

Chapter 5

Meditation Related Metabolomic Changes -
Endosymbiotic Actinidic Archaeal Synthesis of
Bile Acids from Cholesterol Regulates
Cellular Function

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.

Endomyocardial fibrosis along with the root wilt disease of coconut is endemic to Kerala with its radioactive actinide beach sands. Actinides like rutile as well as organisms like phytoplasmas and viroids have been implicated in the etiology of these diseases.¹⁻⁴ Bile acids are considered as steroidal hormones with endocrine, metabolic and neuroregulatory functions. Bile acids has been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration.⁴ The possibility of endogenous bile acid synthesis by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism was considered.⁵⁻⁸ An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described.^{7,9}

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+ciprofloxacin and doxycycline each in a concentration of 1 mg/ml. Cholesterol substrate was prepared as described by Richmond.¹⁰ Aliquots were withdrawn at zero time immediately after mixing and after incubation at 37 °C for 1 hour. The following estimations were carried out: - Cytochrome F420 and bile acids.¹¹⁻¹³ Cytochrome F420 was estimated fluorimetrically (excitation wavelength 420 nm and emission wavelength 520 nm). 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 table 1 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 archaeal bile acids.

Group	CYT F420 % (Increase with Rutile)		CYT F420 % (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	4.48	0.15	18.24	0.66	4.29	0.18	18.15	0.58
Schizo	23.24	2.01	58.72	7.08	23.20	1.87	57.04	4.27
Seizure	23.46	1.87	59.27	8.86	22.61	2.22	66.62	4.99
AD	23.12	2.00	56.90	6.94	22.12	2.19	62.86	6.28
MS	22.12	1.81	61.33	9.82	21.95	2.11	65.46	5.79
NHL	22.79	2.13	55.90	7.29	22.98	2.19	64.96	5.64
DM	22.59	1.86	57.05	8.45	22.87	2.58	64.51	5.93
Meditation	22.29	1.66	59.02	7.50	22.29	1.47	64.35	5.58
CJD	22.06	1.61	57.81	6.04	23.30	1.88	62.49	7.26
Autism	21.68	1.90	57.93	9.64	22.21	2.04	63.84	6.16
EMF	22.70	1.87	60.46	8.06	23.41	1.41	58.70	7.34
F value	306.749		130.054		290.441		203.651	
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.^{6, 14} 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.¹⁵ The archaeal cholesterol hydroxylase activity indicating bile acid synthesis were increased.⁸

The archaeal bile acids are steroidal hormones which can bind GPCR and modulate D2 regulating the conversion of T4 to T3 which activates uncoupling proteins reducing redox stress. Bile acids can activate the transcription factor NRF ½ inducing NQO1, GST, HOI reducing redox stress. Bile acids are neuroprotective and help to prevent neurodegenerative process. Bile acids can bind FXR regulating insulin receptor sensitivity. Bile acid deficiency leads to insulin resistance and metabolic syndrome x. Bile acids can bind PXR inducing the bile acid shunt pathway of cholesterol detoxification.²⁵ Bile acid deficiency leads to cholesterol toxicity. Bile acid binding to FXR receptor inhibits tissue fibrosis and connective tissue deposition. Bile acid deficiency may play a role in MPS and collagen deposition in EMF, CCP, MNG and mucoid angiopathy. Bile acids can bind macrophage GPCR and VDR producing immunosuppression and inhibiting NFkB. This helps to modulate the archaea and viroid induced chronic immune activation. Bile acid deficiency can contribute to autoimmune disease. The archaeal bile acids are chemically diverse and structurally different from human bile acids. The archaeal bile acids can bind olfactory GPCR receptors and stimulate the limbic lobe producing a sense of social identity. The dominance of archaeal bile acids over human bile acids in stimulating the olfactory GPCR - limbic lobe pathway leads to loss of social identity and schizophrenia/autism. Bile acids tend to have an inhibitory effect on cell proliferation and prevent malignant transformation.¹⁶

Archaeal bile acids binding to VDR has a inhibitory effect on cell proliferation. Bile acids thus can modulate cell death and cell proliferation. It can regulate metabolism via modulating mitochondrial uncoupling proteins and

via FXR insulin receptor sensitivity. Bile acids can modulate limbic lobe and brain functions via GPCR receptor and olfactory pathways. Bile acids can regulate immune function via macrophage GPCR and VDR. Archaeal bile acids can produce neuro-immuno-endocrine integration. Archaeal bile acids deficiency can contribute to the pathogenesis 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.

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