Multiple Sclerosis - Stress Activated (STY-1) DIY

When several studies made reference to stress activated multiple sclerosis and the epigenetic molecule STY1 was linked as a likely cause, it was subjected to quantum biology (QB) using the tools outlined in the following illustration.

QB identified three forms of STY; i.e., 1- 3 with 1 being calnexin related and 2 being driven by calmodulin. The following DIY models will enable computational biologists and other interested parties to utilize bioinformatics to verify the QB findings; for calnexin and then calmodulin.

Multiple Sclerosis: Stress Activated - DIY	Multiple Sclerosis: Stress Activated - DIY
TNF-Alpha: TGF- Alpha: VEGF-A (Calnexin) Density (Th1, CD-4 and p63) Calcium - threonine - magnesium E-Cadherin p16 Calcium - serine - magnesium N-Cadherin p18 Calcium - cysteine - magnesium P-Cadherin p19	TNF-Alpha: TGF- Alpha: VEGF-A (Calnexin) Density (Th1, CD-4 and p63)Calcium - threonine - magnesium E-Cadherinp16 p18 p18 Calcium - cysteine - magnesium P-CadherinP19
TNF-Beta: TGF-Beta: VEGF-B (Calmodulin) Motility (Th2, CD-8 and p73) Calcium - phenylalanine - magnesium (HRas) p21 Calcium - tyrosine - magnesium (KRas) p27 Calcium - tryptophan - magnesium (NRas) p57	TNF-Beta: TGF-Beta: VEGF-B (Calmodulin) Motility (Th2, CD-8 and p73)Calcium - phenylalanine - magnesium (HRas)p21Calcium - tyrosine - magnesium (KRas)p27Calcium - tryptophan - magnesium (NRas)p57
Using bioinformatic search for multiple sclerosis, insert each of the following as variables individually to verify its relationship to calnexin; Th1, CD4, p63, STY-1, E- Cadherin, p16, N-Cadherin, p18, TGFa, and TNFa.	Using bioinformatic search for multiple sclerosis, insert each of the following as variables individually to verify its relationship to calmodulin; Th2, CD8, p73, STY-2. HRas, p21, KRas, p27, TGFβ, and TNFβ.

Spanning the fifteen years required to create the QB algorithm, MS has been our most difficult model because the primary cause is stress related and a major neurohormone imbalance behavioral health challenge.

QB has identified three epigenetic factors that create the aforementioned imbalances between the calcium and magnesium dependent calnexin and calmodulin activities; one is IFN γ calcineurin, a second one is the enzyme created by the imbalance of the calpains (p53 - p63 and p73) with the third issue being complex and not suited for an easy DIY exercise; i.e. imbalances between the neuropeptides Y (NPY) and pancreatic polypeptide (PP) with PYY providing enzymatic activity.

Anyone can conduct a basic DIY for the first two factors using bioinformatic search with calcineurin or p53 as the variables.

Note: The calnexin - calmodulin illustrations already identified p63 and p73 interactions. However, the neuropeptide verification will only be provided to computational biologists who become knowledgeable in QB to understand the cellular physiology whereby repetitive thoughts can be stored in the glial cells of the hippocampus as subliminal memory; e.g. chronic pain.

Reference is made to quantum biology (QB). QB is an algorithm for epigenetic activity. A scientifically verifiable non-commercial explanation for the algorithm is provided here for application by the global biomedical research community. Particular attention must be placed on the 1st, 7th and 8th links in the following document. <u>https://www.mcfip.net/Quantum-Biology.html</u>

Note: The following article is provided for discussion relative to MS be driven from "inside the brain."

https://neurosciencenews.com/inflammatory-cells-ms-16385/