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Archaeal Symbiosis and Digoxin Status Modulates Evolution of Homo Sapiens and Homo Neanderthalis - Digoxin Is a Neanderthal Hormone

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Abstract

Introduction: The climate change and global warming/ ice age results in endosymbiotic actinidic archaeal growth in the human system and cholesterol catabolism resulting in endogenous digoxin synthesis. The increased endosymbiotic archaeal growth detected in autism and matrilineal communities with increased incidence of autism and neanderthalic origin leads to the conclusion that digoxin acts as neanderthalic hormone. The increased endosymbiotic archaeal growth and resultant endogenous digoxin synthesis in relation to climate change and global warming results in neanderthalisation of homo sapiens and human disease resulting in homo sapien extinction. Digoxin can inhibit reverse transcriptase activity and RNA editing resulting in suppression of endogenous retroviral growth. This produces inhibition of HERV expression and jumping gene phenomena producing in adynamicity of the human genome. HERV related jumping genes are crucial in synaptic diversity, HLA expression and immunomodulation as well as metabolic diversity. Digoxin produces alteration in sodium-hydrogen exchange producing an acidic pH and acts like a growth factor producing stem cell transformation of adult cells. Stem cells have a distinct metabolism with increased glycolysis and suppression of PDH and mitochondrial function. This can result in cancer and metabolic syndrome. The digoxin interference with RNA editing can lead to mutated RNA viruses and wide spread RNA viral epidemics. The digoxin interference with HERV expression and RNA editing and resultant inhibition of genomic, metabolic, neural and immune diversity produces autoimmune disease, cancer, metabolic syndrome, degenerations, schizophrenia and autism which are increasing at epidemic rates in human population.

Materials and Methods: Endogenous digoxin levels and serum cytochrome F420 levels as a marker of archaeal growth were estimated in matrilineal communities, SLE, multiple sclerosis, parkinson's disease, alzheimer's disease, CNS glioma, multiple myeloma, metabolic syndrome x with CAD and CVA, schizophrenia and autism. 15 numbers were included in each group and each patient had an age and sex matched control. Endogenous digoxin was estimated by Elisa and cytochrome F420 estimated by spectrophotometry. The statistical analysis was done by ANOVA.

Results: Endogenous digoxin levels and cytochrome F420 levels were elevated in matrilineal neanderthalic communities, SLE, multiple sclerosis, parkinson's disease, alzheimer's disease, CNS glioma, multiple myeloma, metabolic syndrome x with CAD and CVA, schizophrenia and autism. Endogenous digoxin and cytochrome F420 levels were low in non-matrilineal homo sapien population.

Conclusion: Homo sapiens tend to have low levels of endosymbiotic actinidic archaea and low digoxin synthesis. Homo sapiens have low incidence of autoimmune disease, cancer, schizophrenia, autism and metabolic syndrome. The neanderthalisation of homo sapiens consequent to endosymbiotic actinidic archaeal growth and digoxin synthesis produces human pathology and extinction. Homo neanderthalis have higher rates of actinidic archaeal symbiosis and digoxin synthesis with higher incidence of autoimmune disease, cancer, schizophrenia, autism and metabolic syndrome. Actinidic archaeal secreted digoxin functions as a Neanderthal hormone.

Key words: Global warming; Actinidic archaea; Digoxin; Hormone; Evolution; Extinction

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INTRODUCTION

The climate change and global warming/ice age results in endosymbiotic actinidic archaeal growth in the human system and cholesterol catabolism resulting in endogenous digoxin synthesis. The increased endosymbiotic archaeal growth detected in autism and matrilineal communities with increased incidence of autism and neanderthalic origin leads to the conclusion that digoxin acts as neanderthalic hormone. The increased endosymbiotic archaeal growth and resultant endogenous digoxin synthesis in relation to climate change and global warming results in neanderthalisation of homo sapiens and human disease resulting in homo sapien extinction. Digoxin can inhibit reverse transcriptase activity and RNA editing resulting in suppression of endogenous retroviral growth. This produces inhibition of HERV expression and jumping gene phenomena producing in adynamicity of the human genome. HERV related jumping genes are crucial in synaptic diversity, HLA expression and immunomodulation as well as metabolic diversity. Digoxin produces alteration in sodium-hydrogen exchange producing an acidic pH and acts like a growth factor producing stem cell transformation of adult cells. Stem cells have a distinct metabolism with increased glycolysis and suppression of PDH and mitochondrial function. This can result in cancer and metabolic syndrome. The digoxin interference with RNA editing can lead to mutated RNA viruses and wide spread RNA viral epidemics. The digoxin interference with HERV expression and RNA editing and resultant inhibition of genomic, metabolic, neural and immune diversity produces autoimmune disease, cancer, metabolic syndrome, degenerations, schizophrenia and autism which are increasing at epidemic rates in human population. Homo sapiens tend to have low levels of endosymbiotic actinidic archaea and low digoxin synthesis. Homo sapiens have low incidence of autoimmune disease, cancer, schizophrenia, autism and metabolic syndrome. The neanderthalisation of homo sapiens consequent to endosymbiotic actinidic archaeal growth and digoxin synthesis produces human pathology and extinction.[1-16]

MATERIALS AND METHODS

Endogenous digoxin levels and serum cytochrome F420 levels as a marker of archaeal growth were estimated in matrilineal communities, SLE, multiple sclerosis, parkinson's disease, alzheimer's disease, CNS glioma, multiple myeloma, metabolic syndrome x with CAD and CVA, schizophrenia and autism. 15 numbers were included in each group and each patient had an age and sex matched control. Endogenous digoxin was estimated by Elisa and cytochrome F420 estimated by spectrophotometry. The statistical analysis was done by ANOVA.

RESULTS

Endogenous digoxin levels and cytochrome F420 levels were elevated in matrilineal neanderthalic communities, SLE, multiple sclerosis, parkinson's disease, alzheimer's disease, CNS glioma, multiple myeloma, metabolic syndrome x with CAD and CVA, schizophrenia and autism. Endogenous digoxin and cytochrome F420 levels were low in non-matrilineal homo sapien population.

Table 1
Digoxin Levels

Group	Digoxin (ng/ml) (increase with cerium)		Digoxin (ng/ml) (decrease with doxy+cipro)	
	Mean	± *SD*	Mean	± *SD*
Homo sapiens	0.11	0.00	0.054	0.003
Schizo	0.55	0.06	0.219	0.043
Autism	0.51	0.05	0.199	0.027
AD	0.55	0.03	0.192	0.040
MS	0.52	0.03	0.214	0.032
Glioma	0.54	0.04	0.210	0.042
DM	0.47	0.04	0.202	0.025
Myeloma	0.56	0.05	0.220	0.052
PD	0.53	0.06	0.212	0.045
Autism	0.53	0.08	0.205	0.041
Neanderthals	0.51	0.05	0.213	0.033
	F value 135.116 *P* value < 0.001		*F* value 71.706 *P* value < 0.001	

Table 2
Cytochrome F420 levels

Group	Cytochrome F420 % (increase with cerium)	
	Mean	± *SD*
Homo sapiens	4.48	0.15
Schizo	23.24	2.01
Autism	23.46	1.87
AD	23.12	2.00
MS	22.12	1.81
Glioma	22.79	2.13
DM	22.59	1.86
SLE	22.29	1.66
PD	22.06	1.61
Autism	21.68	1.90
Neanderthals	22.70	1.87
	F value 306.749 *P* value < 0.001	

DISCUSSION

The increased endosymbiotic archaeal growth detected in autism and matrilineal communities with increased incidence of autism and neanderthalic origin leads to the conclusion that digoxin acts as neanderthalic

hormone. The increased endosymbiotic archaeal growth and resultant endogenous digoxin synthesis in relation to climate change and global warming results in neanderthalisation of homo sapiens and human disease resulting in homo sapiens extinction. Homo sapiens tend to have low levels of endosymbiotic actinidic archaea and low digoxin synthesis. Homo sapiens have low incidence of autoimmune disease, cancer, schizophrenia, autism and metabolic syndrome. The neanderthalisation of homo sapiens consequent to endosymbiotic actinidic archaeal growth and digoxin synthesis produces human pathology and extinction.

The climate change and global warming/ice age results in endosymbiotic actinidic archaeal growth in the human system and cholesterol catabolism resulting in endogenous digoxin synthesis. Cholesterol catabolism can produce endogenous digoxin synthesis. Endogenous digoxin can modulate RNA metabolism. Digoxin can inhibit reverse transcriptase activity and RNA editing resulting in suppression of endogenous retroviral growth. High endogenous digoxin levels can produce retroviral resistance. This produces inhibition of HERV expression and jumping gene phenomena producing in adynamicity of the human genome. HERV can act as jumping genes producing genomic dynamicity. HERV related jumping genes are crucial in synaptic diversity, HLA expression and immunomodulation as well as metabolic diversity. The digoxin interference with HERV expression and RNA editing and resultant inhibition of genomic, metabolic, neural and immune diversity produces autoimmune disease, cancer, metabolic syndrome, degenerations, schizophrenia and autism which are increasing at epidemic rates in human population. The HERV jumping genes produces changes in the genome resulting in synaptic diversity and neural network specialisation. The absence of HERV expression results in prefrontal cortex atrophy and cerebellar dominance. The cerebellum is supposed to have cognitive functions. Cerebellar dysfunction results in the cerebellar cognitive affective syndrome. Cerebellar dominance results in speech dysfunction and development of music and dance as a form of expression. Cerebellum in concerned with intuition and extra sensory perception. Cerebellum also mediates hypnotic trances and spiritual experiences. The cerebellum is concerned with impulsive behavior and the fear, flight, fight responses. Cerebellum is also the site of intuitive creativity. Cerebellum modulates our interaction with the internet. The resulting cerebellar dominance results in schizophrenia, autism, ADHD, addiction, criminality, autistic savant phenomena, introverted behavior and alternate sexuality. It results in an epidemic frontal lobe syndrome and cerebellar cognitive affective syndrome. The inhibition of HERV expression results in decreased diversity of HLA gene expression and autoimmune disease. There is increasing incidence of autoimmune disease in this century.

Digoxin produces alteration in sodium-hydrogen exchange producing an acidic pH and acts like a growth factor producing stem cell transformation of adult cells. Stem cells have a distinct metabolism with increased glycolysis and suppression of PDH and mitochondrial function. The stem cell metabolonomics results in metabolic syndrome x and diabetes mellitus with increased incidence of CVA and CAD. Digoxin converts adult cells to the stem cells. The adult cells envelope is of archaeal origin. This results in regression to endosymbiotic archaeal state. The human body is reduced to archaeal colony network or zombie. The conversion to stem cells results in cellular proliferation and cancer. Cancer and metabolic syndrome x is rising in epidemic proportions in the present century. There is increased incidence of degenerations like alzheimer's disease and parkinson's disease. Increased digoxin can increase cellular calcium producing mitochondrial cell death by activating the caspase cascade. The conversion of adult cells to archaeal stem cells by endogenous digoxin can alter cellular metabolonomics and produce mitochondrial dysfunction resulting in degenerations.

Global warming results in increased carbon dioxide the atmosphere, acidic pH and archaeal growth. Archaea are extremophiles. Neanderthalisation of homo sapiens is a symbiotic transformation due to archaeal growth. The increased endosymbiotic actinidic archaeal growth the human system as well as the conversion of adult cells to stem cells/archaeal forms of cells results in neanderthalisation of homo sapiens. This results in increased incidence of systemic diseases in homo sapiens and their extinction. The digoxin interference with RNA editing can lead to mutated RNA viruses and wide spread RNA viral epidemics. There is increased incidence of RNA viral epidemics in relation to global warming. H1N1 epidemics, the SARS syndrome and increasing dengue epidemics are part of the phenomena. The RNA viral epidemics can result in homo sapiens extinction. The increased actinidic archaeal growth in the ocean beds releases methane which shifts the ocean continental crusts resulting in earthquakes and tsunamis. This can lead to widespread catastrophies and extinction of homo sapiens human population as such. This phenomenon is inevitable as the homo sapiens civilisation expands and technology grows. The increased production of greenhouse gases as a part of civilisational growth leads to global warming, actinidic archaeal growth, neanderthalisation of humans and archaeal related oceanic tsunamis and earthquakes resulting in catastrophic human extinction. This can be described as the Cassandra hypothesis.

Homo sapiens tend to have low levels of endosymbiotic actinidic archaea and low digoxin synthesis. Homo sapiens have low incidence of autoimmune disease, cancer, schizophrenia, autism and metabolic syndrome. The neanderthalisation of homo sapiens consequent to

endosymbiotic actinidic archaeal growth and digoxin synthesis produces human pathology and extinction. Homo neanderthalis have higher rates of actinidic archaeal symbiosis and digoxin synthesis with higher incidence of autoimmune disease, cancer, schizophrenia, autism and metabolic syndrome. Actinidic archaeal secreted digoxin functions as a Neanderthal hormone.

The extinction of homo neanderthalis could be attributed to archaeal synthesised compounds including digoxin. The archaeal digoxin can regulate the nervous system including the NMDA/GABA thalamocorticothalamic pathway mediating conscious perception. NMDA/GABA receptors can be modulated by digoxin induced calcium oscillations resulting NMDA/GAD activity induction. 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. 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. The increased integration of archaea into the neuronal genome can produce increased digoxin mediated NMDA transmission producing schizophrenia and autism. Digoxin induced calcium oscillations can activate NFKB producing immune activation and cytokine secretion. The archaeal digoxin induced chronic immune activation can lead on to autoimmune disease. Archaeal digoxin can induce the host AKT PI3K, AMPK, HIF alpha and NFKB producing the Warburg metabolic phenotype. There is induction of glycolysis, inhibition of PDH activity and mitochondrial dysfunction resulting in inefficient energetics and metabolic syndrome. The archaeal digoxin generated cytokines can lead to TNF alpha induced insulin resistance and metabolic syndrome x. Digoxin induced sodium potassium ATPase inhibition can lead to increase in HMG CoA reductase activity and increased cholesterol synthesis. The increased cholesterol substrate also leads to increased archaeal growth and digoxin synthesis due to metabolic channeling to the mevalonate pathway. Digoxin can produce sodium-potassium ATPase inhibition and inward movement of plasma membrane cholesterol. This produces defective SREBP sensing, increased HMG CoA reductase activity and cholesterol synthesis. The digoxin induced inward movement of plasma membrane cholesterol can alter membrane cholesterol/sphingomyelin ratio producing modified lipid microdomains. The digoxin induced lipid microdomain modulation can regulate the GPCR couple adrenaline, noradrenaline, glucagon and neuropeptide receptors as well as protein tyrosine kinase linked insulin receptor. The digoxin mediated inhibition of nuclear membrane sodium-potassium ATPase can modulate nuclear membrane lipid microdomains and steroidal/thyroxine DNA receptor function. Thus endogenous digoxin can modulate all the endocrine receptors by regulating lipid microdomains. Hyperdigoxinemia is important in the pathogenesis of atherogenesis and metabolic syndrome X. Digoxin induced sodium-potassium ATPase inhibition results in an ATP sparing effect. Eighty percent of the ATP generated is used to run the sodium-potassium ATPase pump. The digoxin inhibition of the sodium-potassium ATPase spares this ATP which is then used for lipid synthesis. Thus endogenous digoxin and the shadow biosphere generated Warburg phenotype can produce increased lipid synthesis and obesity important in metabolic syndrome X. Fat fuels insulin resistance by binding to the toll receptor and producing immune activation and immune infiltration of the adipose tissue. The archaeal digoxin induced monocyte activation and Warburg phenotype induced increased cholesterol synthesis leads to atherogenesis. The Warburg phenotype induced increased mitochondrial PT pore hexokinase can lead on to malignant transformation. The digoxin induced increased intracellular calcium can lead to PT pore dysfunction, cell death and neuronal degeneration. The digoxin mediated transcribed HERV RNA can get encapsulated in microvesicles contributing to the retroviral state. The prion protein conformation is modulated by HERV RNA binding producing prion disease. The archaeal digoxin and rutile induced magnesium depletion can lead MPS deposition and produce EMF, CCP, MNG and mucoid angiopathy. Thus the archaeal digoxin can produce neuro-immune-metabolic-endocrine-genetic integration. The increased archaeal cholesterol catabolism and digoxin secretion can lead to diverse pathological states of neuronal degeneration, metabolic syndrome X, autoimmune disease, malignancy and psychiatric disorders. Thus the archaeal synthesised compounds would have led to neanderthalisation of homo sapiens and their eventual extinction and death of these great civilisations.

REFERENCES

Bastir, M., O'Higgins, P., & Rosas, A. (2007). Facial ontogeny in neanderthals and modern humans. *Proc. Biol. Sci*.; 274:1125–1132.

Bruner, E., Manzi, G., & Arsuaga, J. L. (2003). Encephalization and allometric trajectories in the genus homo: Evidence from the neandertal and modern lineages. *Proc. Natl. Acad. Sci*, *100*, 15335-15340.

Courchesne, E., & Pierce, K. (2005). Brain overgrowth in autism during a critical time in development: Implications for frontal pyramidal neuron and interneuron development and connectivity. *Int. J. Dev. Neuro. Sci*, *23*, 153-170.

Eswaran, V., Harpending. H., & Rogers, A. R. (2005). Genomics refutes an exclusively african origin of humans. *Journal of Human Evolution*, *49*(1), 1-18.

Gooch, S. (2006). *The dream culture of the neanderthals: guardians of the ancient wisdom.* Wildwood House, London: Inner Traditions.

Gooch, S. (2008). *The neanderthal legacy: Reawakening our genetic and cultural origins.* Wildwood House, London: Inner Traditions.

Graves, P. (*1991)*. New models and metaphors for the neanderthal debate. *Current Anthropology, 32*(5), 513-541.

Green, R. E., Krause, J., Briggs, A. W., Maricic, T., Stenzel, U., Kircher, M., Patterson, N., Li, H., …Svante P. (2010). A draft sequence of the neandertal genome. *Science*, *328*, 710-722.

Kurtén, B. (1978). *Den svarta tigern*. Stockholm, Sweden: ALBA Publishing.

Kurup, R. A., & Kurup, P. A. (2012). Endosymbiotic actinidic archaeal mediated warburg phenotype mediates human disease state. *Advances in Natural Science*, *5*(1), 81-84.

Mithen, S. J. (2005). *The singing neanderthals: The origins of music, language, mind and body*. London: Weidenfeld and Nicolson. ISBN 0-297-64317-7.

Morgan, E. (2007). *The Neanderthal theory of autism,* Asperger and ADHD; Restrieved from http://www.rdos.net/eng/asperger.htm.

Neubauer, S., Gunz, P., & Hublin, J. J. (2010). Endocranial shape changes during growth in chimpanzees and humans: A morphometric analysis of unique and shared aspects. *J. Hum. Evol*, *59*, 555-566.

Sawyer, G. J., & Maley, B. (2005). Neanderthal reconstructed. *The Anatomical Record Part B: The New Anatomist*, *283B*(1), 23-31.

Spikins, P. (2009). Autism, the integrations of 'difference' and the origins of modern human behaviour. *Cambridge Archaeological Journa*, *19*(2), 179-201.

Weaver, T. D., & Hublin, J. J. (2009). Neandertal birth canal shape and the evolution of human childbirth. *Proc. Natl. Acad. Sci*, *106*, 8151-8156.

