

## Infection Control in Medicine with Relevance to Mitophagy and Organ Survival

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The association between infections, antibiotic use and mitophagy has raised concerns with relevance to the doses of antibiotics in mitochondrial dynamics and antibiotic induced mitochondrial apoptosis in these chronic diseases with irreversible cell death associated antibiotics and multisystem organ disease. The anti-aging gene Sirt 1 is a NAD<sup>+</sup> dependent class III histone deacetylase activity involved in the regulation of metabolic activity, insulin resistance and inflammatory processes and is now important to drug therapy and metabolism with relevance to prevention of drug-drug interactions. Sirt 1 is involved in drug metabolism and linked to hepatic glucose, fatty acid and caffeine metabolism. Nutritional interventions that regulate Sirt 1 have become critical to maintain antibiotic use and drug therapy with relevance to non alcoholic fatty liver disease (NAFLD), cardiovascular disease and neurodegeneration. Consumption activators of Sirt 1 (magnesium, leucine, alpha-lipoic acid, pyruvic acid, resveratrol) are important to maintain antibiotic use and drug therapy/metabolism.

In the developing world Sirt 1 may be repressed with relevance to defective xenobiotic metabolism and mitophagy. Increased plasma levels of bacterial lipopolysaccharides (LPS) in developing world individuals may repress Sirt 1 and inactivate antibiotic and drug therapy. The use of Indian spices and antibiotic use need to be carefully controlled with relevance to effective mitochondrial dynamics and to maintain mitochondrial biogenesis. Infection prevention and control by antibiotics may become ineffective with diet, drug and inhibitors (drug-drug interactions) that may disrupt Sirt 1 with relevance to autoimmune disease and rejection of the pancreas, thyroid, kidneys, liver, heart and brain. Magnesium defi-

ciency may inactivate antibiotic and drug therapy with irreversible cell death associated antibiotic inactivation, polypharmacy and organ suicide [1-13].

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