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Previous reports suggested that adipose tissue macrophages are involved in maintaining insulin sensitivity adipocytes along with improvement in metabolic genes. Nonetheless, it is largely unknown how depletion o M2-like macrophages regulates insulin sensitivity and adipocyte progenitor (AP) proliferation. To understand the role of M2-like macrophages in white adipose tissue (WAT), we generated CD206DTR mice based on transge expression of diphtheria toxin receptor under the control of the CD206(+) promoter to speci cally deplete CD206 M2-like macrophages. Partial depletion of CD206 M2-like macrophages resulted in the generation of smalle adipocytes, upregulated expression of metabolically favorable genes and enhanced insulin sensitivity in both ch and high-fat diet-fed CD206-reduced mice. In vivo and in vitro studies revealed that Tgf 1, abundantly expresse in CD206 M2-like macrophages, regulate AP di erentiation and proliferation. Flow cytometry analysis revealed tha the number of APs was increased and cyclin gene expression levels in the AP fraction were up-regulated. To valid this hypothesis, we generated genetically engineered mice in which CD206 speci c Tgf 1 was knocked out a e tamoxifen treatment. Increased number of APs and smaller adipocytes were observed in the CD206 speci c Tgf knockout mice, suggesting that CD206 cell-speci c deletion of Tgf 1 resulted in the enhanced proliferation of AP Previous studies had shown that type 2 cytokines and M2 macrophages induce cold-induced browning in inguin WAT (ingWAT) by producing catecholamines. Exactly how the conditional and partial depletion of CD206 M2-like macrophages regulates the cold-induced browning of ingWAT, however, remains unknown. We also examine the role of CD206 M2-like macrophages in the cold-induced browning of WAT and found that partial depletion of CD206 M2-like macrophages caused an increase in the number of beige progenitors and also enhanced th proliferation in ingWAT in response to cold. us, we concluded that CD206 M2-like macrophages inhibit the proliferation of white and beige progenitors.

K d : Adipocyte progenitors, adipose tissue macrophages, beige adipocyte, insulin sensitivity.