Food Quality and Advances in Pharmacological Management Prevent Mitochondrial Apoptosis and Epilepsy Induced Stroke

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Editorial

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EDITORIAL

Major interests in the promotion of neuron mitochondrial biogenesis versus mitochondrial apoptosis has accelerated with relevance to impaired mitochondrial function as a causative factor in various neurodegenerative diseases [1-3]. Evidence from various research groups have reported impaired mitochondrial dynamics (shape, size, fission-fusion, distribution, movement) in various neurodegenerative diseases. In the current global stroke epidemic [4-6] the concern for nutritional interventions and lifestyle changes has accelerated with relevance to maintenance of neuron mitochondrial biogenesis [7] and the prevention of accelerated brain ageing. Individuals with mitochondrial epilepsy [8,9] have been reported with the risk of epilepsy induced stroke [10,11].

The use of drug therapy to control epilepsy has been the focus of the modern era with systematic screening of many thousands of compounds in rodent seizure models [12,13] with advances in pharmacological management achieved over the last 20 years. The discovery of the heat shock gene *Sirtuin 1* (*Sirt 1*) is connected to neuron and mitochondrial biogenesis that is now important to neurodegeneration and epilepsy induced stroke [6,14]. The repression of *Sirt 1* indicates changes in core body temperature **(Figure 1)** that may be critical to toxic immune reactions with mitochondrial apoptosis [15] and sensitive to hyperthermia induced seizure [16,17]. *Sirt 1* is repressed in non-alcoholic fatty liver disease (NAFLD) and diabetes with inactivation of drug/xenobiotic therapy [18] that may lead to inactivation of antimicrobial/antiepileptic therapy (drug-drug interactions) **(Figure 1)** with relevance to neurodegeneration and epilepsy induced stroke [14]. Heat therapy in diabetics with epilepsy should be carefully regulated to prevent heat stress induced inactivation of the nuclear receptor *Sirt 1* [18].

Appetite control to maintain anti-epileptic drug therapy and neuron mitochondrial biogenesis has been the focus of nutritional research [14]. The current global chronic disease epidemic indicates that mitophagy [18,19] has become of major concern to drug treatment programs. Ingestion of a healthy diet is essential to prevent epilepsy and maintain the appetite gene *Sirt 1* [14]. Overnutrition represses *Sirt 1* with effects of food quality [14] that contain either bacterial lipopolysaccharides (LPS), mycotoxin or xenobiotics involved in interference [14,18] of the pharmacological management of patients with neurodegenerative disease and epilepsy. In individuals that consume food/drink that contains caffeine the effects of caffeine as a *Sirt 1* modulator [14] may be overridden with delayed hepatic caffeine metabolism associated with caffeine/drug interactions in the central nervous system and associated with inactivation of antimicrobial/antiepileptic therapy and drug induced mitochondrial toxicity. Pharmacological

management of neurodegenerative and epilepetic patients requires ingestion of *Sirt 1* activators and magnesium [20] when food quality and nutrition guidelines are compromised **(Figure 2)**.

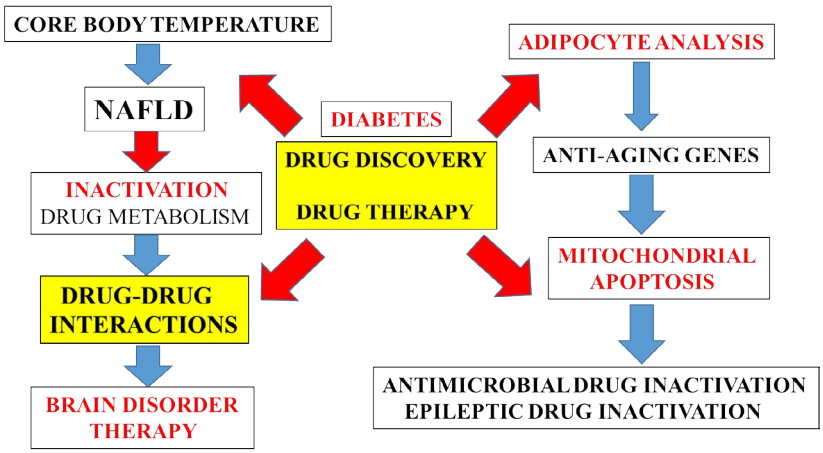

Figure 1. Core body temperature and hepatic drug metabolism are connected to drug therapy with *Sirt 1* and non-alcoholic fatty liver disease (NAFLD) involved in defective drug metabolism with induction of mitochondrial apoptosis in neurodegeneraton and epilepsy induced stroke.

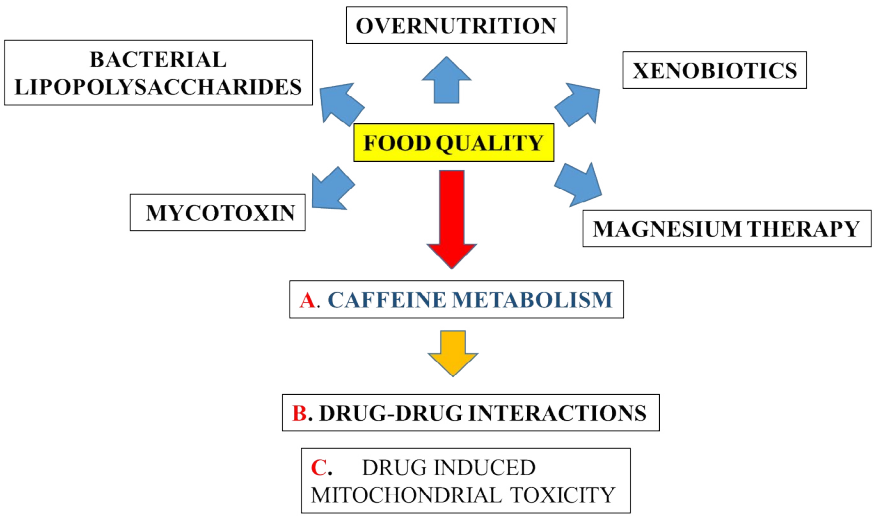

Figure 2. Food quality and overnutrition inactivate caffeine metabolism with relevance to the pharmacological management of antimicrobial and epileptic drugs with relevance to drug induced mitophagy.

CONCLUSION

The consumption of healthy diets that activate *Sirt 1* is essential to maintain drug therapy in patients with neurodegenerative disease and epilepsy induced stroke. Advances in pharmacological management in epileptic patients are essential with food quality and caffeine intake connected to mitochondrial biogenesis and induction of epilepsy induced stroke. Core body temperature regulation is essential to prevent hypethermia induced epilepsy and interference with anti-epileptic drug therapy.

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