Chapter 1

Meditation Related Metabolonomic Changes - Endosymbiotic Actinidic Archaeal Cholesterol Catabolic Syndrome

Introduction

Meditation can induce heme oxygenase activity. Heme oxygenase induction suppresses ALA synthase. Thus heme is depleted from the system. There is increased porphyrin synthesis leading onto porphyrinuria and porphyria. The stimulus for porphyrin synthesis comes from heme deficiency. Porphyrins can organize into self replicating supramolecular structures called porphyrions which are induced by meditative practices. The porphyrins can self organize to form macromolecular structures which can self replicate to form a porphyrin organism. The photon induced transfer of electrons along the macromolecule can lead to light induced ATP synthesis. The porphyrins can form a template on which RNA and DNA can form generating viroids. The porphyrins can also form a template on which prions can form. They all can join together - RNA viroids, DNA viroids, prions - to form primitive archaea. Thus the archaea are capable of self replication on porphyrin templates. This leads to the generation of endosymbiotic nanoarchaea and viroids consequent to meditation.

Actinidic archaea have been implicated in the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration.[1-9] Actinide based primitive organism like archaea have a mevalonate pathway and cholesterol catabolism. Cholesterol catabolism by actinidic archaea can lead to cholesterol depletion and a hypocholesterolemic state contributing to the pathogenesis of these disorders.[10-17]

Archaea can use cholesterol as a carbon and energy source. Archaeal cholesterol catabolism can lead to multiple systemic disease. Low cholesterol values in populations have been related to high mortality. The archaeal cholesterol catabolizing enzymes were studied and the results in presented in

this paper. This can be described as the endosymbiotic actinidic archaeal cholesterol catabolic syndrome.[10-17]

Materials and Methods

The following groups were included in the study: - meditation group, endomyocardial fibrosis, Alzheimer's disease, multiple sclerosis, non-Hodgkin's lymphoma, metabolic syndrome x with cerebrovascular thrombosis and coronary artery disease, schizophrenia, autism, seizure disorder, Creutzfeldt Jakob's disease and acquired immunodeficiency syndrome. There were 10 patients in each group and each patient had an age and sex matched healthy control selected randomly from the general population. The blood samples were drawn in the fasting state before treatment was initiated. Plasma from fasting heparinised blood was used and the experimental protocol was as follows (I) Plasma+phosphate buffered saline, (II) same as I+cholesterol substrate, (III) same as II+rutile 0.1 mg/ml, (IV) same as II+ciprofloxacine and doxycycline each in a concentration of 1 mg/ml. Cholesterol substrate was prepared as described by Richmond.[18] 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, polycyclic aromatic hydrocarbon, digoxin, bile acid, cholesterol oxidase activity measured by hydrogen peroxide liberation, pyruvate, butyrate and propionate were estimated.[19-21] 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. Informed consent of the subjects and the approval of the ethics committee were obtained for the study. The statistical analysis was done by ANOVA.

Results

Plasma of control subjects showed increased levels of the above mentioned parameters with after incubation for 1 hour and addition of cholesterol substrate resulted in still further significant increase in these parameters. The plasma of patients showed similar results but the extent of increase was more. The addition of antibiotics to the control plasma caused a decrease in all the parameters while addition of rutile increased their levels. The addition of antibiotics to the patient's plasma caused a decrease in all the parameters while addition of rutile increased their levels but the extent of change was more in patient's sera as compared to controls. The results are expressed in tables 1-4 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time.

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
Meditation	22.29	1.66	59.02	7.50	23.23	1.97	65.89	5.05
CJD	22.06	1.61	57.81	6.04	23.46	1.91	61.56	4.61
Autism	21.68	1.90	57.93	9.64	22.61	1.42	64.48	6.90
EMF	22.70	1.87	60.46	8.06	23.73	1.38	65.20	6.20
F value	306.749		130.054		391.318		257.996	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 2. *Effect of rutile and antibiotics on butyrate and propionate generation from cholesterol.*

Group	Butyrate % change (Increase with Rutile)		Butyrate % change (Decrease with Doxy+Cipro)		Propionate % change (Increase with Rutile)		Propionate % change (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
Meditation	23.32	1.71	63.15	7.62	23.45	1.79	66.32	3.63
CJD	22.86	1.91	63.66	6.88	23.17	1.88	68.53	2.65
Autism	23.52	1.49	63.24	7.36	23.20	1.57	66.65	4.26
EMF	23.29	1.67	60.52	5.38	22.29	2.05	61.91	7.56
F value	380.721		171.228		372.716		556.411	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 3. *Effect of rutile and antibiotics on digoxin and bile acids.*

Group	Digoxin (ng/ml) (Increase with Rutile)		Digoxin (ng/ml) (Decrease with Doxy+Cipro)		Bile Acids % change (Increase with Rutile)		Bile Acids % change (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	0.11	0.00	0.054	0.003	4.29	0.18	18.15	0.58
Schizo	0.55	0.06	0.219	0.043	23.20	1.87	57.04	4.27
Seizure	0.51	0.05	0.199	0.027	22.61	2.22	66.62	4.99
AD	0.55	0.03	0.192	0.040	22.12	2.19	62.86	6.28
MS	0.52	0.03	0.214	0.032	21.95	2.11	65.46	5.79
NHL	0.54	0.04	0.210	0.042	22.98	2.19	64.96	5.64
DM	0.47	0.04	0.202	0.025	22.87	2.58	64.51	5.93
Meditation	0.56	0.05	0.220	0.052	22.29	1.47	64.35	5.58
CJD	0.53	0.06	0.212	0.045	23.30	1.88	62.49	7.26
Autism	0.53	0.08	0.205	0.041	22.21	2.04	63.84	6.16
EMF	0.51	0.05	0.213	0.033	23.41	1.41	58.70	7.34
F value	135.116		71.706		290.441		203.651	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 4. *Effect of rutile and antibiotics on pyruvate and hydrogen peroxide.*

Group	Pyruvate % change (Increase with Rutile)		Pyruvate % change (Decrease with Doxy+Cipro)		H_2O_2 % (Increase with Rutile)		H_2O_2 % (Decrease with Doxy+Cipro)	
	Mean	**±SD**	**Mean**	**±SD**	**Mean**	**±SD**	**Mean**	**±SD**
Normal	4.34	0.21	18.43	0.82	4.43	0.19	18.13	0.63
Schizo	20.99	1.46	61.23	9.73	22.50	1.66	60.21	7.42
Seizure	20.94	1.54	62.76	8.52	23.81	1.19	61.08	7.38
AD	22.63	0.88	56.40	8.59	22.65	2.48	60.19	6.98
MS	21.59	1.23	60.28	9.22	21.14	1.20	60.53	4.70
NHL	21.19	1.61	58.57	7.47	23.35	1.76	59.17	3.33
DM	20.67	1.38	58.75	8.12	23.27	1.53	58.91	6.09
Meditation	21.21	2.36	58.73	8.10	23.32	1.71	63.15	7.62
CJD	21.07	1.79	63.90	7.13	22.86	1.91	63.66	6.88
Autism	21.91	1.71	58.45	6.66	23.52	1.49	63.24	7.36
EMF	22.29	2.05	62.37	5.05	23.29	1.67	60.52	5.38
F value	321.255		115.242		380.721		171.228	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Discussion

There was increase in cytochrome F420 indicating archaeal growth. The archaea can synthesize and use cholesterol as a carbon and energy source.[22-24] 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.[22-24] The archaeal beta hydroxyl steroid dehydrogenase activity indicating digoxin synthesis and archaeal cholesterol hydroxylase activity indicating bile acid synthesis were increased.[22-24] The archaeal cholesterol oxidase activity was increased resulting in generation of pyruvate and hydrogen peroxide.[22-24] The pyruvate gets converted to glutamate and ammonia by the GABA shunt pathway. The archaeal

aromatisation of cholesterol generating PAH was also detected.[22-24] This indicates archaeal cholesterol aromatase activity. The archaeal cholesterol side chain oxidase activity generates butyrate and propionate. Thus archaeal cholesterol oxidase, cholesterol aromatase, cholesterol side chain oxidase, cholesterol hydroxylase and beta hydroxyl steroid dehydrogenase activity were detected in high levels in the patient population of endomyocardial fibrosis, Alzheimer's disease, multiple sclerosis, non-Hodgkin's lymphoma, metabolic syndrome x with cerebrovascular thrombosis and coronary artery disease, schizophrenia, autism, seizure disorder, Creutzfeldt Jakob's disease and acquired immunodeficiency syndrome. The archaeal cholesterol catabolizing enzymes were actinide dependent. The archaea can undergo magnetite and calcium carbonate mineralization and can exist as calcified nanoforms.[25] This leads to a cholesterol depleted state and hypocholesterolemic syndrome in patients with schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration.

Low cholesterol has been related to multiple systemic diseases. Low cholesterol is detected in patients with autism and schizophrenia. Low cholesterol is also associated with neuronal degenerations like Alzheimer's disease and Parkinson's disease. Cholesterol is required for the formation of synaptic connectivity in neuronal cultures. Depletion of cholesterol from the brain results in loss of synaptic connectivity in multiple neuronal circuits contributing to neuropsychiatric disorders and neuronal degeneration. Low cholesterol has also been related to malignancy. Cholesterol is required for contact inhibition. Absence of cholesterol results in loss of contact inhibition and uncontrolled cell proliferation. Low cholesterol has been related to autoimmune disease.[10-17]

The gut endotoxins and lipopolysaccharides are absorbed along with fat producing the syndrome of metabolic endotoxaemia. The endotoxins and lipopolysaccharides can combine with lipoproteins and are detoxified. Metabolic endotoxaemia produces chronic immune activation and generation of superantigens. This has been related to the genesis of autoimmune disease. Metabolic endotoxaemia results in immune activation and generation of TNF alpha which modulates the insulin receptor producing insulin resistance. Insulin resistance is related to metabolic syndrome x and vascular thrombosis. Metabolic endotoxaemia has been related to neuronal degenerations like Alzheimer's disease and Parkinson's disease. Metabolic endotoxaemia related chronic immune activation drives the retroviral state. Metabolic endotoxaemia can induce NFKB which can drive malignant cell transformation. Thus hypocholesterolemia leads to non-detoxification of endotoxins and lipopolysaccharides resulting in metabolic syndrome x, neuronal degnerations and autoimmune disease.[10-17]

Infections have been related to schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. H. pylori infection and nocardiosis has been related to Parkinson's disease. Chlamydial infection and actinomycosis has been related to Alzheimer's disease. Clostridial infection has been related to motor neuron disease. Atypical mycobacterial infection had been related to malignancy like lymphoma. Staphylococal infections have been related to carcinoma of the breast. Gut bacterial infections had been related to rheumatoid disease. Toxoplasmosis has been related to schizophrenia. Gut bacteria with increase in gut firmicutes and decrease in bacteroides have been related to metabolic syndrome x. Chlamydial infections have been related to vascular disease. Low cholesterol leads to lack of lipoprotein binding to endotoxins.[10-17] The endotoxins and lipopolysaccharides are not detoxified.

Viral diseases have been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. The virus binds to lipid microdomains in the cell membrane. Cholesterol depletion leads to alteration in lipid microdomains and increased entry of virus in the cell. Herpes virus infection and borna virus disease leads to schizophrenia. Enterovirus infection has been associated with motor neuron disease. Corona virus infection predisposes to Parkinson's disease. Herpes virus infection is implicated in Alzheimer's disease. Herpes virus infection and EBV infections predisposed to SLE. Retroviral infection - exogenous and endogenous have been related to schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. CMV infection and herpes infection has been related to atherogenesis. Prion disease has been related to alterations in cholesterol metabolism. Thus a cholesterol depleted state can lead to increased predilection to viral infection and systemic disease.[10-17]

The actinidic archaea uses cholesterol catabolism to generate energy. The cholesterol catabolizing enzymes of the archaea are dependent on actinides. The archaeal cholesterol catabolism leads to a cholesterol depleted state and systemic disease. Cholesterol depleted state have been related to high mortality. This can be described as the endosymbiotic actinidic archaeal cholesterol catabolic syndrome.[10-17]

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