Response to Comment on: Cani et al. (2007) Metabolic Endotoxemia Initiates Obesity and Insulin Resistance: *Diabetes* 56:1761–1772

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We thank Saito and Reiko (1), who point out an important fact with regard to the potential sources of circulating lipopolysaccharide (LPS) in metabolic endotoxemia and its correlation with obesity. This relationship has been shown in humans and animal models (2,3). In the light of our recent data (4), which causally demonstrate the role of intestinal bacterial LPS on the onset of metabolic diseases, Saito and Reiko remind us that changes in plasma LPS, driven by periodontitis, may be a causative factor of the relationship between periodontitis and metabolic diseases. We agree that, in addition to intestinal microflora, periodontitis could be an important source of endotoxemia. Interestingly, regarding Saito and Reiko's hypothesis, our group has previously shown an interaction between the soluble CD14 receptor (the main LPS receptor), a CD14 gene polymorphism, and cigarette smoking (5), in which the deleterious role of buccal microbiota and periodontitis have been well established (6). The next step to pursue these hypotheses will be to demonstrate whether a reduction of metabolic endotoxemia, by means of a dietary intervention or treatment of periodontal disease, impact the subsequent risk for metabolic disease.

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