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Sirtuin 3 Modulates Skeletal Muscle Proteomic Remodeling Under Chronic Kidney Disease

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Objectives : Sarcopenia is a common complication of chronic kidney disease (CKD). Mitochondrial dysfunction and metabolic dysregulation are thought to play key roles in CKD-associated muscle wasting. Sirtuin 3 (SIRT3), a mitochondrial deacetylase, regulates mitochondrial metabolism and oxidative stress. However, its role in CKD-associated skeletal muscle dysfunction remains incompletely understood. This study demonstrated the contribution of SIRT3 to CKD-induced skeletal muscle proteomic alterations.
Methods : Chronic kidney disease (CKD) was induced in C57BL/6 mice by adenine feeding for 8 weeks. Serum from control and CKD mice was applied to differentiated C2C12 myotubes to assess circulating effects on SIRT3 expression. Gastrocnemius tissues from four groups (control, CKD, SIRT3 knockout(KO), and SIRT3KO CKD) were pulverized and subjected to tandem mass tag (TMT)–based quantitative proteomics followed by LC-MS/MS analysis. Raw mass spectrometry data were processed using the FragPipe pipeline (MSFragger, PeptideProphet, ProteinProphet, and IonQuant), followed by differential protein expression and pathway enrichment analyses.
Results : CKD mice exhibited sarcopenic phenotypes compared with control mice. C2C12 myotubes treated with serum from CKD mice reduced SIRT3 expression. Proteomic profiling of gastrocnemius tissue from four groups (control, CKD, SIRT3KO, and SIRT3KO CKD) revealed that CKD was the dominant driver of proteomic remodeling. CKD induced mitochondrial metabolic disturbance characterized by reduced metabolic and mitochondrial protein networks, accompanied by altered cytoskeleton organization, increased extracellular matrix (ECM) remodeling, and inflammatory infiltration pathways. SIRT3 deficiency showed relatively modest effects under basal conditions. However, under CKD condition, SIRT3 loss markedly reshaped the proteomic landscape, further suppressing mitochondrial metabolic pathways while enhancing pathways related to cytoskeletal organization, ECM remodeling, and inflammatory responses.
Conclusions : These findings indicate that CKD induces extensive muscle proteomic alterations and that SIRT3 deficiency amplifies these changes under CKD condition. SIRT3 serves as a context-dependent modulator of CKD-associated skeletal muscle remodeling and may represent a potential therapeutic target for CKD-associated sarcopenia.