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The Differential Roles of Leptin and Adiponectin on Phenotype Transition of Human Peritoneal Mesothelial Cells

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Objectives: Functional and structural integrity of peritoneal membrane is essential for successful peritoneal dialysis (PD). Residential cells in peritoneal cavity are mesothelial cells (MCs), endothelial cells, fibroblasts, and adipocytes (ACs) located in submesothelial tissue. Previous studies focused on the role of peritoneal MCs in the development of peritoneal fibrosis, however the role of neighboring adipose tissue and adipokines released from ACs has not been investigated. Since there are now compelling evidences suggesting that ACs exert important metabolic and proinflammatory effects on peripheral tissue, we examined the effect of 2 representative adipokines, leptin and adiponectin on phenotype transition of MCs

Methods: The effect of co- and pre-treatment of leptin (10~100 ng/mL) or adiponectin (0.5~2 μg/mL) on epithelial-to-mesenchymal transition (EMT) of cultured human peritoneal MCs was investigated. The effect of leptin and adiponectin gene silencing using siRNA technique on TGF-β-induced EMT was examined. EMT was assessed by the changes in morphology and markers of epithelial and mesenchymal cells both in mRNA and protein levels.

Results: Neither leptin nor adiponectin per se induced EMT of MCs up to 72 hours. Leptin (100 ng/mL) aggravated TGF-β (1 ng/mL)-induced EMT whereas adiponectin (2 μg/mL) ameliorated TGF-β-induced EMT of MCs at 48 hours of stimulation. Leptin gene silencing alleviated TGF-β-induced EMT and adiponectin siRNA treatment enhanced TGF-β-induced EMT. EMT induced by 24-hour exposure to TGF-β was reversible upon the removal of TGF-β, and this reversal of EMT was further prominent with co-treatment of adiponectin for additional 48 hours. Reversibility of EMT after removal of TGF-β stimulation was blocked by leptin treatment.

Conclusions: Our results suggest the differential role of leptin and adiponectin in phenotype transition of MCs and peritoneal fibrosis. Modulation of the expression/activity of adipokines can be a novel target for prevention and treatment of peritoneal fibrosis.