GLOBAL WARMING

Archaea and Viroid Induced Symbiotic Human Evolution - The Human Mind - Conscious and Quantal Perception - Mind Downloading/Whole Brain Emulation on Brain Archaeal Colony Networks

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ISBN: 978-1-941926-97-0

Published in 2016 by Open Science Publishers
228 Park Ave., S#45956, New York, NY 10003, U.S.A.
http://www.openscienceonline.com

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Chapter 1

An Actinide Dependent Shadow Biosphere of Archaea and Viroids and Its Messenger Digoxin - Role in Schizophrenia, Autism and Seizure Disorder

Introduction

Global warming induces a genomic change in humans. Global warming induces endosymbiotic archaeal and RNA viroidal growth. The porphyrins form a template for the formation of RNA viroids, DNA viroids, prions, isoprenoids and polysaccharides. They can symbiose together to form primitive archaea. The archaea can further induce HIF alpha, aldose reductose and fructolysis resulting in further porphyrinogenesis and archaeal self-replication. The primitive archaeal DNA is integrated along with RNA viroids which are converted to their corresponding DNA by the action of redox stress induced HERV reverse transcriptase into the human genome by the redox stress induced HERV integrase. The archaeal DNA sequences that are integrated into the human genome forms endogenous archaeal human genomic sequences akin to HERV sequences and can function as jumping genes regulating genomic DNA flexibility. The integrated endogenous genomic archaeal sequences can get expressed in the presence of redox stress forming endosymbiotic archaeal particles which can function as a new organelle called the archaeaons. The archaeaon can express the fructolytic pathway constituting an organelle called the fructosome, cholesterol catabolic pathway and digoxin synthetic forming an organelle called the steroidelle, the shikimic acid pathway forming an organelle called the neurotransminoid, antioxidant vitamin E and vitamin C synthetic organelle called the vitaminocyte as well as the glycosaminoglycan synthetic organelle called glycosaminoglycoid. The archaeaon secreting RNA viroids is called the viroidelle.

The endosymbiotic actinidic archaea forms the basis of life and can be considered as the third element in the cell. It regulates the cell, the neuro-immune-endocrine system and the conscious / unconscious brain. The endosymbiotic actinidic archaea can be called as the elixir of life. A definite

population of endosymbiotic actinidic archaea is required for the existence and survival of life. A higher density of endosymbiotic actinidic archaeal population can lead to human disease. Thus actinidic archaea are important for survival of human life and can be considered as crucial to it. Symbiosis by actinidic archaea is the basis of evolution of humans and primates. The increase in endosymbiotic archaeal growth can lead to the induction of homo neanderthalis. This endosymbiotic archaea induced neanderthalisation of the species leads to human disease like metabolic syndrome X, neurodegenerations, schizophrenia and autism, autoimmune disease and cancer. The reduction in endosymbiotic archaeal growth by a high fibre, high medium chain triglyceride and legume protein ketogenic diet, antibiotics from higher plants like Curcuma longa, Emblica officianalis, Allium sativum, Withania somnifera, Moringa pterygosperma and Zingeber officianalis and transplantation of colonic microflora from normal homo sapien population can lead to deneanderthalisation of species and treatment of the above mentioned diseased states. The colonic microflora of neanderthalised diseased states like metabolic syndrome X, neurodegenerations, schizophrenia and autism, autoimmune disease and cancer when transferred to the normal homo sapien species leads to generation and induction of homo neanderthalis. Thus primate and human evolution is symbiotic event which can be induced the modulating symbiotic archaeal growth. Human populations can be divided into matrilineal Neanderthal population in South Indian Dravidians, Celts, Basques, Jews and Berbers and the Cro-Magnon population seen in Africa and Europe. The symbiotic archaeal colonization decides which species - Neanderthal or Cro-Magnon to which the society belongs to. It is tempting to postulate symbiotic microflora and archaea determining the family behavior and traits as well as societal and caste behavior and traits. The cell has been postulated by Margulis to be a symbiotic association of bacteria and viruses. Similarly, the

family, the caste, the community, nationalities and the species itself is determined by archaeal and other bacterial symbiosis.

Symbiosis by microorganisms especially archaea drives the evolution of the species. In such a case symbiosis can be induced by transfer of microflora symbionts and evolution induced. Endosymbiosis by archaea as well as archaeal symbionts in the gut can modulate the genotype, the phenotype, the social class and the racial group of the individual. The symbiotic archaea can have horizontal and vertical transmission. Endosymbiotic archaeal growth leads to neanderthalisation of the species. The neanderthalised species is matrilineal society and includes the Dravidians, the Celts, the Basques and the Berbers. The inhibition of the endosymbiotic archaeal growth leads to evolution of the homo sapiens. This includes the Africans, Aryan invaders of North India and the Aryan derived European population. Symbiosis mediated evolution depends on the gut flora and the diet. This has been demonstrated in the drosophila pseudoobscura. The drosophila mates only with other individuals eating the same diet. When the drosophila gut microflora is altered by feeding antibiotics they mate with other individuals eating different diets. The diet consumed by the drosophila regulates its gut microflora and mating habits. The combination of the human genome and the symbiotic microbial genome is called the hologenome. The hologenome especially its symbiotic microbial component drives human evolution as well as animal evolution. The evolutionary distance between species of wasp depends on the gut microflora. The human gut microflora regulates the endocrine, genetic and neuronal systems. Humans and primate evolution depends on endosymbiotic archaea and gut microflora. The endosymbiotic archaeal growth determines the racial differences between the matrilineal Harappan / Dravidian societies and the patriarchal Aryan society. The matrilineal Harappan / Dravidian society was neanderthalic and had

increased endosymbiotic archaeal growth. Endosymbiotic archaeal growth and neanderthalisation can lead to autoimmune disease, metabolic syndrome X, neurodegeneration, cancer, autism and schizophrenia. The Neanderthal gut flora and endosymbiotic archaea was determined by the non vegetarian ketogenic high fat high protein diet consumed by them in the Eurasian steppes. The homo sapiens including the classical Aryan tribes and African ate a high fibre diet and had lower archaeal growth both endosymbiotic and gut. The dietary fibre intake determines the microbial diversity of the gut. The high fibre intake is associated with increased generation of short chain fatty acids - butyric acid by the gut flora. Butyrate is a HDAC inhibitor and leads to increased generation and incorporation of endogenous retroviral sequences. The high dietary fibre intake related increased HERV sequences leads to increased synaptic connectivity and a dominant frontal cortex as seen in homo sapien species. The neanderthalic species consume a ketogenic non vegetarian high fat high protein low fibre diet. This leads to decreased generation of endogenous HERV sequences and reduced genomic flexibility in neanderthalic species. This produces smaller cerebral cortex and a dominant cerebellar cortex in the neanderthalic brain. The homo neanderthalic species by the low dietary fibre intake starve their microbial self. This leads to increased endosymbiotic and gut archaeal growth. The mucous membrane lining the gut becomes thinned out as the gut bacteria eats up the mucous lining of the gut. This results in leakage of endotoxin and archaea from the gut to the blood breaching the barrier and produces a chronic immunostimulatory inflammatory state which forms the basis of autoimmune disease, metabolic syndrome, neurodegeneration, oncogenic and psychiatric disorders. The Neanderthal species eat a low fibre diet and have a deficiency of microbiota accessed carbohydrate generating short chain fatty acid. There is a deficiency of butyrate generated in the gut from the dietary fibre which can produce suppression of the chronic inflammatory process. The Neanderthals

have got the fermentation by-product deficiency syndrome. The induction of neanderthalic species depends on the low fibre intake induced high archaeal density endosymbiotic and the gut microflora. The homo sapiens species consume a high fibre diet generating large amounts of short chain fatty acid butyrate which inhibits endosymbiotic and gut archaeal growth. The microbial self of the homo sapien species is more diverse than that of the neanderthalic species and the archaeal population density is less. This results in a protection against chronic inflammation and the induction of diseases like autoimmune disease, metabolic syndrome, neurodegeneration, oncogenic and psychiatric disorders. The homo sapien species have a higher intake of dietary fibre contributing to around 40 g/day and a diverse microbial gut flora with less of archaeal population density. The butyrate generated from dietary fibre produces an immunosuppressive state. Thus the symbiotic microflora with less of archaeal density induces a homo sapien species. This can be demonstrated by experimental induction of evolution. A high fibre high MCT diet as well as antibiotics derived from higher plants and fecal microbiota transfer from sapien species can inhibit the Neanderthal metabolonomics and phenotype and induce the evolution of homo sapiens. A low fibre high fat high protein diet as well as fecal microbiota transfer from the Neanderthal species can produce Neanderthal metabolonomics and phenotype inducing the evolution of homo neanderthalis. Transfer of colonic microflora predominantly archaea and modulation of endosymbiotic archaea by a paleo diet and antibiotics from higher plants can lead to interconversion of human species between homo neanderthalis and homo sapiens. The hologenome especially the microbial flora endosymbiotic/gut drives human and animal evolution and can be experimentally induced. Symbiotic microflora drives evolution. Every animal, every human species, different communities, different races and different caste have their signature endosymbiotic and gut microflora which can be transmitted

vertically and horizontally. Thus symbiosis drives human and animal evolution. The colonic and endosymbiotic archaea and other microbes like clostridial clusters determine the species, race, caste, community and personal identity of the individual. The identity of the individual - personal, community, caste, race, nationality and species is determined by the colonic and endosymbiotic archaeal and clostridial clusters. Predominent archaeal symbiosis produces homo neanderthalis and less prominent archaeal symbiosis and dominant clostridial clusters in the gut produces the homo sapien species. Each individual, race, nationality, caste, creed and community have the endosymbiotic and colonic microbiota signature. This colonic and endosymbiotic microbiota signature is transferable by the change of endosymbiotic and colonic microbiota from one group to another. Thus the evolution and identity based on individuality, race, nationality, caste and creed can be induced.

This can be interpreted on the basis of Villarreal hypothesis of group identity and cooperativity of RNA collectives. Archaeal symbiosis in the gut and in the tissue spaces determines speciation of human beings as homo sapiens and homo neanderthalis. The endosymbiotic archaea can secrete RNA viroids and viruses and there is a viroid-archaeal host relationship between the two. A dynamic state of virus lysis and persistence can occur in archaea suggesting that viral addiction can occur in archaea. The RNA viroids in the archaea coordinate their behavior by information exchange, modulation and innovation generating new sequence based content. This occurs due to a phenomenon of symbiosis in contrast to the concept of survival of the fittest. The generation of new RNA viroidal sequences is a result of practical competence of living agents to generate new sequences by symbiosis and sharing. This represents highly productive RNA viroidal quasi-species consortia for the evolution, conservation and plasticity of genomic environments. The behavioural motives of the RNA

are single stem loop structures. They have self folding and group building capabilities depending upon functional needs. The evolution process depends upon what Villareal calls RNA stem loop consortia. The whole entity can function only if participatory groups of RNA viroids can get their function coordinated. There is competent denovo generation of new sequences by cooperative action and not by competition. These RNA viroidal group consortia can contribute to the host identity, group identity and group immunity. The term used for this is RNA viroidal sociological behavior. The RNA viroids can build groups that invade the archaea and compete as a group for limited resources such host genomes. A key behavioural motif is able to integrate a persistent life style into the archaeal colony with the addiction module forming competing viroidal groups that are counter balancing each other together with the archaeal/host immune system. This leads to creation of an identity for the archaeal colony and the homo neanderthalis host. Viroids can kill their host and also colonize their host without disease and protect the host from similar viruses and viroids. Together with lysis and protection we see a viroid colonized host that is both symbiotic and innovative acquiring new competent codes. Thus the viroid-host relationship is a pervasive, ancient force in the origin and evolution of life. Cumulative evolution at the level of RNA viroids is like a ratchet effect used for transmission of cultural memes. This learning accumulates so that every new generation must not repeat all innovative thoughts and techniques. Quasi-species of RNA viroids are cooperative and exclusive of other quasi-species. They have group recognition differentiating self-groups and non-self-groups allowing for quasi-species to promote the emergence of group identity. With group identity via counter related addiction modules two opposing components must be present and work coherently and define the group as a whole. Biological identity is constituted by dynamic interaction of cooperative groups. Virus addiction module is an essential strategy for existence

of life in the virosphere. Viruses are transmissible and can persist in specific host population leading to a form of group immunity / identity since identical but uncolonized host population remains susceptible to a killing action of lytic viruses. In this way we see that viruses are necessary providing opposing functions for addiction (persistence/protection and lytic/killing). Viroids can function as consortia, an essential interacting group and provide a mechanism from which consortial function could emerge in the origin of protobiotic life. Genetic parasites can act as a group (qs-c). But for this group to be coherent they must attain group identity and this is typically via an addiction strategy. Antiviral and proviral system in the archaea will themselves emerge in the host from virus derived information. The archaeal viruses themselves provide the critical function required for antiviral defence. The opposing functions are the basis of addiction modules. Thus the emergence of group identity becomes an essential and early event in the emergence of life. This is coherent to the basically group behavior of RNA viroids in archaea. This group selection and group identity are needed to create information coherence and network formation and to establish a system of communication - code competent interactions. This identity serves as information also for the ones that do not share this identity. This is the beginning of self/non-self differentiating capability. In this way viroids promote the emergence of group identity in archaeal colonies and host humans. The archaeal colony identity depends upon the colonizing set of RNA viroids producing a coherent network that is inclusive opposing functions and favours the persistence of parasite derived new information. On the basis of population-based functions of RNA DNA can be considered as a habitat for consortia RNA. Thus RNA viroids of the archaea are involved in complex multicellular identity. This is called as the Gangen hypothesis by Villarreal. The Gangen describes the emergence of commonly shared code use, group membership and collective living function of RNA

viroids. Communication is a code depended interaction and transmission of infectious code defines the origin of the virosphere. This issue refers to the idea of collective of RNA viroids with inherent toxic and antitoxic features should be able to transmit or communicate these agents and their features to a nearby competing population. It strongly favours the survival of RNA viroidal population with compatible addiction modules that will inhibit agent toxicity and allow persistence of new agents. This is thus the survival of the persistently colonized set which is an inherently symbiotic and consortial process. It also promotes increasing complexity and identity/immunity of the host collective via a new agent colonization, and stable addition. Thus the transmission of RNA agents attains both communication and recognition of group membership. In this way the emergence of the virosphere must had been an early event in the origin of life and group identity. Viruses and viroids are genetic parasites and the most abundant living entities on earth. The virosphere is a network of infectious genetic agents. Evolution, conservation and plasticity of genetic identities are the result of cooperative consortia of RNA viroids that are competent to communicate. Thus the archaeal viroidal consortia can symbiotically share and communicate producing new sequences and give an identity to the archaeal colony. The low fibre diet and extreme temperatures of the Eurasian steppes leads to archaeal multiplication and induction of the homo neanderthalis species. The archaeal colony's characteristics are determined by the cooperative consortia of RNA viroids in the archaea and the archaeal colony identity determines the homo neanderthalis identity. Thus the archaeal colonies with their quasi-species consortia of RNA viroids determine the homo neanderthalis identity. The new sequence generation by the RNA viroidal consortia's symbiotic sharing character contributes to the diversity in the behavior and creativity of the homo neanderthalis population. The archaeal RNA viruses and viroids and the archaeal colonies themselves protect the homo

neanderthalis population from retroviral infections. Thus the homo neanderthalis population is retroviral resistant and the quasi-species consortia of archaea and archaeal viroids gives them a group identity as retroviral resistant. Thus the quasi-species consortia of archaea and RNA viroids give homo neanderthalis colonies their identity and idea of self. The homo neanderthalis is resistant to retroviral infection like the Australian aboriginals and the endogenous retroviral sequences in the Neanderthal genome are limited. This leads to lack of plasticity and dynamicity of the human genome and the cerebral cortex in ill-developed with a dominant impulsive cerebellar cortex in the homo neanderthalis population. This produces the impulsive creative surrealistic spiritual neanderthalic brain. As the extreme of temperature goes off and the ice age ends the archaeal population density also comes down. This also can result from the consumption of a high fibre diet in the African continent. The high fibre diet digested by clostridial clusters in the colon promotes butyrate synthesis and butyrate will induce HDAC inhibition and expression of retroviral sequences in the primate genome. This leads to increase in endogenous retroviral sequences in the human genome, increasing genomic dynamicity and the evolution of complicated cerebral cortex dominant brain with its complex synaptic connectivity in the homo sapiens. This leads onto a logical, commonsensical, pragmatic and practical homo sapien brain. The homo sapiens due to lack of archaea and the RNA viroids are susceptible retroviral infection. Thus the archaeal colonies and RNA viroidal quasi-species consortia determine the evolution of the human species and the brain networks. Thus extremes of temperature, fibre intake, archaeal colony density, RNA viroidal quasi-species, group identity and retroviral resistance decides on the evolution of homo sapiens and homo neanderthalis as well as the brain networks. The present extremes of temperature and low fibre intake in civilized society can lead to increase in archaeal population densities and quasi-species RNA viroidal

networks generating a new homo neanderthalis in a new neanderthalic anthropocene age as opposed to the present homo sapien anthropocene age. The archaeal population densities and quasi-species RNA viroidal networks determine homo sapien / homo neanderthalis species, racial, caste, community, national, sexual, metabolic, phenotypic, neuronal, psychiatric, psychological, immune, genotypic and individual identity. The archaea secretes the trephone digoxin which can edit the RNA viroids and generate new sequences. Archaeal dipolar magnetite and porphyrins in the setting of digoxin induced membrane sodium potassium ATPas inhibition can produce a pumped phonon system mediated quantal perceptive state and quantal communication in the RNA viroidal symbiotic system generating new sequences by steroidal digoxin enzymatic editing action. This gives rise to archaeal RNA viroidal quasi-species symbiotic diversity and identity to species, race, caste, sex, culture, individual and national identity.

The human brain can be considered as a modified archaeaon colony network. The archaeaon are eternal and can last for billions of years. The human brain is basically an information storage system. The archaeaon has got dipolar magnetite and porphyrins and can function as quantal computer. The archaeal colony with its dipolar magnetite and porphyrin in the setting of archaeal digoxin induced membrane sodium potassium ATPase inhibition can function as a pumped phonon system mediating quantal perception. The archaeaon in the brain is capable of information storage at a point in time and space. The experiences and information stored in the archaeaon is immortal and eternal. The archaeaon can have a wave particle existence and can exist in multiple quantal possible states and can inhabit multiple quantal multiverses. The interaction between information stored in quantal computers in multiple different archaeaon systems all over the universe by the quantal interactions

results in eternal existence of information in quantal multiverses. The information in the quantal multiverses can have a particulate existence creating a newer mode by quantal interactions between information stored at multiple points of time. This creates the particulate mythic world of human existence. These are what are called as Samsaras. The mind is uploaded into information in the neuronal archaeal colony network and its quantal computers. The information stored in the archaeal colony network mediated quantal state is eternal and can be considered as a digital version of the brain, a mind downloading technique or whole brain emulation. The archaeal colony network stores the human experiences in an eternal manner and can contribute to biological reincarnation.

The roots of Western civilisational disease can be related to the starvation of the colonic microflora. The colonic microflora depends upon complex carbohydrates derived from dietary fibre. The processed food of high protein, fat and sugars is digested and absorbed in the stomach and small intestine. A very little of it reaches the colon and widespread use of antibiotics in medicine has produced mass extinction of the colonic microflora. The colonic microflora is extremely diverse and the diversity is lost. There are 100 trillion bacteria in the colon belonging to 1200 species. They regulate the immune system by inducing the T-regulatory cells. A high fibre diet contributes to colonic microbiota diversity. Interaction with farm animals like cows and dogs also contributes to the colonic microflora diversity. The typical Western diet of high fat, high protein and sugars decreases the colonic microbiota diversity and increase colonic/endosymbiotic archaea producing methanogenesis. The colonic archaea feed upon the mucous lining of the colon and produces leakage of archaea into the blood and tissue system producing endosymbiotic archaea. This results in a chronic inflammatory state. The high fibre diet of Africans, South

Americans and Indians produces increased colonic microbiota diversity and increase in clostridial clusters generating SCFA in the gut. High fibre diet is protective against metabolic syndrome and diabetes mellitus. Metabolic syndrome is related to degeneration, cancer, neuropsychiatric illness and autoimmune disease. A high fibre diet of upto 40 g/day can be called as a gut diet. The colonic microflora especially the clostridial cluster digests the fibre generating short chain fatty acids which regulates immunity and metabolism. High fibre diet increases the colonic mucus secretion and the thickness of the mucus lining. A high fibre diet produces increase in clostridial clusters and mucous secretion. This produces a strong gut blood barrier and prevents metabolic endotoxemia which produces a chronic inflammatory response. High dietary fibre intake and the diversity of the colonic microflora with prominent SCFA producing clostridial clusters are interrelated. The clostridial clusters metabolise the complex carbohydrate in dietary fibre to short chain fatty acids butyrate, propionate and acetate. They increase the T-regulatory function. A high fibre diet increases the bacteroides and reduces the firmecutes of the colonic microflora. A high fibre diet is associated with a low body-mass index. A low fibre diet produces increase in colonic archaeal growth as well as endosymbiotic tissue and blood archaea. This produces more of methanogenesis rather than short chain fatty acid synthesis contributing to immune activation. A low fibre diet is associated a high body-mass index and chronic systemic inflammation. Germ-free mice show cardiac, pulmonary and liver atrophy. Gut microflora is required for the generation of organ systems. The gut microflora is also required for generation of T-regulatory cells. High fibre intake produces more colonic microbiota diversity and increase in clostridial clusters and fermentation by products like butyrate which suppresses inflammation and increases T-regulatory cells. A low fibre diet produces increase in archaeal growth, methanogenesis, destruction of the mucus lining and leakage of the

colonic archaea producing endosymbiotic tissue and blood archaea. This produces an immune hyperreactivity contributing to the modern plagues of civilization - metabolic syndrome, schizophrenia, autism, cancer, autoimmunity and degenerations. The gut microbiota drives human evolution. The humans don't host the gut microbiota but the gut microbiota host us. The human system forms an elaborate culture laboratory for the propagation and survival of the microbiota. The human system is induced by the microbiota for their survival and growth. The human system exists for the microbiota and not the other way round. The same mechanism holds good in plant systems. Plant started the colonized earth as they started symbiosing with bacteria in the roots systems which can derive nutrients from the soil. Human beings form a mobile culture laboratory for the more effective propagation and survival of the microbiota. The microbiota induces the formation of specialized immune cells called innate lymphoid cells. The innate lymphoid cells will direct the lymphocytes not to attack the beneficial bacteria. Thus the endosymbiotic archaea and the gut archaea induce human, primate and animal evolution to generate structures for them to survive and propagate. The source of endosymbiotic archaea, the third element of life is the colonic archaea that leaks into the tissue spaces and blood systems due to breach in the gut blood barrier. The increase in colonic archaea is due to the starvation of the gut microbiota consequent to a low fibre diet. This results in increase in colonic archaeal growth and destruction of clostridial clusters and bacteroides. The increase colonic archaeal growth in the presence of gut starvation due to low fibre diet eats up the mucus lining and produces breakages in the gut blood barrier. The colonic archaea enters the blood stream and produces endosymbiosis generating endosymbiotic archaea and various new organelle - fructosoids, steroidelle, vitaminocyte, viroidelle, neurotransminoid, porphyrinoids and glycosaminoglycoids.

The increase in endogenous EDLF, a potent inhibitor of membrane Na^+-K^+ ATPase, can decrease this enzyme activity. The results showed increased endogenous EDLF synthesis as evidenced by increased HMG CoA reductase activity, which functions as the rate limiting step of the isoprenoid pathway. Studies in our laboratory have demonstrated that EDLF is synthesized by the isoprenoid pathway. The endosymbiotic archaeal sequences in the human genome get expressed by redox stress and osmotic stress of global warming. This results in induction of HIF alpha which will upregulate fructolysis and glycolysis. In the setting of redox stress all glucose gets converted to fructose by the induction of enzymes aldose reductase and sorbitol dehydrogenase. Aldose reductase converts glucose to sorbitol and sorbitol dehydrogenase converts sorbitol to fructose. Since fructose is preferentially phosphorylated by ketohexokinases the cell is depleted of ATP and glucose phosphorylation comes to a halt. Fructose becomes the dominant sugar that is metabolized by fructolysis in expressed archaeal particles in the cell functioning as organelle called fructosoids. The fructose is phosphorylated to fructose 1-phosphate which is acted upon by aldolase B which converts it into glyceraldehyde 3-phosphate and dihydroxy acetone phosphate. Glyceraldehyde 3-phosphate is converted to D 1,3-biphosphoglycerate which is then converted to 3-phosphoglycerate. The 3-phosphoglycerate is converted to 2-phosphoglycerate. 2-phosphoglycerate is converted to phosphoenol pyruvate by the enzyme enolase. Phosphoenol pyruvate is converted to pyruvate by the enzyme pyruvic kinase. The archaeaon induces HIF alpha which upregulates fructolysis and glycolysis but inhibits pyruvate dehydrogenase. The forward metabolism of pyruvate is stopped. The dephosphorylation of phosphoenol pyruvate is inhibited in the setting of pyruvic kinase inhibition. Phosphoenol pyruvate enters the shikimic acid pathway where it is converted to chorismate. The shikimic acid is synthesized by a pathway starting from glyceraldehyde

3-phosphate. Glyceraldehyde 3-phosphate combines with the pentose phosphate pathway metabolite sedoheptulose 7-phosphate which is converted to erythrose 4-phosphate. The pentose phosphate pathway is upregulated in the presence of the suppression of glycolytic pathway. Erythrose 4-phosphate combines with phosphoenol pyruvate to generate shikimic acid. Shikimic acid combines with another molecule of phosphoenol pyruvate to generate chorismate. The chorismate is converted to prephenic acid and then to parahydroxy phenyl pyruvic acid. Parahydroxy phenyl pyruvic acid is converted to tyrosine and tryptophan as well as neuroactive alkaloids. The shikimic acid pathway is structured in expressed archaeaon organelle called the neurotransminoid. The fructolytic intermediates glyceraldehydes 3-phosphate and pyruvate are the starting points of the DXP pathway of cholesterol synthesis. Glyceraldehyde 3-phosphate combines with pyruvate to form 1-deoxy D-xylulose phosphate (DOXP) which is then converted to 2-C methyl erythritol phosphate. 2-C methyl erythritol phosphate can be synthesized from erythrose 4-phosphate a metabolite of the shikimic acid pathway. DXP combines with MEP to form isopentenyl pyrophosphate which is converted to cholesterol. Cholesterol is catabolized by archaeal cholesterol oxidases to generate digoxin. The digoxin sugars digitoxose and rhamnose are synthesized by the upregulated pentose phosphate pathway. Glycolytic suppression leads to upregulation of the pentose phosphate pathway. The expressed archaeaon organelle concerned with cholesterol catabolism and digoxin synthesis is called the steroidelle. The suppression of glycolysis and stimulation of fructolysis results in upregulation of the hexosamine pathway. Fructose is converted to fructose 6-phosphate by ketohexokinases. The fructose 6-phosphate is converted to glucosamine 6-phosphate by the action of glutamine fructose 6-phosphate amidotransferase (GFAT). Glucosamine 6-phosphate is converted to UDP N-acetyl glucosamine which is then converted to N-acetyl glucosamine and various amino sugars.

UDP glucose is converted to UDP D-glucuronic acid. UDP D-glucuronic acid is converted to glucuronic acid. This forms the uronic acid synthetic pathway. Uronic acids and hexosamines form repeating units of glycosaminoglycans. In the setting of glycolytic suppression and fructolytic metabolism fructolysis leads to increase synthesis of hexosamines and GAG synthesis. The GAG synthesizing archaeaon particles are called the glycosaminoglycoids. The expressed archaeaon particles are capable of synthesizing antioxidant vitamin C and E. The UDP D-glucose is converted to UDP D-glucuronic acid. UDP D-glucuronic acid is converted to D-glucuronic acid. D-glucuronic acid is converted to L-gulonate by enzyme aldoketoreductases. L-gulonate is converted to L-gulonolactone by lactonase. L-gulonolactone is converted to ascorbic acid by the action of archaeal L-gulo oxidase. The vitamin E is synthesized from shikimate which is converted to tyrosine and then to parahydroxy phenyl pyruvic acid. Parahydroxy phenyl pyruvic acid is converted to homogentisate. Homogentisate is converted to 2-methyl 6-phytyl benzoquinone which is converted to alpha tocopherol. 2-methyl 6-phytyl benzoquinone is converted to 2,3-methyl 6-phytyl benzoquinone and gamma tocopherol. Vitamin E can also be synthesized by the DXP pathway. Glyceraldehyde 3-phosphate and pyruvate combined to form 1-deoxy D-xylulose 5-phosphate which is converted to 3-isopentenyl pyrophosphate. 3-isopentenyl pyrophosphate and dimethyl allyl pyrophosphate combined to form 2-methyl 6-phytyl benzoquinone which is converted to tocopherols. The ubiquinone another important membrane antioxidant and part of the mitochondrial electron transport chain is synthesized by the shikimic acid pathway and DXP pathway. The isoprenoid moiety of ubiquinone is contributed from the DXP pathway and the rest of it by tyrosine catabolism. The tyrosine is generated by the shikimic acid pathway. The archaeaon particles concerned with the synthesis of vitamin C, vitamin E and ubiquinone which are all antioxidants are called the vitaminocyte.

The human brain synthesizes an endogenous membrane sodium-potassium ATPase inhibitor digoxin which plays a role in neuro-immuno-endocrine integration and pathogenesis of several neuropsychiatric diseases. Endomyocardial fibrosis (EMF) along with the root wilt disease of coconut is endemic to Kerala with its radioactive actinide beach sands. Actinides like cerium producing intracellular magnesium deficiency due to cerium-magnesium exchange sites in the cell membrane have been implicated in the etiology of EMF.[1] Endogenous digoxin, a steroidal glycoside has also been related to the etiology of EMF due to the intracellular magnesium deficiency it produces.[2] Organisms like phytoplasmas and viroids have also been demonstrated to play a role in the etiology of these diseases.[3, 4] Endogenous digoxin has also been related to the pathogenesis of schizophrenia, autism and primary seizure disorder.[2] The possibility of endogenous digoxin synthesis by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism was considered.[5-7] Davies has put forward the concept of a shadow biosphere of organisms with alternate biochemistry present in earth itself.[8] An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described.[6]

Methods

Informed consent of the subjects and the approval of the Ethics committee of the Institute 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+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.[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, muramic acid, polycyclic aromatic hydrocarbon, hydrogen peroxide, dopamine, pyruvate, ammonia, glutamate, cytochrome C, hexokinase, ATP synthase, 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 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 1-7 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time.

Table 1. *Effect of cerium and antibiotics on muramic acid and dopamine.*

Group	DOPAMINE % (Increase with Cerium)		DOPAMINE % (Decrease with Doxy)		Muramic acid % change (Increase with Cerium)		Muramic acid % 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 2. *Effect of cerium and antibiotics on free DNA and RNA.*

Group	DNA % change (Increase with Cerium)		DNA % change (Decrease with Doxy)		RNA % change (Increase with Cerium)		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 3. *Effect of cerium and antibiotics on HMG CoA reductase and PAH.*

Group	HMG CoA R % change (Increase with Cerium)		HMG CoA R % change (Decrease with Doxy)		PAH % change (Increase with Cerium)		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 4. *Effect of cerium and antibiotics on digoxin and bile acids.*

Group	Digoxin (ng/ml) (Increase with Cerium)		Digoxin (ng/ml) (Decrease with Doxy+Cipro)		Bile acids % change (Increase with Cerium)		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 5. *Effect of cerium and antibiotics on pyruvate and hexokinase.*

Group	Pyruvate % change (Increase with Cerium)		Pyruvate % change (Decrease with Doxy)		Hexokinase % change (Increase with Cerium)		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 6. *Effect of cerium and antibiotics on hydrogen peroxide and delta amino levulinic acid.*

Group	H_2O_2 % (Increase with Cerium)		H_2O_2 % (Decrease with Doxy)		ALA % (Increase with Cerium)		ALA % (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 7. *Effect of cerium and antibiotics on ATP synthase and cytochrome F420.*

Group	ATP synthase % (Increase with Cerium)		ATP synthase % (Decrease with Doxy)		CYT F420 % (Increase with Cerium)		CYT F420 % (Decrease with Doxy)	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	4.40	0.11	18.78	0.11	4.48	0.15	18.24	0.66
Schizo	23.67	1.42	67.39	3.13	23.24	2.01	58.72	7.08
Seizure	23.09	1.90	66.15	4.09	23.46	1.87	59.27	8.86
Autism	22.60	1.64	66.86	4.21	21.68	1.90	57.93	9.64
F value	449.503		673.081		306.749		130.054	
P value	< 0.001		< 0.001		< 0.001		< 0.001	

Table 8

	Serum fructose		Serum fructokinase		Aldolase B		Total GAG	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	2.50	0.195	8.50	0.405	3.50	1.304	3.50	0.707
Schizo	31.14	4.446	22.19	2.634	11.63	3.081	21.50	1.714
Autism	28.66	5.089	24.09	2.146	12.30	1.621	22.60	3.054
Bipolar	29.88	5.150	22.29	1.641	10.87	1.895	23.47	2.878
F value	17.373		13.973		13.903		21.081	
p value	< 0.01		< 0.01		< 0.01		< 0.01	

Table 9

	Total TG		Serum ATP levels		Uric acid		Anti-aldolase	
	Mean	± SD	Mean	± SD	Mean	± SD	Mean	± SD
Normal	124.00	3.688	2.50	0.405	5.70	0.369	7.50	1.704
Schizo	244.00	31.383	0.72	0.102	8.65	0.701	1.35	0.319
Autism	284.30	19.743	0.87	0.072	8.14	0.538	1.35	0.218
Bipolar	289.89	23.406	0.74	0.115	9.59	0.783	1.80	0.402
F value	16.378		59.169		14.166		55.173	
p value	< 0.01		< 0.01		< 0.01		< 0.01	

Table 10

	Anti-enolase		Anti-pyruvatekinase		Anti-GAPDH	
	Mean	**± SD**	**Mean**	**± SD**	**Mean**	**± SD**
Normal	1.50	0.358	50.40	5.960	5.20	0.363
Schizo	0.40	0.142	22.02	11.954	1.31	0.235
Autism	0.20	0.060	19.27	2.201	1.20	0.205
Bipolar	0.39	0.124	18.93	6.447	1.78	0.355
F value	14.091		21.073		58.769	
p value	< 0.01		< 0.01		< 0.01	

Discussion

Archaeal Cholesterol Catabolism in Relation to Psychiatric Disease

The archaeaon steroidelle DXP pathway and the upregulated pentose phosphate pathway contribute to digoxin synthesis. 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 cerium 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 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]

Archaeal-Viroidal Human Genomic Sequences and Psychiatric Disease

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 noncoding 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 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 and primary seizure disorder.

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.[23] 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.[24] The isoprenoidal clade prokaryotes develop into other groups of prokaryotes via viroidal / virus as well as eukaryotic horizontal gene transfer producing bacterial speciation.[25] 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 ebstein 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.[26, 27] 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 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, 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. Bacteria and viruses have been related to the pathogenesis of schizophrenia and primary seizure disorder. Borrelia, toxoplasma, chlamydia, mycoplasma, retroviruses, herpes virus, influenza virus and borna virus contribute to the neuropathogenesis of schizophrenia.[29-31] Herpes virus is implicated in the pathogenesis of primary seizure disorder. The change in the length and grammar of the noncoding region produces eukaryotic speciation and individuality.[32] Changes in the length of noncoding region can lead onto disorders of consciousness like schizophrenia and autism.[33] The human endogenous retroviruses and change in the length and grammar of the noncoding region has been described in schizophrenia. 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 diseases like schizophrenia and primary seizure disorder. The microchimeras formed can lead to polyploidy. Neuronal polyploidy and microchimeras have been described in schizophrenia.

Archaeal Digoxin and Disorders of Consciousness

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 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] Schizophrenia and autism are described as disorders of consciousness and increased integration of archaea and viroids into the genome can contribute to its neuropathogenesis.

Archaeal and Viroidal Sequences in Human Genome, Hemispheric Dominance and Consciousness

The archaea can regulate limbic lobe transmission with archaeal cholesterol aromatase / ring oxidase generated norepinephrine, dopamine, serotonin and acetyl choline.[17] 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 resulting in autism and schizophrenia.[35] 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 signaling 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, autism 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 and autism by augmenting the bacterial shikimic acid pathway. The upregulated glycolysis consequent to the Warburg phenotype produces phosphoenol pyruvate, a basic substrate for the bacterial shikimic acid pathway which can synthesize monoamines and neuroactive alkaloids. The shikimic acid pathway can generate dopamine and serotonin producing the increased monoaminergic transmission in schizophrenia and autism. 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.

Archaeal and Viroidal Sequences in Human Genome - A New Neuronal Phenotype

In schizophrenia, autism and primary seizure disorder the paper presents data on: (1) Detection of a shadow biosphere of archaea, viroid and mevalonate pathway bacteria, (2) Bacterial cholesterol synthesis and catabolism are important in pathogenesis, and (3) The integration of viroid and archaea into the neuronal genome creates a new phenotype.

References

[1] Valiathan MS, Somers K, Kartha CC. *Endomyocardial fibrosis*. Delhi: Oxford University Press; 1993.

[2] Kurup R, Kurup PA. *Hypothalamic digoxin, cerebral dominance and brain function in health and diseases*. New York: Nova Science Publishers; 2009.

[3] Hanold D, Randies JW. Coconut cadang-cadang disease and its viroid agent. *Plant Disease*, 1991; 75(1): 330-335.

[4] Edwin BT, Mohankumaran C. Kerala wilt disease phytoplasma: Phylogenetic analysis and identification of a vector, *Proutista moesta*. *Phys Mol Plant Path.*, 2007; 71(1-3): 41-47.

[5] Eckburg PB, Lepp PW, Relman DA. Archaea and their potential role in human disease. *Infect Immun.*, 2003; 71: 591-596.

[6] Adam Z. Actinides and Life's Origins. *Astrobiology*, 2007; 7: 6.

[7] Schoner W. Endogenous cardiac glycosides, a new class of steroid hormones. *Eur J Biochem.*, 2004; 269: 2440-2448.

[8] Davies PCW, Benner SA, Cleland CE, Lineweaver CH, McKay CP, Wolfe-Simon F. Signatures of a Shadow Biosphere. *Astrobiology*, 2009; 1: 241-249.

[9] Richmond W. Preparation and properties of a cholesterol oxidase from nocardia species and its application to the enzymatic assay of total cholesterol in serum. *Clin Chem.*, 1973; 19(2): 1350-1356.

[10] Snell ED, Snell CT. *Colorimetric Methods of Analysis*, Vol. 3A. New York: Van Nostrand; 1961.

[11] Glick D. *Methods of Biochemical Analysis*, Vol. 5. New York: Interscience Publishers; 1971.

[12] Colowick, Kaplan NO. *Methods in Enzymology*, Vol. 2. New York: Academic Press; 1955.

[13] Maarten AH, Marie-Jose M, Cornelia G, van Helden-Meewsen, Fritz E, Marten PH. Detection of muramic acid in human spleen. *Infect Immun*., 1995; 63(5): 1652-1657.

[14] Smit A, Mushegian A. Biosynthesis of isoprenoids via mevalonate in Archaea: the lost pathway. *Genome Res*., 2000; 10(10): 1468-84.

[15] Van der Geize R, Yam K, Heuser T. A gene cluster encoding cholesterol catabolism in a soil actinomycete provides insight into Mycobacterium tuberculosis survival in macrophages. *Proc Natl Acad Sci USA*, 2007; 104(6): 1947-52.

[16] Francis AJ. Biotransformation of uranium and other actinides in radioactive wastes. *J Alloys Comp*., 1998; 271-273: 78-84.

[17] Probian C, Wülfing A, Harder J. Anaerobic mineralization of quaternary carbon atoms: Isolation of denitrifying bacteria on pivalic acid (2,2-Dimethylpropionic acid). *Appl Environ Microbiol.,* 2003; 69(3): 1866-1870.

[18] Vainshtein M, Suzina N, Kudryashova E, Ariskina E. New Magnet-Sensitive Structures in Bacterial and Archaeal Cells. *Biol Cell.* 2002; 94(1): 29-35.

[19] Tsagris EM, de Alba AE, Gozmanova M, Kalantidis K. Viroids. *Cell Microbiol*., 2008; 10: 2168.

[20] Horie M, Honda T, Suzuki Y. Endogenous non-retroviral RNA virus elements in mammalian genomes. *Nature,* 2010; 463: 84-87.

[21] Hecht M, Nitz N, Araujo P. 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,* 2010; 5: 2.

[22] Flam F. Hints of a language in junk DNA. *Science,* 1994; 266: 1320.

[23] Horbach S, Sahm H, Welle R. Isoprenoid biosynthesis in bacteria: two different pathways? *FEMS Microbiol Lett.,* 1993; 111: 135-140.

[24] .Gupta RS. Protein phylogenetics and signature sequences: a reappraisal of evolutionary relationship among archaebacteria, eubacteria, and eukaryotes. *Microbiol Mol Biol Rev*., 1998; 62: 1435-1491.

[25] Hanage W, Fraser C, Spratt B. Fuzzy species among recombinogenic bacteria. *BMC Biology,* 2005; 3: 6-10.

[26] Webb JS, Givskov M, Kjelleberg S. Bacterial biofilms: prokaryotic adventures in multicellularity. *Curr Opin Microbiol.,* 2003; 6(6): 578-85.

[27] Whitchurch CB, Tolker-Nielsen T, Ragas PC, Mattick JS. Extracellular DNA Required for Bacterial Biofilm Formation. *Science,* 2002; 295(5559): 1487.

[28] Chen Y, Cai T, Wang H. Regulation of intracellular cholesterol distribution by Na/K-ATPase. *J Biol Chem.,* 2009; 284(22): 14881-90.

[29] Fritzsche M. Seasonal correlation of sporadic schizophrenia to Ixodes ticks and Lyme borreliosis. *Int J Health Geogr.*, 2002; 1(1): 2.

[30] Waltrip RW 2nd, Buchanan RW, Summerfelt A, Breier A, Carpenter WT Jr, Bryant NL, Rubin SA, Carbone KM. Borna disease virus and schizophrenia. *Psych Res.*, 1995; 56(1): 33-44.

[31] Torrey EF, Yolken RH. Toxoplasma gondii and schizophrenia. *Emerg Infect Dis.*, 2003; 9(11): 1375-80.

[32] Poole AM. Did group II intron proliferation in an endosymbiont-bearing archaeon create eukaryotes? *Biol Direct*, 2006; 1: 36-40.

[33] Villarreal LP. How viruses shape the tree of life. *Future Virology*, 2006; 1(5): 587-595.

[34] Lockwood M. *Mind, Brain and the Quantum*. Oxford: Blackwell, 1989.

[35] Lefebvre P, Cariou B, Lien F, Kuipers F, Staels B. Role of bile acids and bile acid receptors in metabolic regulation. *Physiol Rev.*, 2009; 89(1): 147-191.

[36] Eberl M, Hintz M, Reichenberg A, Kollas A, Wiesner J, Jomaa H. Microbial isoprenoid biosynthesis and human γδ T cell activation. *FEBS Lett.*, 2010; 544(1): 4-10.

[37] Wallace DC. Mitochondria and Cancer: Warburg Addressed. *Cold Spring Harbor Symp Quant Biol.*, 2005; 70: 363-374.

Chapter 2

Archaeal Digoxin and the Model of the Mind - Digoxin Mediated Model of Conscious Perception and Quantal Perception

Archaeal digoxin a membrane Na^+-K^+ ATPase inhibitor may probably regulate conscious perception. The elements of conscious perception include perceptual binding, focussed attention and short-term memory. The evidence of increased hypothalamic archaeal digoxin in schizophrenia points to a role for the hypothalamus. The hypothalamus is connected to the thalamus by the mamillothalamic tract and digoxin may play a role in regulating these synapses. There are two-way connections between the cerebral cortex and the thalamic nucleus. There are also two-way connections between the cerebral cortex and the hypothalamus and digoxin may also regulate these synapses. The hypothalamus-thalamus-cerebral cortex reverberatory circuit would play a role in mediating conscious perception.

Perceptual binding important in consciousness occurs when all the neurons associated with any one object's perceptual map in layer 5 of the cerebral cortex fire in bursts and in a synchronised pattern but out of synchrony with those representing other objects. When an object is perceived there is a simultaneous activation of the cerebral cortex-hypothalamic two-way connections and liberation of digoxin from the hypothalamus to stimulate the widely dispersed cerebral cortical neurons receiving the incoming perception and their resultant synchronised burst firing. Digoxin and the sodium potassium ATPase inhibition it produces can lead on to a paroxysmal depolarisation shift resulting in sustained synchronised burst firing of cerebral cortical neurons.

Short-term memory, important in conscious perception, depends on the hypothalamic-thalamic-cerebral cortex reverberatory circuit as well as the phenomena of sustained svnchronised burst firing of neurons in layer 5 of the cerebral cortex. Sustained synchronised burst firing produced by digoxin can temporarily strengthen the relevant synapses so that this particular pattern of firing is recalled quickly similar to a type of short-term memory. Transient

synaptic changes of this type are due to an alteration in the presynaptic neuronal calcium produced by digoxin. The thalamic-cerebral cortex reverberatory circuit mediating short term memory is glutamatergic and digoxin could amplify the circuit by its inhibitory effect on glial uptake of glutamate and increasing synaptic glutamate content.

All axons that pass either way between the cerebral cortex and thalamic nucleus must go through the thalamic reticular nucleus and all give off collateral excitatory glutamatergic abranches that innervate the reticular nucleus. The reticular nucleus in turn provides an inhibitory GABAergic innervation back to the thalamic nucleus that provides the input. Reticular nucleus is involved in mediating selective attention by intensifying or detaching a particular active thalamic input into the cortex. The amplification or focussing and detachment of attention occurs by digoxin's effect in promoting glutamatergic transmission in the collaterals to the reticular nucleus by inhibiting the glial uptake of the glutamate and increasing its synaptic content. The back projections from the cerebral cortical perceptual map of external world to hypothalamus decides whether hypothalamic archaeal digoxin should act on the glutamatergic collaterals to reticular nucleus and thus focus or detach attention.

A quantal perception model for brain function and consciousness has been proposed by several groups of workers. The brain is hypothesized to function as a quantum computer. Reiki like healing practices involving the transfer of a low level electromagnetic force from the healer to the patient has been in use in patients with seizure disorders. The Reiki like treatment practices, if effective are hypothesized to act via quantal perception since the electromagnetic force is too weak to be transferred by normal sensory perceptive mechanisms. The present study was conducted to assess the efficacy of such treatment protocols in epileptic patients. The seizure frequency was used as the end point to assess

the efficacy of treatment. Previous reports have demonstrated an endogenous membrane Na^+-K^+ ATPase inhibition related biochemical cascade in primary generalised epilepsy. Elevated levels of a hypothalamic archaeal endogenous membrane Na^+-K^+ ATPase inhibitor digoxin has been reported in epilepsy. It was considered pertinent to study the changes in the membrane Na^+-K^+ ATPase inhibition cascade in seizure patients undergoing Reiki like treatment practices. The results are discussed in this paper and a hypothalamic archaeal digoxin mediated quantal/extra sensory perceptive model of brain function is proposed.

The human brain can be considered as a modified archaeaon colony network. The archaeaon are eternal and can last for billions of years. The human brain is basically an information storage system. The archaeaon has got dipolar magnetite and porphyrins and can function as quantal computer. The archaeal colony with its dipolar magnetite and porphyrin in the setting of archaeal digoxin induced membrane sodium potassium ATPase inhibition can function as a pumped phonon system mediating quantal perception. The archaeaon in the brain is capable of information storage at a point in time and space. The experiences and information stored in the archaeaon is immortal and eternal. The archaeaon can have a wave particle existence and can exist in multiple quantal possible states and can inhabit multiple quantal multiverses. The interaction between information stored in quantal computers in multiple different archaeaon systems all over the universe by the quantal interactions results in eternal existence of information in quantal multiverses. The information in the quantal multiverses can have a particulate existence creating a newer mode by quantal interactions between information stored at multiple points of time. This creates the particulate mythic world of human existence. These are what are called as Samsaras. The mind is uploaded into information in the neuronal archaeal colony network and its quantal computers. The

information stored in the archaeal colony network mediated quantal state is eternal and can be considered as a digital version of the brain, a mind downloading technique or whole brain emulation. The archaeal colony network stores the human experiences in an eternal manner and can contribute to biological reincarnation.

Materials and Methods

Fifteen patients with refractory primary generalized epilepsy (patients with persistent seizures on three or more antiepileptic drugs in full dosage and total compliance over a period of 3 years) were chosen for the study. The age of the patients ranged from 30 to 50 years. An equal number of age and sex matched healthy subjects served as controls. All patients and controls were on the same dietary regimen, which gave adequate amounts of trace elements especially magnesium throughout the course of the study. They were not on any drugs like digoxin and lithium. The weight of the patient population was all in the normal range as they were all freshly diagnosed cases. The general health status of the patient population was normal and they did not suffer from any malnutrition. An equal number of age and sex matched healthy subjects served as controls. The control group was free from all systemic diseases and was selected randomly from the generation population of Trivandrum City. The patient and control groups were fed the same normal hospital diet for a period of 2 weeks after admission to the metabolic ward of the hospital. The blood samples were drawn from the control and patient groups after they were on the same hospital diet for 2 weeks. The dietary samples were analysed and were confirmed to be free of lithium contamination by atomic absorption spectrophotometry. They underwent daily Reiki like healing hand therapy for one hour, where the healer meditates, reaches a trance like state and transfers his low level of body EMF by the touch of his hand to the patient. They were clinically assessed with seizure

frequency counts at the end of 3 months of therapy. The following biochemical parameters were assessed at the start of the therapy and at the end of three months: plasma HMG CoA reductase, serum digoxin, serum magnesium and RBC membrane Na^+-K^+ ATPase activity. The serum levels of tyrosine, dopamine, noradrenaline, tryptophan, serotonin and quinolinic acid were also assessed. Fasting blood was collected from each of the patients for various estimations. RBCs were separated within 1 hour of collection of blood for the estimation of membrane Na^+-K^+ ATPase. Serum was used for the estimation of HMG CoA reductase activity. Plasma / serum was used for the estimation of the other parameters. All biochemicals used in this study were obtained from M/s Sigma Chemicals, USA. Activity of HMG CoA reductase of the plasma was determined using the method of Rao and Ramakrishnan by determining the ratio of HMG CoA to mevalonate. For the determination of the Na^+-K^+ ATPase activity of the erythrocyte membrane, the procedure described by Wallach and Kamat was used. Digoxin in the plasma was determined by the HPLC procedure described by Arun, Ravikumar, Leelamma and Kurup. Magnesium and lithium in the plasma was estimated by atomic absorption spectrophotometry. Tryptophan was estimated by the method of Bloxam and Warren and tyrosine by the method of Wong, O'Flynn and Inouye. Serotonin was estimated by the method of Curzon and Green and catecholamines by the method of Well-Malherbe. Quinolinic acid content of plasma was estimated by HPLC (C_{18} column micro Bondapark™ 4.6 x 150 mm), solvent system 0.01 M acetate buffer (pH 3.0) and methanol (6:4), flow rate - 1.0 ml/minute and detection - UV (250 nm). Statistical analysis was done by ANOVA.

Results

(1) Pre-therapy the activity of HMG CoA reductase, concentration of serum digoxin and lithium were increased and RBC Na^+-K^+ ATPase activity and

serum magnesium were reduced in patients with refractory primary generalised epilepsy. Post-therapy the activity of HMG CoA reductase, the concentration of digoxin and lithium were reduced and RBC Na^+-K^+ ATPase activity and serum magnesium were increased in these patients.

(2) The concentration of serum tryptophan, quinolinic acid and serotonin was increased in the plasma while that of lyrosine, dopamine and noradrenaline was decreased in the pre-therapy group. Post-therapy the concentration of serum tryptophan, quinolinic acid and serotonin was reduced in the plasma while that of tyrosine, dopamine and noradrenaline was increased.

(3) The post-therapy seizure frequency showed a significant decrease.

Discussion

Archaeal Digoxin and Epileptogenesis

The results showed that plasma HMG CoA reductase activity and serum digoxin were increased in primary generalised epilepsy. Previous studies in this laboratory have demonstrated the incorporation of ^{14}C-acetate into digoxin in rat brain indicating that acetyl CoA is the precursor for digoxin biosynthesis in mammals also. The elevated HMG CoA reductase activity correlates well with elevated digoxin levels and reduced RBC membrane Na^+-K^+ ATPase activity. The increase in endogenous digoxin, a potent inhibitor of membrane Na^+-K^+ ATPase, can decrease this enzyme activity. The inhibition of Na^+-K^+ ATPase by digoxin is known to cause an increase in intracellular calcium resulting from increased Na^+-Ca^{++} exchange, increased entry of calcium via the voltage gated calcium channel and increased release of calcium from intracellular endoplasmic reticulum calcium stores. This increase in intracellular calcium by

displacing magnesium from its binding sites, causes a decrease in the functional availability of Mg^{++}. This decrease in the availability of magnesium can cause decreased mitochondrial ATP formation, which along with low magnesium can cause further inhibition of Na^{+}-K^{+} ATPase, since ATP magnesium complex is the actual substrate for this reaction. Cytosolic free calcium is normally buffered by two mechanisms, ATP dependent calcium extrusion from cell and ATP dependent sequestration of calcium within the endoplasmic reticulum. The magnesium related mitochondrial dysfunction results in defective calcium extrusion from the cell. Thus is there a progressive inhibition of Na^{+}-K^{+} ATPase activity firstly triggered by digoxin. Low intracellular magnesium and high intracellular calcium consequent to Na^{+}-K^{+} ATPase inhibition appear to be crucial to the pathogenesis of primary generalised epilepsy. Serum magnesium was found to be reduced in refractory primary generalized epilepsy and serum lithium was increased. Lithium is also a membrane Na^{+}-K^{+} ATPase inhibitor. The generation of endogenous lithium can lead on to further added membrane Na^{+}-K^{+} ATPase inhibition. Membrane Na^{+}-K^{+} ATPase inhibition can produce defective neuronal membrane repolarisation and a paroxysmal depolarisation shift resulting in epileptogenesis.

The digoxin, apart from affecting cation transport, is also reported to influence the transport of various metabolites across cellular membranes, including amino acids and various neurotransmitters. The present study shows that the concentration of tryptophan, quinolinic acid, and serotonin were higher in the plasma of epilepsy patients while that of tyrosine, dopamine, and norepinephrine were lower. Thus there is an increase in tryptophan and its catabolites and a reduction in tyrosine and its catabolites in the patient's serum. This could be due to the fact that digoxin can regulate neutral amino acid transport system with a preferential promotion of tryptophan transport over

tyrosine. The decrease in the membrane Na^+-K^+ ATPase activity in primary generalised epilepsy could also be due to the fact that the hyperpolarising neurotransmitters (dopamine and noradrenaline) are reduced and the depolarising neuroactive compounds (serotonin and quinolinic acid) are increased. Dopamine deficiency in primary generalised epilepsy and dopamine receptor blockade producing epileptogenesis have been documented in literature. The neurotransmitter pattern of reduced dopamine and noradrenaline and increased serotonin could contribute to epilepsy related psychosis. Quinolinic acid, an NMDA agonist can contribute to NMDA excitotoxicity reported in epilepsy. In the presence of hypomagnesemia, the magnesium block on the NMDA receptor is removed leading to NMDA excitotoxicity. The increased presynaptic neuronal calcium can produce cyclic AMP dependent phosphorylation of synapsins resulting in an increased glutamate release into the synaptic junction and vesicular recycling. Increased intracellular calcium in the post-synaptic neuron can also activate the calcium dependent NMDA signal transduction. The plasma membrane glutamate transporter (on the surface of the glial cell and presynaptic neuron) is coupled to a Na^+ gradient, which is disrupted by the inhibition of Na^+-K^+ ATPase, resulting in decreased clearance of glutamate by presynaptic and glial uptake at the end of synaptic transmission. By these mechanisms, inhibition of Na^+-K^+ ATPase can promote glutamatergic transmission and excitotoxicity contributing to epileptogenesis.

Quantal Perception Model of Brain Function

A quantal perception model of brain function has been postulated by several groups of workers. Though conscious perception is the dominant form of perception in the brain, external world information is also gained by quantal perception for integration into the conscious conical perceptual data bank. The low level of EMF from the healer is probably transferred to the recipient patient

by quantal perception. The perceived element in quantal or subliminal perception could be the quanta of matter dependent upon electric and magnetic fields. The brain functions as a quantum computer with the quantum computer memory elements constituted of superconducting quantum interference devices - the SQUIDS can exist as superpositions of macroscopic states. Bose condensation, the basis of superconductivity is achievable at room temperature in the Frohlich model in biological systems. The dielectric protein molecules and polar sphingolipids of the neuronal membrane, nucleosomes (which are a combination of basic histones and nucleic acid) and cytoplasmic magnetite molecules are excellent electric dipole oscillators, which exist under a steep neuronal membrane voltage gradient. The individual oscillators are energised with a constant source of pumping energy, by the digoxin binding to the membrane Na^+-K^+ ATPase and producing a paroxysmal depolarisation shift in the neuronal membrane. This prevents the dipole oscillators from over settling into thermal equilibrium with the cytoplasm and interstitial fluid which is always kept at a constant temperature. This results in a neuronal quantal state. There are direct connections between the hypothalamus and cerebral cortex and digoxin may serve as neurotransmitter for these hypothalamo-cortical synapses. Bose condensed states produced by digoxin mediated dielectric protein molecular pumped phonon system could be used to store information which might be encoded, - all within the lowest collective frequency mode - by appropriately adjusting the amplitudes of and phase relations between the dipole oscillators. The external world sensory impressions exist in the cortical dipole oscillators as probabilistic multiple superimposed patterns - the U phase of quantum mechanics. The part of the incoming quantal data maps of the external world built by quantal perception in logical sequence and corollary to the preexisting cortical external world maps built by conscious perception is chosen. Hypothalamo-cortical connections mediated by digoxin acting on the neuronal

membrane help to magnify the chosen map to one graviton criteria and to the threshold required for the neuronal network to fire and consciousness. It is then integrated into the cortical conscious perceptual external world map. The comparison between quantal perceptive maps and conscious perceptual maps of the external world occurs by a quantal non-local quasicrystal tiling effect which mediates the activation and deactivation of synapses through the contraction and growth of dendritic spines. This model of quantal perception gives a mechanism for extrasensory or subliminal perception. The R part of quantal subthreshold perception is not deterministic and it introduces a completely random element into the time evolution, and in the operation of R there might be a role for free will, an important component of conscious perception.

Quantal Perception, Biological Transmutation and Reiki Like Healing Practices

Reiki like healing practices may affect neuronal function via quantal perception. Post - therapy there was an increase in RBC Na^+-K^+ ATPase activity and serum magnesium and a reduction in HMG CoA reductase activity and digoxin synthesis. Also the level of tyrosine and its hyperpolarising catabolites (dopamine and noradrenaline) increased while that of tryptophan and its depolarising catabolites (serotonin and quinolinic acid) decreased. Reiki like healing practices can transmit low level EMF from the healer to the recipient by a quantal perceptive mechanism. A low level of EMF can stabilise the neuronal membrane and increase the neuronal membrane Na^+-K^+ ATPase activity. The stimulation of Na^+-K^+ ATPase is known to cause a decrease in intracellular calcium resulting from decreased Na^+-Ca^{++} exchange, decreased entry of calcium via the voltage gated calcium channel and decreased release of calcium from intracellular endoplasmic reticulum calcium stores. This decrease in intracellular calcium causes an increase in the functional availability of

intraneuronal Mg^{++}. Magnesium excess is known to inhibit HMG CoA reductase activity. Digoxin is synthesized by the isoprenoid pathway and HMG CoA reductase is the rate limiting enzyme of this pathway. This leads to reduced digoxin synthesis. Reduced levels of digoxin can stimulate membrane Na^{+}-K^{+} ATPase further and increase intraneuronal magnesium to a greater extent. This starts off a cascade, which stimulates the membrane Na^{+}-K^{+} ATPase further and stabilises the neuronal membrane. The stimulation of membrane Na^{+}-K^{+} ATPase can promote neuronal membrane repolarisation and inhibit the generation of a paroxysmal depolarisation shift and epileptogenesis. Thus, stabilisation of the neuronal membrane leads to a reduction in seizure count and seizure frequency. Digoxin is known to promote tryptophan transport over tyrosine. Low levels of digoxin can lead to an increase in serum tyrosine levels and a decrease in serum tryptophan. This leads to an increase in the levels of tyrosine catabolites and a decrease in the levels of tryptophan catabolites. The increased levels of noradrenaline and dopamine have an antiepileptic action. The increase in serum Mg^{++} also helps to downregulate glutamatergic transmission and inhibits epileptogenesis. The Mg^{++} block on the glutamate NMDA receptor is strengthened.

The decrease in serum magnesium and the increase in serum lithium in the pre-therapy group and the increase in serum magnesium and the reduction in serum lithium in the post - therapy group could also be due to the phenomena of biological transmutation. The increase / decrease in serum magnesium / lithium in the pre - / post-therapy group is significant in the presence of similar normal dietary regime and magnesium / lithium intake for the pre and post-therapy groups. Serum magnesium / lithium levels are increased / decreased, suggesting an increase/decrease in total body magnesium / lithium rather than a functional replacement of calcium with magnesium. Biological transmutation has been

postulated by several groups of workers. Hypothalamic archaeal digoxin induced pumped phonon system produces a quantal state within the neuron and in the cell membrane. In this quantal state biological transmutation between lithium and magnesium can happen leading to an increase / decrease in serum magnesium / lithium levels despite only adequate or normal intake. The evidence for this is the correlation between the increase / decrease in serum magnesium and lithium in the pre / post-therapy groups. In the pre-therapy group serum magnesium is reduced and serum lithium is increased. In the post-therapy group serum magnesium is increased and serum lithium is reduced.

Transmutation has been described in biological systems. Low temperature transmutation or cold fusion has been described. For the first time the experimental study of cold nuclear transmutation of isotopes was carried out by Vysotskii in a growing microbiological culture with controlled conditions of growth. With the help of the Mossbauer effect the formation of Fe isotope from Mn in nutrient medium based on heavy water was observed. It can be shown that quantizing structures of optimal size and shape are necessary for such non-barrier nuclear interaction. The exact parameters of these structures are very hard to calculate. The situation substantially improves when the hole parameters are slowly changing inevitably passing through optimal value. This situation is realized in growing microbiological cultures. In the growth process the replication of DNA and other biomolecules takes place. In this case, in the region of growth, the interatomic potential holes with slowly changing sizes are constantly appearing. If Mn atom and a deuton are in such a changing hole, conditions for a new Fe isotope fusion could appear. If all the above-mentioned conditions are met, the quantizing of the deuton in the hole independent from Mn nucleus takes place. In this case the wave function of deuton $\psi_n(r)$ in this optimal hole does not depend on the position of Mn in the hole and in all states

of its quantized motion can differ from zero at the point r_{Mn} in the hole where the Mn nucleus is located. This leads to a high probability of nuclear fusion $\lambda = C\ |\psi_n(r_{Mn})|^2$, C is the constant of purely nuclear Mn-d^2 interactions. The mechanisms provide a short term elimination of the Coulomb barrier of the pair interaction in optimal micro-potential hole with the structure that is close to parabolic. For such a system the diagonal elements of interaction energy matrix of Mn+d complex [quasimolecule $(MnD)^+$] should be small and the probability of interlayer transition because of this interaction should also be small.

Thus the effect of Reiki like treatment practices on seizure count and frequency as well as on biochemical pathways related to membrane Na^+-K^+ ATPase stimulation provides evidence regarding quantal perception and brain function. It also provides evidence on the regulation of metabolic processes by quantally perceived low levels of EMF induced changes in neuronal transmission. The phenomena of psycho-neuro-molecular biological and an environmental low level of EMF mediated regulation of metabolic processes needs further study.

Quantal Perception and Extrasensory Phenomena

(1) *Out of body experience* - This model of quantal perception gives a mechanism for extrasensory or subliminal perception. This model of quantal perception gives an explanation for phenomena like out of body experience, psychokinesis, mind travel and reincarnation.

(2) *Universal mind* - Because of quantal perception different brain information storage systems in different individuals can function as one single quantal supercomputer. This could be the basis of a universal mind or of the experience of God.

(3) *Hypnosis and quantal perception* - Hypnotic suggestions could also work by the phenomena of quantal perception which can alter brain function.

(4) *Inter/intra-galactic fields and behaviour / astrology* - Quantal perception could also result in low strength intra and intergalactic electromagnetic fields of the universe affecting brain function. Solar flares can change the electromagnetic fields of the earth resulting in altered brain function. Inter/intra-galactic fields can also affect brain function. This is exemplified by diurnal and seasonal changes in disease incidence as well as human behaviour.

(5) *Mind travel* - The spooky phenomena of communication of two points spread over a long distance but not in physical continuity as described in the Einstein-Podolskly phenomena occurring in the quantal state could form the basis of mind travel. Mind travel from one part of the galaxy to the other could be done via wormholes in the quantal state. There could also be the phenomena of quantal teleportation. The quantal foam is riddled with worm holes.

(6) *The mind-body-universe interrelationship* - Digoxin by modulating conscious perception contributes to the observer function of human consciousness. Human consciousness depends on the information perceived from the external world by conscious or subliminal perception and is momentary. Consciousness or human personality thus depends upon the external world and does not have an independent existence. If there is no external world to observe there is no human consciousness. This has been proved by sensory deprivation studies. It is consciousness that converts the quantal world of probabilities into the classical objective real world of matter by the act of making an observation. The external world comes into existence because of the observer function of human

consciousness. Thus human consciousness and the external world have an interrelated existence. In real terms there is neither the external world nor human consciousness. Both exist as an interrelated mirage more aptly described by the philosophical term of maya. The evidence of this comes from the delusions and hallucinations observed in schizophrenia where elevated levels of digoxin have been demonstrated.

(7) *Reincarnative experiences* - Reincarnative experiences have been described in the first three years of life. This would involve the phenomena of quantal perception. The information stored in the preexisting neuronal quantal computer systems of deceased individuals are perceived and transferred to other developing foetal/neonatal neuronal quantal computer systems. This transferred reincarnative information is integrated with the information perceived quantally during the individual's lifespan. This may be one method of information transfer from generation to generation akin to the genetic transfer of information.

Quantal Perception and the Evolution of the Universe

A quantal perception model of brain function has been postulated by several groups of workers. The human endosymbiotic archaea produces an endogenous membrane Na^+-K^+ ATPase inhibitor digoxin. Archaeal digoxin probably plays a role in mediating quantal perception and consciousness. The observer function role of human consciousness in the existence of matter is important in this respect. An archaeal digoxin mediated model for quantal perception / consciousness is postulated in the article. This article deals with a hypothesis linking the quantal perception of information stored in other individual neuronal networks to the development of synaptic networks in the fetal / infant brain.

Also in the quantal state, self-replication of macromolecules is possible leading to their later self organisation and formation of cellular organelle. The organelle/independent unicellular bacteria cluster together symbiotically to form the neuronal / cell. This leads as to the serial endosymbiotic theory of evolution where the individual cell is thought of as a symbiotic collection of bacteria. This article takes the theory further to postulate that the human brain and other neuronal networks / multicellular organism are a symbiotic collection of flagellated bacteria forming a large colony with later formation of synaptic connections between the individual bacteria. The role of intergalactic magnetotactic bacterial networks in the evolution of the universe is also highlighted in this context. The details of the hypothesis are described in the article.

Digoxin, Quantal Perception and Observer Function of Consciousness

The perceived element in quantal or subliminal perception which could play a role in schizophrenic symptomatology could be the quanta of light, sound, vibration pressure and matter dependent electric and magnetic fields. The brain functions as a quantum computer with the quantum computer memory elements constituted of superconducting quantum interference devices - the SQUIDS can exist as superpositions of macroscopic states. Bose condensation, the basis of superconductivity is achievable at room temperature in the Frohlich model in biological systems. The dielectric protein molecules and polar sphingolipids of the neuronal membrane nucleosomes, which are a combination of basic histones, and nucleic acid, and cytoplasmic magnetite molecules are excellent electric dipole oscillators which exist under a steep neuronal membrane voltage gradient. The individual oscillators are energised with a constant source of pumping energy from outside, by digoxin binding to membrane sodium potassium

ATPase and producing a paroxysmal depolarisation shift in the neuronal membrane. This prevents the dipole oscillators from ever settling into thermal equilibrium with the cytoplasm and the interstitial fluid, which is always kept at a constant temperature. There are connections between the hypothalamus and cerebral cortex and digoxin may serve as a neurotransmitter for these synapses. Bose condensed states produced by a digoxin mediated dielectric protein molecular pumped phonon system could be used to store information which might be encoded - all within the lowest collective frequency mode - by appropriately adjusting the amplitudes of and phase relations between the dipole oscillators. The external world sensory impressions exist in the cortical dipole oscillators as probabilistic multiple superimposed patterns-the U phase of quantum mechanics. The part of the incoming quantal data maps of the external world built by subliminal perception in logical sequence and corollary to the cerebral cortical external world maps built by conscious perception is chosen. Hypothalamo-cerebral cortical connections mediated by digoxin acting on the neuronal membrane help to magnify the chosen map to one graviton criteria and to the threshold required for the neuronal network to fire and consciousness. It is then integrated in to the cerebral cortical conscious perceptual external world map. The comparison occurs by quantal non-local quasicrystal tiling effect, which mediates the activation and deactivation of synapses through the contraction and growth of dendritic spines.

This model of quantal perception gives a mechanism for extrasensory or subliminal perception. The R part of quantal subthreshold perception is not deterministic and it introduces a completely random element into the time evolution and also in the operation of R there might be a role for free will, an important component of conscious perception. It is consciousness that converts the world of probabilities into the classical objective real world of matter by the

act of making an observation. Quantal perception leads to foetal / infant brain quantally perceiving the information stored in other individual neuronal networks. This leads to the initiation of information storage in the fetal synaptic networks and development of synaptic connectivity in the fetal / infant brain. This could be a possible mechanism of information transfer between generations of human populations.

Digoxin by modulating conscious perception contributes to the observer function of human consciousness. Human consciousness depends on the information perceived from the external world by conscious or subliminal perception and is momentary. It is consciousness that converts the quantal world of probabilities into the classical objective real world of matter by the act of making an observation. Thus human consciousness and the external world have an interrelated existence.

The Symbiotic Theory, Isoprenoid Organism and Evolution

Symbiogenesis is evolutionary change, brought about through long-term physical contact between members of different species. A lichen is a partnership of two entirely different kinds of life: a fungus and a photosynthesizer - either a green alga or a cyanobacterium. Symbiogenesis has been instrumental not only for the evolution of the lichens, but also for all plant and animal cells - including, of course, the cells of human beings. Serial endosymbiosis theory holds that all cells with nuclei are composites formed from the mergers of as many as four different kinds of bacteria. Margulis set out the central tenets of the theory: that certain present-day cell components were once free-living bacteria, and that any live being larger than a bacterium is a superorganism whose cells evolved by symbiogenesis through bacterial corporeal mergers. The host cell itself is probably related to Thermoplasma, a heat and acid tolerant

archaebacterium that lacks a cell wall. Mitochondria are related to the proteobacteria, a very common oxygen-breathing walled bacteria that inhabit water of all kinds. Chloroplasts began as photosynthetic bacteria that live in microbial mats, muds, pools and rivers and at the surface of the ocean. In the course of becoming cell organelles, they lost their cell walls and much other equipment needed for independent life. The DNA governing those features - they shed as well, or relinquished to the nucleus of the host. The fourth former bacterium, the calonymphids in the nucleated cells, descended from a spirochete: the former spirochete is a little dark-staining dot called a centriole-kinetosome. This gave the organism the capacity to move and reproduce efficiently. The centriole kinetisome is important in the evolution of dividing nucleated cells.

In the archaeal digoxin induced neuronal quantal state individual dielectric molecules like proteins, nucleic acid, mucopolysaccharides and isoprenoidal lipids can store information and undergo self replication on a preexisting template. Such macromolecules would have been the initial form of life on earth. The isoprenoid macromolecule could have been the initial self replicating organism at the beginning of evolution. Information could have been stored in the isoprenoid-repeating units. It would be tempting to speculate on a role for self-replicating macromolecules like proteins, nucleic acid, mucopolysaccharide and isoprenoids in human diseases. Prions are self replicating proteins and have been implicated in neurodegenerative disorders.

Cellular organelle can be considered as symbiotic conglomeration of these macromolecules, which would have undergone self-organisation. The cell can be considered as a symbiotic collection of these organelle. Each organelle may evolutionally represent an organism - like mitochondria and nucleic acids. The cell may be considered as a symbiotic merger of different bacteria representing individual organelle. It is possible to visualise functional clusterings of such

bacterial cells. The brain with its axonal and dendritic connections can be viewed as a colony of such flagellated bacterial organisms with its interlinking connections. The axons and dendrites have a similarity with the flagella of bacteria. Synaptic connections would have formed in the bacterial colony over a period of time leading on to the formation of the primitive neuronal networks. Such bacterial networks would have evolved into the human brain and various organs with the neural networks controlling the rest of the cluster. The same holds good for all multicellular organisms. Thus there has been no evolution in the real sense only symbiotic clustering of unicellular organism. In this theoretical model there is no evolution but only a different conglomeration of the initially existing macromolecules - anevolution. The clinical evidence for such a theory is seen in immune mediated neurological disorders like acute disseminated encephalomyelitis and systemic disorders like rheumatic fever. The phenomena of molecular mimicry between the bacterial / viral and human antigens would depend on this evolutionary phenomenon.

Quantal Perception and Origin of the Universe

The universe can probably evolve in the quantal perceptive stage of the brain or in the super conscious meditative state. To begin with there were quantal electromagnetic traces of the past, present and future existing, which unfolded into matter as the universe, evolved. Initially in the intergalactic magnetic fields there existed protons, neutrons and electrons. They combined to form the hydrogen, nitrogen, carbon and oxygen atom.

Fusion of this nuclei resulted in the formation of amino acids and isoprenoid lipids. The amino acids later self organised to form proteins and enzymes. The isoprenoid unit self organised to form isoprenoidal macromolecules. The initial organism was a proteolipid organism formed of proteins and isoprenoid

macromolecules. The membrane Na^+-K^+ ATPase, which could also function as an ATP synthesizing enzyme functioned as the initial energy reservoir. A primitive mitochondria formed of the isoprenoidal compound ubiquinone and proteins with iron sulphur centers evolved later. All the macromolecules of the proteolipid organism underwent self-replication. The isoprenoidal compound digoxin would have functioned as an endogenous membrane N&-K ATPase inhibitor regulating intracellular calcium / magnesium ratios and the function of various organelle. Digoxin would also induce a cellular quantal perceptive state.

Later dUTP, dATP, dGTP and dCTP were synthesized and RNA was formed leading to the second generation RNA organism. The RNA was formed initially on a poly-amino acid chain serving as a template. The RNA started to function as the information storage structure of the organism. The ribosomes evolved as the next stage and protein synthesis was initiated.

Later on with RNA serving as a template the DNA was formed and the DNA organisms came into existence. Thus the initial unicellular bacterial organisms came into existence.

The initial organisms in the intergalactic magnetic fields were the magnetotactic bacteria. The networks of magnetotactic bacteria served as an observer for the creation of the universe according to anthropomorphic principle. The universe came into existence because there was an initial observer in the magnetotactic bacterial networks. 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 hypothesis the cosmic dust magnetotactic bacteria play 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. This magnetic field can be used to trace the spiral arms of the galaxy

following a pattern of field lines that essentially connect young stars and dust in which new stars are forming at a rapid rate. Studies have shown that a fraction of the dust particle has 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 effect 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 supplies of 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 skated before produces water, crucial for bacterial growth. Cosmic biology of magnetotactic bacteria and star formation are thus closely interlinked. Systems like solar system 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 evolve. The key to maintaining control over the rotation seems to lie in the intergalactic magnetic fields 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 - a cosmic intelligence 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 like magnetotactic bacteria would have interacted with the earth throughout its entire history of 4.5 million years. A thin skin of graphitised material around a single bacteria or clumps of bacteria can shield the interior form destruction by UV light. The sudden surges of evolution and diversification of species of plants and animals and their equally sudden extinctions evident in fossil record point to sporadic evolution produced by induction of fresh cometary genes with the arrival of each major new crop of comets. Cometary genes would get grafted on to preexisting biological stock leading to dramatically new lines. The most likely route to ground level for an extra terrestrial microorganism that comes to be dispersed in the stratosphere is via the rain. The micro-organism would effectively serve as a nucleus around which a particle of water ice could grow. The cosmic dust grains of bacteria have been hypothesized to seed the clouds to produce rain.

The evolution of neural networks in living things would have been from self-organised clumps of magnetotactic bacteria. The primitive magnetotactic bacterial clumps would have evolved into higher life forms. The clumps of magnetotactic bacteria would have been the origin or progenitor of all neural organisations in living beings. It would have evolved initially to the

polysynaptic neural networks of the initial multicellular organisms and by progressive cephalisation to organised brains. This could be the evolutionary origin of the magnetoreceptors and magnetoperceptive functions of organisms.

The general systems theory shows that living organisms tend naturally to self organise towards increasingly complex and hierarchal structures described by what is known as creative evolution. Consciousness itself, the highest fruit of such biological systems and typical of the highest tiers of hierarchal systems in general, exerts downward control over the various level beneath. For the evolution of order and complexity as described, the pattern of already created assemblies is conserved and used over again, this suggests the existence of memory in nature or a memory field.

The self is a collection of momentary images of the external world stored in the neuronal networks mediating short-term memory. The sum total of information in various neuronal networks in living selves/organism taken as a collective universal whole represents a memory field serving the purpose of information storage for self organisation to higher levels of complexity. This collective mind represents the memory field of nature. The memory field information is conserved by an interneural network transfer of informational traces by quantal perception.

The role of this collective mind or memory field in self organisation or evolution of complex life forms in earth is comparable to the intergalactic magnetotactic bacterial clumps related magnetic fields playing a role in formation of galaxies and solar systems. The intergalactic magnetic field of magnetotactic bacteria, the cosmic brain communicates with animal and human brains via quantal perception. The intergalactic magnetic field and geomagnetic field of earth can thus regulate human brain function. It is a well defined

non-locally interconnected closely knit system of interacting intergalactic, geomagnetic - and neuronal magnetic fields.

Digoxin and Morphogenesis / Embryogenesis - Reincarnative Experiences - Archaeal Digoxin and Fertilisation

The fertilisation between the ovum and the sperm results in the formation of the embryo. The mechanism of fertilisation has been worked out in the sea urchin embryo. There is an increase in the enzyme nitric oxide synthase within the sperm head prior to fertilisation, Nitric oxide synthase catalyses the formation of nitric oxide. This nitric oxide is injected in to the ovum. There is an increase in calcium in the embryo before embryogenesis starts. The same mechanisms are postulated to occur in the human embryo also before fertilisation.

Studies with rabbit embryos have demonstrated elevated endogenous digoxin in the embryos after fertilisation. This digoxin is synthesized by the human hypothalamus as well as locally in the sperm head and probably in the ovum by the isoprenoid pathway. Digoxin produces membrane Na^+-K^+ ATPase inhibition and this results in an increase in intracellular calcium and reduction in intracellular magnesium. Magnesium can regulate the function of DNA polymerase, RNA dependent DNA polymerase, DNA ligase and ribosomal integrity. Magnesium can thus regulated the DNA replication, translation and protein transcription process. In the presence of magnesium deficiency the ribosome will disintegrate. The alteration in embryonal calcium / magnesium ratios can regulate all this cell function. Thus embryogenesis depends upon hypothalamic archaeal digoxin secretion.

Reincarnative experiences could result from a transfer of information by quantal perception to virgin newborn minds to create an initial bank of

information to which new information is added by experiences in life. This could create a continuous process of information transfer from generation to generation creating different personalities as an end product of interactions occurring in forward directions through years and centuries. Human almanacs have been described in several civilisations describing the past, the present and future births of individuals. The basis of it could be by transfer of information by quantal perception. In the quantal state the past, the present and future exists at the same time. This could be the basis of mind travel to the future and to the past.

Neuronal network independent electromagnetic traces can get impregnated within the embryo during the early stage of morphogenesis. Digoxin can produce a dielectric protein molecule pumped phonon system. This can produce a Bose condensed state at normal temperature resulting in quantal perception of neuronal network independent electromagnetic traces by the embryonal neuronal network developing in the brain. This could be the basis of reincarnative experiences. The NIMHANS study has shown evidenced for reincarnative experiences during the early stage of human development especially in childhood. The existence of spontaneous activity in foetal brain even before patterned sensory experience could be in part due to the reception of neuronal net work independent electromagnetic information traces of deceased individuals.

References

[1] Kurup RK, Kurup PA. *Hypothalamic Digoxin, Cerebral Dominance and Brain Function in Health and Diseases*. New York: Nova Medical Books, 2009.

Chapter 3

Archaeal Digoxin - A Model for Conscious and Subliminal Perception, Cerebral Dominance, Neuro-Immuno-Endocrine Integration and Regulation of Cellular Functions - Relation to Brain Evolution and Systemic Disease / Psychological / Physiological States

Introduction

There is a specialization of function in the right and left hemispheres of the brain as manifested in cognitive dysfunctions noticed in lesions of the same. Typical cerebral lateralization is associated with left cerebral dominance for language, praxis and serial processing, whereas the right cerebral hemisphere is dominant for externally directed attention, visuospatial tasks and gestalt processing. The right hemisphere is also dominant for emotional stimuli, and patients with right cerebral lesions may exhibit hypoarousal and emotional indifference. Geschwind postulated a relationship between cerebral lateralisation and immune function. For example, they observed a higher frequency of left handedness in patients with some immune disorders. Differences in natural killer cell activity have been reported in women as a function of asymmetries in frontal EEG activation. Bardos et al. demonstrated that lesions of the left neocortex in mice depress T-cell immunity, whereas right lesions enhance T-cell immunity. There is no data as of now on neurotransmitter differences between right and left hemispheres though functional differences have been noticed as described above. The hypothalamus produces an endogenous membrane Na^+-K^+ ATPase inhibitor digoxin. Digoxin being a steroidal glycoside is synthesized by the isoprenoid pathway. Studies using ^{14}C labelled acetate has demonstrated the synthesis of digoxin by the isoprenoid pathway. Digoxin can regulate synaptic transmission of multiple neurotransmitter systems. The other products of the isoprenoid pathway are also important in cellular functions. Cholesterol is an important component of cellular membranes. Ubiquinone is a component of the mitochondrial electron transport chain and also functions as a free radical scavenger. Dolichol is important in N-glycosylation of protein and processing of proteins. The present study assessed the changes in the synthesis of an endogenous membrane Na^+-K^+

ATPase inhibitor, digoxin and neurotransmitter changes in right hemispheric dominant and left hemispheric dominant individuals. The pathological and psychological correlates of cerebral dominance in relation to endogenous digoxin synthesis have also been documented in this paper. The metabolic differences in the setting of glycoconjugate metabolism membranogenesis and free radical metabolism between right hemispheric dominant and left hemispheric dominant individuals are documented and its relation to systemic and neuropsychiatric diseases is stressed. A model of conscious and subliminal perception mediated by digoxin and its role in regulating interface between the consciousness and universe is postulated. A hypothesis implicating membrane Na^+-K^+ ATPase inhibition in cellular and brain evolution is also put forward.

Materials and Methods

I. The parameters listed below were assessed in right handed / left hemispheric dominant, left handed / right hemisphere dominant and amphidextrous / bihemispheric dominant individuals chosen by the dichotic listening test. There were 15 normal individuals in each group. The age of the individuals chosen ranged from 20 to 30 yrs. None of the subjects studied were on medication at the time of blood removal. They were all non-smokers (active or passive). (1) The isoprenoid pathway - HMG CoA reductase, serum digoxin, dolichol and ubiquinone, (2) RBC Na^+-K^+ ATPase activity and serum magnesium, (3) Neurotransmitter patterns - tryptophan, serotonin, dopamine, noradrenaline, tyrosine, quinolinic acid, strychnine, nicotine and morphine, (4) Lysosomal enzymes, (5) Total glycosaminoglycans and different GAG fractions, (6) Hexose, fucose and sialic acid content of serum glycoproteins, (7) Free radicals and scavenging enzymes, and (8) RBC membrane composition.

II. Serum digoxin levels and RBC Na^+-K^+ ATPase activity was assessed in different pathological conditions - Parkinson's disease (PD), Huntington's disease, CNS glioma, Multiple sclerosis (MS), schizophrenia, primary generalised epilepsy, idiopathic basal ganglia calcification, migraine, addiction, healthy aging, obsessive compulsive disorder, depression, recurrent respiratory infections, osteoporosis, essential hypertension, syndrome X, low body mass index, anorexia nervosa, bulimia nervosa, osteoarthritis, spondylosis (cervical and lumbar), acute coronary artery disease, idiopathic hypotensive states, subacute sclerosing panencephalitis (SSPE), neurolupus (SLE), rheumatoid arthritis, acquired immunodeficiency syndrome (AIDS), acid peptic disease (APD), irritable bowel syndrome (IBS), gall stones, bronchial asthma, cirrhosis liver, mesenteric artery occlusion, inflammatory bowel disease (IBD), interstitial lung disease (ILD), sarcoidosis, chronic bronchitis emphysema, lone atrial fibrillation with embolic stroke, chronic renal failure (CRF), nephrotic syndrome, and nephrolithiasis. There were 15 patients in each of the disease groups mentioned above. 15 individuals who were bihemispheric dominant served as controls. None of the subjects studied was under medication at the time of removal of blood. They were all non-smokers (active or passive).

III. Serum digoxin levels and RBC Na^+-K^+ ATPase activity was assessed in different psychological conditions - spiritually inclined individuals, creative individuals, addiction, promiscuous individuals, anorexic, gastronomic, insomniac, individuals with increased bonding and affection. They were also assessed in individuals with the opposite psychological tendencies. There were 15 normal individuals in each of the above group. 15 individuals who were bihemispheric dominant served as controls. None

of the subjects studied were on medication at the time of blood removal. They were all non-smokers (active or passive).

Fasting blood was removed in citrate tubes from each of the number of patients mentioned above. RBCs were separated within one hour of collection of blood for the estimation of membrane Na^+-K^+ ATPase. Plasma was used for the analysis of various parameters. The methodology used in the study was as follows: All biochemicals used in this study were obtained from M/s Sigma Chemicals, USA. Activity of HMG CoA reductase of the plasma was determined by the method of Rao and Ramakrishnan by determining the ratio of HMG CoA to mevalonate. For the determination of the Na^+-K^+ ATPase activity of the erythrocyte membrane, the procedure described by Wallach and Kamat was used. Digoxin in the plasma was determined by the procedure described by Arun et al. For an estimation of ubiquinone and dolichol in the plasma, the procedure described by Palmer et al was used. Magnesium in the plasma was estimated by atomic absorption spectrophotometry. Tryptophan was estimated by the method of Bloxam and Warren and tyrosine by the method of Wong et al. Serotonin was estimated by the method of Curzon and Green and catecholamines by the method of Well-Malherbe. Quinolinic acid content of plasma were estimated by HPLC (C_{18} column micro Bondapak^{TM} 4.6 x 140 mm), solvent system 0.01 M acetate buffer (pH 3.0) and methanol (6:4), flow rate 1.0 ml/minute and detection UV (250 mm). Morphine, strychnine and nicotine were estimated by the method described by Arun et al. Details of the procedure used for the estimation of total and individual GAG, carbohydrate component of glycoproteins, activity of enzymes involved in the degradation of GAG (beta glucuronidase, beta N-acetyl hexosaminidase, hyaluronidase and cathepsin-D) and activity of glycohydrolases (beta galactosidase, beta fucosidase and beta glucosidase) are described previously. Serum glycolipids (gangliosides,

glycosyl diglycerides, cerebrosides and sulphatides) were estimated as described in methods in enzymology. Cholesterol was estimated by using commercial kits supplied by M/s Sigma Chemicals, USA. SOD was assayed by the method of Nishikini et al. as modified by Kakkar et al. Catalase activity was estimated by the method of Maehly and Chance, glutathione peroxidase by the method of Paglia and Valentine as modified by Lawrence and Burk and glutathione reductase by the method of Horn and Bums. MDA was estimated by the method of Wills and conjugated dienes and hydroperoxides by the procedure of Brien. Reduced glutathione was estimated by the method of Beutler et al. Nitric oxide was estimated in the plasma by the method of Gabor and Allon. Statistical analysis was done by the student's 't' test.

Results

I. The isoprenoid pathway and related biochemical cascade in relation to cerebral dominance

(1) The results showed that HMG CoA reductase activity serum digoxin and dolichol were increased and ubiquinone reduced in left handed / right hemispheric dominant individuals. The results also showed that HMG CoA reductase activity, serum digoxin and dolichol were decreased and ubiquinone increased in right handed / left hemispheric dominant individuals.

(2) The results showed that the concentration of tryptophan, quinolinic acid serotonin, strychnine and nicotine was found to be higher in the plasma of left handed / right hemispheric dominant individuals while that of tyrosine, dopamine, morphine and norepinephrine was lower. The results also showed that the concentration of tryptophan, quinolinic acid serotonin, strychnine and nicotine was found to be lower in the plasma of right

handed / left hemispheric dominant individuals while that of tyrosine, dopamine, morphine and norepinephrine was higher.

(3) There was an increase in lipid peroxidation as evidenced from the increase in the concentration of MDA, conjugated dienes, hydroperoxides and NO with decreased antioxidant protection as indicated by a decrease in ubiquinone and reduced glutathione in left handed / right hemispheric dominant individuals. The activity of enzymes involved in free radical scavenging like superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase and catalase is decreased in left handed / right hemispheric dominant individuals. There was decrease in lipid peroxidation as evidenced from the decreased a concentration of MDA, conjugated dienes, hydroperoxides and NO with increased antioxidant protection as indicated by an increase in ubiquinone and reduced glutathione in right handed / left hemispheric dominant individuals. The activity of enzymes involved in free radical scavenging like superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase and catalase is increased in right handed / left hemispheric dominant individuals.

(4) The results show an increase in the concentration of serum total GAG, glycolipids (ganglioside, glycosyl diglyceride, cerebrosides and sulphatides) and carbohydrate components of glycoproteins (hexose, fucose and sialic acid) in left handed / right hemispheric dominant individuals. The increase in the carbohydrate components - total hexose, fucose and sialic acid-in the disorders studied was not to the same extent in all cases suggesting a qualitative change in glycoprotein structure. The individual GAG fractions - heparan sulphate, hyaluronic acid, heparin, chondroitin sulphates and dermatan sulphate, increased in left handed / right hemispheric dominant individuals. The activity of GAG degrading enzymes (beta glucuronidase,

beta N-acetyl hexosaminidase, hyaluronidase and cathepsin-D) and that of glycohydrolases (beta galactosidase, beta fucosidase and beta glucosidase) showed a significant increase in the serum in left handed / right hemispheric dominant individuals. The results show a decrease in the concentration of serum total GAG, glycolipids (ganglioside, glycosyl diglyceride, cerebrosides and sulphatides) and carbohydrate components of glycoproteins (hexose, fucose and sialic acid) in right handed / left hemispheric dominant individuals. The decrease in the carbohydrate components - total hexose, fucose and sialic acid - in the disorders studied was not to the same extent in all cases suggesting qualitative change in glycoprotein structure. The individual GAG fractions heparin, dermatan sulphate, heparan sulphate (HS), hyaluronic acid and chondroitin sulphates (ChS) decreased in right handed / left hemispheric dominant individuals. The activity of GAG degrading enzymes (beta glucuronidase, beta N-acetyl hexosaminidase, hyaluronidase and cathepsin-D) and that of glycohydrolases (beta galactosidase, beta fucosidase and beta glucosidase) showed a significant decrease in the serum in right handed / left hemispheric dominant individuals.

(5) The cholesterol: phospholipid ratio of the RBC membrane was increased in left handed / right hemispheric dominant individuals. The concentration of total GAG, hexose and fucose of glycoprotein decreased in the RBC membrane and increased in the serum suggesting their reduced incorporation into the membrane and defective membrane formation in left handed / right hemispheric dominant individuals. The cholesterol: phospholipid ratio of the RBC membrane was decreased in right handed / left hemispheric dominant individuals. The concentration of total GAG, hexose and fucose of glycoprotein increased in the RBC membrane and

decreased in the serum suggesting their increased incorporation into the membrane and defective membrane formation in right handed / left hemispheric dominant individuals.

II. Serum digoxin levels and RBC Na^+-K^+ ATPase activity was assessed in different pathological conditions

(1) In Parkinson's disease, Huntington's disease, idiopathic basal ganglia calcification, CNS glioma, multiple sclerosis, schizophrenia, primary generalized epilepsy, syndrome X, migraine, addiction, anorexia nervosa, osteoarthritis, spondylosis, acute coronary artery disease / acute cerebral thrombosis, essential hypertension, SSPE, acquired immunodeficiency syndrome, bronchial asthma, neurolupus, acid peptic disease, irritable bowel syndrome, gall stones, cirrhosis liver, inflammatory bowel disease, sarcoidosis, chronic bronchitis emphysema, interstitial lung disease, chronic renal failure, nephrotic syndrome, nephrolithiasis, lone atrial fibrillation and Fahr syndrome - serum digoxin levels were elevated, RBC membrane Na^+-K^+ ATPase activity reduced and serum magnesium decreased.

(2) In healthy aging, obsessive compulsive disorder, depression, recurrent respiratory infections, osteoporosis, familial hypotension, low body mass index and bulimia nervosa - serum digoxin levels were reduced, RBC membrane Na^+-K^+ ATPase activity increased and serum magnesium increased.

III. Serum digoxin levels and RBC Na^+-K^+ ATPase activity was assessed in different psychological conditions.

(1) In spiritually inclined individuals, creative individuals, addiction, promiscuous individuals, homosexuals, anorexic, insomniac, individuals with reduced bonding and affection and detached behaviour - serum

digoxin levels were elevated, RBC Na^+-K^+ ATPase activity reduced and serum magnesium reduced.

(2) In spiritually non-inclined individuals, non-creative individuals, individuals without addictive behaviour, non-promiscous individuals, individuals with gastronomic tendency, somnolent individuals and individuals with increased bonding and affection - serum digoxin levels were reduced, RBC Na^+-K^+ ATPase activity increased and serum magnesium increased.

Discussion

Archaeal Digoxin and Membrane Na^+-K^+ ATPase Inhibition - Cerebral Dominance

The increase in endogenous digoxin, a potent inhibitor or membrane Na^+-K^+ ATPase, can decrease this enzyme activity in left handed / right hemispheric dominant individuals and in Parkinson's disease, CNS glioma, multiple sclerosis, acquired immunodeficiency syndrome, schizophrenia, primary generalised epilepsy, syndrome X, migraine, addiction, anorexia nervosa, osteoarthritis, spondylosis, acute coronary artery disease, hypertension, SSPE, neurolupus, acid peptic disease, irritable bowel syndrome, cirrhosis liver, inflammatory bowel disease, chronic bronchitis emphysema, interstitial lung disease, sarcoidosis, bronchial asthma, chronic renal failure, nephrotic syndrome, nephrolithiasis, lone atrial fibrillation, gall stones and Fahr syndrome. In all the disorders studied, there was significant inhibition of the RBC membrane Na^+-K^+ ATPase and this inhibition appears to be a common feature for these neuropsychiatric and systemic disorders. In creative individuals, addiction, promiscuous individuals, homosexuals, anorexic, insomniac and individuals with reduced bonding / affection and detached behaviour also serum

digoxin levels are increased and RBC membrane Na^{+}-K^{+} ATPase activity reduced decreased. In all these pathological and psychological states there is chemical right hemispheric dominance. The inhibition of Na^{+}-K^{+} ATPase by digoxin is known to cause an increase in intracellular calcium resulting from increased Na^{+}-Ca^{++} exchange, increased entry of calcium via the voltage gated calcium channel and an increased release of calcium from intracellular endoplasmic reticulum calcium stores. This increase in intracellular calcium by displacing magnesium from its binding sites causes a decrease in the functional availability of magnesium. This decrease in the availability of magnesium can cause decreased mitochondrial ATP formation, which along with low magnesium can cause further inhibition of Na^{+}-K^{+} ATPase, since ATP-magnesium complex is the actual substrate for this reaction. Cytosolic free calcium is normally buffered by two mechanisms, ATP dependent calcium extrusion from cell and ATP dependent sequestration of calcium within the endoplasmic reticulum. The magnesium related mitochondrial dysfunction results in defective calcium extrusion from the cell and is a progressive inhibition of Na^{+}-K^{+} ATPase activity first triggered by digoxin. Low intracellular magnesium and high intracellular calcium consequent to Na^{+}-K^{+} ATPase inhibition appear to be crucial to the pathophysiology of these disorders. The intracellular positive calcium signal and negative magnesium signal can regulate diverse cellular process. Calcium on entry into the cell is used to charge up the internal endoplasmic reticulum stores, which then release a burst of signal calcium responsible for activating a large variety of calcium dependent cellular processes. The information processing capability of the calcium signalling system is enhanced by the amplitude and frequency modulation. The calcium is released from channels on internal ER individually or in small groups (bip/quark and puffs/sparks). Further diversity of calcium signalling is produced by compartmentalization such as a cytosolic calcium signal and

nuclear calcium signal. There is evidence for increased digoxin synthesis in these groups of diseases from the increase in HMG CoA reductase activity that is noticed. HMG CoA reductase is the rate limiting enzyme of the isoprenoid pathway. In this connection, incorporation of ^{14}C-acetate into digoxin in rat brains has been shown by us indicating that acetyl CoA is the precursor for digoxin biosynthesis in mammals. Serum magnesium was assessed in left handed / right hemispheric dominant individuals and in Parkinson's disease, CNC glioma, multiple sclerosis, schizophrenia, primary generalised epilepsy, syndrome X, migraine, addiction, idiopathic basal ganglia calcification, anorexia nervosa, osteoarthritis, spondylosis, acute coronary artery disease, essential hypertension, SSPE, neurolupus, acquired immunodeficiency syndrome, acid peptic disease, irritable bowel syndrome, gall stones, cirrhosis liver, inflammatory bowel disease, chronic bronchitis emphysema, interstitial lung disease, chronic renal failure, lone atrial fibrillation and bronchial asthma and was found to be reduced. In all these pathological and psychological states there is chemical right hemispheric dominance. Increases intracellular calcium can bring about basal ganglia calcification. Increased in intracellular calcium can lead on to increased calcium load in the bone and degenerative bone disease like cervical spondylosis. Increased digoxin can also contribute to the pathophysiology of CRF (chronic renal failure). Digoxin by the membrane Na^+-K^+ ATPase inhibition that it produces can lead to inhibition of the outward sodium flux, and inhibition of the inward potassium flux, and also an increased inward flux of calcium. This abnormally high intracellular sodium concentration and hence to osmotically induced overhydration of the cell, whereas the same cells are relatively deficient in potassium. Digoxin can alter the conduction of the cardiac SA node and AV node as well as the conducting tissue contributing to lone atrial fibrillation. Increases in bronchial smooth muscle calcium can contribute to bronchospasm in bronchial asthma. Similarly an increase in

intestinal smooth muscle cell calcium can lead to irritable bowel syndrome by producing intestinal smooth muscle contraction. Increased intracellular parietal cell calcium and reduced intracellular magnesium can lead on to increased gastric acid secretion. Increased intracellular calcium can also activate the G-protein coupled receptor - histamine, which can cause increased gastric acid secretion. An upregulated isoprenoid pathway and increased cholesterol synthesis can lead on to the formation of gallstones. Hypomagnesemia can lead to inhibition of gall bladder contraction and decreased water content of the bile contributing to the formation of gall bladder sludge. Increased renal tubular cell calcium and accumulation of shed renal tubular cell in the renal pelvis and ureter can bring about the formation of renal stones.

The decrease in the activity of HMG CoA reductase in right handed individuals / left hemispheric dominant and in healthy aging, obsessive compulsive disorder, depression, recurrent respiratory infections, osteoporosis, familial hypotension, low body mass index and bulimia nervosa suggests a downregulation of the isoprenoid pathway. In spiritually non - inclined individuals, non-creative individuals, individuals without addictive behaviour, non - promiscuous individuals, individuals with gastronomic tendency, somnolent individuals and individuals with increased bonding and affection there is also a reduction in HMG CoA reductase activity and down regulation of the isoprenoid pathway. In all these psychological states there is chemical left hemispheric dominance. There is a marked decrease in plasma digoxin and dolichol and this decrease may be a consequence of decreased channelling of intermediates of the isoprenoid pathway for their biosynthesis. The decrease in endogenous digoxin, a potent inhibitor of membrane Na^+-K^+ ATPase, can increase this enzyme activity. In all these cases there was significant stimulation of the RBC membrane Na^+-K^+ ATPase. The stimulation of Na^+-K^+ ATPase by

the decrease in digoxin synthesis is known to cause a decrease in intracellular calcium resulting from decreased Na^{+}-Ca^{++} exchange, decreased entry of calcium via the voltage gated calcium channel and a decreased release of calcium from intracellular endoplasmic reticulum calcium stores. The increased intracellular magnesium related mitochondrial ATP synthesis results in increased calcium extrusion from cell and therefore a progressive stimulation of Na^{+}-K^{+} ATPase activity. High intracellular magnesium and low intracellular calcium consequent to Na^{+}-K^{+} ATPase stimulation appear to be crucial to the pathophysiology of these diseases. Serum magnesium was assessed in right handed / left hemispheric dominant individuals and the above-mentioned psychological and pathological state and was found to be increased. A decrease in bone calcium load can lead on to osteoporosis.

There are three different neurological states, which correlates with various systemic diseases and psychological profiles. The hyperdigoxinemic right hemispheric dominant state, the hypodigoxinemic left hemispheric dominant state and the normodigoxinemic bihemispheric dominant / fluctuating dominant state.

Archaeal Digoxin and Regulation of Neurotransmitter Synthesis and Function - Cerebral Dominance

There is an increase in tryptophan and its catabolites and a reduction in tyrosine and its catabolites in the serum of left handed / right hemispheric dominant individuals. This could be due to the fact that digoxin can regulate a neutral amino acid transport system with preferential promotion of tryptophan transport over tyrosine. The decrease in membrane Na^{+}-K^{+} ATPase activity in all the above psychological and pathological states could be due to the fact that the hyperpolarising neurotransmitters (dopamine, morphine and noradrenaline) are reduced and the depolarising neuroactive compounds (serotonin, strychnine, nicotine and quinolinic acid) are increased. The schizoid neurotransmitter

pattern of reduced dopamine, noradrenaline and morphine and increased serotonin, strychnine and nicotine is common to left handed / right hemispheric dominant individuals and to all these pathological and psychological states and could predispose to their development. Quinolinic acid, a NMDA agonist can contribute to NMDA excitotoxicity reported in schizophrenia. Strychnine, by blocking glycinergic transmission can contribute to the decreased inhibitory transmission in schizophrenia. Recent data suggest, the initial abnormality in schizophrenia involves a hypodopaminergic state and the low dopamine levels now observed agrees with this. Nicotine by interacting with nicotinic receptors can facilitate the release of dopamine, promoting the dopaminergic transmission in the brain. This can explain the increased dopaminergic transmission in the presence of decreased dopamine levels.

The increased serotoninergic activity and reduced noradrenergic outflow from locus coeruleus reported earlier in schizophrenia agrees with our finding of elevated seratonin and reduced noradrenaline levels. A schizophreniform type of psychosis is important in the genesis of irritable bowel syndrome, inflammatory bowel disease, bronchial asthma, acid peptic disease and immune mediated disorders like multiple sclerosis and SLE. In the presence of hypomagnesemia, the magnesium block on the NMDA receptor is removed leading to NMDA excitotoxicity. The increased presynaptic neuronal calcium can produce cyclic AMP dependent phosphorylation of synapsins resulting in an increased neurotransmitter release into the synaptic junction and vesicular recycling. Increased intracellular calcium in the post synaptic neuron can also activate the calcium dependent NMDA signal transduction. The plasma membrane neurotransmitter transporter (on the surface of the glial cell and presynaptic neuron) is coupled to a sodium gradient which is disrupted by the inhibition of Na^{+}-K^{+} ATPase, resulting in a decreased clearance of glutamate by

presynaptic and glial uptake at the end of synaptic transmission. By these mechanisms, inhibition Na^{+}-K^{+} ATPase can promote glutamatergic transmission. The elevated levels of quinolinic acid, strychnine and serotonin can also contribute to NMDA excitotoxicity. Strychnine displaces glycine from its binding sites and inhibits glycinergic inhibitory transmission in the brain. The glycine is free to bind to the strychnine insensitive site of the NMDA receptor and promote excitatory NMDA transmission. Quinolinic acid and serotonin are also positive modulators of the NMDA receptor. Increased glutamatergic transmission resulting in excitotoxicity has been implicated in neuronal degeneration observed in Parkinson's disease, primary generalised epilepsy, schizophrenia and AIDS dementia. Inhibition of Na^{+}-K^{+} ATPase can also result in defective neuronal membrane repolarisation and a paroxysomal depolarization shift resulting in epileptogenesis. Increased nicotine synthesis can contribute to the pathophysiology of chronic bronchitis emphysema. Elevated levels of serotonin and nitric oxide production could contribute to increased incidence of migraine in right hemisphere dominant left handed individuals. Increased intracellular calcium can activate the gastrin and acetyl choline related gastric acid secretion. Increased intracellular calcium in the presynaptic neuron can promote cholinergic transmission. The increased presynaptic neuronal Ca^{++} can produce cyclic AMP dependent phosphorylation of synapsins resulting in increased neurotransmitter release into the synaptic junction and vesicular recycling. This promotes cholinergic vagal transmission promoting acid secretion and peptic ulcer formation. These neurotransmitter patterns can also lead to irritable bowel syndrome. The increase in serotonin can contribute to altered bowel motility in IBS. Serotonin blockers are useful in the treatment of IBS. Reduced morphine and dopamine levels can contribute to the pathogenesis of IBS. Studies have shown that there is endogenous synthesis of morphine from tyrosine and dopamine. Kappa and opioid agonist are useful in

the treatment of bowel motility disorders. The particular neurotransmitter patterns can inhibit gall bladder contractility contributing to the formation of gallstones. Therefore in the right hemisphere dominant hyperdigoxinemic state there is upregulated serotoninergic, cholinergic and glutamatergic transmission and downregulated dopaminergic, glycinergic and noradrenergic transmission. These neurotransmitter patterns could also be correlated with psychological states. There was an increased tendency for spirituality in hyperdigoxinemic individuals. Temporal lobe epileptic phenomenon has been described in spiritual individuals. Increased glutamatergic transmission is associated with memory and intelligence. This can contribute to increased creativity and tendency towards reduced appetite and eating behaviour. Increased serotoninergic transmission can lead to reduced appetite. There was also hypersexual behaviour, homosexuality and promiscuity in hyperdigoxinemic individuals. This could be related to the increased production of nitric oxide in hyperdigoxinemic individuals consequent to the induction of nitric oxide synthase by increased intracellular calcium. Nitric oxide has been related to erectile function. There was an increased tendency to addictive behaviour in hyperdigoxinemic individuals. Endogenous morphine deficiency has been related to addiction. Morphine synthesis is low because of low tyrosine levels. There was tendency to insomnia and reduced sleep. This could be related to reduced levels of morphine. There was less of bonding and affectionate behaviour. Bonding and affectionate behaviour has been related to dopamine and morphine. Dopamine and morphine deficiency in hyperdigoxinemic individuals could contribute to less of bonding and affectionate behaviour.

The results showed that the concentration of tryptophan, quinolinic acid, strychnine, and serotonin was found to be lower in the plasma of right handed / left hemispheric dominant individuals while that of tyrosine, morphine,

dopamine and norepinephrine was higher. Thus there is a decrease in tryptophan and its catabolites and increase in tyrosine and its catabolites in the serum of right handed / left hemispheric dominant individuals and the above described psychological / pathological states. This could be due to the fact digoxin can regulate the neutral amino acid transport system with preferential promotion of tryptophan transport over tyrosine and that digoxin levels are low in right handed / left hemispheric dominant individuals and in the above mentioned pathological / psychological states. The increase in membrane Na^+-K^+ ATPase activity in these cases could be due to the fact that the hyperpolarising neurotransmitters (dopamine, morphine and noradrenaline) are increased and the depolarising neuroactive compounds (serotonin, strychnine, nicotine and quinolinic acid) are decreased. The low level of quinolinic acid, serotonin and strychnine can contribute to reduced excitatory glutamatergic transmission as they are all positive modulators of the NMDA receptor. In the presence of hypermagnesemia, the magnesium block on the NMDA receptor is strengthened leading on to reduced NMDA transmission. The decreased presynaptic neuronal calcium can produce reduced cyclic AMP dependent phosphorylation of synapsins resulting in decrease in glutamate release into the synaptic junction and vesicular recycling. The plasma membrane glutamate transporter (On the surface of the glial cell and presynaptic neuron) is coupled to the sodium gradient, which is activated by the stimulation of Na^+-K^+ ATPase, resulting in increased clearance of glutamate by presynaptic and glial uptake at the end of synaptic transmission. By these mechanisms, stimulation of Na^+-K^+ ATPase can inhibit glutamatergic transmission. Reduced glutamatergic transmission can lead on to healthy aging and protect the brain from neuronal degeneration. The depressive syndrome noted could be due to low serotonin. Decreased serotoninergic transmission has been related to depression. The presence of OCD syndrome could also be related to serotonin depletion. Deficiency of

serotonin can lead to increased appetite and eating behaviour resulting in bulimia nervosa. Dopamine and morphine has been related to bonding behaviour. Increased morphine and dopamine could lead to increased bonding and affectionate behaviour. Increased synthesis of morphine can also lead on to lack of addictive behaviour. Morphine deficiency has been related to addiction. The reduced glutamatergic transmission noted could be related to the average to normal IQ and creativity noticed. Dementia has also been related to depression and the phenomenon of pseudomentia has been described. Decreased production of nitric oxide can lead on to hyposexual behaviour. Synthesis of NO has been related to erectile function. These behavioural patterns are suggestive of left hemispheric dominance.

Archaeal Digoxin and Conscious Perception

The increase in serum digoxin levels in schizophrenia is significant. It has been postulated that there is an underlying generalised disorder of consciousness or self awareness that impairs the ability to think with metarepresentations in schizophrenia. Digoxin, a membrane Na^+-K^+ ATPase inhibitor may probably regulate conscious perception. The elements of conscious perception include perceptual binding, focussed attention and short term memory. The evidence of increased hypothalamic archaeal digoxin points to a role for the hypothalamus. The hypothalamus is connected to the thalamus by the mamillothalamic tract and digoxin may play a role in regulating these synapses. There are two way connections between the cerebral cortex and the thalamic nucleus. There are also two way connections between the cerebral cortex and hypothalamus and digoxin may possibly regulate these synapses also. The hypothalamus-thalamus-cerebral cortex reverberatory circuit would play a role in mediating conscious perception.

Perceptual binding important in consciousness occurs when all the neurons associated with any one object's perceptual map in layer 5 of cerebral cortex fire in bursts and in a synchronised pattern but out of synchrony with those representing other objects. When an object is perceived there is a simultaneous activation of the cerebral cortex-hypothalamic two-way connections and liberation of digoxin from the hypothalamus to stimulate the widely dispersed cerebral cortical neurons receiving the incoming perception and their resultant synchronised burst firing. Digoxin by the sodium potassium ATPase inhibition it produces can lead on to a paroxysmal depolarisation shift resulting in sustained synchronised burst firing of cerebral cortical neurons.

Short-term memory important in conscious perception depends on the Hypothalamic-thalamic-cerebral cortex reverberatory circuit as well as the phenomena of sustained synchronised burst firing of neurons in layer 5 of the cerebral cortex. Sustained synchronised burst firing produced by digoxin can temporarily strengthen the relevant synapses so that this particular pattern of firing is recalled quickly - a type of short-term memory. Transient synaptic changes of this type are due to alteration in the presynaptic neuronal calcium produced by digoxin. The thalamic-cerebral cortex reverberatory circuit mediating short term memory is glutamatergic and digoxin could amp1if the circuit by its inhibitory effect on glial uptake of glutamate and increasing synaptic glutamate content.

All axons that pass either way between the cerebral cortex and thalamic nucleus must go through the thalamic reticular nucleus and all give off collateral excitatory glutamatergic abranches that innervate the reticular nucleus. The reticular nucleus in turn provides an inhibitory GABAergic innervation back to the thalamic nucleus that provides the input. Reticular nucleus is involved in mediating selective attention by intensifying or detaching a particular active

thalamic input into the cortex. The amplification or focussing and detachment of attention occurs by digoxin's effect in promoting glutamatergic transmission in the collaterals to the reticular nucleus by inhibiting the glial uptake of the glutamate and increasing its synaptic content. The back projections from the cerebral cortical perceptual map of the external world to the hypothalamus decides whether hypothalamic archaeal digoxin should act on the glutamatergic collaterals to reticular nucleus and thus focus or detach attention.

In schizophrenia hypersensitivity to perceptual stimulae is noticed as a deficit and patients find it difficult to screen out various stimuli and to focus on one piece of information. The defective stimulus barrier causes difficulty throughout every phase of development. The increased secretion of digoxin produces a hyperconscious state with increased focussed attention, perceptual binding and short-term memory. The altered glycoconjugates in schizophrenia lead to disordered synaptic connectivity in the hypothalamic-thalamic-cerebral cortical circuit leading to disordered conscious perception. Cortical cytoarchitectural disorganization of the temporolimbic cortex has been reported in schizophrenia.

Archaeal Digoxin and Quantal Perception

The perceived element in quantal or subliminal perception which could play a role in schizophrenic symptomatology could be the quanta of light, sound, vibration pressure and matter dependent electric and magnetic fields. The brain functions as a quantum computer with the quantum computer memory elements constituted of superconducting quantum interference devices - the SQUIDS which can exist as superpositions of macroscopic states. Bose condensation, the basis of superconductivity is achievable at room temperature in the Frohlich model in biological systems. The dielectric protein molecules and polar sphingolipids of the neuronal membrane, nucleosomes which are a combination

of basic histones and nucleic acid and cytoplasmic magnetite molecules are excellent electric dipole oscillators which exist under a steep neuronal membrane voltage gradient. The individual oscillators are energized with a constant source of pumping energy from outside, by digoxin binding to membrane sodium potassium ATPase and producing a paroxysmal depolarisation shift in the neuronal membrane. This prevents the dipole oscillators from ever settling into thermal equilibrium with the cytoplasm and the interstitial fluid which is always kept at constant temperature. There are connections between the hypothalamus and cerebral cortex and digoxin may serve as a neurotransmitter for these synapses. Bose condensed states produced by the digoxin mediated dielectric protein molecular pumped phonon system could be used to store information which might be encoded - all within the lowest collective frequency mode - by appropriately adjusting the amplitudes of and phase relations between the dipole oscillators. The external world sensory impressions exist in the cortical dipole oscillators as probabilistic multiple superimposed patterns - the U-phase of quantum mechanics. The part of the incoming quantal data maps of the external world built by subliminal perception in logical sequence and corollary to the cerebral cortical external world maps built by conscious perception is chosen. Hypothalamic-cerebral cortical connections mediated by digoxin acting on the neuronal membrane help to magnify the chosen map to I graviton criteria and to the threshold required for the neuronal network to fire and consciousness. It is then integrated into the cerebral cortical conscious perceptual external world map. The comparison occurs by quantal non-local quasicrystal tiling effect which mediates the activation and deactivation of synapses through the contraction and growth of dendritic spines.

This model of quantal perception gives a mechanism for extrasensory or subliminal perception. Hallucination could be due to subliminal extrasensory perception. Paranoid delusions of persecution and alien control could be due to subliminal perception of thoughts of other persons. Normally quantal subliminal perception plays a minor role being a primitive form of perception and is subservient to conscious perception. Hypothalamic archaeal digoxin induced altered synaptic glycoproteins can lead to synaptic connectivity defects in the hypothalamic-thalamic-cerebral cortical circuit mediating conscious perception and disrupt conscious perceptive mechanism in schizophrenia. But increased hypothalamic archaeal digoxin secretion also leads to a hyperfunctional digoxin mediated dielectric protein molecular pumped phonon system and hypersensitive subliminal quantal perception which is also defectively integrated into conscious perception and is not regulated by conscious perception in schizophrenia. The R part of quantal subthreshold perception is not deterministic and it introduces a completely random element into the time evolution and in the operation of R there might be a role for free will, an important component of conscious perception. It is consciousness that converts the world of probabilities in to the classical objective real world of matter by the act of making an observation. This process is deranged if the observer or human consciousness is dysfunctional owing to a disordered hypothalamic-thalamic-cerebral cortical circuit. This would lead to defective perception of the external world and delusions such as seeing a rope as a snake. ECT produces loss of consciousness and benefit in schizophrenia by interfering with the system of biological dipole oscillator.

In the quantal perceptive state there is no past, present or future. All of them can exist together. This gives an explanation for premonitions and visions of the past. Also in the quantal state action at a distance is possible. This can explain

psychokinesis and mind travel. Quantal perceptive modal of the brain function also gives an explanation for hypnosis. In the quantal state depending on the observer function of consciousness matter can be created. The information store in one brain can be quantally transferred to other brains raising the possibility of reincarnative experiences.

Archaeal Digoxin - Golgi Body / Lysosomal Function - Hemispheric Dominance

The elevation in the level of dolichol in right hemispheric dominance may suggest its increased availability for N-glycosylation of proteins. Magnesium deficiency can lead on to defective metabolism of sphinganine producing its accumulation, which may lead to increased cerebroside and ganglioside synthesis. In magnesium deficiency the glycolysis, citric acid cycle and oxidative phosphorylation are blocked and more glucose 6-phosphate is channeled for the synthesis of glycosaminoglycans (GAG). The concentration of total GAG, different GAG fractions, the carbohydrate component of the glycoproteins and glycolipids are increased in right hemispheric dominant individuals. Intracellular magnesium deficiency also results in defective ubiquitin dependent proteolytic processing of glycoconjugates as it requires magnesium for its function. The increase in the activity of glycohydrolases and GAG degrading enzymes could be due to reduced lysosomal stability and consequent leakage of lysosomal enzymes into the serum. The increase in the concentration of carbohydrate components of glycoproteins and GAG in spite of increased activity of many glycohydrolases may be due to their possible resistance to cleavage by glycohydrolases consequent to qualitative change in their structure. Proteoglycan complexes formed in the presence of altered calcium / magnesium ratios intracellularly may be structurally usually abnormal and resistant to lysosomal enzymes and may accumulate.

Previous reports of alteration in glycoproteins in this connection include alteration in alpha acid glycoprotein (AAG) and beta amyloid precursor protein in epilepsy and Alzheimer's disease and alpha synuclein in Parkinson's disease. Structurally abnormal glycoproteins resist catabolism by lysosomal enzymes and accumulate in neuronal degeneration. Interaction between HS-proteoglycan and ChS-proteoglycan with proteins like beta amyloid, tau protein, parkin and alpha synuclein and reduced proteolytic digestion of these complexes leading on to their accumulation in the neurons have been reported in neurodegenerative diseases like Alzheimer's disease and Parkinson's disease. Alteration in the sulphated proteoglycan matrix of the synaptic vesicles can alter neurotransmitter release into the synapse and produce a functional disorder like schizophrenia and epilepsy. Membrane Na^+-K^+ ATPase inhibition can lead to defective notch signalling. Notch is a transmembrane protein that acts as a signal receptor and is important in neurogenesis. Neuronal growth by extending neurites and forming connections is regulated by the notch signalling pathway. The notch signalling inhibits extension of neurites and keep them stable in the mature brain. A notch ligand known as delta regulates neurogenesis by binding to notch in membranes of embryonal cells and prevents them from developing along the neuronal pathway. Notch activation by the ligand causes notch to be cleaved releasing the notch intracellular domain. This then passes in to the nucleus and activates transcription as part of the DNA binding complex with CSL protein. Intracellular cleavage of the notch is regulated by presenilin and also depends upon the lysosomal protease. In the presence of a lysosomal instability consequent to defective lysosomal membranes notch cleavage by protease is defective leading on to functional disorders consequent to defective synaptic connectivity. The defective notch signalling pathway can lead to neuronal degeneration. Altered glycoproteins, glycolipids and GAG of the neuronal membrane can also contribute to schizophrenia and epilepsy by

producing disordered synaptic connectivity. The protein processing defect can result in defective glycosylation of endogenous myelin glycoprotein antigens and exogenous viral glycoproteins antigens with consequent defective formation of MHC class-1 glycoprotein antigen complex. The MHC linked peptide transporter, a P-glycoprotein which transports MHC class-1 glycoprotein antigen complex to the antigen presenting cell surface, has an ATP binding site. The peptide transporter is dysfunctional in the presence of magnesium deficiency. This results in defective transport of the MHC class 1 glycoprotein antigen complex to the antigen presenting cell surface for recognition by the CD_4 or CD_8 cell. Defective presentation of the endogenous myelin glycoprotein antigen can explain the immune dysregulation in MS. A CD_8 MHC class-1 restricted immune dysregulatory defect has been described in MS. This can also explain the immune dysregulation in interstitial lung disease, nephrotic syndrome, inflammatory bowel disease sarcoidosis, rheumatoid arthritis and SLE (systemic lupus erythematosis). Defective presentation of exogenous viral antigens can produce immune evasion by the virus as in AIDS dementia and SSPE. Viral persistence has been implicated in the development of tumours (ebstein barr virus and lymphoma), multiple sclerosis (retro virus), degenerations (Parkinson's disease and corona virus) and schizophrenia (borna virus disease). Altered myelin glycoprotein due to defective glycosylation and alteration in GAG of proteoglycans of myelin can affect the structural integrity of myelin leading onto demyelination. A number of fucose and sialic acid containing natural ligands are involved in trafficking of leukocytes and similar breaches in the blood brain barrier and adhesion of the lymphocyte producing leukocyte trafficking and extravasation in to the perivascular space as has been described in MS. Similar changes can explain the immune infiltration in bronchial asthma, sarcoidosis, interstitial lung disease, inflammatory bowel disease and SLE. A number of fucose and sialic acid containing natural ligands

have been implicated in neoplastic transformation and metastasis. Abnormally glycosylated tumour antigens can lead to defective tumour antigen presentation and loss of immunosurveillance by the natural killer cells. Altered cell surface glycoproteins, glycolipids and GAG can lead to defective contact inhibition and oncogenesis. The MHC glycoproteins are involved in formation of synaptic connectivity during neuronal development. Defective formation and presentation of the MHC class-1 neuronal glycoprotein complex can lead on to disordered synaptic connectivity and functional disorders like schizophrenia and epilepsy. Altered glycoproteins can affect the synaptic connectivity in the nerve plexus of the bowel wall contributing to irritable bowel syndrome. Magnesium deficiency can upregulate collagen and elastin synthesis along with glycoconjugates. This can contribute to the pathogenesis of fibrosis in ILD and cirrhosis of the liver. Increased glycoconjugate synthesis can interfere with the structure of the alveolar basement membrane contributing to the increased alveolar leakiness leading on to the formation of the intra-alveolar hyaline membrane in interstitial lung disease. Increased synthesis of sulphated glycosaminoglycans and alteration in the glomerular basement membrane can contribute to the pathogenesis of nephrotic syndrome by interfering with the glomerular filtration barrier. Altered mucoproteins can affect the gastric mucosal barrier leading on to acid peptic disease. Non-mucin glycoproteins are pro-nucleating factors with regard to gallstone formation. Urine glycoproteins on the other hand have an inhibitory effect on renal stone formation. Altered glycoproteins lead to removal of these particular effects either inhibitory or stimulatory contributing to gallstones and renal stone formation. Altered proteoglycans of the articular surface of the joint can lead on to osteoarthritis as well as degenerative spondylosis of the spine. Thus in the hyperdigoxinemic right hemisphere dominant state there is reduced lysosomal stability, defective ubiquitin dependent proteolytic processing of proteins and alteration in

glycoconjugate structure leading on to their defective catabolism and accumulation. There is also a defect in the MHC antigen presenting pathway leading on to immunodysregulation and viral persistence.

The decrease in the level of dolichol in right handed / left hemispheric dominant individuals and in healthy aging, obsessive compulsive disorder, depression, recurrent respiratory infections, osteoporosis, familial hypotension, patients with low body mass index and bulimia nervosa may suggest its decreased availability for N-glycosylation of proteins. Magnesium excess can lead on to increased catabolism of sphinganine leading on to decreased cerebroside and ganglioside synthesis. In magnesium excess the glycolysis, citric acid cycle and oxidative phosphorylation are activated and less of glucose 6-phosphate is channelled for the synthesis of glycosaminoglycans (GAG). The results show a decrease in the concentration of serum total GAG, glycolipids (ganglioside, glycosyl diglyceride, cerebrosides and sulphatides) and carbohydrate components of glycoproteins (hexose, fucose and sialic acid). The individual GAG fractions in the serum-heparan sulphate (HS), chondroitin sulphates (ChS), heparin (H), hyaluronic acid (HA) and dermatan sulphate (DS) are decreased in left hemisphere dominant individuals (pathological / psychological). The activity of GAG degrading enzymes (beta glucuronidase, beta N-acetyl hexosaminidase, hyaluronidase and cathepsin-D) and that of glycohydrolases (beta galactosidase, beta fucosidase and beta glucosidase) showed significant decrease in the serum in hypodigoxinemic left hemisphere dominant states. Intracellular magnesium excess also results in increased ubiquitin dependent proteolytic processing of glycoconjugates as it requires magnesium for its function. The decrease in the activity of glycohydrolases and GAG degrading enzymes could be due to increased lysosomal stability. Defective lysosomal stability and defective degradation of glycoprotein - GAG complexes as in the case of tau protein /

amyloid - HS proteoglycan complexes in Alzheimer's disease can lead on to brain aging. Membrane Na^+-K^+ ATPase stimulation could protect against neuronal aging. A number of fucose and sialic acid containing natural ligands have been implicated in inflammatory responses and neoplastic transformation. The decrease in fucose and sialic acid noted in these cases could inhibit a protective inflammatory response to the virus or bacteria leading on to recurrent respiratory infection. Decrease in fucose and sialic acid could also protect against malignant transformation. The reduction in glycoconjugate could also result in increased osteoporosis as it affects the structure of the bone matrix. Thus in the hypodigoxinemic left hemisphere dominant state there is increased lysosomal stability, increased ubiquitin dependent proteolytic processing of proteins and alteration in glycoconjugate metabolism leading to decrease in the levels of glycolipids, the carbohydrate component of glycoproteins and glycosaminoglycans. There is no viral persistence but a resulting hypoimmune state contributing to recurrent respiratory infections.

Archaeal Digoxin and Alteration in Membrane Structure and Membrane Formation - Relation to Hemispheric Dominance

The alteration in the isoprenoid pathway specifically, cholesterol as well as changes in glycoproteins and GAG can affect cellular membranes. The upregulation of the isoprenoid pathway in right hemispheric dominant individuals can lead to increased cholesterol synthesis and magnesium deficiency can inhibit phospholipid synthesis. Phospholipid degradation is increased owing to increase in intracellular calcium activating phospholipases A_2 and D. The cholesterol: phospholipid ratio of the RBC membrane was increased in right hemispheric dominance individuals. The concentration of total GAG, hexose and fucose of glycoprotein decreased in the RBC membrane and increased in the serum suggesting their reduced incorporation into the membrane and defective

membrane formation. The glycoproteins, GAG and glycolipids of the cellular membrane are formed in the endoplasmic reticulum, which is then budded of as a vesicle, which fuses with the golgi complex. The glycoconjugates are then transported via the golgi channel and the golgi vesicle fuses with the cell membrane. This trafficking depends upon GTPases and lipid kinases, which are crucially dependent on magnesium and are defective in magnesium deficiency. The change in membrane structure produced by alteration in glycoconjugates and the cholesterol: phospholipid ratio can produce changes in the conformation Na^+-K^+ ATPase resulting in further membrane Na^+-K^+ ATPase inhibition. The same changes can affect the structure of the organelle membrane. This results in defective lysosomal stability and leakage of glycohydrolases and GAG degrading enzymes into the serum. Increased release of lysosomal enzymes can contribute to proteolytic destruction in chronic bronchitis and emphysema, osteoarthritis and rheumatoid arthritis. Defective peroxisomal membranes lead to catalase dysfunction which has been documented in these disorders. Alteration in the alveolar basement membrane can contribute to ILD and the glomerular basement membrane and filtration barrier to nephrotic syndrome. Similar changes in the membrane of the cardiac conducting tissue can contribute to lone atrial fibrillation. Changes in the composition of the neuronal membranes can predispose to epilepsy and functional disorders like schizophrenia. Thus in the hyperdigoxinemic right hemisphere dominant state there is defective membrane formation, membrane structure and function.

The downregulation of the isoprenoid pathway in right handed / left hemispheric dominant individuals and in healthy aging, obsessive compulsive disorder, depression, recurrent respiratory infections, osteoporosis, familial hypotension, patients with low body mass index and bulimia nervosa can lead to decreased cholesterol synthesis and magnesium excess can stimulate

phospholipid synthesis. Phospholipid degradation is decreased owing to decrease in intracellular calcium inhibiting phospholipase A_2 and D. The cholesterol: phospholipid ratio of the RBC membrane was decreased in hypodigoxinemia. The concentration of total GAG, hexose and fucose of glycoprotein increased in the RBC membrane and decreased in the serum suggesting their increased incorporation into the membrane and defective membrane formation. The membrane trafficking depends upon GTPases and lipid kinases which are crucially dependent on magnesium and are activated in magnesium excess. The change in membrane structure produced by alteration in glycoconjugates and the cholesterol: phospholipid ratio can produce changes in the conformation of Na^+-K^+ ATPase resulting in further membrane Na^+-K^+ ATPase stimulation. The same changes can affect the structure of the organelle membrane. This results in increased lysosomal stability. Altered peroxisomal membranes could lead to catalase hyperactivity noticed in hypodigoxinemic states. Thus there is increased membrane formation and increased stability of membrane of the cellular organelle in the left hemisphere dominant hypodigoxinemic state.

Archaeal Digoxin and Mitochondrial Function - Relation to Cerebral Dominance

The concentration of ubiquinone decreased significantly in left handed / right hemispheric dominant individuals which may be the result of low tyrosine levels, reported in most of the disorders, consequent to digoxin's effect in preferentially promoting tryptophan transport over tyrosine. The aromatic ring portion of ubiquinone is derived from tyrosine. Ubiquinone, which is an important component of the mitochondrial electron transport chain, is a membrane antioxidant and contributes to free radical scavenging. The increase in intracellular calcium can open the mitochondrial PT pore causing a collapse of the hydrogen gradient across the inner membrane and uncoupling of the

respiratory chain. Intracellular magnesium deficiency can lead to a defect in the function of ATP synthase. All this leads to defects in mitochondrial oxidative phosphorylation, incomplete reduction of oxygen and generation of the superoxide ion which produces lipid peroxidation. Ubiquinone deficiency also leads to reduced free radical scavenging. The increase in intracellular calcium may lead to increased generation of NO by inducing the enzyme nitric oxide synthase which combines with the superoxide radical to form peroxynitrite. Increased calcium also can activate phospholipase A_2 resulting in increased generation of arachidonic acid which can undergo increased lipid peroxidation. Increased generation of free radicals like the superoxide ion, and hydroxyl radical can produce lipid peroxidation and cell membrane demage which can further inactivate Na^+-K^+ ATPase, triggering the cycle of free radical generation once again. Magnesium deficiency can affect glutathione synthetase and glutathione reductase function. The mitochondrial superoxide dismutase leaks out and becomes dysfunctional with calcium related opening of the mitochondrial PT pore and Outer membrane rupture. The peroxisomal membrane is defective owing to the membrane Na^+-K^+ ATPase inhibition related defect in membrane formation and leads to reduced catalase activity. Mitochondrial dysfunction related free radical generation has been implicated in the pathogenesis of the neuronal degeneration, oncogenesis and immune mediated disorders. Increased free radical generation can lead on to immune activation important in immune mediated diseases like interstitial lung disease, bronchial asthma, sarcoidosis, inflammatory bowel disease, systemic lupus erythematosis, rheumatoid arthritis, nephrotic syndrome and multiple sclerosis. Mitochondrial dysfunction can lead on to Reye's syndrome. The increased intracellular calcium and ceramide related opening of the mitochondrial PT pore also leads to volume dysregulation of the mitochondria, causing hyperosmolality of the matrix and expansion of the matrix space. The outer

membrane of the mitochondria ruptures and releases apoptosis inducing factor and cytochrome C into the cytoplasm. This results in activation of caspase-9 and caspase-3. Caspase-9 can produce apoptosis of the cell. Apoptosis has been implicated in neuronal degeneration. Apoptosis can produce defective synaptogenesis and synaptic connectivity contributing to functional disorders like schizophrenia and epilepsy. Apoptosis of the CD_4 cell can contribute to CD_4 depletion in the acquired immunodeficiency syndrome. Oligodendrocyte (the myelin forming cell) apoptosis is crucial to the pathogenesis of MS. Hepatocyte apoptosis can contribute to cell death in cirrhosis of the liver. Caspase-3 activation can cleave P_{21} involved in linking DNA duplication to cell division resulting in a polyploid cell and oncogenesis. We have been able to demonstrate neuronal degeneration and apoptosis in the digoxin injected rat brain. Thus in the hyperdigoxinemic right hemisphere dominant state there is defect in mitochondrial function and increased free radical generation and reduced scavenging. There is also increased apoptosis.

The concentration of ubiquinone increased significantly in right handed / left hemispheric dominant individuals which may be the result of increased tyrosine levels, consequent to digoxin deficiency promoting tyrosine transport over tryptophan. The decrease in intracellular calcium can stabilize the mitochondrial PT pore and improve mitochondrial function. Intracellular magnesium excess can lead to an increase in the activity of ATP synthase. All this leads to improved efficiency in mitochondrial oxidative phosphorylation and reduced free radical generation. Ubiquinone excess also leads to increased free radical scavenging. The decrease in intracellular calcium may lead to decreased generation of NO by inhibiting the enzyme nitric oxide synthase and reduced peroxynitrite formation. Decreased calcium also can inhibit phospholipase. A_2 resulting in decreased generation of arachidonic acid and free radical formation.

Decreased generation of free radicals like the superoxide ion and hydroxyl radical can stabilise the cell membrane and stimulate membrane Na^+-K^+ ATPase. There was decrease in lipid peroxidation as evidenced from the decrease in the concentration of MDA, conjugated dienes, hydroperoxides and NO with increased antioxidant protection as indicated by the increase in ubiquinone and increased reduced glutathione in hypodigoxinemic left hemisphere dominant individuals The activity of enzymes involved in free radical scavenging like superoxide dismutase, catalase, glutathione peroxidase and glutathione reductase is increased suggesting increased free radical scavenging. The peroxisomal membrane is stabilised owing to membrane Na^+-K^+ ATPase stimulation related alteration in membrane formation and this leads to increased catalase activity. Glutathione is synthesized by the enzyme glutathione synthetase which needs magnesium and ATP. The high intracellular magnesium consequent to Na^+-K^+ ATPase stimulation and the resulting increased ATP can result in increased synthesis of glutathione. Glutathione peroxidase, selenium containing enzyme oxidases reduced glutathione (GSH) to oxidised glutathione (GSSG) which is rapidly reduced to GSH by glutathione reductase. There is also a concomitant conversion of H_2O_2 to H_2O. The activity of glutathione reductase needs NADPH for the regeneration of GSH. This NADPH comes mostly from the pentose phosphate pathway. Intracellular magnesium excess due to membrane Na^+-K^+ ATPase stimulation leads to increased formation of glucose 6-phosphate and upregulation of the pentose phosphate pathway with consequent increased generation of NADPH. Thus glutathione system of free radical scavenging is activated in the presence of membrane Na^+-K^+ ATPase stimulation. Superoxide dismutase exists in a mitochondrial and cytoplasmic form. The stabilization of the mitochondrial PT pore consequent to reduced intracellular calcium produces increased efficiency of superoxide dismutase activity. The increase in catalase, superoxide dismutase (SOD), glutathione

peroxidase and glutathione reductase suggests increased free radical protection. This leads to decreased incidence of neuronal degeneration and oncogenesis in the hypodigoxinemic individuals. Free radicals are required for lymphocyte activation and this leads to a hypoimmune response and increased respiratory infection owing to immunodeficiency. The decreased intracellular calcium and ceramide related stabilisation of the mitochondrial PT pore also leads to down regulation of the apoptotic program and reduced apoptosis. The stabilisation of the mitochondria leads to reduced release of apoptosis inducing factor and cytochrome C into the cytoplasm. This results in inactivation of caspase-9 and caspase-3. Inhibition of apoptosis protects against neuronal aging. Caspase-3 inactivation inhibits P_{21} cleavage and protects against oncogenesis. Thus the hypodigoxinemic left hemisphere dominant state has improved efficiency of mitochondrial oxidative phosphorylation, reduced generation of free radicals, increased free radical scavenging and reduced apoptosis.

Archaeal Digoxin and Immunoregulation - Relation to Hemispheric Dominance

In left handed / right hemispheric dominant individuals increased intracellular calcium activates the calcium dependent calcineurin signal transduction pathway which can produce T-cell activation and secretion of interleukin - 3, 4, 5, 6 and TNF alpha. TNF alpha binds to its receptor TNFR1 and activates the transcription factors NFKB and AP-1 leading to the induction of proinflammatory and immunomodulatory genes. This can also explain the immune activation in MS. TNF alpha can also bring about apoptosis of the cell. It binds to its receptor and activates caspase-9, an ICE protease which converts IL-1 beta precursor to IL-1 beta. IL-1 beta produces apoptosis of the neurons (in Alzheimer’s disease and AIDS dementia), the oligodendrocyte - the myelin forming cell in MS and the CD_4 cell in HIV infection. IL-1 beta and TNF alpha

induce HIV protein expression by the transcription related mechanism and contribute to the pathogenesis of AIDS dementia. Similar digoxin mediated immune activation can play a role in migraine, interstitial lung disease, sarcoidosis, bronchial asthma, inflammatory bowel disease, nephrotic syndrome and immune complex diseases like SLE. Membrane Na^+-K^+ ATPase inhibition can produce immune activation and is reported to increase CD_4/CD_8 ratios as exemplified by the action of lithium. The hyperdigoxinemic right hemisphere dominant state results in immune activation.

In the hypodigoxinemic left hemisphere dominant state decreased intracellular calcium inactivates the calcium dependent calcineurin signal transduction pathway involved in T-cell activation and resulting in decreased secretion of interleukin - 3, 4, 5, 6 and TNF alpha. TNF alpha can also bring about apoptosis of the cell and this is inhibited. Low levels of TNF alpha can lead to immunosuppression. This can explain the immunosuppression and increased rate of respiratory infection. In the hypodigoxinemic left hemisphere dominance there is a tendency for immunosuppression.

Archaeal Digoxin and Regulation of Cell Division, Genomic Function, Cell proliferation and Neoplastic Transformation - Relation to Hemispheric Dominance

Intracellular magnesium depletion can produce defective phosphorylation of MAP (microtubule associated proteins). This results in defective microtubule related spindle fibre dysfunction and chromosomal non-disjunction probably contributing to trisomy 21 and polyploidy. Intracellular magnesium depletion can lead on to defect in the proof reading function of DNA polymerase. This leads on to the genesis of trinucleotide repeats in Huntington's disease. In intracellular magnesium deficiency there is also defective protein transcription owing to ribosomal dysfunction. Thus the hyperdigoxinemic state is associated

with genomic instability owing to the intracellular hypomagnesemia it produces. The reverse holds good for the hypodigoxinemic left hemisphere dominant state. Because of increase in intracellular magnesium there is genomic stability.

In the hyperdigoxinemic right hemisphere dominant state increased intracellular calcium activates phospholipase C beta which results in increased production of diacyglycerol (DAG) with resultant activation of protein kinase C. The protein kinase C (PKC) activates the MAP kinase cascade resulting in cellular proliferation. The decreased intracellular magnesium can produce dysfunction of GTPase activity of the alpha-subunit of G-protein. This results in ras oncogene activation, as more of the ras is bound to GTP rather than GDP. Phosphorylation mechanisms are required for the activation of the tumours suppressor gene P_{53}. The activation of P_{53} is impaired owing to intracellular magnesium deficiency producing a phosphorylation defect. Upregulation of the isoprenoid pathway can result in increased production of farnesyl phosphate which can farnesylate the ras oncogene producing its activation. The ubiquitin system of catabolic processing of processing of proteins is important in the DNA repair mechanism. In the presence of intracellular magnesium deficiency ubiquitin protein catabolic processing and DNA repair mechanisms are defective and this could contribute to oncogenesis. In the hyperdigoxinemic right hemisphere dominant state there is oncogene activation and increased cell proliferation.

In the hypodigoxinemic left hemisphere dominant state high intracellular magnesium and low intracellular calcium consequent to Na^+-K^+ ATPase stimulation appears to be crucial to protection against oncogenesis. Decreased intracellular calcium inactivates phospholipase C beta which results in decreased production of diacylglycerol (DAG) with resultant inactivation of protein kjnase C. The protein kinase C (PKC) activation of the MAP kinase cascade is inhibited

resulting in blockade of cellular proliferation. The increased intracellular magnesium can produce increase in the GTPase activity of the alpha-subunit of G-protein. This results in ras oncogene inactivation, as more of the ras is bound to GDP rather than GTP. Phosphorylation mechanisms required for the activation of the tumour suppressor gene P_{53} are increased owing to intracellular magnesium excess producing increased phosphorylation. Downregulation of the isoprenoid pathway can result in decreased production of farnesyl phosphate which is required for ras oncogene activation. Therefore the ras oncogene is inactivated. In the hypodigoxinemic left hemisphere dominant state there is a tendency for oncogene inactivation and inhibition of cellular proliferation.

Archaeal Digoxin and the Metabolic Regulation - Relation to Hemispheric Dominance

In the hyperdigoxinemic right hemisphere dominant state there is inhibition of Na^+-K^+ ATPase which can explain the pathogenesis of syndrome X. Increased TNF alpha as mentioned above consequent to Na^+-K^+ ATPase inhibition related T-cell activation can contribute to insulin resistance in syndrome X at the receptor level. Decrease in intracellular magnesium can block the phosphorylation reactions involved in protein tyrosine kinase receptor activity leading to insulin resistance. Increase in beta cell calcium can contribute to increased insulin release from beta cells and hyperinsulinemia. Increased intracellular calcium can activate the G-protein coupled signal transduction of the contra insulin hormones (growth hormone and glucagon) leading to hyperglycemia. Decreased intracellular magnesium can lead on to a mitochondrial ATP synthase defect. Increased intracellular calcium can open up the mitochondrial PT pore, disrupt the hydrogen gradient across the inner membrane and block mitochondrial oxidative phosphorylation. Also this leads to defective glucose utilisation and hyperglycemia. Increase in intracellular

calcium can activate the G-protein coupled angiotensin receptor producing hypertension and the G-protein coupled thrombin receptor and platelet activating factor producing thrombosis observed in syndrome X. Na^+-K^+ ATPase inhibition related increased smooth muscle calcium and decreased magnesium can contribute to vasospasm and ischaemia observed in stroke, coronary artery disease and mesenteric artery occlusion. Na^+-K^+ ATPase inhibition related altered glycoprotein and GAG can contribute to the microangiopathy and macroangiopathy observed in syndrome X. Metabolic syndrome X could be visualised as being due to hypothalamic archaeal digoxin hypersecretion. In hypomagnesemia there is inhibition of lipoprotein lipase and decrease in catabolism of triglyceride rich lipoprotein resulting in hypertriglyceridemia. Also magnesium deficiency leads to inhibition of lecithin cholesterol acyl transferase (LCAT) producing decreased formation of cholesterol esters in HDL. This leads on to the dyslipidemia of syndrome X with elevated triglyceride and low HDL cholesterol levels. Digoxin induced hyperinsulinemia and hypertriglyceridemia produces the trunkal obesity in syndrome X. In the hyperdigoxinemic right hemisphere dominant state glucose metabolism and utilisation is impaired consequent to insulin resistance as there is also a tendency for vasospasm and thrombosis.

In the hypodigoxinemic left hemisphere dominant state stimulation Na^+-K^+ ATPase can also lead to metabolic abnormalities. Hypermagnesemia consequent to membrane Na^+-K^+ ATPase stimulation can lead on to increased cell membrane transport of glucose. Increase in intracellular magnesium can activate the phosphorylation reactions involved in protein tyrosine kinase receptor activity leading to increased insulin receptor activity. Increase in intracellular magnesium can lead on to stimulation of glycolysis causing increased glucose utilization. Decrease in intracellular calcium can stabilise the

mitochondrial PT pore and stimulate mitochondrial oxidative phosphorylation. Intracellular magnesium excess can also lead to ATPase synthase hyperactivity. This leads to increased glucose utilisation. Decrease in beta cell calcium can contribute to decreased insulin release from beta cells and hypoinsulinemia. Hypermagnesemia has been reported to markedly decreased glucose stimulated insulin secretion by the perfused pancreas. Increased intracellular magnesium can produce hyperactivity of lipoprotein lipase producing increased catabolism of triglycerides rich lipoproteins and hypotriglyceridemia. In hypermagnesemia lecithin cholesterol acyl transferase is activated and there is increased formation of cholesterol esters in HDL. This results in increased HDL cholesterol. Magnesium excess has been reported to decrease LDL cholesterol levels also. Low insulin levels and increased triglyceride catabolism can be correlated with low body mass index. Decrease in intracellular calcium can inactivate the G-protein coupled angiotensin receptor producing hypotension and the G-protein coupled thrombin receptor and platelet activating factor producing decreased thrombosis observed in the hypodigoxinemic state. Increased intracellular magnesium can lead to decreased thrombin and ADP / collagen induced platelet aggregation. Na^+-K^+ ATPase stimulation related decreased smooth muscle calcium and increased magnesium can contribute to vasodilatation and protect from ischaemia due to stroke and coronary artery disease. This can also lead on to a hypotensive state and familial hypotension. Na^+-K^+ ATPase stimulation induced hypermagnesemia related altered glycoprotein and glycosaminoglycan synthesis can contribute to the decreased atherosclerosis. Thus in the hypodigoxinemic left hemisphere dominant state there is increased efficiency of mitochondrial oxidative phosphorylation, increased glucose utilisation with hypercatabolism of triglyceride rich lipoproteins, low body mass index and decreased vascular thrombosis. This leads on to healthy aging. In the left hemisphere dominant hypodigoxinemic

state there is an endogenous morphine excess syndrome. Morphine has been reported to have an effect on glucose metabolism. In mice, subcutaneous administration of morphine has been shown to produce a dose dependent hyperglycemia, while intrathecal administration of a much lower concentration in the lumbar region caused a dose dependent hypoglycemia. These effects are thought to be due to an insulin independent mechanism mediated through spinal opiate and central alpha-adrenergic receptor stimulation. The effect of morphine on pancreatic glucagon release has been hypothesized to result from suppression of somatostatin and concurrent release of the alpha cell from tonic inhibition leading to an increase in glucagon secretion. Glucagon is the most potent mediator of morphine induced hyperglycemia. Morphine can regulate insulin release from the beta cells with both an inhibitory effect and stimulatory effect being reported. Morphine induced hyperglycemia would involve activation of the pituitary adrenal axis, endocrine pancreas and endogenous opioid peptides. Morphine can also act as a vasodilator contributing to hypotension. Morphine also has an immunosuppressive action. This could contribute to increased incidence of respiratory infections in the left hemispheric dominant state.

Archaeal Digoxin and Regulation of the Immune Response to Viral Infection

The same biochemical Na^+-K^+ ATPase related cascade described above could contribute to the acquired immunodeficiency syndrome in the hyperdigoxinemic right hemisphere dominant state. There is increased incidence of neoplasms like non-Hodgkin’s lymphomas and vasculitis in the acquired immunodeficiency syndrome. Neuronal degenerations like AIDS dementia has been related to glutamate excitotoxicity. An AIDS related schizophreniform psychosis has been described. Polyclonal beta-cell proliferation and lymphadenopathy have been described in AIDS. Digoxin induced calcineurin signal transduction mediated

T-cell activation and polyclonal B-cell proliferation can contribute to HIV-l replication. Digoxin induced T-cell activation can lead on to a secretion of TNF-alpha which induces the immunomodulatory transcription factor NFKB. Chief among the inducible cellular proteins that promote the growth of HIV-1 is transcription factor NFKB. HIV-1 has incorporated two such NFKB binding-enhancer elements into its own genome, which allows the triggering of HIV-l transcription in the presence of nuclear NFKB. Digoxin induced protein glycosylation defects can also lead to defective glycosylation of HIV glycoprotein antigens leading on to defective formation of the HIV glycoprotein antigen-MHC complex for presentation to the CD_4 cell. This results in immune evasion by the virus and could also contribute to the persistence of the herpes virus and Epstein Barr virus producing Kaposi's sarcoma and non-Hodgkin's lymphoma respectively. In hyperdigoxinemia the intracellular magnesium excess results in Z to B transition of DNA and defective methylation of DNA bases leading on to retroviral transposon expression. Hypothalamic structural abnormalities have been described in homosexuals predisposed to the development of acquired immunodeficiency syndrome. In the hyperdigoxinemic right hemisphere dominant state there is a tendency for viral persistence consequent to defective processing of viral proteins and defective immune response to the virus.

The Isoprenoid Organism and Evolution

In the hypothalamic archaeal digoxin induced neuronal quantal state individual dielectric molecules like proteins, nucleic acid, mucopolysaccharides and lipids can store information and undergo self-replication on a preexisting template. The cellular organelle can be considered as symbiotic conglomeration of these macromolecules. The cell can be considered as a symbiotic collection of these organelle. Each organelle may evolutionally represent an organism-like

mitochondria and nucleic acids. The brain with its axonal and dendritic connections can be visualised as a colony of such organisms with its interlinking connections. In this theoretical model there is no evolution but only different conglomeration of the initially existing macromolecules - anevolution. The isoprenoid macromolecule could have been the initial self-replicating organism at the beginning of evolution. Information could have been stored in the isoprenoid-repeating units. It would be tempting to speculate on a role for self-replicating macromolecules like proteins, nucleic acid, mucopolysaccharide and isoprenoid in human diseases. Prions are self-replicating proteins and have been implicated in neurodegenerative disorders.

Archaeal Digoxin and Integration of Brain and Cellular Function

Hypothalamic archaeal digoxin can thus integrate multiple brain and cellular functions. It can integrate the function of multiple cellular organelle - golgi body, lysosome, nucleus and genomic function, mitochondria and cell membrane. It can regulate cell death, cell differentiation and cell proliferation. Digoxin can regulate neuronal transmission and conscious perception in the brain by its effect on neutral amino acid and neurotransmitter transport. Digoxin can also play a role in endocrine integration. The hypothalamic hormone secretion is regulated by the biogenic amines noradrenaline, dopamine and serotonin. Digoxin by regulating the release and uptake of these neurotransmitters can control hypothalamic hormone secretion. Digoxin, by its lithium like action in modulating G-protein function and by facilitating calcium induced signal transduction consequent to increased sodium-calcium exchange, can regulate the function of these hormones. Digoxin can act as an immunomodulator owing to its effect on calcineurin signal transduction in the lymphocyte and subsequent immune activation. Digoxin can thus produce neuro-immuno-endocrine integration in the brain.

The Mind-Body Universe

Digoxin by modulating conscious perception contributes to the observer function of human consciousness. Human consciousness depends on the information perceived from the external world by conscious or subliminal perception and is momentary. It is consciousness that converts the quantal world of probabilities in to the classical objective real world of matter by the act of making an observation. Thus human consciousness and the external world have an interrelated existence. Hypothalamic archaeal digoxin can produce integration and coordination of cellular organelle function. It can also function as the principal conductor of the neuro-immuno-endocrine orchestra.

References

[1] Kurup RK, Kurup PA. *Hypothalamic Digoxin, Cerebral Dominance and Brain Function in Health and Diseases*. New York: Nova Medical Books, 2009.

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