Chapter 1

The Dietary Fibre, Species Evolution and Neuro-Immuno-Genomic-Endocrine Integration - Relation to Internet Exposure

Dietary fibre deficiency can lead to increased endosymbiotic and colonic archaeal overgrowth. Increased endosymbiotic archaeal growth in the brain leads to perception of low level EMF related to internet use. This leads to induction of heme oxygenase, heme deficiency, induction of ALA synthase and porphyrin synthesis. The porphyrins can form supramolecular self replicating organisms called porphyrions. The porphyrions can form a template for the formation of RNA viroids, DNA viroids, prions and isoprenoid organism which symbiose to form primitive nanoarchaea. The primitive nanoarchaea have a abiogenetic self replication. The increased growth of archaea consequent to exposure to low level EMF fields in internet users leads to neanderthalisation of the brain. This produces structural changes in the human brain with prefrontal cortical atrophy and cerebellar dominance. The internet exposure and the low level EMF perception lead to modulation of the feeding behaviour leading to dietary fibre deficiency.

Dietary fibre deficiency leads to increased endosymbiotic as well as colonic archaeal growth. Dietary fibre can affect body and cell function. The original evidence linking dietary fibre and body metabolism in relation to systemic disorders came from the work of Kurup *et al*. where it was shown that the dietary fibre regulates cholesterol metabolism in the body and contributes to the genesis of metabolic syndrome X.[1-7] Dietary fibre deficiency produces predominant small intestinal digestion and dietary fibre excess leads to colonic digestion. Small intestinal digestion in the presence of dietary fibre deficiency leads to alteration in colonic flora and archaeal overgrowth. A high fibre diet produces predominantly colonic digestion and leads to suppression of archaeal growth. The colonic archaea seeps through the gut blood barrier producing archaeal endosymbiosis. Thus small intestinal digestion due to dietary fibre deficiency versus colonic digestion due to dietary fibre excess determines the density of archaea in the colon as well as the endosymbiotic compartment.

Dietary fibre is the single most important component of the human diet more important than dietary protein, fats, carbohydrates, vitamins and minerals. Dietary fibre is the substrate that determines symbiosis and symbiotic evolution.

The endosymbiotic archaea regulates human functions and species type and depends upon the colonic archaea whose density is determined by the fibre intake. The colonic archaeal population density depends upon dietary fibre intake. Populations with low fibre intake have lesser density of colonic archaeal microflora and endosymbiotic archaea. Endosymbiotic archaea contributes to neanderthalisation of the species. Populations consuming a high saturated fat and protein diet with low fibre intake tend to get increased endosymbiotic archaeal growth and are neanderthalised. Populations with high fibre intake up to 80 g/day tend to have reduced archaeal density in the colon and reduced archaeal endosymbiosis contributing to homo sapienisation of the population. Thus fibre intake regulates the endosymbiotic archaeal density and type of human species.

Dietary fibre can affect brain function. The colonic digestion of dietary fibre by the microflora generates short chain fatty acids. The short chain fatty acid propionate can produce an autistic brain pathology. The short chain fatty acids can bind to GPCR increasing sympathetic activity. The SCFA acetate, propionate and butyrate can be metabolized by the mitochondria generating ATP. The SCFA butyrate is a HDAC inhibitor and modulates genomic transmission.

Butyrate can modulate cognition and increase cognitive function. The acetate is channelled to the glutamate glutamine cycle and modulates neurotransmitter in the synapse. Butyrate can produce histone hyperacetylation and increase BDNF activity. The SCFA can bind to G-protein coupled FFA receptor producing immunosuppression. The short chain fatty acids are anti-inflammatory. Because the SCFA are anti-inflammatory it can modulate insulin resistance. Dietary fibre

deficiency can lead to metabolic syndrome and autoimmune disease. Butyrate by producing HDAC inhibition is antioncogenic and inhibits oncogenesis. Butyrate can produce HDAC inhibition and alter protein conformation and folding producing modulation and amelioration of genetic disorders. Butyrate can increase BDNF activity in the brain and produces neuroprotection. Thus a high fibre diet protects against civilisational diseases. Butyrate promotes stem cell transformation and converts fibroblast to pluripotent embryonic stem cells. Thus the fibre derived butyrate is a regenerative molecule. Thus dietary fibre deficiency can lead to cancer, metabolic syndrome, stroke, coronary artery disease, neurodegeneration, genetic disorders, autoimmune diseases and protein folding diseases. Dietary fibre is regulatory substance for the neuronal, immune, genomic and endocrine system.

Dietary fibre can alter the colonic microflora. A high fibre intake suppresses colonic archaeal growth and archaeal endosymbiosis. A high fibre diet suppresses endosymbiotic archaeal growth leading onto homo sapienisation of the species. A high fibre diet leads to increased generation of butyrate and HDAC inhibition leading onto expression of the HERV genes and their reintegration into the genome. The HERV jumping genes contributes to the dynamicity of the genome and is important in the evolution of synaptic connectivity and the homo sapien neocortex. A low fibre diet increases colonic archaeal growth and archaeal endosymbiosis contributing to neanderthalisation of the species and the brain. A low fibre diet and reduced levels of butyrate contributes to modulation of histone acetylation and reduced generation of HERV sequences. This contributes to rigidity of the genome and reduced synaptic connectivity. This leads to cerebral cortical suppression and cerebellar dominance contributing to neanderthalisation of the brain and species. Thus fibre intake in the diet alters symbiotic microflora especially archaeal endosymbiosis and human evolution.

Table 1. *Dietary fibre intake.*

Groups	Fibre content of diet
Homo Sapiens	High fibre 80%
Homo Neanderthalis	Low fibre 70%

*Low fibre < 5 g/day; high fibre > 20 g/day

Table 2. *Dietary fibre intake.*

Groups	Fibre content of diet
Normal	High fibre 80%
Schizophrenia	Low fibre 70%
Autism	Low fibre 60%

*Low fibre < 5 g/day; high fibre > 20 g/day

References

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