Chronic Inflammation Resolution in Obese Women: Biomarkers of Sustained Immune Remodeling Post-RYGB

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Obesity-driven inflammation is primarily caused by pro-inflammatory immune cell infiltration, which leads to an increased secretion of pro-inflammatory cytokines like TNF and IL-6. This drives a cycle of metabolic dysfunction disproportionately affecting obese women. Roux-en-Y gastric bypass (RYGB), one of the most common types of bariatric surgery, is successful in resolving inflammation long-term with metabolic improvements linked to weight loss and decreased inflammatory markers. Past research has studied short-term reductions of pro-inflammatory serum biomarkers post-RYGB, but few studies have identified genetic biomarkers associated with long-term inflammation resolution. This study aimed to identify candidate genetic biomarkers that mediate long-term suppression of pro-inflammatory immune cells from baseline to 5 years post-RYGB in white adipose tissue (WAT) of obese women. A differential gene expression analysis pipeline with FDR corrections was conducted to determine the statistical significance of genes with differentiation between chosen time points. Then, pathway enrichment analysis was performed to determine significant biological pathways. The study identified 50 highly differentially expressed genes and 13 downregulated genes as candidate biomarkers for sustained WAT inflammation resolution. Pathway enrichment analysis validated the roles of biomarkers in neutrophil degranulation, macrophage activation, and inflammatory responses. This biomarker panel offers a molecular framework for predicting long-term patient responses post-RYGB, with implications for personalized post-surgical care and potential non-invasive therapies for immune cell infiltration. Further studies will functionally validate biomarkers and determine sex-specificity in broader cohorts.

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