



Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care

**KSN** SEOUL, KOREA  
**2026**

The 46th Annual Meeting of the Korean Society of Nephrology



Jun 11 (Thu) – 14 (Sun), 2026

COEX, Seoul, Korea

**Abstract Type : Oral presentation**

**Abstract Submission No.: A-0940**

**Abstract Topic : Diabetic Kidney Disease + Metabolic Abnormality-related Kidney Disease**

**MOTS-c pretreatment ameliorates the cardioprotective effects of mesenchymal stem/stromal cells harvested from patients with obesity**

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**Objectives :** Mitochondrial open-reading frame of the 12S ribosomal RNA type-c (MOTS-c) has anti-inflammatory and metabolic benefits. Mesenchymal stem/stromal cells (MSCs) offer treatment potential via direct cellular interactions and paracrine effects, including extracellular vehicles (EVs). However, MSC and EV potency in subjects with obesity is blunted, partly due to mitochondrial impairment. We hypothesized that a fall of MOTS-c expression in adipose tissue-derived (AT)-MSC from obese patients contributes to their dysfunction in renovascular hypertensive (RVH) mice.

**Methods :** AT-MSCs were isolated from subcutaneous fat of obese and age-matched lean subjects during bariatric or kidney-donation surgeries, respectively. EVs were obtained from AT-MSC preconditioned with 10  $\mu$ M MOTS-c or vehicle for 48h. Two weeks after RVH intra-aortic AT-MSC or EVs were delivered in mice. Two weeks later cardiac function was assessed in-vivo by MRI, and hearts studied ex-vivo . .

**Results :** MOTS-c expression was markedly decreased in Obese-MSCs, associated with impaired mitochondrial function. Co-incubation restored MOTS-c levels. RVH-induced cardiac hypertrophy and myocardial damage was attenuated by lean but not obese-MSCs or obese-EVs. MOTS-c preconditioning partially restored obese-AT-MSCs functions, preventing blood pressure elevation, reducing hypertrophy, and improving myocardial vascularization and redox status.

**Conclusions :** Downregulation of MOTS-c in obese-MSCs in part impairs the reparative capacity of obesity-derived AT-MSCs. MOTS-c replenishment may enhance endogenous repair and autologous cell-based therapies in obesity.