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AMP-activated protein kinase inhibitor compound C inhibits adipocyte conditioned medium-induced macrophage chemotaxis and inflammation through targeting focal adhesion kinase and I κ B kinase

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Macrophage infiltration and inflammation in adipose tissue are well established to cause obesity-linked insulin resistance and type 2 diabetes. AMP-activated protein kinase (AMPK) has pleiotropic effects for energy metabolism, but its role for adipocyte-promoted macrophage chemotaxis has not been explored. Here, we report that an AMPK inhibitor, compound C (CC), significantly inhibited adipocyte conditioned medium (CM)-induced macrophage chemotaxis in RAW 264.7 cells in a concentration dependent manner, and this inhibitory effect was accompanied by the inhibition of focal adhesion kinase (FAK), AKT and inhibitory κ B kinase (IKK) in macrophages, which all are important regulators of cell migration. Inhibition of macrophage chemotaxis by CC was not prevented but potentiated by co-treatment with AMPK activator 5-aminoimidazole-4-carboxamide ribonucleotide (AICAR), indicating that CC inhibition of AMPK is not involved in its suppression of chemotaxis. Instead, FAK phosphorylation was additively reduced by combined treatment of CC and AICAR, suggesting that CC and AICAR prevent adipocyte macrophage chemotaxis through targeting FAK pathway. CC treatment also prevented the expression of proinflammatory genes in RAW 264.7 macrophages when stimulated with either CM or lipopolysaccharide (LPS) and this effect was mediated by inhibition of IKK/NF κ B pathway. Lastly, we demonstrated that CC functioned as a repressor of macrophage-mediated insulin resistance in adipocytes. Our results suggest that CC might serve as a useful molecule in research on adipocyte-mediated macrophage chemotaxis and as a potential lead compound for the treatment of obesity-linked insulin resistance.

Biography

Seong Ji Woo has completed her PhD at 2014 from Chonbuk National University Medical School. She is a post-doc in the same school and has a interest in the the role of macrophage in the obesity-related inflammation and insulin resistance.

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