

Recent work has focused on the role of the microflora in the etiology of obesity. In a rodent model, we have shown that an increase in gastrointestinal (GI) permeability is associated with weight gain whereas resistance diet-induced obesity is characterized by an intact intestinal barrier. A leaky gut allows bacterial products translocation to the circulation, notably pro-inflammatory lipopolysaccharide, LPS. Chronic treatment with LPS leads to weight gain. In this study we investigated the pathways behind the obesigenic effect of LPS and notably found that LPS can alter gut-brain satiety signaling.