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The Extinction of Homo Sapiens and Symbiotic Neanderthalisation: Relation to Archaeal Mediated RNA Viroidsand Amyloidosis

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Abstract

Introduction: Prion proteins have been implicated in systemic disorders like neurodegenerations, cancer and metabolic syndrome. The beta amyloid in Alzheimer's disease, alpha synuclein in parkinson's disease, the TAR protein in frontotemporal dementia and copper zinc dismutase in motor neuron disease behaves like prion proteins. Prion proteins like behavior is also seen in the tumour suppressor P_{53} protein in cancer and the islet cell associated amyloid in diabetes mellitus. Prion diseases are conformational diseases. The abnormal prion protein seeded into the system converts the normal proteins with prion like domains to abnormal configuration. This abnormal protein resists digestion by lysosomal enzymes after its half-life is over and results in deposition of amyloid plaques. This produces organ dysfunction. Prion phenomena were initially described for Creutzfeldt Jakob's disease, but now it is found to be wide spread in chronic disease pathogenesis. Ribonucleoproteins are well known to behave like prion proteins and form amyloid. We have demonstrated actinidic archaea which secretes RNA viroids in metabolic syndrome, neurodegenerations, cancer, autoimmune disease, schizophrenia, autism and CJD. The RNA viroids can bind with normal proteins with prion like domains eg., superoxide dismutase and produce a ribonucleoprotein resulting in prion phenomena and amyloidogenesis.

Materials and Methods: The following groups were included in the study:- 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+cerium 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. 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, Cytochrome F420 was estimated flourimetrically (excitation wavelength 420 nm and emission wavelength 520 nm).

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 cerium increased their levels. The addition of antibiotics to the patient's plasma caused a decrease in all the parameters while addition of cerium increased their levels but the extent of change was more in patient's sera as compared to controls.

Discussion: The actinidic archaeal growth results in increased digoxin synthesis and phenotypic conversion of homo sapiens to homo Neanderthals as reported earlier. The increased actinidic archaeal growth is due to global warming and these results in neanderthalisation.

Homo neanderthalis tend to have more of civilisational diseases like metabolic syndrome, neurodegenerations, cancer, autoimmune disease, schizophrenia, autism and CJD. Actinidic archaeal secreted RNA viroids may play a crucial role in amyloid formation and pathogenesis of these disorders. The evolution and origin of homo sapiens and homo neanderthalis is on the basis of actinidic archaeal symbiosis and digoxin synthesis. Extreme climate change related increased actinidic archaeal symbiosis and digoxin synthesis results in homo neanderthalis. The homo sapien species results from inhibition of actinidic archaeal symbiosis and endogenous digoxin synthesis by normal global climate. Thus there are evolutionary swings between the two human species depending on extremes of climate and archaeal symbiosis. Endogenous digoxin can be considered as a Neanderthal hormone. The results show that there was increase in cytochrome F420 in CJD and other disease groups indicating increased archaeal growth. There was also an increase in free RNA indicating self-replicating RNA viroids in CJD and other disease groups. The RNA viroid generation was catalysed by actinides. The RNA viroids can bind with proteins having prion like domains forming ribonucleoproteins. These ribonucleoproteins can give an abnormal conformation to the protein resulting in generation of abnormal prions. The abnormal prions can act as a template to convert normal proteins with normal configuration to abnormal conformation. This can result in amyloidogenesis. The abnormal configured proteins will resist lysosomal digestion and accumulate as amyloid producing disease states and extinction.

Key words: Actinidic archaea, Prions, Amyloid, Systemic disease, Infectivity

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INTRODUCTION

Prion proteins have been implicated in systemic disorders like neurodegenerations, cancer and metabolic syndrome. The beta amyloid in Alzheimer's disease, alpha synuclein in parkinson's disease, the TAR protein in frontotemporal dementia and copper zinc dismutase in motor neuron disease behaves like prion proteins. Prion proteins like behavior is also seen in the tumour suppressor P_{53} protein in cancer and the islet cell associated amyloid in diabetes mellitus. Prion diseases are conformational diseases. The abnormal prion protein seeded into the system converts the normal proteins with prion like domains to abnormal configuration. This abnormal protein resists digestion by lysosomal enzymes after its half-life is over and results in deposition of amyloid plaques. This produces organ dysfunction. Prion phenomena were initially described for Creutzfeldt Jakob's disease, but now it is found to be wide spread in chronic disease pathogenesis. Ribonucleoproteins are well known to behave like prion proteins and form amyloid. We have demonstrated actinidic archaea which secretes RNA viroids in metabolic syndrome, neurodegenerations, cancer, autoimmune disease, schizophrenia, autism and CJD. The RNA viroids can bind with normal proteins with prion like domains eg., superoxide dismutase and produce a ribonucleoprotein resulting in prion phenomena and amyloidogenesis. The actinidic archaeal growth results in increased digoxin synthesis and phenotypic conversion of homo sapiens to homo Neanderthals as reported earlier. The increased actinidic archaeal growth is due to global warming and these results in neanderthalisation. Homo neanderthalis tend to have more of civilisational diseases like metabolic syndrome, neurodegenerations, cancer, autoimmune disease, schizophrenia, autism and CJD. Actinidic archaeal secreted RNA viroids may play a crucial role in amyloid formation and pathogenesis of these disorders.[1-16]

MATERIALS AND METHODS

The following groups were included in the study:- 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+cerium 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. Aliquots were withdrawn at zero time immediately after mixing and after incubation at 37^{o}C for 1 hour. The following estimations were carried out:- Cytochrome F420, free RNA, Cytochrome F420 was estimated flourimetrically (excitation wavelength 420 nm and emission wavelength 520 nm). 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

cerium increased their levels. The addition of antibiotics to the patient's plasma caused a decrease in all the parameters while addition of cerium increased their levels but the extent of change was more in patient's sera as compared to controls. The results are expressed in tables 1-2 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time.

RESULTS

The results show that there was increase in cytochrome F420 in CJD and other disease groups indicating increased archaeal growth. There was also an increase in free RNA indicating self-replicating RNA viroids in CJD and other disease groups. The RNA viroid generation was catalysed by actinides. The RNA viroids can bind with proteins having prion like domains forming ribonucleoproteins. These ribonucleoproteins can give an abnormal conformation to the protein resulting in generation of abnormal prions. The abnormal prions can act as a template to convert normal proteins with normal configuration to abnormal conformation. This can result in amyloidogenesis. The abnormal configured proteins will resist lysosomal digestion and accumulate as amyloid.

Table 1
Effect of Cerium and Antibiotics on Cytochrome F420

Group	CYT F420 %(increase with cerium)		CYT F420 % (decrease with doxy+cipro)	
	Mean	**± *SD***	**Mean**	**± *SD***
Normal	4.48	0.15	18.24	0.66
Schizo	23.24	2.01	58.72	7.08
Seizure	23.46	1.87	59.27	8.86
AD	23.12	2.00	56.90	6.94
MS	22.12	1.81	61.33	9.82
NHL	22.79	2.13	55.90	7.29
DM	22.59	1.86	57.05	8.45
AIDS	22.29	1.66	59.02	7.50
CJD	22.06	1.61	57.81	6.04
Autism	21.68	1.90	57.93	9.64
	F value 306.749 *P* value < 0.001		*F* value 130.054 *P* value < 0.001	

Table 2
Effect of Cerium and Antibiotics on Free RNA

Group	RNA % change (increase with cerium)		RNA % change (decrease with doxy+cipro)	
	Mean	**± *SD***	**Mean**	**± *SD***
Normal	4.37	0.13	18.38	0.48
Schizo	23.59	1.83	65.69	3.94
Seizure	23.08	1.87	65.09	3.48
AD	23.29	1.92	65.39	3.95
MS	23.29	1.98	67.46	3.96

To becontinued

Continued

Group	RNA % change (increase with cerium)		RNA % change (decrease with doxy+cipro)	
	Mean	**± *SD***	**Mean**	**± *SD***
NHL	23.78	1.20	66.90	4.10
DM	23.33	1.86	66.46	3.65
AIDS	23.32	1.74	65.67	4.16
CJD	23.11	1.52	66.68	3.97
Autism	23.33	1.35	66.83	3.27
	F value 427.828 *P* value < 0.001		*F* value 654.453 *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. The archeal origin of the self replicating RNA 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 cerium induced increase in enzyme activities. There was an increase in free RNA indicating self replicating RNA viroids. 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. The RNA viroids can bind with proteins having prion like domains forming ribonucleoproteins. These ribonucleoproteins can give an abnormal conformation to the protein resulting in generation of abnormal prions. The abnormal prions can act as a template to convert normal proteins with normal configuration to abnormal conformation. This can result in amyloidogenesis. The abnormal configured proteins will resist lysosomal digestion and accumulate as amyloid.

Amyloidogenesis has been implicated in systemic disorders. The beta amyloid in Alzheimer's disease, alpha synuclein in parkinson's disease, the TAR protein in frontotemporal dementia and copper zinc dismutase in motor neuron disease behaves like prion proteins. Prion proteins like behavior is also seen in the tumour suppressor P53 protein in cancer and the islet cell associated amyloid in diabetes mellitus. Prion diseases are conformational diseases.

The RNA viroids generated from actinidic archaea can bind to proteins with prion like domains resulting in generation of ribonucleoproteins. Ribonucleoproteins with abnormal conformation can act as a template for normal proteins with prion like domains to change to abnormal conformation. This results in generation of prion proteins with abnormal conformation resisting lysosomal digestion and generating amyloid. These systemic diseases are

due to actinidic archaeal generated RNA viroid induced prion protein generation and amyloidogenesis. Prion proteins have been implicated in systemic disorders like neurodegenerations, cancer and metabolic syndrome. The beta amyloid in Alzheimer's disease, alpha synuclein in parkinson's disease, the TAR protein in frontotemporal dementia and copper zinc dismutase in motor neuron disease behaves like prion proteins. Prion proteins like behavior is also seen in the tumour suppressor P_{53} protein in cancer and the islet cell associated amyloid in diabetes mellitus. The present study shows that the same prion protein mechanism can operate in schizophrenia, autism and autoimmune diseases. Sporadic CJD is also induced by actinidic archaea induced RNA viroids. Actinidic archaeal induced RNA viroids generated prions can be transferred between individuals indicating the infective nature of neurodegenerations, cancer, metabolic syndrome, autoimmune disease and neuropsychiatric diseases.

The archaeal porphyrins can modulate amyloid formation. The archaeal cholesterol oxidase activity was increased resulting in generation of pyruvate and hydrogen peroxide. 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. Glycine and succinyl CoA are the substrates for ALA synthesis. 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. Protoporphyrine binds to the peripheral benzodiazepine receptor regulating steroid and digoxin synthesis. Increased porphyrin metabolites can contribute to hyperdigoxinemia. Digoxin can modulate the neuroimmunoendocrine system.

The global warming results in increased growth of actinidic archaea and neanderthalisation of the homo sapien species. The actinidic archaea secreted viroids can generate ribonucleoproteins by binding to proteins with prion like domains. This generates amyloidogenesis and systemic diseases like neurodegenerations, cancer, metabolic syndrome, autoimmune disease and neuropsychiatric diseases. The widespread incidence of these systemic diseases leads to extinction of the neanderthalised species.

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