16	6.51	42.50	3.23	267.81	64.05	401.49	50.73
Seizure	79.28	4.55	68.28	6.02	46.54	4.55	290.44	57.65	436.71	52.95
HD	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
MS	80.18	5.67	64.00	7.33	46.85	3.49	277.36	15.48	440.35	25.34
SLE	81.03	6.22	65.01	5.42	48.55	3.81	294.51	58.62	447.39	39.84
NHL	77.98	5.68	63.21	6.55	47.17	4.86	310.25	40.44	495.98	39.11
Glio	79.18	5.88	67.67	5.69	46.84	4.43	304.19	14.16	479.35	58.86
DM	78.36	6.68	64.72	6.81	48.15	3.36	285.46	29.46	422.27	33.86
CAD	78.15	3.72	66.66	7.77	47.00	3.81	314.01	17.82	426.14	24.28
CVA	77.59	5.24	69.02	4.86	46.33	4.01	320.85	24.73	402.16	33.80
AIDS	79.17	5.88	67.78	4.41	48.03	3.64	306.61	22.47	429.72	24.97
CJD	80.41	5.70	66.99	3.71	47.94	5.33	317.92	29.63	429.24	18.29
Autism	76.71	5.25	68.16	4.92	42.04	2.38	318.84	82.90	423.29	47.57
DS	80.30	6.65	64.99	6.72	45.69	4.18	258.33	37.85	421.52	36.57
Cerebral Palsy	80.02	6.82	65.56	6.28	44.58	4.52	280.16	26.14	431.39	28.88
CRF	81.36	5.37	67.61	5.55	46.81	4.62	301.78	48.22	427.57	33.55
Cirr/Hep Fail	77.61	4.42	66.28	6.55	48.23	2.36	276.51	16.66	436.44	25.65
Muc Angio	79.38	5.14	67.86	5.65	44.08	2.81	303.86	13.91	441.58	25.51
EMF	80.04	4.69	64.76	5.23	44.82	3.46	300.90	31.96	443.22	38.14
CCP/MNG	80.34	4.73	66.68	4.14	48.70	3.35	287.09	15.63	442.85	49.61
Low level background radiation	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
Schizo	44.30	2.66	12.82	2.40	1.74	0.08	0.073	0.013	2.66	0.58
Seizure	49.59	1.70	13.03	0.70	1.84	0.07	0.070	0.015	3.09	0.65
HD	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
MS	50.81	3.21	11.87	1.84	1.81	0.10	0.079	0.007	3.05	0.52
SLE	52.94	3.67	12.95	1.53	1.82	0.08	0.061	0.006	2.85	0.34
NHL	54.80	4.04	11.76	1.37	1.84	0.08	0.077	0.011	3.01	0.55
Glio	53.73	5.34	13.68	1.67	1.76	0.11	0.073	0.012	2.70	0.62
DM	49.80	4.01	12.83	2.07	1.77	0.19	0.067	0.014	3.19	0.89
CAD	49.51	2.27	11.39	1.10	1.75	0.12	0.080	0.007	2.99	0.65
CVA	46.74	4.28	11.26	0.95	1.82	0.10	0.079	0.009	2.98	0.78
AIDS	49.32	5.13	11.60	1.23	1.79	0.08	0.072	0.013	3.29	0.63
CJD	50.02	4.58	11.76	1.32	1.82	0.09	0.066	0.009	3.21	0.95
Autism	47.50	2.87	12.37	2.09	1.83	0.16	0.072	0.014	2.67	0.80
DS	50.97	7.07	11.81	1.14	1.85	0.07	0.071	0.015	3.15	0.73
Cerebral Palsy	49.23	3.91	11.61	1.36	1.85	0.09	0.069	0.012	3.14	0.46
CRF	49.66	4.41	12.03	1.40	1.76	0.22	0.070	0.012	3.14	0.57
Cirr/Hep Fail	50.56	1.63	11.92	1.33	1.81	0.10	0.076	0.009	3.01	0.47
Muc Angio	47.86	3.34	12.13	1.10	1.78	0.24	0.067	0.014	2.92	0.55
EMF	51.37	4.86	12.61	2.00	1.79	0.07	0.074	0.009	3.12	0.60
CCP/MNG	50.36	3.49	12.01	1.53	1.84	0.07	0.073	0.011	3.15	0.46
Low level background radiation	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)	
	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
RHCD	2.24	0.44	12.39	1.23	25.99	8.10	100.51	12.32
LHCD	0.02	0.01	1.21	0.38	2.75	0.41	23.79	2.51
Schizo	1.26	0.19	11.58	0.90	22.07	1.06	96.54	9.96
Seizure	1.66	0.56	12.06	1.09	21.78	0.58	90.46	8.30
HD	1.27	0.26	12.65	1.06	24.28	1.69	95.44	12.04
AD	2.06	0.19	11.94	0.86	22.04	0.64	97.26	8.26
MS	1.63	0.26	11.81	0.67	23.32	1.10	102.48	13.20
SLE	1.59	0.22	11.73	0.56	23.06	1.49	100.51	9.79
NHL	1.73	0.26	11.91	0.49	22.83	1.24	95.81	12.18
Glio	1.48	0.32	13.00	0.42	22.20	0.85	96.58	8.75
DM	1.97	0.11	12.95	0.56	25.56	7.93	96.30	10.33
CAD	1.57	0.37	11.51	0.47	22.83	0.82	97.29	12.45
CVA	1.49	0.27	12.74	0.80	23.03	1.26	103.25	9.49
AIDS	1.59	0.38	12.29	0.89	24.87	4.14	95.55	7.20
CJD	1.69	0.43	12.19	1.22	23.02	1.61	96.50	5.93
Autism	2.03	0.12	12.48	0.79	21.95	0.65	92.71	8.43
DS	1.17	0.11	12.79	1.15	23.69	2.19	91.81	4.12
Cerebral Palsy	1.56	0.39	12.14	1.30	23.12	1.81	95.33	11.78
CRF	1.53	0.33	12.66	1.01	23.42	1.20	97.38	10.76
Cirr/Hep Fail	1.32	0.26	12.81	0.90	26.20	5.29	97.77	13.24
Muc Angio	1.35	0.29	12.84	0.74	23.64	1.43	96.19	12.15
EMF	1.56	0.48	12.72	0.92	25.35	5.52	103.32	13.04
CCP/MNG	1.51	0.38	12.23	0.94	23.66	1.64	94.36	8.06
Low level background radiation	1.37	0.27	12.26	1.00	23.31	1.46	103.28	11.47
F value	67.588		445.772		162.945		154.701	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 5

Group	RBC hexokinase (ug glu phos/hr/mgpro)		ACOA (mg/dl)		ACH (ug/ml)		Glutamate (mg/dl)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
NO/BHCD	1.66	0.45	8.75	0.38	75.11	2.96	0.65	0.03
RHCD	5.46	2.83	2.51	0.36	38.57	7.03	3.19	0.32
LHCD	0.68	0.23	16.49	0.89	91.98	2.89	0.16	0.02
Schizo	7.69	3.40	2.51	0.57	48.52	6.28	3.41	0.41
Seizure	6.29	1.73	2.15	0.22	33.27	5.99	3.67	0.38
HD	9.30	3.98	1.95	0.06	35.02	5.85	3.14	0.32
AD	8.46	3.63	2.19	0.15	42.84	8.26	3.53	0.39
MS	8.56	4.75	2.03	0.09	39.99	12.61	3.58	0.36
SLE	8.02	3.01	2.54	0.38	49.30	7.26	3.37	0.38
NHL	7.41	4.22	2.30	0.26	50.58	3.82	3.48	0.46
Glio	7.82	3.51	2.34	0.43	42.51	11.58	3.28	0.39
DM	7.05	1.86	2.17	0.40	41.31	10.69	3.53	0.44
CAD	8.88	3.09	2.37	0.44	49.19	6.86	3.61	0.28
CVA	7.87	2.72	2.25	0.44	37.45	7.93	3.31	0.43
AIDS	9.84	2.43	2.11	0.19	38.40	7.74	3.45	0.49
CJD	8.81	4.26	2.10	0.27	34.97	4.24	3.94	0.22
Autism	6.95	2.02	2.42	0.41	50.61	6.32	3.30	0.32
DS	8.68	2.60	2.01	0.08	39.34	8.15	3.30	0.48
Cerebral Palsy	7.92	3.32	2.06	0.35	40.79	9.34	3.24	0.34
CRF	7.75	3.08	2.24	0.32	37.52	4.37	3.26	0.43
Cirr/Hep Fail	8.99	3.27	2.13	0.17	46.20	4.95	3.25	0.40
Muc Angio	10.12	1.75	2.51	0.42	45.51	7.56	3.11	0.36
EMF	9.44	3.40	2.19	0.19	42.48	8.62	3.27	0.39
CCP/MNG	8.53	2.64	2.04	0.10	37.95	8.82	3.33	0.25
Low level background radiation	7.58	3.09	2.14	0.19	37.75	7.31	3.47	0.37
F value	18.187		1871.04		116.901		200.702	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 6

Group	Se. ammonia (ug/dl)		HMG CoA (HMG CoA/MEV)		Bile acid (mg/ml)	
	Mean	± SD	Mean	± SD	Mean	± SD
NO/BHCD	50.60	1.42	1.70	0.07	79.99	3.36
RHCD	93.43	4.85	1.16	0.10	25.68	7.04
LHCD	23.92	3.38	2.21	0.39	140.40	10.32
Schizo	94.72	3.28	1.11	0.08	22.45	5.57
Seizure	95.61	7.88	1.14	0.07	22.98	5.19
HD	94.60	8.52	1.08	0.13	28.93	4.93
AD	95.37	4.66	1.10	0.07	26.26	7.34
MS	93.42	3.69	1.13	0.08	24.12	6.43
SLE	101.18	17.06	1.14	0.07	19.62	1.97
NHL	91.62	3.24	1.12	0.10	23.45	5.01
Glio	93.20	4.46	1.10	0.09	23.43	6.03
DM	93.38	7.76	1.09	0.12	22.77	4.94
CAD	93.93	4.86	1.07	0.12	24.55	6.26
CVA	103.18	27.27	1.05	0.09	22.39	3.35
AIDS	92.47	3.97	1.08	0.11	23.28	5.81
CJD	93.13	5.79	1.09	0.12	21.26	4.81
Autism	94.01	5.00	1.12	0.06	23.16	5.78
DS	98.81	15.65	1.09	0.11	21.31	4.49
Cerebral Palsy	92.09	3.21	1.07	0.09	22.80	5.02
CRF	98.76	11.12	1.03	0.10	26.47	5.30
Cirr/Hep Fail	94.77	2.86	1.04	0.10	24.91	5.06
Muc Angio	92.40	4.34	1.12	0.08	24.37	4.38
EMF	95.37	5.76	1.08	0.08	25.17	3.80
CCP/MNG	93.42	5.34	1.01	0.09	23.87	4.00
Low level background radiation	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/Bihemispheric chemical dominance

RHCD: Right hemispheric chemical dominance

LHCD: Left hemispheric chemical dominance

HD: Huntington's disease

AD: Alzheimer's disease

MS: Multiple sclerosis

SLE: Systemic lupus erythematosis

NHL: Non-hodgkin's lymphoma

Glio: Glioma

DM: Diabetes mellitus

CAD: Coronary artery disease

CVA: Cerebrovascular accident

AIDS: Acquired immunodeficiency syndrome

CJD: Creutzfeldt's-jakob disease

DS: Down syndrome

CRF: Chronic renal failure

Cirr/Hep Fail: Cirrhosis/Hepatic failure

EMF: Endomyocardial fibrosis

CCP: Chronic calcific pancreatitis

Discussion

Low level background irradiation has been related to psychiatric disorders, autoimmune disease, malignancy, degenerations and metabolic syndrome. Actinidic archaea have been also related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease, neuronal degeneration, endomyocardial fibrosis, chronic calcific pancreatitis, multinodular goiter and mucoid angiopathy. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. Actinidic archaea can induce porphyrin synthesis. Low level background irradiation can also induce porphyrin synthesis. Porphyrins have been related to schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis, Alzheimer's diseases, endomyocardial fibrosis, chronic calcific pancreatitis, multinodular goiter and mucoid angiopathy. Porphyrins can mediate the pathogenesis of low level background radiation inducing the above mentioned disease states. The paper attempts to explore the relationship between low level radioactivity, porphyrin synthesis and disease pathology. 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 terrestrial background radiation 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 background radiation can also induce heme oxygenase depleting heme and inducing ALA synthase. Heme is a negative regulator of porphyrin synthesis and ALA synthase. Low level background irradiation induced heme deficiency induces 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].

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 autooxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Porphyrin autooxidation is modulated by low level of electromagnetic fields and geomagnetic fields. Cellular porphyrins 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 human porphyrin microarrays induced biophotons and quantal fields can modulate the source from which low level radioactivity were generated. Thus the porphyrin generated by extraneous low level radioactivity can interact with the source of low level radioactivity modulating it. Thus porphyrins can serve as a bridge between the human brain and the source of low level environmental radioactivity. The porphyrins can also serve as the source of communication with the environment. Environmental background radiation 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. The low level of radiation enhances porphyrin synthesis and serves the purpose of two way extrasensory perception and environmental communication.

Low level background radiation in the actinidic beach sands of Kerala would have contributed to abiogenesis. 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 micro organism 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].

Low level background irradiation and porphyrins would have contributed to the origin of Neanderthal races. Porphyrins also have evolutionary significance since porphyria is related to Neanderthal 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 Neanderthal 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 Neanderthal 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. The actinidic beach sands in the submerged lemurian supercontinent would have been the origin of Neanderthal races. The above mentioned diseases are more common in Neanderthal races of Kerala.

Low level background irradiation and its porphyrin messengers can regulate the brain mediating conscious and quantal perception. Porphyrin microarrays serve the purpose of quantal and conscious perception. The archaea and viroids via porphyrin synthesis can regulate the nervous system including the NMDA/GABA thalamocorticothalamic 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 thalamocorticothalamic pathway of conscious perception. 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 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 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 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[3, 4, 16]. Thus porphyrins microarrays can function as a quantal brain modulating extrasensory quantal perception. Thus low level of background irradiation can modulate conscious and quantal perception and regulate hemispheric dominance by inducing porphyrinogenesis.

Low level background radiation 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 immune activation, vasospasm and vascular disease. A vagal neuropathy underlines neoplastic and autoimmune processes as well as metabolic syndrome x related to low level background radiation. Low level radiation 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 epileptogenesis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate brain function and cell death[3, 4, 16]. Low level background radiation can contribute to neuronal degeneration and epileptogenesis.

Low level radioactivity by modulating porphyrin metabolism can generate redox stress to regulate cell functions. 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. Low level radioactivity and porphyrins have been related to schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. Low level radioactivity 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 radioactivity 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 radioactivity 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 radioactivity 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 radioactivity 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 photooxidation 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. Thus low level radioactivity and 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].

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 schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. 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. Low level background irradiation inhibits ferrochelatase and stimulates heme oxygenase. Low level background irradiation leads to heme deficiency. 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. Low level electromagnetic fields can induce the Warburg phenotype contributing to human disease.

The role of porphyrins and low level radioactivity in regulation of cell functions and neuro-immuno-endocrine integration is discussed. Low levels of radioactivity can induce digoxin synthesis. Low levels of radioactivity induce heme oxygenase and inhibit ferrochelatase and deplete heme inducing porphyrin 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. Digoxin can produce sodium potassium ATPase inhibition resulting in increase intracellular calcium and reduce intracellular magnesium. Porphyrins can combine with membranes modulating membrane function and produce sodium potassium ATPase inhibition. Increase in intracellular calcium can produce immune activation by inducing NFKB and open the mitochondrial PT pore producing mitochondrial dysfunction. Low level of radioactivity can modulate membrane, nucleic acid and protein structure and function via induction of porphyrin synthesis. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. Low level of radioactivity can modulate protein structure and function via increasing porphyrin synthesis. Porphyrins can complex with DNA and RNA modulating their function. Porphyrin interpolating with DNA can alter transcription and generate HERV expression. Low level radioactivity via porphyrins can modulate HERV expression and DNA transcription. Low level of radioactivity 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. There is thus a mitochondrial dysfunction and redox stress induced by low level background irradiation. The cytochrome P450 enzymes involved in steroid and bile acid synthesis have reduced activity leading to steroid- cortisol, activated vitamin D, sex hormones, bile acid deficiency states. This can lead to adrenal dysfunction and cortisol deficiency. This can also contribute to deficiency of sex hormones, estrogens and testosterone. The low level radioactivity induced activated vitamin D deficiency can produce immune activation. Bile acid deficiency can lead to metabolic syndrome x. Low level radioactivity related 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. Low level of background irradiation can lead to vasospasm and vascular disease. Heme has got cytoprotective, neuroprotective, anti-inflammatory and antiproliferative effects.

Low level irradiation induced heme deficiency can lead to cell death, degeneration, autoimmune disease and cancer. Heme is also involved in the stress response. Heme deficiency leads to metabolic syndrome, immune disease, degenerations and cancer[3-5]. Low level background irradiation can also physiologically modulate cell functions and neuro-immuno-endocrine-genetic integration via induction of porphyrin synthesis.

Low level radioactivity 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 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]. Low level radioactivity and porphyrinogenesis can contribute to autoimmune disease. Low level radioactivity 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 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]. Low level radioactivity can contribute to metabolic syndrome x and vascular thrombosis. Low level radioactivity 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 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]. Low level radioactivity can contribute to oncogenesis. Low level radioactivity 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. Low level radioactivity and porphyrins can contribute to prion disease. Low level radioactivity 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. Low level of radioactivity can contribute to the retroviral state. Low level radioactivity and porphyrins can modulate predilection to infections. The porphyrins in the blood can combine with bacteria and viruses and the photooxidation generated free radicals can kill them. Low level radioactivity by modulating porphyrin metabolism can lead to increase predilection for viral and bacterial infections. The archaeal porphyrins can modulate bacterial and viral infections. The archaeal porphyrins are regulatory molecules keeping other prokaryotes and viruses on check[3, 4]. A porphyrin metabolic defect produces heme deficiency and porphyrin autooxidation produces redox stress. Both of these factors induce the Warburg phenotype and increase glycolysis and inhibit pyruvate dehydrogenase. The increase in glycolysis with blockade of TCA cycle produces increase fructose 1, 6 diphosphate which enters the pentose phosphate pathway generating aminosugars for MPS synthesis. This produces MPS accumulation in tissues and contributes to endomyocardial fibrosis, chronic calcific pancreatitis, multinodular goiter and mucoid angiopathy.

The study elucidates the relationship between low level background irradiation, porphyrin synthesis and disease patterns. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states-metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis is described. The porphyrins can contribute to the role of low level electromagnetic fields on 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. Low level of background irradiation induces porphyrinogenesis and contributes to the pathogenesis of the above mentioned disorders. Low level background irradiation and its messenger 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 porphyrins functions as key regulatory molecules with mitochondrial benzodiazepine receptors playing an important role. The low level background irradiation from the mineral sands of Kerala induces porphyrin synthesis, regulates cell function, induces neuro-immuno-genetic-endocrine integration and leads to disease pathology.

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