**Roles of Immune Cells in Obese Adipose Tissue**

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Systemic low-grade chronic inflammation has been intensively investigated in obese subjects. Recently, various immune cell types, such as macrophages, granulocytes, helper T cells, cytotoxic T cells, and B cells, have been implicated in the pathogenesis of adipose tissue inflammation. However, the roles of invariant natural killer T cells (iNKT cells) and the regulation of iNKT cell activity in adipose tissue are not thoroughly understood. Recently, we have demonstrated that iNKT cells are decreased in number in the adipose tissue of obese subjects. Moreover, iNKT cell-deficient J18 knockout mice become more obese and exhibit increased adipose tissue inflammation at the early stage of obesity. Interestingly, CD1d, a molecule involved in lipid antigen presentation to iNKT cells, is highly expressed in adipocytes and CD1d-expressing adipocytes stimulated iNKT cell activity through physical interaction. iNKT cell population and CD1d expression are reduced in the adipose tissue of obese mice and humans compared to those of lean subjects. To investigate the *in vivo* role of adipocyte CD1d in the regulation of adipose iNKT cell activity and adipose tissue inflammation, we generated adipocyte specific CD1d knockout (adipo-CD1d KO) mice. When adipo-CD1d KO mice were fed with high fat diet, they showed elevated insulin resistance and adipose tissue inflammation. These data suggest that adipocytes regulate iNKT cell activity via CD1d and that the interaction between adipocytes and iNKT cells would modulate adipose tissue inflammation in obesity.