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Cross-talk between Mesothelial Cells (MCs) and Adipocytes (ACs) as a Trigger for Peritoneal Fibrosis in Peritoneal Dialysis (PD)

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Objectives: In peritoneal cavity, adipose tissue is buried under mesothelial monolayer and known to interact with neighboring MCs. Although a majority of previous work has focused on peritoneal MCs as a key player of peritoneal fibrosis, more attention needs to be paid to the role of peritoneal ACs and their interaction with MCs in PD. We investigated the cross-talk of MCs with ACs and its role on epithelial-to-mesenchymal transition (EMT) of MCs.

Methods: Transwell co-culture system was used in which MCs were cultured with mature ACs differentiated from 3T3-L1 preadipocytes. EMT of MCs was evaluated by the changes in morphology and markers of epithelial and mesenchymal cells. Adipokines in supernatant and cell lysate were analyzed by adipokine array, real-time PCR, and western blotting. Effect of stimulation or gene silencing of adipokines on EMT was examined.

Results: Co-culture of MCs and ACs induced EMT of MCs from 48 hours. Co-culture resulted in a decrease of adiponectin (0.4-fold) and plasminogen activator inhibitor-1 (PAI-1) (0.6-fold) and an increase in IL-6 (3.9-fold) and leptin (1.5-fold) in cell culture supernatant. Co-culture also led to an increased expression of monocyte chemoattractant protein-1 (MCP-1) (1.6-fold), vascular endothelial growth factor (VEGF) (2.4-fold), and PAI-1 (1.4-fold) whereas resulted in a decreased expression of adiponectin (0.88-fold) and resistin (0.81-fold). siLeptin or siPAI-1 treatment alleviated EMT of MCs whereas the treatment of IL-6 (50 ng/ml), MCP-1 (10 ng/ml), or VEGF (2 ng/ml) induced EMT of MCs.

Conclusions: ACs could induce phenotype transition of peritoneal MCs and trigger pro-fibrotic signal in peritoneal cavity. Precise mechanism for the pro-fibrotic cross-talk between MCs and ACs and the role of various adipokines which shows the different pattern of up- or down-regulation on co-culture system needs to be further investigated.