

Chapter 2

Fossilised Neanderthal Matrilineal Societies - Neoneanderthal Hybrids, Endosymbiotic Actinidic Archaea and Civilizational Diseases

Introduction

The human genome has been found to have up to 10 percent Neanderthal genes. Neanderthal hybrids with homo sapiens species are common in global population. There is a high incidence of autism, schizophrenia and Neanderthal anthropometric phenotypes in the Nair community of Kerala. The Nair community is matrilineal and is one of the few functional matriarchies in the world and speaks the Dravidian language with similarities to Celtic, Scythian, Berber and Basque societies. The autistic brain is comparable to the large sized Neanderthal brain.¹ Autistic and schizophrenic metabolonomic patterns include low efficiency pyruvate dehydrogenase activity, mitochondrial dysfunction, dominant GABA shunt, Warburg glycolytic phenotype, hyperammonemia, hyperhomocysteinemia, porphyria, low cholesterol and bile acid levels.² Similar pattern of autistic metabolonomics is seen in the normal Nair population of Kerala. Neanderthal metabolonomic patterns include a low efficiency PDH activity.³ Autistic, schizophrenic and matrilineal societies like Nair can be considered as fossilised remnants of the Neanderthal population.⁴ Endosymbiotic actinidic archaea using cholesterol as an energy substrate has been described in systemic disease from our laboratory.² The autistic, schizophrenic and Nair population have increased actinide dependent cytochrome F420 activity suggestive of endosymbiotic archaeal growth. Archaeal induced PDH and mitochondrial suppression results in the autistic and schizophrenic metabolonomic cascade. The increased archaeal growth in extremophilic conditions of the Ice age would have contributed to the evolution of Neanderthal population.⁵ There is a rising epidemic of autism and schizophrenia indicating Neanderthalisation of the human species due to global warming, extreme climate change and archaeal growth. Global warming itself

could be construed as due to increased archaeal growth and methanogenesis. It would indicate the emergence of cultural, linguistic, psychological, neurological, metabolic, immune and anthropometric phenotype - homo archaeax neanderthalis. The aim of the study aimed to detect fossilized Neanderthal matrilineal societies and new Neanderthal hybrids in relation to civilizational diseases.

Materials and Methods

Four groups, 25 numbers in each group were chosen for the study - the autistic population diagnosed according to DSM criteria, the normal Nair population, the normal non-Nair population and civilizational disease group including metabolic syndrome X, Alzheimer's disease, cancer, schizophrenia and multiple sclerosis. The matrilineal characteristics and Neanderthal anthropometric characteristics of normal Nair and non-Nair population as well as autistic and schizophrenic population were studied. The blood samples were drawn in the fasting state before treatment was initiated. The estimations done in the blood samples collected include cytochrome F420 activity, cholesterol oxidase activity - cholesterol ring oxidase activity, cholesterol side chain oxidase activity and cholesterol aromatase activity, digoxin, lactate, pyruvate, ammonia, ATP, glutamate, acetyl CoA, acetyl choline, ALA, homocysteine, cholesterol and bile acid levels as well as cyto C and hexokinase levels activity. Archaeal cholesterol catabolism was studied as follows - 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, and (IV) same as II+ciprofloxacin and doxycycline each in a concentration of 1 mg/ml. Cholesterol substrate was prepared as described by Richmond. Aliquots were withdrawn at zero time immediately after mixing and after incubation at 37 °C for 1 hour. The following

estimations were carried out: - Cytochrome F420, polycyclic aromatic hydrocarbon, hydrogen peroxide, pyruvate, ammonia, glutamate, digoxin, butyrate, propionate and bile acids. Cytochrome F420 was estimated fluorimetrically (excitation wavelength 420 nm and emission wavelength 520 nm). Polycyclic aromatic hydrocarbon was estimated by measuring hydrogen peroxide liberated by using glucose reagent. Informed consent of the subjects and the approval of the ethics committee were obtained for the study. The statistical analysis was done by ANOVA.

Table 1. Incidence of autism in Nair, autistic and non-Nair population.

Groups	Autism	Percentage
Nair	68 cases	68
Non-Nair	32 cases	32
Total	100	

Table 2. Incidence of schizophrenia in nair and non nair population.

Groups	Schizophrenia	Percentage
Nair	30 cases	30
Non-Nair	70 cases	70
Total	100	

(Nair population is 7% of Kerala population)

Table 3. Anthropometric features in Nair, autistic and non-Nair population.

Groups	Neanderthal Anthropometric	Total	Percentage
Nair	72 cases	100	72
Non-Nair	21 cases	100	21
Autism	81 cases	100	81

Table 4. Autistic metabolonomics.

		Nair	Non-Nair	Schizo	AD	MS
RBC Digoxin (ng/ml RBC Susp)	Mean	1.41	0.18	1.38	1.10	1.21
	±SD	0.23	0.05	0.26	0.08	0.21
Cytochrome F420	Mean	4.00	0.00	4.00	4.00	4.00
	±SD	0.00	0.00	0.00	0.00	0.00
H ₂ O ₂ (umol/ml RBC)	Mean	278.29	111.63	274.88	277.47	280.89
	±SD	7.74	5.40	8.73	10.90	11.25
NOX (OD diff/hr/mgpro)	Mean	0.04	0.01	0.04	0.04	0.03
	±SD	0.01	0.00	0.01	0.01	0.01
TNF ALP (pg/ml)	Mean	78.63	9.29	78.23	79.65	80.18
	±SD	5.08	0.81	7.13	5.57	5.67
ALA (umol24)	Mean	63.50	3.86	66.16	67.32	64.00
	±SD	6.95	0.26	6.51	5.40	7.33
SE ATP (umol/dl)	Mean	2.24	0.02	1.26	2.06	1.63
	±SD	0.44	0.01	0.19	0.19	0.26
Cyto C (ng/ml)	Mean	12.39	1.21	11.58	11.94	11.81
	±SD	1.23	0.38	0.90	0.86	0.67
Lactate (mg/dl)	Mean	25.99	2.75	22.07	22.04	23.32
	±SD	8.10	0.41	1.06	0.64	1.10
Pyruvate (umol/l)	Mean	100.51	23.79	96.54	97.26	102.48
	±SD	12.32	2.51	9.96	8.26	13.20
RBC Hexokinase (ug glu phos/hr/mgpro)	Mean	5.46	0.68	7.69	8.46	8.56
	±SD	2.83	0.23	3.40	3.63	4.75
ACOA (mg/dl)	Mean	2.51	16.49	2.51	2.19	2.03
	±SD	0.36	0.89	0.57	0.15	0.09
ACH (ug/ml)	Mean	38.57	91.98	48.52	42.84	39.99
	±SD	7.03	2.89	6.28	8.26	12.61
Glutamate (mg/dl)	Mean	3.19	0.16	3.41	3.53	3.58
	±SD	0.32	0.02	0.41	0.39	0.36
Se. Ammonia (ug/dl)	Mean	93.43	23.92	94.72	95.37	93.42
	±SD	4.85	3.38	3.28	4.66	3.69
Bile Acid (mg/ml)	Mean	25.68	140.40	22.45	26.26	24.12
	±SD	7.04	10.32	5.57	7.34	6.43
Cholesterol (mg/dl)	Mean	129.23	237.36	126.31	130.14	126.67
	±SD	10.03	38.07	6.93	6.64	5.70
Homocysteine (mg/dl)	Mean	37.49	9.18	31.50	31.75	38.39
	±SD	9.17	0.80	4.07	4.62	8.75

Table 4. Continue.

		Cancer	DM	Autism	F value	P value
RBC Digoxin (ng/ml RBC Susp)	Mean	1.27	1.35	1.19	60.288	< 0.001
	±SD	0.24	0.26	0.24		
Cytochrome F420	Mean	4.00	4.00	4.00	0.001	< 0.001
	±SD	0.00	0.00	0.00		
H ₂ O ₂ (umol/ml RBC)	Mean	278.19	280.89	274.52	713.569	< 0.001
	±SD	12.80	10.58	9.29		
NOX (OD diff/hr/mgpro)	Mean	0.04	0.04	0.04	44.896	< 0.001
	±SD	0.01	0.01	0.01		
TNF ALP (pg/ml)	Mean	79.18	78.36	76.71	427.654	< 0.001
	±SD	5.88	6.68	5.25		
ALA (umol24)	Mean	67.67	64.72	68.16	295.467	< 0.001
	±SD	5.69	6.81	4.92		
SE ATP (umol/dl)	Mean	1.48	1.97	2.03	67.588	< 0.001
	±SD	0.32	0.11	0.12		
Cyto C (ng/ml)	Mean	13.00	12.95	12.48	445.772	< 0.001
	±SD	0.42	0.56	0.79		
Lactate (mg/dl)	Mean	22.20	25.56	21.95	162.945	< 0.001
	±SD	0.85	7.93	0.65		
Pyruvate (umol/l)	Mean	96.58	96.30	92.71	154.701	< 0.001
	±SD	8.75	10.33	8.43		
RBC Hexokinase (ug glu phos/hr/mgpro)	Mean	7.82	7.05	6.95	18.187	< 0.001
	±SD	3.51	1.86	2.02		
ACOA (mg/dl)	Mean	2.34	2.17	2.42	1871.04	< 0.001
	±SD	0.43	0.40	0.41		
ACH (ug/ml)	Mean	42.51	41.31	50.61	116.901	< 0.001
	±SD	11.58	10.69	6.32		
Glutamate (mg/dl)	Mean	3.28	3.53	3.30	200.702	< 0.001
	±SD	0.39	0.44	0.32		
Se. Ammonia (ug/dl)	Mean	93.20	93.38	94.01	61.645	< 0.001
	±SD	4.46	7.76	5.00		
Bile Acid (mg/ml)	Mean	23.43	22.77	23.16	635.306	< 0.001
	±SD	6.03	4.94	5.78		
Cholesterol (mg/dl)	Mean	130.52	129.23	125.86	312.947	< 0.001
	±SD	8.01	5.97	7.79		
Homocysteine (mg/dl)	Mean	39.64	39.38	41.55	46.516	< 0.001
	±SD	9.21	7.00	7.62		

Table 5. Cholesterol oxidase activity.

		Nair	Non-Nair	Schizo	AD	MS
CYT F420%	Mean	23.46	4.48	23.24	23.12	22.12
(Increase with Cerium)	±SD	1.87	0.15	2.01	2.00	1.81
CYT F420%	Mean	59.27	18.24	58.72	56.90	61.33
(Decrease with Doxy+Cipro)	±SD	8.86	0.66	7.08	6.94	9.82
PAH % change	Mean	22.67	4.45	23.01	23.26	22.83
(Increase with Cerium)	±SD	2.29	0.14	1.69	1.53	1.78
PAH % change	Mean	57.69	18.25	59.49	60.91	59.84
(Decrease with Doxy+Cipro)	±SD	5.29	0.72	4.30	7.59	7.62
Digoxin (ng/ml)	Mean	0.51	0.11	0.55	0.55	0.52
(Increase with Cerium)	±SD	0.05	0.00	0.06	0.03	0.03
Digoxin (ng/ml)	Mean	0.20	0.05	0.22	0.19	0.21
(Decrease with Doxy+Cipro)	±SD	0.03	0.00	0.04	0.04	0.03
Bile Acids % change	Mean	22.61	4.29	23.20	22.12	21.95
(Increase with Cerium)	±SD	2.22	0.18	1.87	2.19	2.11
Bile Acids % change	Mean	66.62	18.15	57.04	62.86	65.46
(Decrease with Doxy+Cipro)	±SD	4.99	0.58	4.27	6.28	5.79
Pyruvate % change	Mean	20.94	4.34	20.99	22.63	21.59
(Increase with Cerium)	±SD	1.54	0.21	1.46	0.88	1.23
Pyruvate % change	Mean	62.76	18.43	61.23	56.40	60.28
(Decrease with Doxy+Cipro)	±SD	8.52	0.82	9.73	8.59	9.22
H ₂ O ₂ %	Mean	23.81	4.43	22.50	22.65	21.14
(Increase with Cerium)	±SD	1.19	0.19	1.66	2.48	1.20
H ₂ O ₂ %	Mean	61.08	18.13	60.21	60.19	60.53
(Decrease with Doxy+Cipro)	±SD	7.38	0.63	7.42	6.98	4.70
Butyrate %	Mean	22.29	4.41	21.88	23.66	22.92
(Increase with Cerium)	±SD	1.33	0.15	1.19	1.67	2.14
Butyrate %	Mean	65.38	18.63	66.28	65.97	67.54
(Decrease with Doxy+Cipro)	±SD	3.62	0.12	3.60	3.36	3.65
Propionate % change	Mean	22.13	4.34	23.02	23.09	21.93
(Increase with Cerium)	±SD	2.14	0.15	1.65	1.81	2.29
Propionate % change	Mean	66.26	18.24	67.61	65.86	63.70
(Decrease with Doxy+Cipro)	±SD	3.93	0.37	2.77	4.27	5.63
ATP synthase %	Mean	4.40	23.67	23.09	23.58	23.52
(Increase with Cerium)	±SD	0.11	1.42	1.90	2.08	1.76
ATP synthase %	Mean	18.78	67.39	66.15	66.21	67.05
(Decrease with Doxy+Cipro)	±SD	0.11	3.13	4.09	3.69	3.00
Hexokinase %	Mean	4.21	23.01	23.33	22.96	22.81
change (Increase with Cerium)	±SD	0.16	2.61	1.79	2.12	1.91
Hexokinase % change	Mean	18.56	65.87	62.50	65.11	63.47
(Decrease with Doxy+Cipro)	±SD	0.76	5.27	5.56	5.91	5.81

Table 5. Continue.

		Cancer	DM	Autism	F value	P value																																																																																																																																																																																																																																		
CYT F420%	Mean	22.79	22.59	21.68	306.749	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	2.13	1.86	1.90			CYT F420%	Mean	55.90	57.05	57.93	130.054	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.29	8.45	9.64	PAH % change	Mean	22.84	23.40	22.61	391.318	< 0.001	(Increase with Cerium)	±SD	1.42	1.55	1.42	PAH % change	Mean	66.07	65.77	64.48	257.996	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.78	5.27	6.90	Digoxin (ng/ml)	Mean	0.54	0.47	0.53	135.116	< 0.001	(Increase with Cerium)	±SD	0.04	0.04	0.08	Digoxin (ng/ml)	Mean	0.21	0.20	0.21	71.706	< 0.001	(Decrease with Doxy+Cipro)	±SD	0.04	0.03	0.04	Bile Acids % change	Mean	22.98	22.87	22.21	290.441	< 0.001	(Increase with Cerium)	±SD	2.19	2.58	2.04	Bile Acids % change	Mean	64.96	64.51	63.84	203.651	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.64	5.93	6.16	Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001	(Increase with Cerium)	±SD	1.61	1.38	1.71	Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44
CYT F420%	Mean	55.90	57.05	57.93	130.054	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	7.29	8.45	9.64			PAH % change	Mean	22.84	23.40	22.61	391.318	< 0.001	(Increase with Cerium)	±SD	1.42	1.55	1.42	PAH % change	Mean	66.07	65.77	64.48	257.996	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.78	5.27	6.90	Digoxin (ng/ml)	Mean	0.54	0.47	0.53	135.116	< 0.001	(Increase with Cerium)	±SD	0.04	0.04	0.08	Digoxin (ng/ml)	Mean	0.21	0.20	0.21	71.706	< 0.001	(Decrease with Doxy+Cipro)	±SD	0.04	0.03	0.04	Bile Acids % change	Mean	22.98	22.87	22.21	290.441	< 0.001	(Increase with Cerium)	±SD	2.19	2.58	2.04	Bile Acids % change	Mean	64.96	64.51	63.84	203.651	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.64	5.93	6.16	Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001	(Increase with Cerium)	±SD	1.61	1.38	1.71	Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08										
PAH % change	Mean	22.84	23.40	22.61	391.318	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	1.42	1.55	1.42			PAH % change	Mean	66.07	65.77	64.48	257.996	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.78	5.27	6.90	Digoxin (ng/ml)	Mean	0.54	0.47	0.53	135.116	< 0.001	(Increase with Cerium)	±SD	0.04	0.04	0.08	Digoxin (ng/ml)	Mean	0.21	0.20	0.21	71.706	< 0.001	(Decrease with Doxy+Cipro)	±SD	0.04	0.03	0.04	Bile Acids % change	Mean	22.98	22.87	22.21	290.441	< 0.001	(Increase with Cerium)	±SD	2.19	2.58	2.04	Bile Acids % change	Mean	64.96	64.51	63.84	203.651	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.64	5.93	6.16	Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001	(Increase with Cerium)	±SD	1.61	1.38	1.71	Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																						
PAH % change	Mean	66.07	65.77	64.48	257.996	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	3.78	5.27	6.90			Digoxin (ng/ml)	Mean	0.54	0.47	0.53	135.116	< 0.001	(Increase with Cerium)	±SD	0.04	0.04	0.08	Digoxin (ng/ml)	Mean	0.21	0.20	0.21	71.706	< 0.001	(Decrease with Doxy+Cipro)	±SD	0.04	0.03	0.04	Bile Acids % change	Mean	22.98	22.87	22.21	290.441	< 0.001	(Increase with Cerium)	±SD	2.19	2.58	2.04	Bile Acids % change	Mean	64.96	64.51	63.84	203.651	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.64	5.93	6.16	Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001	(Increase with Cerium)	±SD	1.61	1.38	1.71	Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																		
Digoxin (ng/ml)	Mean	0.54	0.47	0.53	135.116	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	0.04	0.04	0.08			Digoxin (ng/ml)	Mean	0.21	0.20	0.21	71.706	< 0.001	(Decrease with Doxy+Cipro)	±SD	0.04	0.03	0.04	Bile Acids % change	Mean	22.98	22.87	22.21	290.441	< 0.001	(Increase with Cerium)	±SD	2.19	2.58	2.04	Bile Acids % change	Mean	64.96	64.51	63.84	203.651	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.64	5.93	6.16	Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001	(Increase with Cerium)	±SD	1.61	1.38	1.71	Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																														
Digoxin (ng/ml)	Mean	0.21	0.20	0.21	71.706	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	0.04	0.03	0.04			Bile Acids % change	Mean	22.98	22.87	22.21	290.441	< 0.001	(Increase with Cerium)	±SD	2.19	2.58	2.04	Bile Acids % change	Mean	64.96	64.51	63.84	203.651	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.64	5.93	6.16	Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001	(Increase with Cerium)	±SD	1.61	1.38	1.71	Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																										
Bile Acids % change	Mean	22.98	22.87	22.21	290.441	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	2.19	2.58	2.04			Bile Acids % change	Mean	64.96	64.51	63.84	203.651	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.64	5.93	6.16	Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001	(Increase with Cerium)	±SD	1.61	1.38	1.71	Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																						
Bile Acids % change	Mean	64.96	64.51	63.84	203.651	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	5.64	5.93	6.16			Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001	(Increase with Cerium)	±SD	1.61	1.38	1.71	Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																		
Pyruvate % change	Mean	21.19	20.67	21.91	321.255	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	1.61	1.38	1.71			Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001	(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66	H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																														
Pyruvate % change	Mean	58.57	58.75	58.45	115.242	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	7.47	8.12	6.66			H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001	(Increase with Cerium)	±SD	1.76	1.53	1.49	H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																										
H ₂ O ₂ %	Mean	23.35	23.27	23.52	380.721	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	1.76	1.53	1.49			H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36	Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																						
H ₂ O ₂ %	Mean	59.17	58.91	63.24	171.228	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	3.33	6.09	7.36			Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001	(Increase with Cerium)	±SD	1.90	1.61	2.20	Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																		
Butyrate %	Mean	23.81	24.10	22.76	403.394	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	1.90	1.61	2.20			Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52	Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																														
Butyrate %	Mean	66.95	65.78	67.63	680.284	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	3.67	4.43	3.52			Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001	(Increase with Cerium)	±SD	1.71	2.46	2.20	Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																																										
Propionate % change	Mean	23.12	22.73	22.79	348.867	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	1.71	2.46	2.20			Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02	ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																																																						
Propionate % change	Mean	65.12	65.87	64.26	364.999	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	5.58	4.35	6.02			ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001	(Increase with Cerium)	±SD	1.17	1.73	1.64	ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																																																																		
ATP synthase %	Mean	24.01	23.72	22.60	449.503	< 0.001																																																																																																																																																																																																																																		
(Increase with Cerium)	±SD	1.17	1.73	1.64			ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001	(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21	Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																																																																														
ATP synthase %	Mean	66.66	66.25	66.86	673.081	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	3.84	3.69	4.21			Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001	change (Increase with Cerium)	±SD	2.41	1.88	1.87	Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																																																																																										
Hexokinase %	Mean	22.53	23.23	22.88	292.065	< 0.001																																																																																																																																																																																																																																		
change (Increase with Cerium)	±SD	2.41	1.88	1.87			Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001	(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																																																																																																						
Hexokinase % change	Mean	64.29	65.11	65.45	317.966	< 0.001																																																																																																																																																																																																																																		
(Decrease with Doxy+Cipro)	±SD	5.44	5.14	5.08																																																																																																																																																																																																																																				

Results

The results of the study were as follows. The Nair, schizophrenic and autistic group had: (1) increased cytochrome F420 activity, cholesterol oxidase activity, ring oxidase activity, aromatase activity and digoxin synthesis, (2) had decreased PDH activity as indicated by increased pyruvate and lactate levels with low acetyl CoA levels, (3) had increased glycolysis as indicated by increased hexokinase activity and mitochondrial dysfunction as noted by increased cyto C activity in the serum and low ATP levels, (4) had low cholesterol and bile acid levels and increased homocysteine levels, (5) had increased GABA shunt pathway as indicated by increased pyruvate, glutamate and ammonia levels, and (6) had increased porphyrin synthesis from substrates glycine and succinyl CoA derived from GABA shunt pathway as indicated by increased ALA levels. The Nair, schizophrenic, autistic and civilizational disease group had features of Neanderthal metabolism as indicated by pyruvate dehydrogenase suppression.

There is an increased incidence of autism and schizophrenia in the Nair community of Kerala with 68 percent of the autistic patient population of 1500 attending the Metabolic Centre belonging to this matrilineal community. The incidence of schizophrenia in the Nair community is around 30 percent. The autistic population, schizophrenic and the Nair population have the Neanderthal anthropometric phenotype with slanting forehead, large face, stubby nose, prominent mandibles, low 2D:4D ratio, large coarse trunk, macrocephaly and longer second toe as compared to big toe.

Discussion

Matrilineal Societies and Neanderthal Hybrids

Reports indicate that the autistic brain is larger and similar in size to the Neanderthal brain.⁶⁻⁸ Neanderthal societies were matrilineal and matriarchal with female dominance. Autistic, schizophrenic and Nair matrilinearity had also similarities with Neanderthal clusters. Matrilineal culture and matriarchy are seen in the Nair societies and they speak a Dravidian language. The language and culture of the matrilineal Nair community is similar to the Celtic, Basque, Berber and Scythian societies. Matrilineal Nair society with its high incidence of autism and Neanderthal anthropometric characteristics would represent fossilized remnants of the Neanderthal population along with the Celtic, Jews, Sumerian, Minoan, Harappan, Scythian, Basque, South African bushmen and Berber societies. These societies are predominantly characterized by the use of Dravidian linguistics. The Neanderthal fossilized remnant societies described above probably inhabitant the mythological Lemurian continent the remnants of which have been described under the Indian ocean. The end of the ice age resulted in floods and break up of Lemuria and the population migrated to the Eurasian land mass creating the Harappan civilization, the Sumerian civilization, the Egyptian civilization, Celtic civilization and Minoan civilization which were all co-terminus Dravidian and matrilineal. They can be compared to the mythological Asuras in the Vedas whose society was also matrilineal. There was gender equality and matriarchal dominance. The asuric society of the Vedas was democratic and more equal. They had extrasensory perceptive capabilities and extreme form of spirituality. The asuric society is represented in the Dravidian South India where festivals like Onam in celebration of the Asura king Mahabali are celebrated. It is anthropological evidence of the asuric origin of the Dravidians. The Dravidians were originally supposed to have evolved in the

continent of Lemuria in the Indian ocean. Traces of this massive supercontinent involving land masses of South India, Southern Africa, Australia and Antarctica have been detected in the oceanic bed of the Indian ocean. Certain diseases like endomyocardial fibrosis, chronic calcific pancreatitis, multinodular goiter and mucoid angiopathy are specific for south India, South Africa and Australian aboriginals. All these communities South Indian including Nairs, bushmen and Australian aboriginals speak Dravidian related languages and are matrilineal. These endemic disease have been related to the actinidic monazite and illmenite seen in the ocean shores of South India, South Africa and Australia. This is further medical anthropological evidence of the origin of matrilineal neanderthalic asuric communities from the Lemurian supercontinent. This supercontinent also encompassed parts of Antarctica. The Neanderthal skin colour was more lighter and fairer to increase UV absorption and correct vitamin D deficiency seen in this groups which would have originated in the Antarctic part of the Lemurian supercontinent. Life originated in the Lemurian supercontinent on actinidic substrates forming the original archaeal cell which evolved to multicellular forms. The Neanderthal origin would be related to massive extremophilic archaeal expansion which occurred in the ice age. The Asuras of Vedas and Rig vedic descriptions would fit in with a Southern polar origin of the epic. The principle God of the Rig veda was Varuna which was an oceanic God and Asura. The other Gods of the Rig Veda - Rudra, Vayu and Agni were also Asuras. This can indicate a Southern Lemurian origin for Vedic mythology and its asuric Vedic Gods. The asuric society was democratic, more social, spiritual, eco-conscious, gender equal, matrilineal and socialistic. The ice age ended and the floods that occurred following it as well as the massive Tsunamis in the Indian ocean broke up the Lemurian land mass. This has been described in Vedic literature on the Dravidian King Manu who survived the flood and migrated north to the Eurasian land mass. The asuric Dravidians who

migrated north developed the modern cities of Harappa and Mohenjo-Daro, Sumeria, Minoan civilization of Crete, the Egyptian civilization, the Basque Celt and Berber societies. The mythology of these matrilineal societies has Siva as their God, identified in different names like Minoan Zeus, the Celtic Cerannos and the Irish Dragda. The language of the societies could be related to Dravidian and the structure of the society was matrilineal like the Asuras. The homo sapien groups evolved in Africa in relation to HERV sequences in the human genome. HERV sequences in the genome contributed to fluidity and dynamic nature of the genome leading to the evolution of the prefrontal cortex dominant homo sapien brain. The homo sapiens migrated from Africa northwards in the central Eurasian landmass. They were a primitive nomadic society without an urban culture, mythology, language or arts. The Devas of the Rig Veda would be these homo sapien groups which migrated out of Africa into Europe at a later stage and settled in central Eurasia with their lighter colour as an adaptation for increased UV absorption and vitamin D synthesis in the colder regions. The battles between the Asuras and Devas were attempts by the central Asian homo sapien population to overcome and subdue the Asuras to inhabit the Indus Valley and created the civilization in Harappa and Mohenjo-Daro. The defeated asuric Dravidians of Mohenjo-Daro and Harappa migrated south and settled in their original home land in South India. The matrilineal Dravidian Nair community with increased autistic rates belongs to this group.

Autistic and Neanderthal Metabolonomics

Autistic, schizophrenic and Nair metabolonomic patterns had similarities with Neanderthals population. Neanderthals have a low efficient pyruvate dehydrogenase activity.⁹ The Neanderthals diet was rich in protein and fat and low in carbohydrate. Ketone body was used as the energy fuel and does not need the insulin receptor for metabolism. Therefore insulin resistance developed

as a part of the Neanderthal diet and the Neanderthal phenotype is akin to the metabolic syndrome phenotype. As there was less need to metabolize glucose owing to an intake of high fat, high protein diet the enzyme pyruvate dehydrogenase would have evolved into a low efficiency system. Insulin resistance would have contributed to lipogenesis as a protective adaptation against the cold climate of the Ice age. Insulin resistance and ketogenic diet would have contributed to the longevity of the Neanderthal population. Insulin resistance has been related to autism. Pyruvate dehydrogenase deficiency leads to low acetyl CoA levels. This leads to a down regulated mevalonate pathway and low cholesterol synthesis. Low cholesterol levels are related to autism. Smith Lemli Opitz syndrome is related to autism and schizophrenia. Low cholesterol values would have contributed to vitamin D deficiency in Neanderthals. Vitamin D deficiency and rickets would explain the skeletal abnormalities and macrocephaly of Neanderthals. Vitamin D deficiency would have led to fairer complexion of the Neanderthals in view of increased need of cutaneous UV ray absorption to promote increased vitamin D synthesis to correct its deficiency. Cholesterol catabolizing endosymbiotic actinidic archaea has been described in systemic and neuropsychiatric disease from our laboratory. There is increased actinide dependent cytochrome F420 activity in autistic, schizophrenic and normal Nair population. This indicates increased endosymbiotic archaeal growth which suppresses pyruvate dehydrogenase activity. Autistic, schizophrenic and nair metabolonomic patterns include low efficiency pyruvate dehydrogenase activity contributing to pyruvic acidemia. Pyruvate is not converted to acetyl CoA. Acetyl CoA deficiency results in mitochondrial oxidative phosphorylation defects and mitochondrial dysfunction. Energy is obtained from glycolysis and this leads to the genesis of the Warburg phenotype. The actinide dependent hexokinase activity and actinide dependent ATP synthase activity were high but the blood ATP levels were low. The cyto C

activity in the blood was high indicating mitochondrial dysfunction. The pyruvate is channeled to the GABA shunt pathway to glutamate. Glutamate is acted upon by glutamate dehydrogenase generating ammonia which acts as a neurotransmitter modulating thalamo-cortico-thalamic GABA/NMDA function and consciousness. The GABA shunt pathway also generates succinyl CoA and glycine which are substrates for porphyrin synthesis contributing to porphyrinuria. Since glycine is utilized for porphyrin synthesis it is not available for cystathionine synthesis. This contributes to hyperhomocysteinemia and hypermethionemia modulating genomic methylation patterns. Hyperhomocysteinemia, hyperammonemia and porphyrinuria are characteristic of autism and schizophrenia. The low acetyl CoA leads to low cholesterol synthesis and low bile acid as well as vitamin D synthesis. Vitamin D and bile acids bind to the VDR producing immunosuppression and their deficiency contributes to the autoimmunity of autism and schizophrenia. Vitamin D and bile acid deficiency can modulate neocortical development and contribute to autism and schizophrenia. Low cholesterol levels can contribute to low sex hormone levels and less well defined gender phenotypes in autism and schizophrenia. Pyruvate dehydrogenase forms part of the enzyme system 2-oxoacid dehydrogenases which were all deficient in Neanderthals, schizophrenic and autistic groups. The other enzymes included are branched chain ketoacid dehydrogenase, glycine cleavage enzyme - glycine decarboxylase which are deficient in autism, schizophrenic and Neanderthals. The branched chain ketoacid dehydrogenase deficiency leads to increase in branched chain amino acids leucine, isoleucine and valine. The increase in branched chain amino acids leads to metabolic syndrome X and diabetes mellitus. The increase in branched chain amino acids can also produce immune activation and autoimmune disease. The increase in branched chain amino acids can affect the transport of tryptophan and tyrosine through the neutral amino

acid transporter leading to deficiency of monoamine transmission. The branched chain amino acids can increase NMDA activation producing neuronal excitability contributing to neurodegenerative disorders. The alteration in NMDA and monoamine transmission can lead to neuropsychiatric disease. The branched chain amino acids can increase the muscle bulk and strength contributing to the Neanderthal phenotype. The deficiency of glycine cleavage enzyme - glycine decarboxylase can lead to accumulation of glycine. The branched chain amino acids itself inhibits the glycine cleavage enzyme. The PDH deficiency leads to increased glycolysis contributing to increased phosphoglycerate, phosphoserine and serine synthesis. L serine is converted to D serine by serine racemase. D serine and glycine can increase NMDA transmission contributing to neuropsychiatric diseases like autism and schizophrenia as well as neurodegeneration. Glycine itself is an inhibitory neurotransmitter in the brain. Serine is immune activating contributing to autoimmune disease. Glycine is immunosuppressive. Serine/glycine ratios can modulate immunity and NMDA transmission. Serine can contribute to cell proliferation and cancer. Glycine on the other hand inhibits cell proliferation. Serine by the action serine palmitoyl transferase can generate sphingolipids. Deoxysphingolipids are atherogenic and contribute to the metabolic syndrome X. Thus the 2 oxoacid dehydrogenases - pyruvate dehydrogenase, branched chain keto acid dehydrogenase and glycine decarboxylase dysfunction in Neanderthals and autism can contribute to neuropsychiatric, neurodegenerative, cancer, autoimmune disease and metabolic syndrome. Alterations in serine/glycine ratios and organic acidurias are seen in autism, schizophrenia, autoimmune disease, tumours, metabolic syndrome and degenerations. As said before the hyperammonemia, porphyria and hyperhomocysteinemia seen in autism and schizophrenia are contributed by Neanderthal genes and Neanderthal metabolism.

Autistic Metabolonomics and Systemic Diseases

The autistic and schizophrenic neanderthalic metabolonomic phenotype is also seen in cancer, autoimmune disease, degeneration, metabolic syndrome X which can coexist with schizophrenia. This is due to a vagal neuropathy due to defective acetyl choline synthesis consequent to lack of substrate acetyl CoA. This also leads to sympathetic overactivity. Vagal neuropathy is associated with immune activation and autoimmune disease. Vagal neuropathy can contribute to insulin resistance and increased sympathetic activity to neoplastic transformation. The cholesterol synthetic defect leads to defective synaptogenesis seen in autism and schizophrenia. Cholesterol derived bile acid and vitamin D deficiency can contribute to schizophrenia and autism. Cholesterol is involved in contact inhibition and when the membranes are defective can lead to cell proliferation. Low cholesterol levels lead to low vitamin D and bile acid levels both of which bind to VDR producing immunosuppression. This can contribute to autoimmunity. Vitamin D deficiency can contribute to insulin resistance and metabolic syndrome phenotype in Neanderthals. Bile acids function as hormones regulating lipid and glucose metabolism and its deficiency can also contribute to syndrome X and insulin resistance. The Warburg phenotype can also contribute to civilizational diseases. The increase in mitochondrial PT pore hexokinase can contribute to cell proliferation and cancer. The increase in GAPD (glyceraldehyde 3 phosphate dehydrogenase) can contribute to its ADP ribosylation and nuclear cell death. The increase in glycolysis can contribute to lymphocytes activation and autoimmune diseases. The MHC genes are of Neanderthal origin and autoimmunity is related to Neanderthal MHC alleles. Autoimmunity and antibrain antibodies are characteristic of autism and schizophrenia. The phosphoglycerate, a glycolytic metabolite can be converted to serine a modulator of NMDA receptor and inhibitory neurotransmitter glycine. The increase in fructose 1,6 diphosphate

results in its channeling to the pentose phosphate pathway generating NADPH stimulating NOX and redox stress contributing to disease. NOX is also involved in NMDA activity. Redox stress and increased NMDA activity contributing to thalamocorticothalamic pathway dysfunction is important in schizophrenia. Thus the generation of atavistic archaeal metabolic, immune and neuronal phenotype can contribute to schizophrenia.

Actinidic Archaea and Neanderthal Hybrids

The further global warming related increase in archaeal growth leads to an atavistic archaeal endosymbiotic colony with its own metabolic phenotype.² The archaea are actinide dependent and use cholesterol as an energy substrate. The increased archaeal cholesterol catabolism produces endogenous digoxin synthesis which inhibits membrane sodium potassium ATPase activity leading to increase in intracellular calcium and reduction in intracellular magnesium. Increase in intracellular calcium produces calcified nanoarchaea which can exist for eternity. The nanoarchaea as in the case of *Ignococcus hospitalis* can produce multicellular tissue forms resulting in a atavistic actinidic archaeal colony network within the cell. Reverse transcriptase activity of HERV origin can integrate archaeal genomes into the human genome as has been demonstrated with regard to trypanosomal genomes in Chagas disease. The increased expression of archaeal genes and integrated into human genes as a consequence of oxidative stress produced by global warming and ice age resulting in HDAC inhibition and demethylation. The endogenous archaeal genomes when expressed can lead to archaeal multiplication in the system. The basis of origin of Neanderthal hybrids is expression and multiplication of endogenous archaeal sequences in the genome. The Neanderthals would have evolved due to changes in the non coding area of the primate genome consequent to integration of archaeal genomes into primate genomes in the ice

age. Global warming and cooling has been postulated to lead to increased propagation of extremophilic archaeal colonies. In fact global warming has been related to increased release of methane from multiplying archaeal colonies in the ocean bed. During periods of extreme climate change the extremophilic archaea undergoes expansion not only in the environment but also in the non coding area of the human genome. This by global warming related oxidative stress related HDAC inhibition of reverse transcriptase activation and integrase expression which reintegrates the multiplied archaeal genomes into the human genomes. Homo neanderthalis would have evolved as a consequence of archaeal expansion in the human genome in the ice age and the present increased tendency for expression of Neanderthal autistic hybrid phenotypes would result from the phenomena of archaeal expansion in the human genome produced by global warming. The archaeal expansion would result from civilizational and industrial activity of homo sapien population. This results in increased green house gas emissions and carbon dioxide production leading to environmental and symbiotic archaeal multiplication. Symbiotic archaeal multiplication results in increased archaeal integration into the non coding region of genome and expression of Neanderthal hybrids. The environmental archaeal multiplication results in methanogenesis which accelerates geometrically the global warming enhancing the process already set in motion. The increase in archaeal multiplication and global warming will melt the polar ice caps triggering massive floods and catastrophic extinctions. The multiplication of archaea in the ocean beds can trigger earth quakes in the ocean beds and massive tsunamis and floods land continental break down. The cycle of Yugas described in vedic mythology would be a consequence of climate change related catastrophic extinctions and subsequent regeneration of life. The actinidic archaea also being extremophilic can inhabit the intergalactic spaces contributing to intergalactic magnetic fields whose rotation which leads to

evolution of star systems. Seeding of life on earth would have come out of asteroids transporting the actinidic archaea into the earth. This would have led to subsequent evolution of the multicellular organism, primates and later on Neanderthal groups. The homo neanderthalis have the APOBEC phenotype which makes them resistant to retroviral infections and the HERV load in the Neanderthal genome is less. The increased archaeal growth and cholesterol catabolism in Neanderthals, schizophrenic and autistic phenotypes lead to increased endogenous digoxin synthesis. Digoxin produces sodium potassium ATPase inhibition and magnesium deficiency intracellularly. Magnesium deficiency inhibits reverse transcriptase activity and HERV expression. Therefore retroviral expression, multiplication and integration into the genome is defective in Neanderthals, autism and schizophrenia. This leads to less dynamicity and fluidity of the Neanderthal genome leading to defective synaptic connectivity, large sized brains and smaller prefrontal cortex. The deficient synaptic connectivity occurs due to two factors. The cholesterol synthesis is less and the glial cholesterol secretion acts as a trophic factor for synaptogenesis. The HERV expression leads onto jumping genes which are responsible for the fluidity and dynamicity of the genome required for the development complex large neuronal networks. This leads to the development of large brain size as in autism and Neanderthals. The cerebral cortex and cerebellum are both large. The cerebellum contains 50 percent of the neurons in the brain. Therefore, in the absence of complex neuronal networks in the cerebral cortex especially prefrontal cortex the cerebellum become dominant and function as the master of the brain. The homo sapiens lack the APOBEC phenotype and retroviral resistance. The homo sapiens did not have archaeal overgrowth, cholesterol catabolism and digoxin synthesis. There was no digoxin induced reverse transcriptase inhibition. The HERV expression and its integration into the genome via reverse transcriptase activity led to increase in non coding region of

the genome. Retroviral epidemics in African primates contributed to the evolution of homo sapiens and their brain in Africa. The homo sapiens evolved consequent to expansion of HERV sequences in the genome consequent to persistent retroviral infections in African primates. The increase in HERV sequences in the primate genome led to increased fluidity and dynamic nature of the genome leading to development of a dominant prefrontal cortex and limbic lobe. The synaptic connectivity required for the formation of complex neuronal networks based on a dynamic genome modulated by HERV jumping genes were present in the homo sapien brain. This resulted in a trim and lean but more efficient and logical brain with dominant prefrontal cortex function. The cerebellar function was inhibited with predominant control over motor functions. The increase in electromagnetic wave pollution due to internet addiction and persistent usage leads to prefrontal cortical atrophy. This leads to reversion to cerebellar dominance in the homo sapien brain and wide spread increasing incidence of autism, schizophrenia, obsessive compulsive neurosis, sexual addiction syndrome, attention deficit hyperactivity disorders and dyslexias. The lack of APOBEC phenotype in the homo sapiens and the development of resistant retroviral strains would lead to extinction of the homo sapiens species. In addition the global warming can lead to oxidative stress, HDAC inhibition, demethylation and HERV expression leading to reconstitution of retroviruses in the system contributing to the acquired immunodeficiency syndrome. HERV expression in the human genome non coding area has been related to autism and schizophrenia. The development of resistant retroviral infections and the global warming related archaeal multiplication would lead to extermination of the homo sapiens species with its non coding area of genome contributed by HERV sequences. They will get replaced by Neanderthal hybrids with the non coding region of the genome contributed by integrated archaeal sequence which multiply an increase in length owing to global warming. The multiplying

symbiotic and environmental archaea will further contribute to increase global warming, further increased archaeal multiplication and dominance of Neanderthal hybrids in the world. The archaeal metabolism of cholesterol results in low cholesterol levels contributing to sex hormone deficiency, falling reproductive rates and extinction of Neanderthal hybrids generated.

Actinidic Archaeal Metabolism and Autism

The actinidic archaea have cholesterol ring oxidase activity generating pyruvate, side chain oxidase activity generating butyrate and propionate, aromatase activity generating the PAH ring and beta hydroxy steroid dehydrogenase activity generating the glycosidic digoxin and steroidal bile acids. The endogenous digoxin is archaeal in origin as the glycosidic sugars are not synthesized by the human cell. The glycoside digoxin can regulate neural function, immune function and endocrine function. Endogenous digoxin produces sodium potassium ATPase inhibition resulting in increase in intracellular calcium and reduction in intracellular magnesium. Digoxin can modulate intracellular calcium/magnesium ratios increasing cellular calcium and depleting cellular magnesium. Magnesium deficiency inhibits the glycolytic enzymes, tricarboxylic TCA cycle enzymes and mitochondrial ATP synthase. The increase in intracellular calcium can modulate mitochondrial PT pore and its function. The magnesium deficiency can inhibit DNA and RNA polymerase function as well as reverse transcriptase activity. The HERV genes are not expressed and this affects the jumping genes contributing to the dynamicity and fluidity of the genome. HERV gene expression mediated genomic fluidity is required for the generation of complex neuronal networks and immune genes especially the HLA genes. This leads to defective development of the prefrontal cortex and its connections as well as immune mechanisms contributing to autoimmune diseases. Thus digoxin can inhibit genomic function. The digoxin

induced intracellular magnesium deficiency results in ribosomal disintegration and defective protein synthesis. The PDH blockade results in defective generation of acetyl CoA resulting in reduced synthesis of cholesterol and fatty acids. Fatty acid oxidation and ketogenesis is also inhibited by magnesium deficiency related mitochondrial ATP synthase dysfunction. The actinidic archaeal multicellular network through digoxin secretion effectively blocks and shuts down all aspects of cell metabolism. The cellular energetic depends upon sodium potassium ATPase mediated membrane ATP synthesis. The cell requirement of ATP comes down as the membrane sodium pump is inhibited and all metabolic pathways are blocked. The cell goes into hibernation. The human cell, tissues and organ systems functions as a zombie. The cell is taken over by the atavistic multicellular actinidic archaeal colony. The actinidic archaeal metabolism survives. As fatty acid, glucose and amino acid metabolism is inhibited the glucose, fatty acids and amino acids accumulate in the cell and is used for actinidic archaeal metabolic pathways. This is exemplified by increase in actinide catalysed hexokinase activity, mitochondrial ATP synthase activity, membrane sodium potassium ATPase mediated ATP synthesis and cholesterol oxidase - side chain oxidase, ring oxidase, ring aromatase, beta hydroxy steroid dehydrogenase and cholesterol 7 alpha hydroxylase activity. The archaeal shikimic acid pathway synthesizes tyrosine and tryptophan derived neurotransmitters and neuroalkaloids. The shikimic acid pathway can synthesize dopamine, norepinephrine and serotonin as well as neuroalkaloids - morphine, nicotine and strychnine as has been demonstrated from this laboratory. The atavistic archaeal metabolism using cholesterol as energy substrates and actinides as catalyst takes over the cell. The human cell which goes into hibernation functions as a zombie with the multicellular actinidic archaeal colony taking over the cell and the body. Digoxin can produce cell death by calcium mediated mitochondrial PT pore dysfunction and cell proliferation by

increased intracellular calcium activating ras oncogene. Digoxin by modulating sodium potassium ATPase can regulate cell membrane and nuclear membrane transport. Digoxin can modulate NFkB function by increase in intracellular calcium and produce immune activation. Digoxin by altering intracellular calcium/magnesium ratios can modulate G protein coupled and protein tyrosine kinase related neurotransmitter and endocrine receptors. Hyperdigoxinemia has been related to autism. Butyrate functions as a HDAC inhibitor regulating genomic function and also producing immunosuppression. Butyrate mediated altered genomic function can contribute to autism. Propionate can contribute to organic acidurias. Propionate can produce NMDA activation, increased monoamine transmission produce immunosuppression and modulate synaptic transmission. Pyruvate is also immunosuppressive, regulates insulin secretion and functions as an antioxidant. PAH can modulate AHR receptor function regulating cell proliferation and immunity. PAH and AHR receptor activation can affect brain function leading onto autism and ADHD. Cholesterol oxidase activity can generate H_2O_2 and redox stress modulating cell function. Redox stress is related to autism. The archaea can generate magnetite modulating magnetoperception and extrasensory perception important in autism. Thus the archaeal cholesterol catabolism can regulate genetic, immune, metabolic, endocrine and neural functions producing an atavistic phenotype. This atavistic archaeal colony functions as a new phenotype leading to autism. Climate change leads to global warming and increase in extremophilic archaeal growth. This leads onto autistic and schizophrenic metabolic patterns and increased incidence of civilizational diseases. The human body is taken over by the atavistic archaeal colonial phenotype leading to a zombie syndrome. There is a body change, mind change and cultural change akin to climate change. This leads onto neanderthalisation of the human species.

Autism, Schizophrenia and Neanderthal Hybrid Brains

The increase in archaean growth and autistic metabolic patterns leads to autistic, cultural, neural and linguistic atavistic phenotypes. Low cholesterol values are characteristic of autistic brains. Low cholesterol levels can contribute to defective synaptogenesis as cholesterol is a trophic factor for synaptogenesis. This leads to reactive brain hypertrophy and neocortical dysfunction. The Neanderthals had large stout bodies and motor movements were an important part of their hunter gatherer life style. This also was associated with larger eyes and a highly defined visual system important in their hunter gatherer life style. This would also have been associated with a prominent pineal gland with its retinal connections for regulation of diurnal rhythms and geomagnetic field modulation of body function. The Neanderthal brain was larger in size but the major part of the brain was associated with regulation of motor movements and vision crucial for their hunter gatherer life style. The importance of motor movements and the large body size of the Neanderthals contributed to a prominent motor cortex and parietal lobe. The visual cortex also occupied a major part of the cerebral cortex in view of the importance of vision for hunter gatherer lifestyle. The visual, gustatory, auditory and sensory cortex were dominant leading to a predominance of sensory perception regulating life or a civilization of senses. Sensual satisfaction becomes the dominant theme in life. The bile acids important in forming large social groups were binding to olfactory GPCR receptors producing limbic lobe stimulation was deficient. The limbic lobe areas of hippocampus, and prefrontal cortex were ill developed. The prefrontal cortex concerned with social interaction, executive decisions, judgment and social networking was small. Therefore the Neanderthals never formed large social clusters but only small matriarchal groups. The Neanderthals never formed large national groups as the prefrontal cortex concerned with logical higher level executive interactions was small. The

language area of the brain was not developed and the linguistic substrates of the nation states was also lacking. This results in lack of nation states among Neanderthal population and states of war. The motor cortex, the cerebellar cortex controlling coordination and the visual cortex were dominant. The cerebellar cortex was more dominant as compared to the cerebral cortex. The Neanderthal brain had cerebellar dominance. The bulk of the cerebellar function was cognitory and motor regulation. The cerebellum is concerned with impulsive behavior, disinhibited states, obsessive compulsive states, paranoid states, childish naive behavior, ritualized behavior and stereotyped repetitive behavior. The cerebellum is concerned with hypometric and hypermetric states and produces dysmetria of thought. The cerebellar vermis is concerned with emotional behavior. The posterior cerebellum is predominantly cognitory. The anterior cerebellum is concerned with motor regulation. Right cerebellum is connected to the left cerebral hemisphere and left cerebellum is connected to the right cerebral hemisphere. Through the phenomena of diaschisis cerebral cortical atrophy leads to cerebellar atrophy. Thus if the cerebellum is not developed in the fetus the cerebral cortex does not develop. The dorsolateral prefrontal cortex development depends upon cerebellar development. In the context of defective cerebellar development the prefrontal cortex fails to develop. The cerebellum is in fact more important than the cerebral cortex and contains 50 percent of the neurons of the brain. The cerebral cortical and cerebellar function can be compared as conscious versus unconscious, dream versus wake and logical versus intuitive. It can also be compared as patriarchal cerebral cortex versus matriarchal cerebellar cortex as well as commonsensical cerebral cortex versus magical cerebellar cortex. The cerebral cortex can be considered as the HERV modulated brain and the cerebellar cortex can be considered as archaeal modulated brain. As said before, the atavistic archaeal colony network secretes digoxin and neuronal cell goes into metabolic and

functional hibernation. The atavistic actinidic archaeal colony network functions as an information sensing and processing network which also has a capacity of social intelligence. The archaeal colony network has got magnetite capable of magnetoperception and quantal perception. Actinidic archaeal colony mediated quantal perception becomes the dominant form of perception as the neuronal cells goes into metabolic and functional hibernation induced by digoxin. The conscious perception modulated by the thalamocorticothalamic pathway becomes dysfunctional and is replaced by magnetoperception/quantal perception mediated by digoxin induced pumped phonon system involving in dipolar magnetite and porphyrins. The porphyrin and magnetite induced quantal perception can contribute to wave forms of the atavistic archaeal colony network generating macromolecular quantal states. The porphyrins and magnetite are dipolar molecules and can lead onto macroscopic quantal states. Extrasensory perceptual modes are dominant in autism and schizophrenia. The magnetite and archaeal porphyrins are dipolar and in the presence of digoxin induced sodium potassium ATPase inhibition can create pumped phonon states required for quantal perception. The porphyrins which are synthesized more in autism and schizophrenia contribute to extrasensory perception. Extrasensory quantal perception is dominant in autism and schizophrenia. In the quantal state everything exists as unlimited probabilities and it is the conscious observer that brings one of the probabilities into one graviton criteria and consciousness. The multiple probabilities in the quantal states according to the many world interpretation can exist in multiple universes or multiverses at the same time. Thus the quantal brain modulated by the actinidic archaeal colony is eternal and can exist for ever. This forms the basis of the biocentric theory of the universe producing a unified explanation for all phenomena. The world exists because of consciousness. The universe is basically biological. The actinidic nano archaea are extremophilic and can exists in the intergalactic space contributing to the

spiral intergalactic magnetic fields whose rotation leads to the evolution of star systems and planets. Life itself would have an actinidic origin formed on actinidic substrates by abiogenesis. The quantal brain function and quantal phenomena like quantal crystal diffraction gradient can lead onto the origin of the material world.

The cerebellum is concerned with extrasensory perception and trance-like hypnotic states. The cerebellum is involved in out of the body experience and magical states. Spiritual experiences and magical experiences as well as dream like states are also mediated via the cerebellum. The cerebellum is dominant for intuition. Intuitive phenomenon is the basis of creativity and can be called as sixth sense. The cerebellum is involved in telepathy, telekinesis and poltergeist phenomena. Quantal perception is also dominant in the cerebellum as 50 percent of the neurons in the brain are in the cerebellum and the atavistic actinidic archaeal colony network is basically lodged in cerebellum. Quantal perception can lead to communication with the animals and plants. Magnetoperception and quantal perception would have generated a feeling of oneness of humans, nature and animals contributing to a spiritual experience. Magnetoperception and porphyrins are involved in sensing of geomagnetic fields. This leads onto a feeling of oneness with nature and group. This leads onto group consciousness, group identity and group motherhood characteristic of Neanderthal clusters. There is no individual identity which is replaced with group identity. This would have contributed to a magical civilization of dreams. This would have generated a pagan culture. The prominent pineal gland would have led to dominant geomagnetic and solar perception leading to a greater level of spirituality. Thus the dominant extrasensory quantal perceptive modes in the Neanderthal brain would have led to a world of dreams in quantal foam where the material world merged with the world of quantal waves. This would have led to a sense of oneness with the world or a feeling of God which can be aptly described as the world of Maya. This can lead onto increase sense of spirituality in the

Neanderthal groups. Since the prefrontal and temporal cognitive cortex was small and dysfunctional extrasensory perception dominated. The Neanderthal brain had an atavistic archaean colony network. The archaean magnetite induced magnetoperception and group consciousness. The atavistic archaean colony network has magnetite and actinide mediated magnetoperception in autism. They also had non local communication and telepathic abilities. Quantal perception was more dominant compared to conscious perception. This leads onto dominance of unconscious over conscious function. This contributes to a dreamy shamanic trance-like states leading to spiritual experience. Magnetoperception and quantal perception can contribute to perceiving nature and environmental consciousness. Neocortical function is defective due to defective synaptogenesis. Brain function is more intuitive than logical. There is more of emotional behavior than logical behaviour. There is more of dreamy trance-like spiritual states than wakeful states. The population lives in dreamy, hallucinatory state. Extrasensory perception contributes to spiritual experience in autism and Neanderthals. The conversion of ketone bodies derived from ketogenic diet to the neurotransmitter GABA and hydroxybutyric acid would have contributed to stimulation of inhibitory transmission in the brain and docile, spiritual behavior of Neanderthal societies. Quantal perception and magnetoperception leads to the phenomena of social networking with equality among all people participating in the network and without a leader. Such social networking behaviour has led to rapid social revolutions in recent times as in Egypt and northern Africa. Social networking groups linked by quantal perceptive modes become the basis of society. The family, the caste and religious hierarchies dissolves giving way to more gender equal and social equal networking groups based on quantal perception or magnetoperception.

Neocortical dysfunction contributes to defective vocalization in Neanderthals. They also had a highly placed larynx contributing to disordered symmetry

between swallowing and breathing leading to evolution of linguistics characteristic of Dravidian language lacking quantal vowels. Language development and communication skills decline with more of gestural and extrasensory communication. Vocal language spoken and written becomes less and less widely used. The use of gestural and communicative music and dance becomes dominant in replacement to written and spoken speech. The cerebellum is important with regard to speech. Word selection, grammar, prosody and gestures depend on the cerebellum. Cerebellar dominance leads to defective language usage, autism and dyslexias. Symbolic gestural communicative forms and trances have been described in art forms of Kerala exemplified by Kathakali and Theyyams.¹⁰ Speech defects are hallmark of autism. This leads onto widespread generation of autistic brain phenotypes in the community. The cerebellum though was large was predominantly cognitory. This leads to decreased efficiency of motor function of the cerebellum leading onto a functional cerebellar syndrome. The Neanderthal movements were clumsy owing to cerebellar dysfunction as happens in autism. The cerebellar speech staccato, explosive, incoordinate and slurred. This can lead onto a musical quality for speech. The frontal cortical dysfunction leads to ecolalia and repetitive. This would have lead to the origin of music. The Neanderthal language would have been predominantly musical. The appendicular incoordination leads to appendicular ataxia. This leads onto the creation of vague abstract forms of drawing. This would have been the genesis of the abstract art. The written language of the Neanderthals as in the case of Dravidian Harappans was predominantly as pictorial scripts or hieroglyphics. Abstract art originated in the basque community with leading figures like Picasso and Dali generated from them. The cerebellar appendicular ataxia also leads to ataxic gait leading to generation of dance forms. Symbolic dance forms of theyyam and kathakali in Kerala are representative of this. The frontal

cortical dysfunction also leads to ecopraxia or repetition of motor acts. Repetitive cerebellar and frontal cortical dysfunction related ataxic movements would have been the origin of dance forms. Dominant cerebellar function contributes to the development of religious rituals, music and dance. The archetypes of the unconscious common to all civilizations also have their substratum in the cerebellum. Neanderthal music, art and dance were a form of spiritual worship in communion with nature as a part of environmental consciousness. Repetitive and ritualized motor acts as a part of spiritual worship would have been generated by prefrontal cortex and cerebellar dysfunction. The increased exposure to the low level electromagnetic fields due to increase in internet usage in the current population also leads to atrophy of the prefrontal cortex leading to dominance of parietal, motor and visual cortex. This creates a Neanderthal like brain in people with internet addiction and over usage which is widespread in the modern world. The shrinkage of the prefrontal cortex and its dopaminergic pathways linking to the basal ganglia is the basis of drug, sexual and sugar addiction. Addictive behaviours were common in the Neanderthal population with usage of drugs like ephedra for creating shamanic states. Similar addictive behavior is common in population overexposed to low level electromagnetic fields generated by internet usage and resultant prefrontal cortex shrinkage. The cerebellar dominance leads to increased incidence of schizophrenia, autism, dyslexia, ADHD, obsessive compulsive disorder and sexual addiction syndromes. The cerebellar size is related to estrogen and testosterone levels and cerebellar dysfunction can contribute to sexual deviant obsessive traits. Thus cerebellar dominance leads to dysmetria of motion and dysmetria of thought leading to dominant quantal perceptive mode. Cerebellar dominant individuals are creative, autistic savants and geniuses but are clumsy with routine motor acts due to dysmetria of motion.

Increasing Incidence of Autism, Actinidic Archaea and Global Warming

The rising incidence of autism can be related to global warming related archaeal growth in the brain and low EMF exposure due to increased internet usage. The increase in homo sapien growth and increased industrial pollution and global warming leads to archaeal overgrowth and neanderthalisation of the brain leading to return of the magical world. This also would result from increased electromagnetic pollution and internet usage leading onto prefrontal cortex atrophy and autistic brain dominance. There would be a return to the dreamy world of the Neanderthals. The increase in archaeal growth in the oceans would also increase methanogenesis and global warming as also contribute to quakes in the ocean bed, leading to Tsunamis. The global warming would lead to melting of the ice caps of the earth and flooding leading to eventual extinction of the world population. In addition the low cholesterol levels and low sex hormone levels would lead to an asexual gender equal world with aberrant sexual behavior and decreased reproductive rates contributing to population extinction. This would be the basis of the theories of Kali yuga, and end of the world in mythologies. The quantal magical world of the Neanderthals would persist. Vitamin D deficiency can produce abnormalities in brain synaptogenesis and growth. Macrocephaly and large sized brains are seen in autism and Neanderthals.¹¹ The Neanderthal have been postulated to have the APOBEC3G phenotype producing retroviral resistance as in Dravidian related Australian aboriginals.⁹ The Neanderthal hybrids are resistant to retroviral infections and have less of HERV load in the genome. The homo sapiens lack the apobec phenotype and are more susceptible to retroviral infections producing increased integration of HERV into the genome. HERV integration into the genome produces jumping genes and a dynamic genome. This dynamic genome is important in generation of complex synaptic networks and HLA

phenotypes. This leads to the smaller size brain with increase in prefrontal cortex and autoimmunity in the homo sapiens unlike the Eurasian Neanderthal phenotype. The homo sapien brain with its prefrontal cortex dominance and smaller size is a consequence of HERV expression in contrast to the large sized Neanderthal brain with smaller prefrontal cortex which is induced by endosymbiotic archaeal over growth. The increased cholesterol levels and bile acid levels in homo sapiens resulted in bile acid binding to olfactory GPCR receptors and limbic lobe stimulation. This resulted in prefrontal cortex and temporal cortex hypertrophy. The homo sapien brain was dominated by the large prefrontal cortex which was required for executive, logical, reasoning and questioning ability. This led onto the world of logic and reason. The homo sapien brain was dominated by a web of synaptic connections produced by HERV expression mediated dynamic genome. The prefrontal cortical dominance led to the evolution of large social groups and nation states. The evolution of language areas in the frontoparietal cortex developed into linguistic substrates of nation states. This resulted in lack of global consciousness and genesis of the idea of war between nations and persecution of linguistic groups or nations. This was a logical brain as compared to the intuitive and spiritual brain of the Neanderthals. The loss of extrasensory quantal perceptive modes of the homo sapien brain led to decreased communion with plants, animals and nature leading to decreased environmental consciousness in the Western homo sapien civilization. Homo sapiens alone were considered to have the life force of soul and the plant and animal kingdom was outside the pale of spirituality. The loss of environmental consciousness and spirituality resulted in environmental destruction and global warming. The communion with nature was lost and life became mechanical, logical and commonsensical. The magical dreamy trance-like world of the Neanderthal brain was lost. This arose with the dominance of the Western Christian civilization. The dreamy trance-like world

of the hermetic faiths - Kabbala, Shamanism, Paganism, Hinduism, Taoism, Shitoism and Gnostic Christianity was lost with loss of the Neanderthal structure of the brain. The archaeal overgrowth related changes in the brain and development of Neanderthal hybrids contribute to schizophrenia and autism.

Neanderthal Hybrids and Endocrine Function

Low cholesterol leads to low testosterone and estrogen levels and defective sex hormone modulation of brain function and growth. This would lead to defective stress response and sexual reproductive rates leading to eventual extinction of the Neanderthal population. Low testosterone levels and estrogen levels would lead to less defined asexual phenotypes, lack of male dominance, gender equality and matriarchal societies with group motherhood. This is the basis of the matriarchal cultural phenotype with lack of male dominance. The low sex hormone levels would lead to low maturity rates seen in fossil specimens of characteristic of Neanderthals. Bile acids bind to the olfactory receptors and lead to limbic lobe stimulation and family bonding as well as bonding between individual mother and child. The group motherhood characteristic of matriarchy would be a reflection of low bile acid levels. The low bile acid levels leads to less family bonding. This contributes to autistic behaviour. There is no family bonding which gets replaced with common motherhood. This fits in with the grandmother hypothesis with dominant females regulating the society. The society becomes more gender equal with its astereotyped asexual behavioural patterns common in autism. These phenomena can lead to globalization, loss of national identity, loss of sexual identity and universalisation of behavior and thought.^{12, 13, 14, 15} The homo sapiens had higher cholesterol levels leading to higher levels of sex hormone synthesis - testosterone and estrogen. This lead to the development of a male dominant patriarchal society in homo sapiens. The females were suppressed and were not

allowed any rights and subjected to the rigid social codes enforced by the male dominant patriarchy. The sexual behavior was also more towards conservative forms with aberrations being considered as illegal. The homo sapien society gender unequal society. The increased cholesterol and bile acid levels led to increase in family bonding and family as a basic structure of society. The child was identified with the father and his family. The concept of nuclear family got strengthened in the homo sapien group. The group community feeling and group motherhood of the matriarchal Neanderthal society was lost. Neanderthal societies with its group motherhood, group consciousness, gender equality and togetherness were akin to a primitive form of communist society. This postulate has been put forward by Engels in his thesis 'The Mothers'. The Neanderthal society because of its group consciousness was more of a primitive communist or socialistic society and paganistic. The lack of sex hormone modulation of brain function in Neanderthal hybrids can contribute to schizophrenia and autism.

Neanderthal Hybrids, Actinidic Archaea and Civilizational Disease - Cancer, Metabolic Syndrome X, Autoimmune Disease and Neurodegeneration

The human cell and tissues go into hibernation mediated by the actinidic archaeal colony secreted digoxin. The DNA polymerase, RNA polymerase, ribosomal function, fatty acid oxidation, glycolysis, TCA cycle, mitochondrial oxidative phosphorylation and cholesterol/fatty acid synthesis gets shut down owing to archaeal digoxin induced magnesium deficiency. The human cell and tissues go into hibernation with the energy for survival produced by membrane sodium potassium ATPase mediated ATP synthesis. The actinidic archaea forms a multicellular colony/network which takes over the human cell and tissues which are reduced to a zombie in hibernation. This produces a human zombie syndrome. The glucose, fatty acids and amino acids accumulate in the cell as the

metabolic and catabolic pathways are blocked. The actinidic archaeal metabolic using actinide catalysis takes over. Actinide dependent hexokinase activity and mitochondrial ATP synthase activity as well as cholesterol oxidase activity has been described in systemic disorders. The hyperglycemia generated due to actinidic archaea secreted digoxin induced block in glucose catabolism leads to diabetes mellitus. Endogenous digoxin leads to increase in vascular smooth muscle calcium, vasospasm and vascular thromobosis. The actinidic archaeal atavistic network and colony grows into neoplasms and cancer. The actinidic archaeal colony generated digoxin shuts down the metabolic machinery of the neuronal cell and over a period of time lead to cell death contributing to neurodegenerative disorders like parkinson's disease, alzhemier's disease and motor neuron disease. The actinidic archaeal colony secreted digoxin shuts down the neuronal metabolic machinery and synaptic networks resulting in dominance of quantal and magnetoperception. Quantal and magnetoperception is mediated by digoxin induced dipolar magnetite and archaeal porphyrins pumped phonon system. As the cerebellum contains 50 percent of the neurons in the brain the cerebellar magnetoperception and quantal perception dominates. The cerebellum becomes dominant. Cerebellar dominance can also occur due to electromagnetic pollution and wider internet usage. Low level of EMF is perceived by magnetite in the brain. This leads to prefrontal cortical atrophy and cerebellar dominance. Cerebellar dominance has been related to autism, schizophrenia, OCD, ADHD, sexual deviant traits and naïve childhood type disinhibited, impulsive behavior. The atavistic archaeal colony network takes over the body and tissues. This leads to immune activation, generation of autoantigens as the human body tries to fight the invading archaeal atavistic colony. This leads to autoimmune disease like lupus, multiple sclerosis and rheumatoid arthritis. The archaeal atavistic colony generated digoxin blocks reverse transcriptase activity and retroviral multiplication and integration. This leads to resistance to retroviral infection. The

defective HERV expression leads to defective jumping genes and HLA genes contributing to autoimmune disease. The MHC genes are of Neanderthal origin and autoimmunity is related to Neanderthal MHC alleles. Autoimmunity and antibrain antibodies are characteristic of autism. Autism and schizophrenia is associated with systemic disorders. The autistic metabolonomic phenotype is also seen in cancer, autoimmune disease, degeneration, metabolic syndrome X and schizophrenia. This is due to a vagal neuropathy due to defective acetyl choline synthesis consequent to lack of substrate acetyl CoA. This also leads to sympathetic overactivity. Vagal neuropathy is associated with immune activation and autoimmune disease. Vagal neuropathy can contribute to insulin resistance and increased sympathetic activity to neoplastic transformation. The cholesterol synthetic defect leads to defective synaptogenesis seen in autism and schizophrenia. Cholesterol derived bile acid and vitamin D deficiency can contribute to schizophrenia and autism. Cholesterol is involved in contact inhibition and when the membranes are defective can lead to cell proliferation. Low cholesterol levels lead to low vitamin D and bile acid levels both of which bind to VDR producing immunosuppression. This can contribute to autoimmunity. Vitamin D deficiency can contribute to insulin resistance and metabolic syndrome phenotype in Neanderthals. Bile acids function as hormones regulating lipid and glucose metabolism and its deficiency can also contribute to syndrome X and insulin resistance. Thus the generation of atavistic archaeal colony/network leads to a new metabolic, immune and neuronal phenotype taking over the human body contributing to civilizational diseases like cancer, degenerations, autoimmune disease and metabolic syndrome X which are showing an epidemic increase in incidence like autism. The human body goes to hibernation and death as a zombie taken over by the actinidic archaeal colony network which rules over the human brain, organ systems, tissues and cell. The age of Neanderthals blooms again with its catastrophic consequences.

Conclusion

The results suggest neanderthalisation of the humans due to global warming and archaeal growth. The Neanderthalisation of the human species is the basis of the global autistic, schizophrenic and civilizational disease epidemic - epidemic Neanderthal hybrid zombie syndrome. The matrilineal societies are fossilized Neanderthal remnants and neoneanderthal hybrids contribute to civilizational diseases. There is a mind change, linguistic change, cultural change, social change and spiritual change akin to climate change owing to increased archaeal growth as a consequence of global warming. The Neanderthal species evolved during periods of extreme climate change of the Ice age which led to increased extremophilic endosymbiotic archaeal growth. A similar extreme climate phenomenon of global warming is a feature of our current existence. This leads to increased extremophilic endosymbiotic archaeal growth and neanderthalisation of the population. Low cholesterol levels and low sex hormone levels would lead to asexual phenotypes and eventual population extinction. A new human species homo archaeax neanderthalis with its new anthropometric, metabolic, cultural, linguistic, neural, psychological and genetic atavistic phenotype is evolving.¹⁶ The Neanderthalisation of the human species is the basis of the global autistic, schizophrenic and civilizational disease epidemic - epidemic Neanderthal hybrid zombie syndrome. The matrilineal societies are fossilized Neanderthal remnants and neoneanderthal hybrids contribute to civilizational diseases. The Neanderthal hybrids will eventually replace the homo sapien species.

References

- [1] Weaver TD, Hublin JJ. Neandertal Birth Canal Shape and the Evolution of Human Childbirth. *Proc. Natl. Acad. Sci. USA* 2009; 106: 8151-8156.

- 86 The Out of South India Origin of Life, Homo Neanderthals and Human Species - the Descent of the Woman and Fertile Backwaters of Kerala - Aquatic Apes, Vanara Tribes, Parthenogenesis, Neanderthals, Amazonians and Male Eunuchs - the Last Refuge of Neanderthals in South India
- [2] Kurup RA, Kurup PA. Endosymbiotic Actinidic Archaeal Mediated Warburg Phenotype Mediates Human Disease State. *Advances in Natural Science* 2012; 5(1): 81-84.
- [3] Morgan E. The Neanderthal theory of autism, Asperger and ADHD; 2007, www.rdos.net/eng/asperger.htm.
- [4] Graves P. New Models and Metaphors for the Neanderthal Debate. *Current Anthropology* 1991; 32(5): 513-541.
- [5] Sawyer GJ, Maley B. Neanderthal Reconstructed. *The Anatomical Record Part B: The New Anatomist* 2005; 283B(1): 23-31.
- [6] Bastir M, O'Higgins P, Rosas A. Facial Ontogeny in Neanderthals and Modern Humans. *Proc. Biol. Sci.* 2007; 274: 1125-1132.
- [7] Neubauer S, Gunz P, Hublin JJ. Endocranial Shape Changes during Growth in Chimpanzees and Humans: A Morphometric Analysis of Unique and Shared Aspects. *J. Hum. Evol.* 2010; 59: 555-566.
- [8] Courchesne E, Pierce K. Brain Overgrowth in Autism during a Critical Time in Development: Implications for Frontal Pyramidal Neuron and Interneuron Development and Connectivity. *Int. J. Dev. Neurosci.* 2005; 23: 153-170.
- [9] Green RE, Krause J, Briggs AW, Maricic T, Stenzel U, Kircher M, Patterson N, Li H, Zhai W, *et al.* A Draft Sequence of the Neandertal Genome. *Science* 2010; 328: 710-722.
- [10] Mithen SJ. *The Singing Neanderthals: The Origins of Music, Language, Mind and Body*; 2005, ISBN 0-297-64317-7.
- [11] Bruner E, Manzi G, Arsuaga JL. Encephalization and Allometric Trajectories in the Genus Homo: Evidence from the Neandertal and Modern Lineages. *Proc. Natl. Acad. Sci. USA* 2003; 100: 15335-15340.
- [12] Gooch S. *The Dream Culture of the Neanderthals: Guardians of the Ancient Wisdom*. Inner Traditions, Wildwood House, London; 2006.
- [13] Gooch S. *The Neanderthal Legacy: Reawakening Our Genetic and Cultural Origins*. Inner Traditions, Wildwood House, London; 2008.

- [14] Kurt  B. *Den Svarta Tigern*, ALBA Publishing, Stockholm, Sweden; 1978.
- [15] Spikins P. *Autism, the Integrations of 'Difference' and the Origins of Modern Human Behaviour*. Cambridge Archaeological Journal 2009; 19(2): 179-201.
- [16] Eswaran V, Harpending H, Rogers AR. Genomics Refutes an Exclusively African Origin of Humans. *Journal of Human Evolution* 2005; 49(1): 1-18.

