

## A Cholesterol and Actinide Dependent Shadow Biosphere of Archaea and Viroids Indicating Cholesterol Based Abiogenesis- Evolution of the Biological Universe

Ravikumar Kurup A.<sup>1,\*</sup>; Parameswara Achutha Kurup<sup>1</sup>

<sup>1</sup>Professor of Metabolic Medicine and Neurology  
The Metabolic Disorders Research Centre, TC 4/1525, Gouri Sadan,  
Kattu Road, North of Cliff House, Kowdiar PO, Trivandrum, Kerala,  
India

\*Corresponding author.  
Email: ravikurup13@yahoo.in

Received 17 September 2011; accepted 19 November 2011

### Abstract

#### Aim

Endomyocardial fibrosis along with the root wilt disease of coconut is endemic to Kerala with its radioactive actinide beach sands. Actinides like rutile, endogenous digoxin as well as organisms like phytoplasmas and viroids have been implicated in the etiology of these diseases. Endogenous digoxin has been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. The possibility of endogenous digoxin synthesis by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism was considered.

#### Methods

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. The following estimations were carried out:- Cytochrome F420, free RNA, free DNA, polycyclic aromatic hydrocarbon, hydrogen peroxide, dopamine, serotonin, pyruvate, ammonia, glutamate, cytochrome C, hexokinase, ATP synthase, HMG CoA reductase, digoxin and bile acids.

#### 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 and rutile to the patient's plasma produced the same changes but the extent of change was more in patient's sera as compared to controls.

#### Conclusion

An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. Metal actinides in beach sands have been postulated to play a role in abiogenesis. A hypothesis of cholesterol as the primal prebiotic molecule synthesised on actinide surfaces with all other biomolecules arising from it and a self replicating cholesterol lipid organism as the initial life form is presented. A cholesterol based theory of abiogenesis and its role in the evolution of universe is elucidated.

**Key words:** Biological Universe; Cholesterol; Actinide

Ravikumar Kurup A., Parameswara Achutha Kurup (2011). A Cholesterol and Actinide Dependent Shadow Biosphere of Archaea and Viroids Indicating Cholesterol Based Abiogenesis- Evolution of The Biological Universe. *Advances in Natural Science*, 4(2), 33-41. Available from: URL: <http://www.cscanada.net/index.php/css/article/view/j.ans.1715787020110402.132> DOI: <http://dx.doi.org/10.3968/j.ans.1715787020110402.132>

### INTRODUCTION

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<sup>[1]</sup>. Endogenous digoxin, a steroidal glycoside which functions as a membrane sodium-potassium ATPase inhibitor has

also been related to its etiology due to the intracellular magnesium deficiency it produces<sup>[2]</sup>. Organisms like phytoplasmata and viroids have also been demonstrated to play a role in the etiology of these diseases<sup>[3,4]</sup>. Endogenous digoxin has been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration<sup>[2]</sup>. The possibility of endogenous digoxin synthesis by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism was considered<sup>[5,6,7]</sup>. Davies has put forward the concept of a shadow biosphere of organisms with alternate biochemistry present in earth itself<sup>[8]</sup>. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described<sup>[6]</sup>. Metal actinides in beach sands have been postulated to play a role in abiogenesis<sup>[6]</sup>. Actinide mineral like rutile, monazite and illmenite by surface metabolism would have contributed to abiogenesis<sup>[9]</sup>. A hypothesis of cholesterol as the primal prebiotic molecule synthesised on actinide surfaces with all other biomolecules arising from it and a self replicating cholesterol lipid organism as the initial life form is presented. The role of actinidic archaea in the genesis of the interstellar polycyclic aromatic hydrocarbons as well as the interstellar magnetic fields important in the evolution of the universe is hypothesized.

---

## 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:- 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 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<sup>[10]</sup>. 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, 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<sup>[11,12,13,14]</sup>. Cytochrome F420 was estimated fluorimetrically (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 1-7 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 Muramic Acid**

Group	CYT F420 % (Increase with Rutile)		CYT F420 % (Decrease with Doxy+Cipro)		Muramic acid % change (Increase with Rutile)		Muramic acid % 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
EMF	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
EMF	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 HMG CoA Reductase and ATP Synthase**

Group	HMG CoA R % change (Increase with Rutile)		HMG CoA R % change (Decrease with Doxy+Cipro)		ATP synthase % (Increase with Rutile)		ATP synthase % (Decrease with Doxy+Cipro)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.30	0.20	18.35	0.35	4.40	0.11	18.78	0.11
Schizo	22.91	1.92	61.63	6.79	23.67	1.42	67.39	3.13
Seizure	23.09	1.69	61.62	8.69	23.09	1.90	66.15	4.09
AD	23.43	1.68	61.68	8.32	23.58	2.08	66.21	3.69
MS	23.14	1.85	59.76	4.82	23.52	1.76	67.05	3.00
NHL	22.28	1.76	61.88	6.21	24.01	1.17	66.66	3.84
DM	23.06	1.65	62.25	6.24	23.72	1.73	66.25	3.69
AIDS	22.86	2.58	66.53	5.59	23.15	1.62	66.48	4.17
CJD	22.38	2.38	60.65	5.27	23.00	1.64	66.67	4.21
Autism	22.72	1.89	64.51	5.73	22.60	1.64	66.86	4.21
EMF	22.92	1.48	61.91	7.56	23.37	1.31	63.97	3.62
	F value 319.332 P value < 0.001		F value 199.553 P value < 0.001		F value 449.503 P value < 0.001		F value 673.081 P value < 0.001	

**Table 4**  
**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
AIDS	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 P value < 0.001		F value 71.706 P value < 0.001		F value 290.441 P value < 0.001		F value 203.651 P value < 0.001	

**Table 5**  
**Effect of Rutile and Antibiotics on Pyruvate and Hexokinase**

Group	Pyruvate % change (Increase with Rutile)		Pyruvate % change (Decrease with Doxy+Cipro)		Hexokinase % change (Increase with Rutile)		Hexokinase % change (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
EMF	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 Delta Amino Levulinic Acid**

Group	H <sub>2</sub> O <sub>2</sub> % (Increase with Rutile)		H <sub>2</sub> O <sub>2</sub> % (Decrease with Doxy+Cipro)		ALA % (Increase with Rutile)		ALA % (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
EMF	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	

**Table 7**  
**Effect of Rutile and Antibiotics on PAH and Serotonin**

Group	PAH % (Increase with Rutile)		PAH % (Decrease with Doxy+Cipro)		5 HT % change (Increase with Rutile)		5 HT % 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
AD	23.66	1.67	65.97	3.36	23.09	1.81	65.86	4.27
MS	22.92	2.14	67.54	3.65	21.93	2.29	63.70	5.63
NHL	23.81	1.90	66.95	3.67	23.12	1.71	65.12	5.58
DM	24.10	1.61	65.78	4.43	22.73	2.46	65.87	4.35
AIDS	23.43	1.57	66.30	3.57	22.98	1.50	65.13	4.87
CJD	23.70	1.75	68.06	3.52	23.81	1.49	64.89	6.01
Autism	22.76	2.20	67.63	3.52	22.79	2.20	64.26	6.02
EMF	22.28	1.52	64.05	2.79	22.82	1.56	64.61	4.95
	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	

## DISCUSSION

There was increase in cytochrome F420 indicating archaeal growth. The archaea can synthesise and use cholesterol as a carbon and energy source<sup>[15,16]</sup>. The archeal 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<sup>[17]</sup>. 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<sup>[17]</sup>. The archaeal cholesterol oxidase activity was increased resulting in generation of pyruvate and hydrogen peroxide<sup>[16]</sup>. The pyruvate gets converted to glutamate and ammonia by the GABA shunt pathway. The archaeal aromatization of cholesterol generating PAH, serotonin and dopamine was also detected<sup>[18]</sup>. 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<sup>[19]</sup>. 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<sup>[20]</sup>. 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<sup>[21]</sup>. 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<sup>[22]</sup>. The integrated viroids and archaea can undergo vertical transmission and can exist as genomic parasites<sup>[21,22]</sup>. This increases the length and alters the grammar of the noncoding region producing memes or memory of acquired characters<sup>[23]</sup>. The viroidal complementary DNA can function as jumping genes producing a dynamic genome important in storage of synaptic information, HLA gene expression and developmental gene expression. The RNA viroids can regulate mrna function by RNA interference<sup>[20]</sup>. The phenomena of RNA interference can modulate T cell and B cell function, insulin signaling lipid metabolism, cell growth and differentiation, apoptosis, neuronal transmission and euchromatin/heterochromatin expression.

The presence of muramic acid, HMG CoA reductase and cholesterol oxidase activity inhibited by antibiotics indicates the presence of bacteria with mevalonate pathway. The bacterial with mevalonate pathway include streptococcus, staphylococcus, actinomycetes, listeria, coxiella and borrelia<sup>[24]</sup>. The bacteria and archaea with mevalonate pathway and cholesterol catabolism had a evolutionarily advantage and constitutes the isoprenoidal clade organism with the archaea evolving into mevalonate pathway gram positive and gram negative organism through horizontal gene transfer of viroidal and virus genes<sup>[25]</sup>. The isoprenoidal clade prokaryotes develop into other groups of prokaryotes via viroidal/virus as well as eukaryotic horizontal gene transfer producing bacterial speciation<sup>[26]</sup>. The RNA viroids and its complementary DNA developed into cholesterol enveloped RNA and DNA viruses like herpes, retrovirus, influenza virus,

borna virus, cytomegalo virus and Epstein Barr virus by recombining with eukaryotic and human genes resulting in viral speciation. Bacterial and viral species are ill defined and fuzzy with all of them forming one common genetic pool with frequent horizontal gene transfer and recombination. Thus the multi and unicellular eukaryote with its genes serves the purpose of prokaryotic and viral speciation. The multicellular eukaryote developed so that their endosymbiotic archaeal colonies could survive and forage better. The multicellular eukaryotes are like bacterial biofilms. The archaea and bacteria with a mevalonate pathway uses the extracellular RNA viroids and DNA viroids for quorum sensing and in the generation of symbiotic biofilm like structures which develop into multicellular eukaryotes<sup>[27,28]</sup>. The endosymbiotic archaea and bacteria with mevalonate pathway still uses the RNA viroids and DNA viroids for the regulation of multicellular eukaryote. Pollution is induced by the primitive nanoarchaea and mevalonate pathway bacteria synthesised 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<sup>[29]</sup>. 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, archaeal and mevalonate pathway bacterial sequences can undergo vertical transmission and can exist as genomic parasites. The genomic integrated archaea, mevalonate pathway bacteria and viroids form a genomic reserve of bacteria and viruses which can recombine with human and eukaryotic genes producing bacterial and viral speciation. The change in the length and grammar of the noncoding region produces eukaryotic speciation and individuality<sup>[30]</sup>. The integration of nanoarchaea, mevalonate pathway prokaryotes 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, prokaryotic 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 disease.

The archaea and viroids can regulate the nervous system including the NMDA/GABA thalamocorticothalamic pathway mediating conscious perception<sup>[2,31]</sup>. NMDA/GABA receptors can be modulated by digoxin induced calcium oscillations resulting NMDA/GAD activity induction, PAH increasing NMDA activity and inducing GAD as well as viroid induced RNA interference<sup>2</sup>. The cholesterol ring oxidase generated

pyruvate can be converted by the GABA shunt pathway to glutamate and GABA. 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<sup>[2,31]</sup>. The archaea can regulate limbic lobe transmission with archaeal cholesterol aromatase/ring oxidase generated norepinephrine, dopamine, serotonin and acetyl choline<sup>18</sup>. 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<sup>[2]</sup>. The increased integration of archaea into the neuronal genome can produce increased cholesterol oxidase and aromatase mediated monoamine and NMDA transmission producing schizophrenia and autism. 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  $\gamma\delta$  TCR and digoxin induced calcium signaling can activate NFKB producing chronic immune activation<sup>[2,32]</sup>. The archaea and viroid induced chronic immune activation and generation of superantigens can lead on to autoimmune disease. Archaea, viroids and digoxin can induce the host AKT PI3K, AMPK, HIF alpha and NFKB producing the Warburg metabolic phenotype<sup>[33]</sup>. 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 and metabolic syndrome. The archaea and viroid generated cytokines can lead to TNF alpha induced insulin resistance and metabolic syndrome x. 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<sup>[33]</sup>. 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. 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, can activate NRF  $\frac{1}{2}$  inducing NQO1, GST, HOI reducing redox stress, can bind FXR regulating insulin receptor sensitivity and bind PXR inducing the bile acid shunt pathway of cholesterol detoxification<sup>[34]</sup>. The archaea and viroid induced monocyte activation and Warburg phenotype induced increased cholesterol synthesis leads to atherogenesis. The Warburg phenotype induced increased mitochondrial PT pore hexokinase, archaeal PAH and viroid induced RNA

interference can lead on to malignant transformation. The digoxin and PAH induced increased intracellular calcium can lead to PT pore dysfunction, cell death and neuronal degeneration<sup>[2]</sup>. The archaeal cholesterol catabolism can deplete the cell membranes of cholesterol resulting in organelle dysfunction and degeneration. The RNA viroids can recombine with HERV sequences and get encapsulated in microvesicles contributing to the retroviral state. The prion protein conformation is modulated by RNA viroid binding producing prion disease. The archaeal digoxin and rutile induced magnesium depletion can lead MPS deposition and produce EMF, CCP, MNG and mucoid angiopathy<sup>[2]</sup>.

The metal actinides provide radiolytic energy, catalysis for oligomer formation and provide a coordinating ion for metalloenzymes all important in abiogenesis<sup>6</sup>. The metal actinide surfaces would by surface metabolism generate acetate which could get converted to acetyl CoA and then to cholesterol which functions as the primal prebiotic molecule self organizing into self replicating supramolecular systems, the lipid organism<sup>[8,9,35]</sup>. Cholesterol by radiolysis by actinides would have formed PAH generating PAH aromatic organism<sup>8</sup>. Cholesterol radiolysis would generate pyruvate which would get converted to amino acids, sugars, nucleotides, porphyrins, fatty acids and TCA acids. Anastase and rutile surfaces can produce polymerization of amino acids, isoprenyl residues, PAH and nucleotides to generate the initial lipid organism, PAH organism, prions and RNA viroids which would have symbiosed to generate the archaeal protocell. The archaea evolved into gram negative and gram positive bacteria with a mevalonate pathway which had a evolutionary advantage and the symbiosis of archaea with gram negative organism generated the eukaryotic cell<sup>36</sup>. The data supports the persistence of an actinide and cholesterol based shadow biosphere which throws light on the actinide based origin of life and cholesterol as the premier prebiotic molecule.

The archaea can synthesise magnetite by biomineralization. The archaeal cholesterol catabolism can generate PAH. The archaea can exist as nanoarchaea and can have calcified nano forms. The actinidic magnetotactic nanoarchaea and its secreted PAH organisms are extremophiles and survive in the interstellar space and can contribute to the interstellar grains and magnetic fields which play a role in the formation of the galaxies and star systems<sup>[37]</sup>. The cosmic dust grains occupy the intergalactic space and are thought to be formed of magnetotactic bacteria identified according to their spectral signatures. According to the Hoyle's hypothesis, the cosmic dust magnetotactic bacteria plays a role in the formation of the intergalactic magnetic field. A magnetic field equal in strength to about one millionth part of the magnetic field of earth exists throughout much of our galaxy. The magnetic files can be used to trace the spiral arms of the galaxy following a pattern

of field lines that connect young stars and dust in which new stars are formed at a rapid rate. Studies have shown that a fraction of the dust particles have elongated shape similar to bacilli and they are systematically lined up in our galaxy. Moreover the direction of alignment is such that the long axes of the dust tend to be at right angles to the direction of the galactic magnetic field at every point. Magnetotactic bacteria have the property to affect the degree of alignment that is observed. The fact that the magnetotactic bacteria appear to be connected to the magnetic field lines that thread through the spiral arms of the galaxy connecting one region of star formation to another support a role for them in star formation and in the mass distribution and rotation of stars. The nutrient supply for a population of interstellar bacteria comes from mass flows out of supernovas populating the galaxy. Giants arising in the evolution of such stars experience a phenomenon in which material containing nitrogen, carbon monoxide, hydrogen, helium, water and trace elements essential for life flows continuously outward into space. The interstellar bacteria need liquid water. Water exists only as vapour or solid in the interstellar space and only through star formation leading to associated planets and cometary bodies can there be access to liquid water. To control conditions leading to star formation is of paramount importance in cosmic biology. The rate of star formation is controlled by two factors: Too high a rate of star formation produces a destructive effect of UV radiation and destroys cosmic biology. Star formation as stated before produces water crucial for bacterial growth. Cosmic biology of magnetotactic bacteria and star formation are thus closely interlinked. Systems like solar systems do not arise in random condensation of blobs of interstellar gas. Only by a rigorous control of rotation of various parts of the system would galaxies and solar system evolved. The key to maintaining control over rotation seems to lie in the intergalactic magnetic field as indeed the whole phenomena of star formation. The intergalactic magnetic fields owes its origin to the lining up of magnetotactic bacteria and the cosmic biology of interstellar bacteria can prosper only by maintaining a firm grip on the interstellar magnetic field and hence on the rate of star formation and type of star system produced. This points to a cosmic intelligence or brain capable of computation, analysis and exploration of the universe at large- of magnetotactic bacterial networks. The origin of life on earth according to the Hoyle's hypothesis would be by seeding of bacteria from the outer intergalactic space. Comets carrying microorganisms would have interacted with the earth. A thin skin of graphitized material around a single bacteria or clumps of bacteria can shield the interior from destruction by UV light. The sudden surge and diversification of species of plants and animals and their equally sudden extinction has seen from fossil records point to sporadic evolution produced by induction of fresh cometary genes with the arrival of each major new crop

of comets<sup>[38,39]</sup>. The interstellar PAH aromatic organism is formed from nanoarchaeal cholesterol catabolism. The PAH and cholesterol are the interconvertible primal prebiotic molecules. PAH aromatic organism and nanoarchaeal magnetite can have a wave particle existence and bridge the world of bosons and fermions. The nanoarchaea can form biofilms and the PAH aromatic organism can form a molecular quantum computing cloud in the biofilm which forms a interstellar intelligence regulating the formation of star systems and galaxies. The magnetite loaded nanoarchaeal biofilms and PAH aromatic organism quantal computing cloud can bridge the wave particle world functioning as the anthropic observer sensing gravity which orchestrates the reduction of the quantal world of possibilities in to the macroscopic world. The actinide based nanoarchaea can regulate the earth's carbon cycle by methanogenesis, nitrogen cycle by ammonia oxidation and rain formation by contributing the seeding nucleus. The earth's temperature and global warming and cooling are regulated by nanoarchaeal synthesised PAH from cholesterol and methanogenesis. The increased nanoarchaeal growth in ocean beds and soil leads to increased methane production and movement of the earth's crust producing tsunamis and massive earthquake leading to catastrophic mass extinction<sup>[40]</sup>. The eternal nanoarchaea survive and start the cycle of evolution once more. The actinide based nanoarchaea regulates the human system and biological universe.

## REFERENCES

- [1] Valiathan, M. S., Somers, K., & Kartha, C. C. (1993). *Endomyocardial Fibrosis*. Delhi, India: Oxford University Press.
- [2] Kurup, R., & Kurup, P. A. (2009). *Hypothalamic Digoxin, Cerebral Dominance and Brain Function in Health and Diseases*. New York: Nova Science Publishers.
- [3] Hanold, D., & Randies, J. W. (1991). Coconut Cadang-Cadang Disease and Its Viroid Agent. *Plant Disease*, 75, 330-335.
- [4] Edwin, B. T., & Mohankumaran, C. (2007). Kerala Wilt Disease Phytoplasma: Phylogenetic Analysis and Identification of a Vector. *Proutista Moesta, Physiological and Molecular Plant Pathology*, 71(1-3), 41-47.
- [5] Eckburg, P. B., Lepp, & P. W., Relman, D. A. (2003). Archaea and their potential role in human disease. *Infect Immun*, 71, 591-596.
- [6] Adam, Z. (2007). Actinides and Life's Origins. *Astrobiology*, 7, 6-10.
- [7] Schoner, W. (2002). Endogenous cardiac glycosides, a new class of steroid hormones, *Eur J Biochem*, 269, 2440-2448.
- [8] Davies, P. C. W., Benner, S. A., Cleland, C. E., Lineweaver, C. H., McKay, C. P., & Wolfe-Simon, F. (2009). Signatures of a Shadow Biosphere. *Astrobiology*, 10, 241-249.
- [9] Wächtershäuser, G. (1988). Before Enzymes and Templates: Theory of Surface Metabolism. *Microbiol Rev*, 52(4), 452-454.
- [10] Richmond, W. (1973). Preparation and Properties of a Cholesterol Oxidase from *Nocardia* Species and its Application to the Enzymatic Assay of Total Cholesterol in Serum. *Clin Chem*, 19, 1350-1356.
- [11] Snell, E. D., & Snell, C. T. (1961). *Colorimetric Methods of Analysis* (Vol. 3A). New York: Van Nostrand.
- [12] Glick D. (1971). *Methods of Biochemical Analysis* (Vol. 5). New York: Interscience Publishers.
- [13] Colowick, Kaplan, N. O. (1955). *Methods in Enzymology* (Vol. 2). New York: Academic Press.
- [14] Maarten A. H., Marie-Jose, M., Cornelia, G., van Helden-Meewsen, Fritz, E., & Marten, P. H. (1995). Detection of Muramic Acid in Human Spleen. *Infection and Immunity*, 63(5), 1652 – 1657.
- [15] Smit A., & Mushegian, A. (2000). Biosynthesis of Isoprenoids via Mevalonate in Archaea: The Lost Pathway. *Genome Res*, 10(10), 1468-84.
- [16] Van der Geize, R., Yam, K., Heuser, T., Wilbrink, M. H., Hara, H., & Anderton, M. C. (2007). A Gene Cluster Encoding Cholesterol Catabolism in a Soil Actinomycete Provides Insight into Mycobacterium Tuberculosis Survival In Macrophages. *Proc Natl Acad Sci USA*, 104(6), 1947-52.
- [17] Francis, A. J. (1998). Biotransformation of uranium and other actinides in radioactive wastes. *Journal of Alloys and Compounds*, 271(273), 78-84.
- [18] Probian, C., Wülfing, A., Harder, J. (2003). Anaerobic mineralization of quaternary carbon atoms: Isolation of denitrifying bacteria on pivalic acid (2,2-Dimethylpropionic acid). *Applied and Environmental Microbiology*, 69(3), 1866-1870.
- [19] Vainshtein, M., Suzina, N., Kudryashova, E., Ariskina, E. (2002). New Magnet-Sensitive Structures in Bacterial and Archaeal Cells, *Biol Cell*, 94(1), 29-35.
- [20] Tsagris, E. M., de Alba, A. E., Gozmanova, M., & Kalantidis, K. (2008). Viroids. *Cell Microbiol*, 10, 2168.
- [21] Horie, M., Honda, T., Suzuki, Y., Kobayashi, Y., Daito, T., & Oshida, T. (2010). Endogenous Non-Retroviral RNA Virus Elements in Mammalian Genomes. *Nature*, 463, 84-87.
- [22] Hecht, M., Nitz, N., Araujo, P., Sousa, A., Rosa, A., & Gomes, D. (2010). Genes from Chagas Parasite can Transfer to Humans and be Passed on to Children. Inheritance of DNA Transferred from American Trypanosomes to Human Hosts. *PLoS ONE*, 5, 2-10.
- [23] Flam, F. (1994). Hints of a Language in Junk DNA. *Science*, 266, 1320.
- [24] Horbach, S., Sahm, H., & Welle, R. (1993). Isoprenoid Biosynthesis in Bacteria: Two Different Pathways? *FEMS Microbiol Lett*, 111, 135-140.
- [25] Gupta, R. S. (1998). Protein Phylogenetics and Signature Sequences: A Reappraisal of Evolutionary Relationship Among Archaeobacteria, Eubacteria, and Eukaryotes. *Microbiol Mol Biol Rev*, 62, 1435-1491.
- [26] Hanage, W., Fraser, C., & Spratt, B. (2005). Fuzzy Species Among Recombinogenic Bacteria. *BMC Biology*, 3, 6-10.

- [27] Whitchurch, C. B., Tolker-Nielsen, T., Ragas, P. C., & Mattick, J. S. (2002). Extracellular DNA Required for Bacterial Biofilm Formation. *Science*, 295(5559), 1487.
- [28] Webb J. S., Givskov, M., & Kjelleberg, S. (2003). Bacterial Biofilms: Prokaryotic Adventures in Multicellularity. *Curr Opin Microbiol*, 6(6), 578–85.
- [29] Chen, Y., Cai, T., Wang, H., Li, Z., Loreaux, E., & Lingrel, J. B. (2009). Regulation of Intracellular Cholesterol Distribution by Na/K-ATPase. *J Biol Chem*, 284(22), 14881-90.
- [30] Poole, A. M. (2006). Did Group Ii Intron Proliferation in an Endosymbiont-Bearing Archaeon Create Eukaryotes? *Biol Direct*, 1, 36-40.
- [31] Lockwood, M. (1989). *Mind, Brain and the Quantum*. Oxford: B. Blackwell.
- [32] Eberl M., Hintz, M., Reichenberg, A., Kollas, A., Wiesner, J., & Jomaa, H. (2010). Microbial Isoprenoid Biosynthesis and Human  $\gamma\delta$  T Cell Activation. *FEBS Letters*, 544(1), 4-10.
- [33] Wallace, D. C. (2005). *Mitochondria and Cancer: Warburg Addressed*. *Cold Spring Harbor Symposia on Quantitative Biology*, 70, 363-374.
- [34] Lefebvre, P., Cariou, B., Lien, F., Kuipers, F., & Staels, B. (2009). Role of Bile Acids and Bile Acid Receptors in Metabolic Regulation. *Physiol Rev*, 89(1), 147 - 191.
- [35] Russell, M. J., & Martin, W. (2004). The Rocky Roots of the Acetyl-CoA Pathway. *Trends in Biochemical Sciences*, 29, 7.
- [36] Margulis, L. (1996). Archaeal-Eubacterial Mergers in the Origin of Eukarya: Phylogenetic Classification of Life. *Proc Natl Acad Sci USA*, 93, 1071-1076.
- [37] Tielens, A. G. G. M. (2008). Interstellar Polycyclic Aromatic Hydrocarbon Molecules. *Annual Review of Astronomy and Astrophysics*, 46, 289-337.
- [38] Wickramasinghe, C. (2004). The Universe: A Cryogenic Habitat for Microbial Life. *Cryobiology*, 48(2), 113-125.
- [39] Hoyle, F., & Wickramasinghe, C. (1988). *Cosmic Life-Force*. London: J.M. Dent and Sons Ltd.
- [40] Dun, D. (2005). *The Black Silent*. New York: Pinnacle Books.