

Metabolic syndrome and melatonin: a tool for prevent obesity-associated abnormalities

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Obesity is a common and complex health problem, which impacts crucial organs; it is also considered an independent risk factor for chronic kidney disease [1]. Few studies have analyzed the consequence of obesity in the renal proximal convoluted tubules, the major section of the reabsorptive process. To best perform its functions, the kidney requires energy primarily provided by mitochondria. Melatonin, indoleamine and antioxidant, has been identified in mitochondria, and overwhelming evidence has documented its essential role in the prevention of oxidative mitochondrial damage [2]. Herein, we evaluated the mechanism(s) of mitochondrial alterations in an animal model of obesity (*ob/ob* mice) and describe the beneficial effects of melatonin treatment on mitochondria morphology and dynamics as influenced by mitofusin-2 and the intrinsic apoptotic cascade. Melatonin was dissolved in 1% ethanol and added to the drinking water from postnatal week 5 to 13; the calculated dose of melatonin intake was 100 mg/kg body weight/day. Compared to control mice, obesity-induced morphological alterations were apparent in the proximal tubules; the tubules contained round mitochondria with irregular, short cristae and the lining cells excited and elevated apoptotic index. Melatonin supplementation in obese mice changed mitochondria shape and cristae organization of proximal tubules, enhanced mitofusin-2 expression, which in turn modulated the progression of the mitochondria-driven intrinsic apoptotic pathway. The results aid in reducing renal failure. The melatonin-mediated changes probably suggest the use of melatonin to protect against renal morphological damage and dysfunction during metabolic disease.

References

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Keywords

Obesity, melatonin, mitochondria, proximal convoluted tubules.