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Resistin exacerbates insulin resistance under the condition of low adiponectin in 3T3-L1 adipocytes

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Adipocytokines such as resistin, TNF-α, and adiponectin, which are adipocyte-derived peptides, play important roles in glucose metabolism. Resistin and TNF-α have been implicated as factors associated with the development of insulin resistance in obesity. In contrast, adiponectin has been shown to improve insulin sensitivity in insulin resistance. However, the interaction among these adipocytokines is still unknown. In this study, we investigated the mechanism of the effects of these 3 adipocytokines (resistin, adiponectin, and TNF-α) on glucose transport in 3T3-L1 adipocytes.

Glucose uptake was evaluated by 2-[3H] deoxy-glucose (DOG) uptake assay in 3T3-L1 adipocytes. Resistin and adiponectin secretion were analyzed by western blotting.

Adenovirus-mediated overexpression of resistin inhibited insulin-stimulated 2-DOG uptake by only 15% compared with control cells. In contrast, pretreatment of cells with 10 ng/mL TNF-α for 3 hrs did not inhibit insulin stimulated 2-DOG uptake compared with control cells, whereas overexpression of resistin led to a ~40 % decrease in insulin stimulated 2-DOG uptake following pretreatment with TNF-α. TNF-α has been shown to suppress the expression and secretion of adiponectin from adipocytes. Therefore, we speculated that this potentiating effect of resistin might be caused by the reduction in adiponectin secretion. We confirmed that the secretion of adiponectin was decreased by ~50 % in TNF-α treated cells compared to control cells. Furthermore, overexpression of adiponectin prevented this additive effect of resistin and TNF-α.

In conclusion, these results suggest that: (1) TNF-α enhances the action of resistin via the reduction of adiponectin, 2) Resistin may cause severe insulin resistance under low adiponectin levels.

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