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Involvement of Cbl-b-mediated macrophage inactivation in insulin resistance

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Abstract

Aging and overnutrition cause obesity in rodents and humans. It is well-known that obesity causes various diseases by producing insulin resistance (IR). Macrophages infiltrate the adipose tissue (AT) of obese individuals and cause chronic low-level inflammation associated with IR. Macrophage infiltration is regulated by the chemokines that are released from hypertrophied adipocytes and the immune cells in AT. Saturated fatty acids (SFAs) are recognized by toll-like receptor 4 (TLR4) and induce inflammatory responses in AT macrophages (ATMs). The inflammatory cytokines that are released from activated ATMs promote IR in peripheral organs, such as the liver, skeletal muscle and AT. Therefore, ATM activation is a therapeutic target for IR in obesity. The ubiquitin ligase Casitas b-lineage lymphoma-b (Cbl-b) appears to potently suppress macrophage migration and activation. Cbl-b is highly expressed in leukocytes and negatively regulates signals associated with migration and activation. Cbl-b deficiency enhances ATM accumulation and IR in aging- and diet-induced obese mice. Cbl-b inhibits migration-related signals and SFA-induced TLR4 signaling in ATMs. Thus, targeting Cbl-b may be a potential therapeutic

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