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Lactobacillus acidophilus KBL409 protects against kidney injury via improving mitochondrial function with chronic kidney disease

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Objectives: Recent advances have led to greater recognition of the role of mitochondrial dysfunction in the pathogenesis of chronic kidney disease (CKD). There has been evidence that CKD is also associated with dysbiosis. Here, we aimed to evaluate whether probiotic supplements can have protective effects against kidney injury via improving mitochondrial function.

Methods: An animal model of CKD was induced by feeding C57BL/6 mice a diet containing 0.2% adenine. KBL409, a strain of *Lactobacillus acidophilus,* was administered via oral gavage at a dose of 1×10^9 CFU daily. To clarify the underlying mechanisms by which probiotics exert protective effects on mitochondria in CKD, primary mouse tubular epithelial cells stimulated with TGF-β and p-cresyl sulfate were administered with butyrate.

Results: In CKD mice, PGC-1α and AMPK, key mitochondrial energy metabolism regulators, were down-regulated. In addition, mitochondrial dynamics shifted toward fission, the number of fragmented cristae increased, and mitochondrial mass decreased. These alterations were restored by KBL409 administration. KBL409 supplementation also improved defects in fatty acid oxidation and glycolysis and restored the suppressed enzyme levels involved in TCA cycle. Accordingly, there was a concomitant improvement in mitochondrial respiration and ATP production assessed by mitochondrial function assay. These favorable effects of KBL409 on mitochondria ultimately decreased kidney fibrosis in CKD mice. *In vitro* analyses with butyrate recapitulated the findings of animal study.

Conclusions: This study demonstrates that administration of the probiotic *Lactobacillus acidophilus* KBL409 protects against kidney injury via improving mitochondrial function.