# Chapter 18

Porphyrions and Human Disease - Dietary Fibre and Pollution Related Antioxidant Deficiency Induced Civilizational Disease - Modulation by Dietary Fibre and Antioxidant Vitamins E and C

## Introduction

Actinidic archaea have been related to the pathogenesis of schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration. An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. Actinidic archaea have a mevalonate pathway and are cholesterol catabolizing. They can use cholesterol as a carbon and energy source. Archaeal cholesterol catabolism can generate porphyrins via the cholesterol ring oxidase generated pyruvate and GABA shunt pathway. Archaea can produce a secondary porphyria by inducing the enzyme heme oxygenase resulting in heme depletion and activation of the enzyme ALA synthase. Porphyrins have been related to schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. Porphyrin metabolic abnormality can be modulated by drugs. In this report correction of the porphyrin metabolic abnormality contributing to disease by high fibre diet and dietary antioxidants vitamin E and C is discussed. High fibre diet generates short chain fatty acid acetate which inhibits ALA synthase. Dietary fibre itself functions as an antioxidant and has anti-archaeal activity. Vitamin E and C functions as modulators of porphyrin metabolism inhibiting ALA synthase. Dietary monosaccharides like glucose and fructose can inhibit ALA synthase. Fructose was used in this study. The above mentioned disorders of metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis are disorders of civilization due to decreased consumption of dietary fibre and environmental pollution induced antioxidant vitamin C and E deficiency. The results are presented in this report.[1-5] A dietary fibre deficiency and environmental pollution induced antioxidant vitamin E and C deficiency civilizational disorder due to

porphyrin metabolic dysfunction with schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration is described. They can function as self replicating supramolecular organisms which can be called as porphyrions.

## Materials and Methods

The following groups were included in the study:- endomyocardial fibrosis, Alzheimer's disease, multiple sclerosis, non-Hodgkin's lymphoma, metabolic syndrome x with cerebrovascular thrombosis and coronary artery disease, schizophrenia, autism, seizure disorder, Creutzfeldt Jakob disease and acquired immunodeficiency syndrome. There were also patients on treatment with high banana fibre diet at doses of 40 g/day, fructose in doses of 50 g/day, high vitamin C 1 g per day and vitamin E 800 mg per day. There were 10 patients in each group and each patient had an age and sex matched healthy control selected randomly from the general population. Plasma from fasting heparinised blood was used and the experimental protocol was as follows (I) Plasma ± phosphate buffered saline, (II) same as I ± cholesterol substrate, (III) same as II ± rutile 0.1 mg/ml, (IV) same as II ± ciprofloxacine and doxycycline each in a concentration of 1 mg/ml. The following estimations were carried out: - Cytochrome F420, free RNA, free DNA, polycyclic aromatic hydrocarbon, hydrogen peroxide, pyruvate, ammonia, glutamate, succinate, glycine, delta aminolevulinic acid and digoxin. The study also involved estimating the following parameters in the patient population- Hexokinase, porphyrins, pyruvate, glutamate, ammonia, succinic acid, serine, glycine, HMG CoA reductase, cytochrome C, blood ATP and heme oxygenase.[6-9] Informed consent of the subjects and the approval of the ethics committee were obtained for the study. The statistical analysis was done by ANOVA.

## Results

Plasma of treatment group showed decreased levels of the above mentioned parameters with after incubation for 1 hour and addition of cholesterol substrate also resulted in decrease in the above parameters. The plasma of patients showed increase in the above mentioned parameters. The addition of antibiotics to the treatment group caused a decrease in all the parameters while addition of rutile increased their levels to some extent. The addition of antibiotics and rutile to the patient's plasma produced the same changes but the extent of change was more in patient's sera as compared to treatment group. The results are expressed in section 1: tables 1-6 as percentage change in the parameters after 1 hour incubation as compared to the values at zero time. There was upregulated archaeal porphyrin synthesis in the patient population which was archaeal in origin as indicated by actinide catalysis of the reactions. The cholesterol oxidase pathway generated pyruvate which entered the GABA shunt pathway. This resulted in synthesis of succinate and glycine which are substrates for ALA synthase.

The study showed the patient's blood had increased heme oxygenase activity, increased serine, glycine, succinic acid and porphyrins. The hexokinase activity was high. The pyruvate, glutamate, ammonia, GABA and succinic acid levels were elevated indicating blockade of PDH activity, and operation of the GABA shunt pathway. The cyto C levels were increased in the serum indicating mitochondrial dysfunction suggested by low blood ATP levels. This was indicative of the Warburg's phenotype. The HMG CoA reductase activity was high indicating cholesterol synthesis. The addition of high fibre diet, high fructose, vitamin E and vitamin C produced inhibition of porphyrin synthesis and reversed the metabolic abnormalities significantly. Acetate derived from dietary fibre, vitamin E, vitamin C and fructose functions as factors inhibiting ALA synthase and modulating porphyrin metabolism.

## Section 1: Experimental Study

***Table 1.*** *Effect of rutile and antibiotics on cytochrome F420 and PAH.*

| Group | CYT F420 % (Increase with Rutile) | | CYT F420 % (Decrease with Doxy ±Cipro) | | PAH % change (Increase with Rutile) | | PAH % change (Decrease with Doxy ±Cipro) | |
|---|---|---|---|---|---|---|---|---|
| | Mean | ± SD | Mean | ± SD | Mean | ± SD | Mean | ± SD |
| Treatment grp. | 4.48 | 0.15 | 18.24 | 0.66 | 4.45 | 0.14 | 18.25 | 0.72 |
| Schizo | 23.24 | 2.01 | 58.72 | 7.08 | 23.01 | 1.69 | 59.49 | 4.30 |
| Seizure | 23.46 | 1.87 | 59.27 | 8.86 | 22.67 | 2.29 | 57.69 | 5.29 |
| AD | 23.12 | 2.00 | 56.90 | 6.94 | 23.26 | 1.53 | 60.91 | 7.59 |
| MS | 22.12 | 1.81 | 61.33 | 9.82 | 22.83 | 1.78 | 59.84 | 7.62 |
| NHL | 22.79 | 2.13 | 55.90 | 7.29 | 22.84 | 1.42 | 66.07 | 3.78 |
| DM | 22.59 | 1.86 | 57.05 | 8.45 | 23.40 | 1.55 | 65.77 | 5.27 |
| AIDS | 22.29 | 1.66 | 59.02 | 7.50 | 23.23 | 1.97 | 65.89 | 5.05 |
| CJD | 22.06 | 1.61 | 57.81 | 6.04 | 23.46 | 1.91 | 61.56 | 4.61 |
| Autism | 21.68 | 1.90 | 57.93 | 9.64 | 22.61 | 1.42 | 64.48 | 6.90 |
| EMF | 22.70 | 1.87 | 60.46 | 8.06 | 23.73 | 1.38 | 65.20 | 6.20 |
| F value | 306.749 | | 130.054 | | 391.318 | | 257.996 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 2.*** *Effect of rutile and antibiotics on free RNA and DNA.*

| Group | DNA % change (Increase with Rutile) | | DNA % change (Decrease with Doxy ±Cipro) | | RNA % change (Increase with Rutile) | | RNA % change (Decrease with Doxy ±Cipro) | |
|---|---|---|---|---|---|---|---|---|
| | Mean | ± SD | Mean | ± SD | Mean | ± SD | Mean | ± SD |
| Treatment grp. | 4.37 | 0.15 | 18.39 | 0.38 | 4.37 | 0.13 | 18.38 | 0.48 |
| Schizo | 23.28 | 1.70 | 61.41 | 3.36 | 23.59 | 1.83 | 65.69 | 3.94 |
| Seizure | 23.40 | 1.51 | 63.68 | 4.66 | 23.08 | 1.87 | 65.09 | 3.48 |
| AD | 23.52 | 1.65 | 64.15 | 4.60 | 23.29 | 1.92 | 65.39 | 3.95 |
| MS | 22.62 | 1.38 | 63.82 | 5.53 | 23.29 | 1.98 | 67.46 | 3.96 |
| NHL | 22.42 | 1.99 | 61.14 | 3.47 | 23.78 | 1.20 | 66.90 | 4.10 |
| DM | 23.01 | 1.67 | 65.35 | 3.56 | 23.33 | 1.86 | 66.46 | 3.65 |
| AIDS | 22.56 | 2.46 | 62.70 | 4.53 | 23.32 | 1.74 | 65.67 | 4.16 |
| CJD | 23.30 | 1.42 | 65.07 | 4.95 | 23.11 | 1.52 | 66.68 | 3.97 |
| Autism | 22.12 | 2.44 | 63.69 | 5.14 | 23.33 | 1.35 | 66.83 | 3.27 |
| EMF | 22.29 | 2.05 | 58.70 | 7.34 | 22.29 | 2.05 | 67.03 | 5.97 |
| F value | 337.577 | | 356.621 | | 427.828 | | 654.453 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 3.*** *Effect of rutile and antibiotics on digoxin and delta aminolevulinic acid.*

| Group | Digoxin (ng/ml) (Increase with Rutile) | | Digoxin (ng/ml) (Decrease with Doxy ±Cipro) | | ALA % (Increase with Rutile) | | ALA % (Decrease with Doxy ±Cipro) | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 0.11 | 0.00 | 0.054 | 0.003 | 4.40 | 0.10 | 18.48 | 0.39 |
| Schizo | 0.55 | 0.06 | 0.219 | 0.043 | 22.52 | 1.90 | 66.39 | 4.20 |
| Seizure | 0.51 | 0.05 | 0.199 | 0.027 | 22.83 | 1.90 | 67.23 | 3.45 |
| AD | 0.55 | 0.03 | 0.192 | 0.040 | 23.67 | 1.68 | 66.50 | 3.58 |
| MS | 0.52 | 0.03 | 0.214 | 0.032 | 22.38 | 1.79 | 67.10 | 3.82 |
| NHL | 0.54 | 0.04 | 0.210 | 0.042 | 23.34 | 1.75 | 66.80 | 3.43 |
| DM | 0.47 | 0.04 | 0.202 | 0.025 | 22.87 | 1.84 | 66.31 | 3.68 |
| AIDS | 0.56 | 0.05 | 0.220 | 0.052 | 23.45 | 1.79 | 66.32 | 3.63 |
| CJD | 0.53 | 0.06 | 0.212 | 0.045 | 23.17 | 1.88 | 68.53 | 2.65 |
| Autism | 0.53 | 0.08 | 0.205 | 0.041 | 23.20 | 1.57 | 66.65 | 4.26 |
| EMF | 0.51 | 0.05 | 0.213 | 0.033 | 22.29 | 2.05 | 61.91 | 7.56 |
| F value | 135.116 | | 71.706 | | 372.716 | | 556.411 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 4.*** *Effect of rutile and antibiotics on succinate and glycine.*

| Group | Succinate % (Increase with Rutile) | | Succinate % (Decrease with Doxy ±Cipro) | | Glycine % change (Increase with Rutile) | | Glycine % change (Decrease with Doxy ±Cipro) | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 4.41 | 0.15 | 18.63 | 0.12 | 4.34 | 0.15 | 18.24 | 0.37 |
| Schizo | 22.76 | 2.20 | 67.63 | 3.52 | 22.79 | 2.20 | 64.26 | 6.02 |
| Seizure | 22.28 | 1.52 | 64.05 | 2.79 | 22.82 | 1.56 | 64.61 | 4.95 |
| AD | 23.81 | 1.90 | 66.95 | 3.67 | 23.12 | 1.71 | 65.12 | 5.58 |
| MS | 24.10 | 1.61 | 65.78 | 4.43 | 22.73 | 2.46 | 65.87 | 4.35 |
| NHL | 23.43 | 1.57 | 66.30 | 3.57 | 22.98 | 1.50 | 65.13 | 4.87 |
| DM | 23.70 | 1.75 | 68.06 | 3.52 | 23.81 | 1.49 | 64.89 | 6.01 |
| AIDS | 23.66 | 1.67 | 65.97 | 3.36 | 23.09 | 1.81 | 65.86 | 4.27 |
| CJD | 22.92 | 2.14 | 67.54 | 3.65 | 21.93 | 2.29 | 63.70 | 5.63 |
| Autism | 21.88 | 1.19 | 66.28 | 3.60 | 23.02 | 1.65 | 67.61 | 2.77 |
| EMF | 22.29 | 1.33 | 65.38 | 3.62 | 22.13 | 2.14 | 66.26 | 3.93 |
| F value | 403.394 | | 680.284 | | 348.867 | | 364.999 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 5.*** *Effect of rutile and antibiotics on pyruvate and glutamate.*

| Group | Pyruvate % change (Increase with Rutile) | | Pyruvate % change (Decrease with Doxy ±Cipro) | | Glutamate (Increase with Rutile) | | Glutamate (Decrease with Doxy ±Cipro) | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 4.34 | 0.21 | 18.43 | 0.82 | 4.21 | 0.16 | 18.56 | 0.76 |
| Schizo | 20.99 | 1.46 | 61.23 | 9.73 | 23.01 | 2.61 | 65.87 | 5.27 |
| Seizure | 20.94 | 1.54 | 62.76 | 8.52 | 23.33 | 1.79 | 62.50 | 5.56 |
| AD | 22.63 | 0.88 | 56.40 | 8.59 | 22.96 | 2.12 | 65.11 | 5.91 |
| MS | 21.59 | 1.23 | 60.28 | 9.22 | 22.81 | 1.91 | 63.47 | 5.81 |
| NHL | 21.19 | 1.61 | 58.57 | 7.47 | 22.53 | 2.41 | 64.29 | 5.44 |
| DM | 20.67 | 1.38 | 58.75 | 8.12 | 23.23 | 1.88 | 65.11 | 5.14 |
| AIDS | 21.21 | 2.36 | 58.73 | 8.10 | 21.11 | 2.25 | 64.20 | 5.38 |
| CJD | 21.07 | 1.79 | 63.90 | 7.13 | 22.47 | 2.17 | 65.97 | 4.62 |
| Autism | 21.91 | 1.71 | 58.45 | 6.66 | 22.88 | 1.87 | 65.45 | 5.08 |
| EMF | 22.29 | 2.05 | 62.37 | 5.05 | 21.66 | 1.94 | 67.03 | 5.97 |
| F value | 321.255 | | 115.242 | | 292.065 | | 317.966 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | e < 0.001 | |

***Table 6.*** *Effect of rutile and antibiotics on hydrogen peroxide and ammonia.*

| Group | $H_2O_2$ % (Increase with Rutile) | | $H_2O_2$ % (Decrease with Doxy ±Cipro) | | Ammonia % (Increase with Rutile) | | Ammonia % (Decrease with Doxy ±Cipro) | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 4.43 | 0.19 | 18.13 | 0.63 | 4.40 | 0.10 | 18.48 | 0.39 |
| Schizo | 22.50 | 1.66 | 60.21 | 7.42 | 22.52 | 1.90 | 66.39 | 4.20 |
| Seizure | 23.81 | 1.19 | 61.08 | 7.38 | 22.83 | 1.90 | 67.23 | 3.45 |
| AD | 22.65 | 2.48 | 60.19 | 6.98 | 23.67 | 1.68 | 66.50 | 3.58 |
| MS | 21.14 | 1.20 | 60.53 | 4.70 | 22.38 | 1.79 | 67.10 | 3.82 |
| NHL | 23.35 | 1.76 | 59.17 | 3.33 | 23.34 | 1.75 | 66.80 | 3.43 |
| DM | 23.27 | 1.53 | 58.91 | 6.09 | 22.87 | 1.84 | 66.31 | 3.68 |
| AIDS | 23.32 | 1.71 | 63.15 | 7.62 | 23.45 | 1.79 | 66.32 | 3.63 |
| CJD | 22.86 | 1.91 | 63.66 | 6.88 | 23.17 | 1.88 | 68.53 | 2.65 |
| Autism | 23.52 | 1.49 | 63.24 | 7.36 | 23.20 | 1.57 | 66.65 | 4.26 |
| EMF | 23.29 | 1.67 | 60.52 | 5.38 | 22.29 | 2.05 | 61.91 | 7.56 |
| F value | 380.721 | | 171.228 | | 372.716 | | 556.411 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

## Section 2: Patient Study

***Table 1.***

| Group | RBC Digoxin (ng/ml RBC Susp) | | Cytochrome F 420 | | HERV RNA (ug/ml) | | $H_2O_2$ (umol/ml RBC) | |
|---|---|---|---|---|---|---|---|---|
| | Mean | ± SD | Mean | ± SD | Mean | ± SD | Mean | ± SD |
| Treatment grp. | 0.18 | 0.05 | 0.00 | 0.00 | 8.70 | 0.90 | 111.63 | 5.40 |
| Schizo | 1.38 | 0.26 | 4.00 | 0.00 | 51.17 | 3.65 | 274.88 | 8.73 |
| Seizure | 1.23 | 0.26 | 4.00 | 0.00 | 50.04 | 3.91 | 278.90 | 11.20 |
| HD | 1.34 | 0.31 | 4.00 | 0.00 | 51.16 | 7.78 | 295.37 | 3.78 |
| AD | 1.10 | 0.08 | 4.00 | 0.00 | 51.56 | 3.69 | 277.47 | 10.90 |
| MS | 1.21 | 0.21 | 4.00 | 0.00 | 47.90 | 6.99 | 280.89 | 11.25 |
| SLE | 1.50 | 0.33 | 4.00 | 0.00 | 48.20 | 5.53 | 278.59 | 11.51 |
| NHL | 1.26 | 0.23 | 4.00 | 0.00 | 51.08 | 5.24 | 283.39 | 10.67 |
| Glio | 1.27 | 0.24 | 4.00 | 0.00 | 51.57 | 2.66 | 278.19 | 12.80 |
| DM | 1.35 | 0.26 | 4.00 | 0.00 | 51.98 | 5.05 | 280.89 | 10.58 |
| CAD | 1.22 | 0.16 | 4.00 | 0.00 | 50.00 | 5.91 | 280.89 | 13.79 |
| CVA | 1.33 | 0.27 | 4.00 | 0.00 | 51.06 | 4.83 | 287.33 | 9.47 |
| AIDS | 1.31 | 0.24 | 4.00 | 0.00 | 50.15 | 6.96 | 278.58 | 12.72 |
| CJD | 1.48 | 0.27 | 4.00 | 0.00 | 49.85 | 6.40 | 286.16 | 10.90 |
| Autism | 1.19 | 0.24 | 4.00 | 0.00 | 52.87 | 7.04 | 274.52 | 9.29 |
| DS | 1.34 | 0.25 | 4.00 | 0.00 | 47.28 | 3.55 | 283.04 | 9.17 |
| Cerebral Palsy | 1.44 | 0.19 | 4.00 | 0.00 | 53.49 | 4.15 | 273.70 | 12.37 |
| CRF | 1.26 | 0.26 | 4.00 | 0.00 | 49.39 | 5.51 | 285.51 | 8.79 |
| Cirr/Hep Fail | 1.50 | 0.20 | 4.00 | 0.00 | 46.82 | 4.73 | 275.97 | 10.66 |
| Muc Angio | 1.40 | 0.32 | 4.00 | 0.00 | 46.37 | 4.87 | 290.37 | 9.10 |
| EMF | 1.51 | 0.29 | 4.00 | 0.00 | 47.47 | 4.34 | 287.49 | 9.81 |
| CCP | 1.35 | 0.22 | 4.00 | 0.00 | 48.54 | 5.97 | 277.50 | 7.51 |
| Exposure to EMF | 1.41 | 0.30 | 4.00 | 0.00 | 51.01 | 4.77 | 276.49 | 10.92 |
| F value | 60.288 | | 0.001 | | 194.418 | | 713.569 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 2.***

| Group | NOX (OD diff/hr/mgpro) | | TNF ALP (pg/ml) | | ALA (umol24) | | PBG (umol24) | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 0.007 | 0.001 | 9.29 | 0.81 | 3.86 | 0.26 | 12.11 | 1.34 |
| Schizo | 0.036 | 0.009 | 78.23 | 7.13 | 66.16 | 6.51 | 42.50 | 3.23 |
| Seizure | 0.038 | 0.007 | 79.28 | 4.55 | 68.28 | 6.02 | 46.54 | 4.55 |
| HD | 0.035 | 0.011 | 82.13 | 3.97 | 67.30 | 5.98 | 47.25 | 4.19 |
| AD | 0.036 | 0.007 | 79.65 | 5.57 | 67.32 | 5.40 | 49.83 | 3.45 |
| MS | 0.034 | 0.009 | 80.18 | 5.67 | 64.00 | 7.33 | 46.85 | 3.49 |
| SLE | 0.038 | 0.008 | 81.03 | 6.22 | 65.01 | 5.42 | 48.55 | 3.81 |
| NHL | 0.041 | 0.006 | 77.98 | 5.68 | 63.21 | 6.55 | 47.17 | 4.86 |
| Glio | 0.038 | 0.007 | 79.18 | 5.88 | 67.67 | 5.69 | 46.84 | 4.43 |
| DM | 0.041 | 0.005 | 78.36 | 6.68 | 64.72 | 6.81 | 48.15 | 3.36 |
| CAD | 0.038 | 0.009 | 78.15 | 3.72 | 66.66 | 7.77 | 47.00 | 3.81 |
| CVA | 0.037 | 0.007 | 77.59 | 5.24 | 69.02 | 4.86 | 46.33 | 4.01 |
| AIDS | 0.039 | 0.010 | 79.17 | 5.88 | 67.78 | 4.41 | 48.03 | 3.64 |
| CJD | 0.039 | 0.006 | 80.41 | 5.70 | 66.99 | 3.71 | 47.94 | 5.33 |
| Autism | 0.036 | 0.006 | 76.71 | 5.25 | 68.16 | 4.92 | 42.04 | 2.38 |
| DS-50 | 0.035 | 0.009 | 80.30 | 6.65 | 64.99 | 6.72 | 45.69 | 4.18 |
| Cerebral Palsy | 0.038 | 0.008 | 80.02 | 6.82 | 65.56 | 6.28 | 44.58 | 4.52 |
| CRF | 0.039 | 0.008 | 81.36 | 5.37 | 67.61 | 5.55 | 46.81 | 4.62 |
| Cirr/Hep Fail | 0.037 | 0.010 | 77.61 | 4.42 | 66.28 | 6.55 | 48.23 | 2.36 |
| Muc Angio | 0.039 | 0.010 | 79.38 | 5.14 | 67.86 | 5.65 | 44.08 | 2.81 |
| EMF | 0.035 | 0.008 | 80.04 | 4.69 | 64.76 | 5.23 | 44.82 | 3.46 |
| CCP | 0.040 | 0.006 | 80.34 | 4.73 | 66.68 | 4.14 | 48.70 | 3.35 |
| Exposure to EMF | 0.038 | 0.007 | 76.41 | 5.96 | 68.41 | 5.53 | 47.27 | 3.42 |
| F value | 44.896 | | 427.654 | | 295.467 | | 183.296 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 3.***

| Group | Uroporphyrin (nmol24) | | Coproporphyrin (nmol/24) | | Protoporphyrin (Ab unit) | | Heme (uM) | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 9.51 | 1.19 | 64.33 | 13.09 | 2.64 | 0.42 | 50.55 | 1.07 |
| Schizo | 267.81 | 64.05 | 401.49 | 50.73 | 44.30 | 2.66 | 12.82 | 2.40 |
| Seizure | 290.44 | 57.65 | 436.71 | 52.95 | 49.59 | 1.70 | 13.03 | 0.70 |
| HD | 286.84 | 24.18 | 432.22 | 50.11 | 49.36 | 4.18 | 11.81 | 0.80 |
| AD | 259.61 | 33.18 | 433.17 | 45.61 | 49.68 | 3.30 | 12.09 | 1.12 |
| MS | 277.36 | 15.48 | 440.35 | 25.34 | 50.81 | 3.21 | 11.87 | 1.84 |
| SLE | 294.51 | 58.62 | 447.39 | 39.84 | 52.94 | 3.67 | 12.95 | 1.53 |
| NHL | 310.25 | 40.44 | 495.98 | 39.11 | 54.80 | 4.04 | 11.76 | 1.37 |
| Glio | 304.19 | 14.16 | 479.35 | 58.86 | 53.73 | 5.34 | 13.68 | 1.67 |
| DM | 285.46 | 29.46 | 422.27 | 33.86 | 49.80 | 4.01 | 12.83 | 2.07 |
| CAD | 314.01 | 17.82 | 426.14 | 24.28 | 49.51 | 2.27 | 11.39 | 1.10 |
| CVA | 320.85 | 24.73 | 402.16 | 33.80 | 46.74 | 4.28 | 11.26 | 0.95 |
| AIDS | 306.61 | 22.47 | 429.72 | 24.97 | 49.32 | 5.13 | 11.60 | 1.23 |
| CJD | 317.92 | 29.63 | 429.24 | 18.29 | 50.02 | 4.58 | 11.76 | 1.32 |
| Autism | 318.84 | 82.90 | 423.29 | 47.57 | 47.50 | 2.87 | 12.37 | 2.09 |
| DS-50 | 258.33 | 37.85 | 421.52 | 36.57 | 50.97 | 7.07 | 11.81 | 1.14 |
| Cerbral Palsy | 280.16 | 26.14 | 431.39 | 28.88 | 49.23 | 3.91 | 11.61 | 1.36 |
| CRF | 301.78 | 48.22 | 427.57 | 33.55 | 49.66 | 4.41 | 12.03 | 1.40 |
| Cirr/Hep Fail | 276.51 | 16.66 | 436.44 | 25.65 | 50.56 | 1.63 | 11.92 | 1.33 |
| Muc Angio | 303.86 | 13.91 | 441.58 | 25.51 | 47.86 | 3.34 | 12.13 | 1.10 |
| EMF | 300.90 | 31.96 | 443.22 | 38.14 | 51.37 | 4.86 | 12.61 | 2.00 |
| CCP | 287.09 | 15.63 | 442.85 | 49.61 | 50.36 | 3.49 | 12.01 | 1.53 |
| Exposure to EMF | 288.21 | 26.17 | 444.94 | 38.89 | 50.59 | 1.71 | 12.36 | 1.26 |
| F value | 160.533 | | 279.759 | | 424.198 | | 1472.05 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 4.***

| Group | Bilirubin (mg/dl) | | Biliverdin (Ab unit) | | ATP Synthase (umol/gHb) | | SE ATP (umol/dl) | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 0.21 | 0.00 | 0.017 | 0.001 | 0.09 | 0.01 | 0.02 | 0.01 |
| Schizo | 1.74 | 0.08 | 0.073 | 0.013 | 2.66 | 0.58 | 1.26 | 0.19 |
| Seizure | 1.84 | 0.07 | 0.070 | 0.015 | 3.09 | 0.65 | 1.66 | 0.56 |
| HD | 1.83 | 0.09 | 0.071 | 0.014 | 3.34 | 0.84 | 1.27 | 0.26 |
| AD | 1.77 | 0.13 | 0.073 | 0.016 | 3.34 | 0.75 | 2.06 | 0.19 |
| MS | 1.81 | 0.10 | 0.079 | 0.007 | 3.05 | 0.52 | 1.63 | 0.26 |
| SLE | 1.82 | 0.08 | 0.061 | 0.006 | 2.85 | 0.34 | 1.59 | 0.22 |
| NHL | 1.84 | 0.08 | 0.077 | 0.011 | 3.01 | 0.55 | 1.73 | 0.26 |
| Glio | 1.76 | 0.11 | 0.073 | 0.012 | 2.70 | 0.62 | 1.48 | 0.32 |
| DM | 1.77 | 0.19 | 0.067 | 0.014 | 3.19 | 0.89 | 1.97 | 0.11 |
| CAD | 1.75 | 0.12 | 0.080 | 0.007 | 2.99 | 0.65 | 1.57 | 0.37 |
| CVA | 1.82 | 0.10 | 0.079 | 0.009 | 2.98 | 0.78 | 1.49 | 0.27 |
| AIDS | 1.79 | 0.08 | 0.072 | 0.013 | 3.29 | 0.63 | 1.59 | 0.38 |
| CJD | 1.82 | 0.09 | 0.066 | 0.009 | 3.21 | 0.95 | 1.69 | 0.43 |
| Autism | 1.83 | 0.16 | 0.072 | 0.014 | 2.67 | 0.80 | 2.03 | 0.12 |
| DS-50 | 1.85 | 0.07 | 0.071 | 0.015 | 3.15 | 0.73 | 1.17 | 0.11 |
| Cerebral Palsy | 1.85 | 0.09 | 0.069 | 0.012 | 3.14 | 0.46 | 1.56 | 0.39 |
| CRF | 1.76 | 0.22 | 0.070 | 0.012 | 3.14 | 0.57 | 1.53 | 0.33 |
| Cirr/Hep Fail | 1.81 | 0.10 | 0.076 | 0.009 | 3.01 | 0.47 | 1.32 | 0.26 |
| Muc Angio | 1.78 | 0.24 | 0.067 | 0.014 | 2.92 | 0.55 | 1.35 | 0.29 |
| EMF | 1.79 | 0.07 | 0.074 | 0.009 | 3.12 | 0.60 | 1.56 | 0.48 |
| CCP | 1.84 | 0.07 | 0.073 | 0.011 | 3.15 | 0.46 | 1.51 | 0.38 |
| Exposure to EMF | 1.75 | 0.22 | 0.073 | 0.013 | 3.39 | 1.03 | 1.37 | 0.27 |
| F value | 370.517 | | 59.963 | | 54.754 | | 67.588 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 5.***

| Group | Cyto C (ng/ml) | | Lactate (mg/dl) | | Pyruvate (umol/l) | | RBC Hexokinase (ug glu phos/ hr/mgpro) | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 1.21 | 0.38 | 2.75 | 0.41 | 23.79 | 2.51 | 0.68 | 0.23 |
| Schizo | 11.58 | 0.90 | 22.07 | 1.06 | 96.54 | 9.96 | 7.69 | 3.40 |
| Seizure | 12.06 | 1.09 | 21.78 | 0.58 | 90.46 | 8.30 | 6.29 | 1.73 |
| HD | 12.65 | 1.06 | 24.28 | 1.69 | 95.44 | 12.04 | 9.30 | 3.98 |
| AD | 11.94 | 0.86 | 22.04 | 0.64 | 97.26 | 8.26 | 8.46 | 3.63 |
| MS | 11.81 | 0.67 | 23.32 | 1.10 | 102.48 | 13.20 | 8.56 | 4.75 |
| SLE | 11.73 | 0.56 | 23.06 | 1.49 | 100.51 | 9.79 | 8.02 | 3.01 |
| NHL | 11.91 | 0.49 | 22.83 | 1.24 | 95.81 | 12.18 | 7.41 | 4.22 |
| Glio | 13.00 | 0.42 | 22.20 | 0.85 | 96.58 | 8.75 | 7.82 | 3.51 |
| DM | 12.95 | 0.56 | 25.56 | 7.93 | 96.30 | 10.33 | 7.05 | 1.86 |
| CAD | 11.51 | 0.47 | 22.83 | 0.82 | 97.29 | 12.45 | 8.88 | 3.09 |
| CVA | 12.74 | 0.80 | 23.03 | 1.26 | 103.25 | 9.49 | 7.87 | 2.72 |
| AIDS | 12.29 | 0.89 | 24.87 | 4.14 | 95.55 | 7.20 | 9.84 | 2.43 |
| CJD | 12.19 | 1.22 | 23.02 | 1.61 | 96.50 | 5.93 | 8.81 | 4.26 |
| Autism | 12.48 | 0.79 | 21.95 | 0.65 | 92.71 | 8.43 | 6.95 | 2.02 |
| DS-50 | 12.79 | 1.15 | 23.69 | 2.19 | 91.81 | 4.12 | 8.68 | 2.60 |
| Cerebral Palsy | 12.14 | 1.30 | 23.12 | 1.81 | 95.33 | 11.78 | 7.92 | 3.32 |
| CRF | 12.66 | 1.01 | 23.42 | 1.20 | 97.38 | 10.76 | 7.75 | 3.08 |
| Cirr/Hep Fail | 12.81 | 0.90 | 26.20 | 5.29 | 97.77 | 13.24 | 8.99 | 3.27 |
| Muc Angio | 12.84 | 0.74 | 23.64 | 1.43 | 96.19 | 12.15 | 10.12 | 1.75 |
| EMF | 12.72 | 0.92 | 25.35 | 5.52 | 103.32 | 13.04 | 9.44 | 3.40 |
| CCP | 12.23 | 0.94 | 23.66 | 1.64 | 94.36 | 8.06 | 8.53 | 2.64 |
| Exposure to EMF | 12.26 | 1.00 | 23.31 | 1.46 | 103.28 | 11.47 | 7.58 | 3.09 |
| F value | 445.772 | | 162.945 | | 154.701 | | 18.187 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 6.***

| Group | ACOA (mg/dl) | | ACH (ug/ml) | | Glutamate (mg/dl) | |
|---|---|---|---|---|---|---|
| | Mean | ± SD | Mean | ± SD | Mean | ± SD |
| Treatment grp. | 16.49 | 0.89 | 91.98 | 2.89 | 0.16 | 0.02 |
| Schizo | 2.51 | 0.57 | 48.52 | 6.28 | 3.41 | 0.41 |
| Seizure | 2.15 | 0.22 | 33.27 | 5.99 | 3.67 | 0.38 |
| HD | 1.95 | 0.06 | 35.02 | 5.85 | 3.14 | 0.32 |
| AD | 2.19 | 0.15 | 42.84 | 8.26 | 3.53 | 0.39 |
| MS | 2.03 | 0.09 | 39.99 | 12.61 | 3.58 | 0.36 |
| SLE | 2.54 | 0.38 | 49.30 | 7.26 | 3.37 | 0.38 |
| NHL | 2.30 | 0.26 | 50.58 | 3.82 | 3.48 | 0.46 |
| Glio | 2.34 | 0.43 | 42.51 | 11.58 | 3.28 | 0.39 |
| DM | 2.17 | 0.40 | 41.31 | 10.69 | 3.53 | 0.44 |
| CAD | 2.37 | 0.44 | 49.19 | 6.86 | 3.61 | 0.28 |
| CVA | 2.25 | 0.44 | 37.45 | 7.93 | 3.31 | 0.43 |
| AIDS | 2.11 | 0.19 | 38.40 | 7.74 | 3.45 | 0.49 |
| CJD | 2.10 | 0.27 | 34.97 | 4.24 | 3.94 | 0.22 |
| Autism | 2.42 | 0.41 | 50.61 | 6.32 | 3.30 | 0.32 |
| DS-50 | 2.01 | 0.08 | 39.34 | 8.15 | 3.30 | 0.48 |
| Cerebral Palsy | 2.06 | 0.35 | 40.79 | 9.34 | 3.24 | 0.34 |
| CRF | 2.24 | 0.32 | 37.52 | 4.37 | 3.26 | 0.43 |
| Cirr/Hep Fail | 2.13 | 0.17 | 46.20 | 4.95 | 3.25 | 0.40 |
| Muc Angio | 2.51 | 0.42 | 45.51 | 7.56 | 3.11 | 0.36 |
| EMF | 2.19 | 0.19 | 42.48 | 8.62 | 3.27 | 0.39 |
| CCP | 2.04 | 0.10 | 37.95 | 8.82 | 3.33 | 0.25 |
| Exposure to EMF | 2.14 | 0.19 | 37.75 | 7.31 | 3.47 | 0.37 |
| F value | 1871.04 | | 116.901 | | 200.702 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | |

***Table 7.***

| Group | Se. Ammonia (ug/dl) | | HMG Co A (HMG CoA/MEV) | | Bile Acid (mg/ml) | |
|---|---|---|---|---|---|---|
| | **Mean** | **± SD** | **Mean** | **± SD** | **Mean** | **± SD** |
| Treatment grp. | 23.92 | 3.38 | 2.21 | 0.39 | 140.40 | 10.32 |
| Schizo | 94.72 | 3.28 | 1.11 | 0.08 | 22.45 | 5.57 |
| Seizure | 95.61 | 7.88 | 1.14 | 0.07 | 22.98 | 5.19 |
| HD | 94.60 | 8.52 | 1.08 | 0.13 | 28.93 | 4.93 |
| AD | 95.37 | 4.66 | 1.10 | 0.07 | 26.26 | 7.34 |
| MS | 93.42 | 3.69 | 1.13 | 0.08 | 24.12 | 6.43 |
| SLE | 101.18 | 17.06 | 1.14 | 0.07 | 19.62 | 1.97 |
| NHL | 91.62 | 3.24 | 1.12 | 0.10 | 23.45 | 5.01 |
| Glio | 93.20 | 4.46 | 1.10 | 0.09 | 23.43 | 6.03 |
| DM | 93.38 | 7.76 | 1.09 | 0.12 | 22.77 | 4.94 |
| CAD | 93.93 | 4.86 | 1.07 | 0.12 | 24.55 | 6.26 |
| CVA | 103.18 | 27.27 | 1.05 | 0.09 | 22.39 | 3.35 |
| AIDS | 92.47 | 3.97 | 1.08 | 0.11 | 23.28 | 5.81 |
| CJD | 93.13 | 5.79 | 1.09 | 0.12 | 21.26 | 4.81 |
| Autism | 94.01 | 5.00 | 1.12 | 0.06 | 23.16 | 5.78 |
| DS-50 | 98.81 | 15.65 | 1.09 | 0.11 | 21.31 | 4.49 |
| Cerebral Palsy | 92.09 | 3.21 | 1.07 | 0.09 | 22.80 | 5.02 |
| CRF | 98.76 | 11.12 | 1.03 | 0.10 | 26.47 | 5.30 |
| Cirr/Hep Fail | 94.77 | 2.86 | 1.04 | 0.10 | 24.91 | 5.06 |
| Muc Angio | 92.40 | 4.34 | 1.12 | 0.08 | 24.37 | 4.38 |
| EMF | 95.37 | 5.76 | 1.08 | 0.08 | 25.17 | 3.80 |
| CCP | 93.42 | 5.34 | 1.01 | 0.09 | 23.87 | 4.00 |
| Exposure to EMF | 102.62 | 26.54 | 1.00 | 0.07 | 22.58 | 5.07 |
| F value | 61.645 | | 159.963 | | 635.306 | |
| P value | < 0.001 | | < 0.001 | | < 0.001 | |

## Abbreviations

Schizo: Schizophrenia
HD: Huntington's disease
AD: Alzheimer's disease
MS: Multiple sclerosis
SLE: Systemic lupus erythematosis
NHL: Non-hodgkin's lymphoma
Glio: Glioma
DM: Diabetes mellitus
CAD: Coronary artery disease
CVA: Cerebrovascular accident
AIDS: Acquired immunodeficiency syndrome
CJD: Creutzfeldt Jakob disease
DS: Down syndrome
CRF: Chronic renal failure
Cirr/Hep Fail - Cirrhosis/Hepatic failure
EMF: Endomyocardial fibrosis
CCP: Chronic calcific pancreatitis

# Discussion

There was increase in cytochrome F420 indicating archaeal growth. The archaea can synthesize and use cholesterol as a carbon and energy source.[2, 10] The archaeal origin of the enzyme activities was indicated by antibiotic induced suppression. The study indicates the presence of actinide based archaea with an alternate actinide based enzymes or metalloenzymes in the system as indicated by rutile induced increase in enzyme activities.[11] The archaeal beta hydroxyl steroid dehydrogenase activity indicating digoxin synthesis.[12] The archaeal cholesterol

oxidase activity was increased resulting in generation of pyruvate and hydrogen peroxide.[10] The pyruvate gets converted to glutamate and ammonia by the GABA shunt pathway. The pyruvate is converted to glutamate by serum glutamate pyruvate transaminase. The glutamate gets acted upon by glutamate dehydrogenase to generate alpha ketoglutarate and ammonia. Alanine is most commonly produced by the reductive amination of pyruvate via alanine transaminase. This reversible reaction involves the interconversion of alanine and pyruvate, coupled to the interconversion of alpha-ketoglutarate (2-oxoglutarate) and glutamate. Alanine can contribute to glycine. Glutamate is acted upon by Glutamic acid decarboxylase to generate GABA. GABA is converted to succinic semialdehyde by GABA transaminase. Succinic semialdehyde is converted to succinic acid by succinic semialdehyde dehydrogenase. Glycine combines with succinyl CoA to generate delta aminolevulinic acid catalysed by the enzyme ALA synthase. There was upregulated archaeal porphyrin synthesis in the patient population which was archaeal in origin as indicated by actinide catalysis of the reactions. The cholesterol oxidase pathway generated pyruvate which entered the GABA shunt pathway. This resulted in synthesis of succinate and glycine which are substrates for ALA synthase. The archaea can undergo magnetite and calcium carbonate mineralization and can exist as calcified nanoforms.[13]

The addition of dietary fibre inhibited porphyrin synthesis, blocked archaeal growth and reverse the biochemical abnormalities. Dietary fibre is converted to acetate by gut bacteria. Dietary fibre is an antioxidant. Acetate and antioxidants can inhibit porphyrin synthesis by blocking ALA synthase activity. Dietary fibre can also have anti-archaeal activity inhibiting archaeal multiplication. Vitamin E and C inhibits ALA synthase and blocks porphyrin metabolism. They function as key factors regulating porphyrin metabolism. Fructose and other monosaccharides like glucose, levulose and mannose can inhibit ALA synthase. The addition of

vitamin C, vitamin E and fructose blocked ALA synthase and porphyrin synthesis and reversed the biochemical abnormalities. Vitamin C, vitamin E and fructose also blocked archaeal multiplication and has anti-archaeal activity.

The porphyrin metabolic dysfunction can contribute to metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis. Actinidic archaea induces porphyrin synthesis and catabolizes cholesterol generating porphyrins. The fibre in the diet is acted upon by gut bacteria generating acetate. Acetate inhibits ALA synthase and porphyrin synthesis. Dietary fibre reduces redox stress and inhibits ALA synthase by modulating redox stress. Dietary fibre has also antioxidant activity. Dietary fibre has anti-archaeal activity. An intake of 30 g of fibre is required for healthy life. The dietary fibre intake of the world population is decreasing and it produces a fibre deficiency syndrome. Dietary fibre deficiency induces porphyrinogenesis and contributes to the pathogenesis of metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis. Dietary fibre is required to modulate porphyrin metabolism and inhibit the above mentioned disease states. The above mentioned diseases states are disorders of civilization due to dietary fibre deficiency. Antioxidant vitamin C and vitamin E deficiency derived from vegetarian sources also contributes to porphyrinogenesis. Environmental pollution and redox stress can induce increased consumption of antioxidant vitamins contributing to vitamin C and vitamin E deficiency. Increased archaeal multiplication produces cholesterol consumption and deficiency of cholesterol in the system. The lipoproteins functions as vitamin E transport proteins. Increased archaeal multiplication leads to vitamin E deficiency. Deficiency of vitamin E and C when corrected by megadose administration leads to correction of porphyrin metabolic abnormalities. Vitamin E and C has got anti-archaeal activity. Intake of monosaccharides especially fructose inhibits ALA synthase and porphyrin

synthesis. Fructose also has got anti-archaeal activity. The above mentioned disorders of metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis are disorders of civilization due to decreased consumption of dietary fibre and environmental pollution induced antioxidant vitamin C and E deficiency.

The possibility of Warburg phenotype induced by actinide based primitive organism like archaea with a mevalonate pathway and cholesterol catabolism was considered in this paper. The Warburg phenotype results in inhibition of pyruvate dehydrogenase and the TCA cycle. The pyruvate enters the GABA shunt pathway where it is converted to succinyl CoA. The glycolytic pathway is upregulated and the glycolytic metabolite phosphoglycerate is converted to serine and glycine. Glycine and succinyl CoA are the substrates for ALA synthesis. The archaea induces the enzyme heme oxygenase. Heme oxygenase converts heme to bilirubin and biliverdin. This depletes heme from the system and results in upregulation of ALA synthase activity resulting in porphyria. Heme inhibits HIF alpha. The heme depletion results in upregulation of HIF alpha activity and further strengthening of the Warburg phenotype. The porphyrin self oxidation results in redox stress which activates HIF alpha and generates the Warburg phenotype. The Warburg phenotype results in channeling acetyl CoA for cholesterol synthesis as the TCA cycle and mitochondrial oxidative phosphorylation are blocked. The archaea uses cholesterol as an energy substrate. Porphyrin and ALA inhibits sodium potassium ATPase. This increases cholesterol synthesis by acting upon intracellular SREBP. The cholesterol is metabolized to pyruvate and then the GABA shunt pathway for ultimate use in porphyrin synthesis. The porphyrins can self organize and self replicate into macromolecular arrays. The porphyrin arrays behave like an autonomous organism and can have intramolecular electron transport generating ATP. The

porphyrin macroarrays can store information and can have quantal perception. The porphyrin macroarrays serves the purpose of archaeal energetics and sensory perception. The Warburg phenotype is associated with malignancy, autoimmune disease and metabolic syndrome x.

The role of archaeal porphyrins in regulation of cell functions and neuro-immuno-endocrine integration is discussed. Protoporphyrine binds to the peripheral benzodiazepine receptor regulating steroid and digoxin synthesis. Increased porphyrin metabolites can contribute to hyperdigoxinemia. Digoxin can modulate the neuro-immuno-endocrine system. Porphyrins can combine with membranes modulating membrane function. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. Porphyrins can complex with DNA and RNA modulating their function. Porphyrin interpolating with DNA can alter transcription and generate HERV expression. Heme deficiency can also result in disease states. Heme deficiency results in deficiency of heme enzymes. There is deficiency of cytochrome C oxidase and mitochondrial dysfunction. The glutathione peroxidase is dysfunctional and the glutathione system of free radical scavenging does not function. The cytochrome P450 enzymes involved in steroid and bile acid synthesis have reduced activity leading to steroid- cortisol and sex hormones as well as bile acid deficiency states. The heme deficiency results in dysfunction of nitric oxide synthase, heme oxygenase and cystathione beta synthase resulting in lack of gasotransmitters regulating the vascular system and NMDA receptor - NO, CO and $H_2S$. Heme has got cytoprotective, neuroprotective, anti-inflammatory and antiproliferative effects. Heme is also involved in the stress response. Heme deficiency leads to metabolic syndrome, immune disease, degenerations and cancer.[3-5] The porphyrins can undergo photo-oxidation and auto-oxidation generating free radicals. The archaeal porphyrins can produce free radical injury.

Free radicals produce NFKB activation, open the mitochondrial PT pore resulting in cell death, produce oncogene activation, activate NMDA receptor and GAD enzyme regulating neurotransmission and generates the Warburg phenotypes activating glycolysis and inhibiting TCA cycle/oxphos. Porphyrins have been related to schizophrenia, metabolic syndrome x, malignancy, systemic lupus erythematosis, multiple sclerosis and Alzheimer's diseases. The porphyrins can complex and intercalate with the cell membrane producing sodium potassium ATPase inhibition adding on to digoxin mediated inhibition. Porphyrins can complex with proteins and nucleic acid producing biophoton emission. Porphyrins complexing with proteins can modulate protein structure and function. Porphyrins complexing with DNA and RNA can modulate transcription and translation. The porphyrin especially protoporphyrins can bind to peripheral benzodiazepine receptors in the mitochondria and modulate its function, mitochondrial cholesterol transport and steroidogenesis. Peripheral benzodiazepine receptor modulation by protoporphyrins can regulate cell death, cell proliferation, immunity and neural functions. The porphyrin photo-oxidation generates free radicals which can modulate enzyme function. Redox stress modulated enzymes include pyruvate dehydrogenase, nitric oxide synthase, cystathione beta synthase and heme oxygenase. Free radicals can modulate mitochondrial PT pore function. Free radicals can modulate cell membrane function and inhibit sodium potassium ATPase activity. Thus the porphyrins are key regulatory molecules modulating all aspects of cell function.[3-5] There was an increase in free RNA indicating self replicating RNA viroids and free DNA indicating generation of viroid complementary DNA strands by archaeal reverse transcriptase activity. The actinides and porphyrins modulate RNA folding and catalyse its ribozymal action. Digoxin can cut and paste the viroidal strands by modulating RNA splicing generating RNA viroidal diversity. The viroids are evolutionarily escaped archaeal group I introns which have retrotransposition and

self splicing qualities. Archaeal pyruvate producing histone deacetylase inhibition and porphyrins intercalating with DNA can produce endogenous retroviral (HERV) reverse transcriptase and integrase expression. This can integrate the RNA viroidal complementary DNA into the noncoding region of eukaryotic non coding DNA using HERV integrase as has been described for borna and ebola viruses. The archaea and viroids can also induce cellular porphyrin synthesis. Bacterial and viral infections can precipitate porphyria. Thus porphyrins can regulate genomic function. The increased expression of HERV RNA can result in acquired immunodeficiency syndrome, autoimmune disease, neuronal degenerations, schizophrenia and malignancy.[14, 15] The porphyrin photo-oxidation can generate free radicals which can activate NFKB. This can produce immune activation and cytokine mediated injury. The increase in archaeal porphyrins can lead to autoimmune disease like SLE and MS. A hereditary form of MS and SLE related to altered porphyrin metabolism has been described. The protoporphyrins binding to mitochondrial benzodiazepine receptors can modulate immune function. Porphyrins can combine with proteins oxidizing their tyrosine, tryptophan, cysteine and histidine residues producing crosslinking and altering protein conformation and function. Porphyrins can complex with DNA and RNA modulating their structure. Porphyrin complexed with proteins and nucleic acids are antigenic and can lead onto autoimmune disease.[3, 4] The porphyrin photo-oxidation mediated free radical injury can lead to insulin resistance and atherogenesis. Thus archaeal porphyrins can contribute to metabolic syndrome x. Glucose has got a negative effect upon ALA synthase activity. Therefore hyperglycemia may be reactive protective mechanism to increased archaeal porphyrin synthesis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can modulate mitochondrial steroidogenesis and metabolism. Altered porphyrin metabolism has been described in the metabolic syndrome x. Porphyrias can lead onto vascular thrombosis.[3, 4] The porphyrin

photo-oxidation can generate free radicals inducing HIF alpha and producing oncogene activation. Heme deficiency can lead to activation of HIF alpha and oncogenesis. This can lead to oncogenesis. Hepatic porphyrias induced hepatocellular carcinoma. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate cell proliferation.[3, 4] The porphyrin can combine with prion proteins modulating their conformation. This leads to abnormal prion protein conformation and degradation. Archaeal porphyrins can contribute to prion disease. The porphyrins can intercalate with DNA producing HERV expression. The HERV particles generated can contribute to the retroviral state. The porphyrins in the blood can combine with bacteria and viruses and the photo-oxidation generated free radicals can kill them. The archaeal porphyrins can modulate bacterial and viral infections. The archaeal porphyrins are regulatory molecules keeping other prokaryotes and viruses on check.[3, 4] Thus the archaeal porphyrins can contribute to the pathogenesis of metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis. Archaeal porphyrin synthesis is crucial in the pathogenesis of these disorders. Porphyrins may serve as regulatory molecules modulating immune, neural, endocrine, metabolic and genetic systems. The porphyrins photo-oxidation generated free radicals can produce immune activation, produce cell death, activate cell proliferation, produce insulin resistance and modulate conscious/quantal perception. The archaeal porphyrins functions as key regulatory molecules with mitochondrial benzodiazepine receptors playing an important role.[3, 4]

The archaea and viroids can regulate the nervous system including the NMDA/GABA thalamo-cortico-thalamic pathway mediating conscious perception. Porphyrin photo-oxidation can generate free radicals which can modulate NMDA transmission. Free radicals can increase NMDA transmission. Free radicals can induce GAD and increase GABA synthesis. ALA blocks GABA

transmission and upregulates NMDA. Protoporphyrins bind to GABA receptor and promote GABA transmission. Thus porphyrins can modulate the thalamo-cortico-thalamic pathway of conscious perception. The dipolar porphyrins, PAH and archaeal magnetite in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated Frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macroscopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrin molecules have a wave particle existence and can bridge the dividing line between quantal state and particulate state. Thus the porphyrins can mediate conscious and quantal perception. Porphyrins binding to proteins, nucleic acids and cell membranes can produce biophoton emission. Porphyrins by autooxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Cellular porphyrins photo-oxidation are involved in sensing of earth magnetic fields and low level biomagnetic fields. Thus prophyrins can mediate extrasensory perception. The porphyrins can modulate hemispheric dominance. There is increased porphyrin synthesis and right hemispherical chemical dominance and decreased porphyrin synthesis in left hemispherical chemical dominance. The increase in archaeal porphyrins can contribute to the pathogenesis of schizophrenia and autism. Porphyria can lead to psychiatric disorders and seizures. Altered porphyrin metabolism has been described in autism. Porphyrin by modulating conscious and quantal perception is involved in the pathogenesis of schizophrenia and autism. Protoporphyrins block acetyl choline transmission producing a vagal neuropathy with sympathetic overactivity. Vagal neuropathy results in immune activation, vasospasm and vascular disease. A vagal neuropathy underlines neoplastic and autoimmune processes as well as metabolic syndrome x. Porphyrin induced

increased NMDA transmission and free radical injury can contribute to neuronal degeneration. Free radicals can produce mitochondrial PT pore dysfunction. This can lead to cyto C leak and activation of the caspase cascade leading to apoptosis and cell death. Altered porphyrin metabolism has been described in Alzheimer's disease. The increased porphyrin photo-oxidation generated free radicals mediated NMDA transmission can also contribute to epileptogenesis. The protoporphyrins binding to mitochondrial benzodiazepine receptors can regulate brain function and cell death.[3, 4, 16] The dipolar porphyrins, PAH and archaeal magnetite in the setting of digoxin induced sodium potassium ATPase inhibition can produce a pumped phonon system mediated Frohlich model superconducting state inducing quantal perception with nanoarchaeal sensed gravity producing the orchestrated reduction of the quantal possibilities to the macroscopic world. ALA can produce sodium potassium ATPase inhibition resulting in a pumped phonon system mediated quantal state involving dipolar porphyrins. Porphyrins by auto-oxidation can generate biophotons and are involved in quantal perception. Biophotons can mediate quantal perception. Cellular porphyrins photo-oxidation are involved in sensing of earth magnetic fields and low level biomagnetic fields. Porphyrins can thus contribute to quantal perception. Low level electromagnetic fields and light can induce porphyrin synthesis. Low level EMF can produce ferrochelatase inhibition as well as heme oxygenase induction contributing to heme depletion, ALA synthase induction and increased porphyrin synthesis. Light also induces ALA synthase and porphyrin synthesis. The increased porphyrin synthesized can contribute to increased quantal perception and can modulate conscious perception. The porphyrin induced biophotons and quantal fields can modulate the source from which low level EMF and photic fields were generated. Thus the porphyrin generated by extraneous low level EMF and photic fields can interact with the source of low level EMF and photic fields modulating it. Thus porphyrins can serve as a bridge between the human brain and the source of low

level EMF and photic fields. This serves as a mode of communication between the human brain and EMF storage devices like internet. The porphyrins can also serve as the source of communication with the environment. Environmental EMF and chemicals produce heme oxygenase induction and heme depletion increasing porphyrin synthesis, quantal perception and two-way communication. Thus induction of porphyrin synthesis can serve as a mechanism of communication between human brain and the environment by extrasensory perception.

An actinide dependent shadow biosphere of archaea and viroids in the above mentioned disease states is described. The archaeal porphyrins can contribute to the pathogenesis of metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis. Archaeal porphyrin synthesis is crucial in the pathogenesis of these disorders. Porphyrins may serve as regulatory molecules modulating immune, neural, endocrine, metabolic and genetic systems. The porphyrins photo-oxidation generated free radicals can produce immune activation, produce cell death, activate cell proliferation, produce insulin resistance and modulate conscious/quantal perception. Porphyrins can regulate hemispheric dominance. The archaeal porphyrins functions as key regulatory molecules with mitochondrial benzodiazepine receptors playing an important role. The porphyrin metabolic abnormality is corrected by acetate derived from bacterial fibre digestion, high dose fructose and high dose antioxidant vitamins C and E. Acetate, vitamin E, vitamin C and fructose inhibits ALA synthase and functions as factors modulating porphyrin metabolism. The above mentioned disorders of metabolic syndrome x, malignancy, psychiatric disorders, autoimmune disease, AIDS, prion disease, neuronal degeneration and epileptogenesis are disorders of civilization due to decreased consumption of dietary fibre and environmental pollution induced antioxidant vitamin C and E deficiency. A dietary fibre deficiency and environmental pollution induced antioxidant vitamin E and C

deficiency civilizational disorder due to porphyrin metabolic dysfunction with schizophrenia, malignancy, metabolic syndrome x, autoimmune disease and neuronal degeneration is described.

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