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Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

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Muscle Energetics and Function in Kidney Disease

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Sarcopenia, defined as decrease in muscle function and mass, is common in patients with moderate to advanced chronic kidney disease (CKD) and is associated with poor clinical outcomes. Muscle mitochondrial dysfunction is proposed as one of the mechanisms underlying sarcopenia. Patients with moderate to advanced CKD have decreased muscle mitochondrial content and oxidative capacity along with suppressed activity of various mitochondrial enzymes such as mitochondrial electron transport chain complexes and pyruvate dehydrogenase, leading to impaired energy production. Other mitochondrial abnormalities found in this population include defective beta-oxidation of fatty acids and mitochondrial DNA mutations. These changes are noticeable from the early stages of CKD and correlate with severity of the disease. Damage induced by uremic toxins, oxidative stress, and systemic inflammation has been implicated in the development of mitochondrial dysfunction in CKD patients. Given that mitochondrial function is an important determinant of physical activity and performance, its modulation is a potential therapeutic target for sarcopenia in patients with kidney disease. Coenzyme Q, nicotinamide, and cardiolipin-targeted peptides have been tested as therapeutic interventions in early studies. Aerobic exercise, a well-established strategy to improve muscle function and mass in healthy adults, is not as effective in patients with advanced kidney disease. This might be due to reduced expression or impaired activation of PGC1- α, the master regulator of mitochondrial biogenesis. Further studies are needed to broaden our understanding of the pathogenesis of mitochondrial dysfunction and to develop mitochondrial-targeted therapies for prevention and treatment of sarcopenia in patients with CKD.

Keywords: nutrition, muscle, mitochondria, sarcopenia