

Abstract

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β -Carotene accumulation in 3T3-L1 adipocytes inhibits the elevation of reactive oxygen species and the suppression of genes related to insulin sensitivity induced by tumor necrosis factor- α .

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OBJECTIVE: β -Carotene is an abundant carotenoid with potent antioxidative activities and accumulates in adipose tissue. However, its physiologic functions are poorly understood. In this study, we examined whether accumulation of β -carotene for 4 d in insulin-resistant 3T3-L1 adipocytes alters the expression of genes related to insulin sensitivity.

METHODS: The 3T3-L1 adipocytes were treated with/without 10 or 20 μ M β -carotene during differentiation for 4 d. The cells treated with 10 μ M β -carotene for 4 d were subsequently incubated with/without 5 ng/mL of tumor necrosis factor- α for 48 h in the medium without β -carotene. The mRNA levels of genes in the cells and adiponectin protein levels in the medium were determined by real-time reverse transcription-polymerase chain reaction and enzyme-linked immunosorbent assay, respectively. Reactive oxygen species levels in the cells were assessed by oxidation of 2',7'-dichlorodihydrofluorescein diacetate.

RESULT: β -Carotene treatment at a concentration of 20 μ M, but not 10 μ M, in 3T3-L1 adipocytes during differentiation for 4 d enhanced the expression of genes related to insulin sensitivity, including adiponectin, adipocyte lipid-binding protein, glucose transporter-4, peroxisome proliferator-activated receptor- γ 2, and adiponectin protein in the medium. Tumor necrosis factor- α treatment repressed the expression of these genes and adiponectin protein in the medium and induced reactive oxygen species levels. In contrast, cells that accumulated β -carotene at a concentration of 10 μ M did not show these alterations.

CONCLUSION: The accumulation of the β -carotene in 3T3-L1 adipocytes restores the expression of genes related to insulin sensitivity and reactive oxygen species levels in insulin-resistant adipocytes.

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