♦♦♦ Chapter 8 ♦♦♦

Endosymbiotic Actinidic Archaea and Viroids Mediated Model of Conscious/Quantal Perception and Regulation of Brain Function

Introduction

An endosymbiotic actinidic archaea and viroid mediated model of conscious and quantal perception is presented. Endomyocardial fibrosis (EMF) along with the root wilt disease of coconut is endemic to Kerala with its radioactive actinide beach sands. Actinides like rutile producing intracellular magnesium deficiency due to rutile-magnesium exchange sites in the cell membrane has been implicated in the etiology of EMF[1, 2]. Organisms like phytoplasmas and viroids have also been demonstrated to play a role in the etiology of these diseases[3, 4]. Actinidic archaea and viroids has been related to the pathogenesis of schizophrenia, autism and primary seizure disorder[2]. Actinidic archaea have a mevalonate pathway and cholesterol catabolism[5-8]. The role of endosymbiotic actinidic archaea and viroids in conscious and quantal perception as well as in regulation of brain function is discussed.

Materials and Methods

Informed consent of the subjects and the approval of the ethics committee were obtained for the study. The following groups were included in the study: – schizophrenia, autism and primary seizure disorder/primary generalized epilepsy. 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[9]. Aliquots were withdrawn at zero time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, dopamine, noradrenaline, serotonin, pyruvate, ammonia, glutamate, acetyl choline, hexokinase, HMG CoA reductase, digoxin and bile acids[10-13]. 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 statistical analysis was done by ANOVA.

Results

The parameters checked as indicated above were: – cytochrome F420, free RNA, free DNA, muramic acid, polycyclic aromatic hydrocarbon, hydrogen peroxide, serotonin, pyruvate, ammonia, glutamate, cytochrome C, hexokinase, ATP synthase, HMG CoA reductase, digoxin and bile acids. 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 1-8 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 F 420 and noradrenaline.*

Group	CYT F420 % (Increase with Rutile)		CYT F420 % (Decrease with Doxy)		Noradrenaline % (Increase with Rutile)		Noradrenaline % (Decrease with Doxy+Cipro)	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Normal	4.48	0.15	18.24	0.66	4.43	0.19	18.13	0.63
Schizo	23.24	2.01	58.72	7.08	22.50	1.66	60.21	7.42
Seizure	23.46	1.87	59.27	8.86	23.81	1.19	61.08	7.38
Autism	21.68	1.90	57.93	9.64	23.52	1.49	63.24	7.36
F value	306.749		130.054		380.721		171.228	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 2 *Effect of rutile and antibiotics on dopamine and Serotonin.*

Group	DOPAMINE % change (Increase with Rutile)		DOPAMINE % change (Decrease with Doxy)		Serotonin % change (Increase with Rutile)		Serotonin % 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	21.88	1.19	66.28	3.60	23.02	1.65	67.61	2.77
Seizure	22.29	1.33	65.38	3.62	22.13	2.14	66.26	3.93
Autism	22.76	2.20	67.63	3.52	22.79	2.20	64.26	6.02
F value	403.394		680.284		348.867		364.999	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 3 *Effect of rutile and antibiotics on free DNA and RNA.*

Group	DNA % change (Increase with Rutile)		DNA % change (Decrease with Doxy)		RNA % change (Increase with Rutile)		RNA % change (Decrease with Doxy)	
	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
Autism	22.12	2.44	63.69	5.14	23.33	1.35	66.83	3.27
F value	337.577		356.621		427.828		654.453	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 4 *Effect of rutile and antibiotics on HMG CoA reductase and PAH.*

Group	HMG CoA R % change (Increase with Rutile)		HMG CoA R % change (Decrease with Doxy)		PAH % change (Increase with Rutile)		PAH % change (Decrease with Doxy)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.30	0.20	18.35	0.35	4.45	0.14	18.25	0.72
Schizo	22.91	1.92	61.63	6.79	23.01	1.69	59.49	4.30
Seizure	23.09	1.69	61.62	8.69	22.67	2.29	57.69	5.29
Autism	22.72	1.89	64.51	5.73	22.61	1.42	64.48	6.90
F value	319.332		199.553		391.318		257.996	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 5 *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)	
	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
Autism	0.53	0.08	0.205	0.041	22.21	2.04	63.84	6.16
F value	135.116		71.706		290.441		203.651	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 6 *Effect of rutile and antibiotics on pyruvate and hexokinase.*

Group	Pyruvate % change (Increase with Rutile)		Pyruvate % change (Decrease with Doxy)		Hexokinase % change (Increase with Rutile)		Hexokinase % change (Decrease with Doxy)	
	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
Autism	21.91	1.71	58.45	6.66	22.88	1.87	65.45	5.08
F value	321.255		115.242		292.065		317.966	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 7 *Effect of rutile and antibiotics on hydrogen peroxide and acetyl choline.*

Group	H_2O_2 % (Increase with Rutile)		H_2O_2 % (Decrease with Doxy)		Acetyl Choline% (Increase with Rutile)		Acetyl Choline % (Decrease with Doxy)	
	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
Autism	23.52	1.49	63.24	7.36	23.20	1.57	66.65	4.26
F value	380.721		171.228		372.716		556.411	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 8 *Effect of rutile and antibiotics on glutamate and ammonia.*

Group	Glutamate % (Increase with Rutile)		Glutamate % (Decrease with Doxy)		Ammonia % (Increase with Rutile)		Ammonia % (Decrease with Doxy)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.34	0.21	18.43	0.82	4.40	0.10	18.48	0.39
Schizo	20.99	1.46	61.23	9.73	22.52	1.90	66.39	4.20
Seizure	20.94	1.54	62.76	8.52	22.83	1.90	67.23	3.45
Autism	21.91	1.71	58.45	6.66	23.20	1.57	66.65	4.26
F value	321.255		115.242		372.716		556.411	
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[14, 15]. 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[16]. There was also an increase in archaeal HMG CoA reductase activity indicating increased cholesterol synthesis by the archaeal mevalonate pathway. The archaeal beta hydroxyl steroid dehydrogenase activity indicating digoxin synthesis and archaeal cholesterol

hydroxylase activity indicating bile acid synthesis were increased[7]. The archaeal cholesterol oxidase activity was increased resulting in generation of pyruvate and hydrogen peroxide[15]. The pyruvate gets converted to glutamate and ammonia by the GABA shunt pathway. The pyruvate can get converted to acetyl CoA and acetyl choline. The archaeal aromatization of cholesterol generating PAH, serotonin and dopamine was also detected[17]. The archaeal glycolytic hexokinase activity and archaeal extracellular ATP synthase activity were increased. The archaea can undergo magnetite and calcium carbonate mineralization and can exist as calcified nanoforms[18].

The endosymbiotic actinidic archaea and viroids have got axonal and transynaptic transport functioning as biological neurotransmitters. The human brain can be compared to a well organised modified archaeal biofilm with archaeal derived viroids serving as messengers. 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 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[19]. Archaeal pyruvate can produce histone deacetylase inhibition resulting in 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[20]. The noncoding DNA is lengthened by integrating RNA viroidal complementary DNA with the integration going on as a continuing event. The archaea genome can also get integrated into human genome using integrase as has been described for trypanosomes[21]. The integrated viroids and archaea can undergo vertical transmission and can exist as genomic parasites[20, 21]. This

increases the length and alters the grammar of the noncoding region producing memes or memory of acquired characters[22]. The viroidal complementary DNA can function as jumping genes producing a dynamic genome important in storage of synaptic information, HLA gene expression and neurodevelopmental gene expression. The alteration in DNA sequences produced by viroidal complementary DNA jumping genes can lead onto schizophrenia, autism and primary seizure disorder. The RNA viroids can regulate mRNA function by RNA interference[19]. The phenomena of RNA interference can modulate T cell and B cell function, neuronal transmission and euchromatin/ heterochromatin expression. The RNA viroid induced mRNA interference can modulate dopaminergic, glutamatergic and serotoninergic synaptic transmission contributing to the pathogenesis of schizophrenia, autism and primary seizure disorder.

Pollution is induced by the primitive nanoarchaea synthesized PAH and methane leading on to redox stress. Redox stress leads to sodium potassium ATPase inhibition, inward movement of plasma membrane cholesterol, defective SREBP sensing, increased cholesterol synthesis and nanoarchaeal/mevalonate pathway bacterial growth[28]. Redox stress leads on to viroidal and archaeal multiplication. Redox stress can also lead to HERV reverse transcriptase and integrase expression. The noncoding DNA is formed of integrating RNA viroidal complementary DNA and archaea with the integration going on as a continuing event. The archaeal pox like dsDNA virus forms evolutionarily the nucleus. The integrated viroidal and archaeal sequences can undergo vertical transmission and can exist as genomic parasites. Bacteria and viruses have been related to the pathogenesis of schizophrenia, autism and primary seizure disorder[23-31].

The change in the length and grammar of the noncoding region produces eukaryotic speciation and individuality[32]. It is the increase in non coding region and HERV sequences of the genome that led to the evolution of the primate and

the human brain and its attendant property of conscious and quantal perception. It is the noncoding region of the genome with its archaeal, RNA viroidal complementary DNA and HERV sequences that makes for the human qualities of the hominid brain. Changes in the length of noncoding region can lead onto disorders of consciousness like schizophrenia[33]. A schizophrenia specific human endogenous retroviruses and change in the length and grammar of the noncoding region has been described in schizophrenia. The integration of nanoarchaea and viroids in to the eukaryotic and human genome produces a chimera which can multiply producing biofilm like multicellular structures having a mixed archaeal, viroidal and eukaryotic characters which is a regression from the multicellular eukaryotic tissue. This results in a new neuronal, metabolic, immune and tissue phenotype leading to human diseases like schizophrenia, autism and primary seizure disorder. The microchimeras formed can lead to polyploidy. Neuronal polyploidy and microchimeras have been described in schizophrenia and autism.

The archaea and viroids can regulate the nervous system including the NMDA/GABA thalamo-cortico-thalamic pathway mediating conscious perception[2, 34]. NMDA/GABA receptors can be modulated by digoxin induced calcium oscillations resulting in NMDA/ glutamic acid decarboxylase (GAD) activity induction, PAH increasing NMDA activity and inducing GAD as well as viroid induced RNA interference modulating NMDA/GABA receptors[2]. The cholesterol ring oxidase generated pyruvate can be converted by the GABA shunt pathway to glutamate and GABA. Increased NMDA transmission has been described in schizophrenia, autism and primary seizure disorder. The dipolar 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[2, 34]. The quantal perception mediated by actinidic archaea

and viroids gives rise to the phenomena of the collective unconscious. This can mediate extrasensory perceptive phenomena in humans. Schizophrenia and autism are described as a disorder of consciousness and increased integration of archaea and viroids into the genome can contribute to its neuropathogenesis. The archaea can regulate limbic lobe transmission with archaeal cholesterol aromatase/ring oxidase generated norepinephrine, dopamine, serotonin and acetyl choline[17]. The archaea can thus regulate the sympathetic and parasympathetic system regulating visceral function. Increased dopaminergic and serotoninergic transmission is important in the pathogenesis of schizophrenia and autism. The higher degree of integration of the archaea into the genome produces increased digoxin synthesis producing right hemispheric dominance and lesser degree producing left hemispheric dominance[2]. Right hemispheric dominance has been described in schizophrenia, autism and primary seizure disorder. The increased integration of archaea into the neuronal genome can produce increased cholesterol oxidase and aromatase mediated monoamine and NMDA transmission producing schizophrenia, autism and primary seizure disorder.

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[35]. The archaeal bile acids are important as modulators of the limbic lobe and gives social, group and racial identity to humans.

Archaea and RNA viroid can bind the TLR receptor induce NFKB producing immune activation and cytokine TNF alpha secretion. The archaeal DXP and mevalonate pathway metabolites can bind γδ TCR and digoxin induced calcium signalling can activate NFKB producing chronic immune activation[2, 36]. The

archaea and viroid induced chronic immune activation and generation of superantigens can lead on to autoimmune disease. Immune activation and autoimmunity is important in the pathogenesis of schizophrenia, autism and primary seizure disorder. Autoantibodies have been described in schizophrenia, autism and primary seizure disorder.

Archaea, viroids and digoxin can induce the host AKT PI3K, AMPK, HIF alpha and NFKB producing the Warburg metabolic phenotype[37]. The increased glycolytic hexokinase activity, decrease in blood ATP, leakage of cytochrome C, increase in serum pyruvate and decrease in acetyl CoA indicates the generation of the Warburg phenotype. There is induction of glycolysis, inhibition of PDH activity and mitochondrial dysfunction resulting in inefficient energetics. Mitochondrial dysfunction can lead onto NMDA excitotoxicity and cell death important in schizophrenia and primary seizure disorder. Cholesterol oxidase activity, increased glycolysis related NADPH oxidase activity and mitochondrial dysfunction generates free radicals important in the pathogenesis of schizophrenia, autism and primary seizure disorder. The accumulated pyruvate enters the GABA shunt pathway and is converted to citrate which is acted upon by citrate lyase and converted to acetyl CoA, used for cholesterol synthesis[37]. The pyruvate can be converted to glutamate and ammonia which is oxidised by archaea for energy needs. The increased cholesterol substrate leads to increased archaeal growth and digoxin synthesis leading to metabolic channeling to the mevalonate pathway. Hyperdigoxinemia is important in the pathogenesis of schizophrenia, autism and primary seizure disorder[2].

The Warburg phenotype can contribute to the pathogenesis of schizophrenia by augmenting the bacterial shikimic acid pathway. The upregulated glycolysis consequent to the Warburg phenotype produces phosphoenolpyruvate, a basic substrate for the bacterial shikimic acid pathway which can synthesise

monoamines and neuroactive alkaloids. The shikimic acid pathway can generate dopamine and serotonin producing the increased monoaminergic transmission in schizophrenia. The shikimic acid pathway can also synthesize the neuroactive alkaloids strychnine, nicotine, morphine, mescaline and LSD important in the pathogenesis of schizophrenia and autism. Endogenous neuroactive alkaloids have been described in schizophrenia, autism and primary seizure disorder by several workers[2]. The upregulated glycolysis can also contribute to increased NMDA and GABA transmission in the thalamo-cortico-thalamic pathway. The glycolytic pathway produces phosphoglycerate which is converted to phosphoserine and then serine which activates the NMDA receptor. The glycolytic enzyme glyceraldehyde-3-phosphate dehydrogenase is a GABA receptor kinase and activates GABA transmission. Thus the archaea and viroid induced Warburg phenotype can contribute to the pathogenesis of schizophrenia, autism and primary seizure disorder. The archaeal cholesterol catabolism can deplete the cell membranes of cholesterol resulting in alteration in lipid microdomains and their related neurotransmitter receptor contributing to the altered NMDA, serotoninergic and dopaminergic transmission in schizophrenia, autism and primary seizure disorder.

Thus the actinidic archaea and viroids can regulate brain function. The actinidic archaea and viroids can modulate multiple neurotransmitter systems – monoaminergic, glutamatergic, GABAergic and cholinergic. An actinidic archaea and viroid mediated model of conscious and quantal perception is postulated. The actinidic archaea and viroids also play a role in the genesis of hemispheric dominance. It is dysfunction of the archaea and viroidal mediated regulatory mechanisms of brain function and consciousness that leads to schizophrenia and autism.

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