The role of NK cells in the development of obesity-induced inflammation and insulin resistance

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Obesity is the major cause of the development of insulin resistance and Type 2 Diabetes. Recently, the notion that obesity-induced inflammation mediates the development of insulin resistance in animal models and humans has been gaining strong support. Furthermore, numerous studies have also shown that immune cells in local tissues, in particular in visceral adipose tissue, play a major role in the regulation of obesity-induced inflammation. It has been shown that obesity disrupts the immune balance by suppressing anti-inflammatory cells (*e.g.,* regulatory T cells [Tregs]) while simultaneously activating pro-inflammatory cells (*e.g.,* adipose tissue macrophages [ATMs]). Many studies from the classical immunology field show that complex cross-regulating interactions between different immune cell types control inflammation. However, the roles these interactions play have not been studied extensively in the metabolism field. We have recently shown that natural killer (NK) cells play a critical role in the development of obesity-induced inflammation and insulin resistance, in part by controlling ATM activation and adipose tissue inflammation. Hence, NK cells play a detrimental role in the regulation of obesity-induced insulin resistance and Type 2 Diabetes, which is a striking contrast that NK cells in cancer plays a positive role in cancer treatments. Thereby, our studies may provide important preclinical implication that obesity-induced inflammation regulated by adipose NK cells could be a therapeutic target for the treatment of insulin resistance and Type 2 Diabetes.