	Percent Abundance (median value)			
<u>Taxon</u>	<u>Diabetic</u>	Normoglycemic	<u>p-value</u>	FDR corrected
Proteobacteria Enterobacteriaceae	23.18	3.8	0.021	0.036
Firmicutes Aerococcus	5.53	0.04	0.001	0.006
Firmicutes Streptococcus	3.74	1.49	0.005	0.02
Firmicutes Clostridiales	2.54	9.82	0.009	0.027
Firmicutes Enterococcus	0.94	0.2	0.012	0.03
Firmicutes Lachnospiraceae	0.8	3.33	0.001	0.008
Bacteroidetes Bacteroides	0.71	2.52	0.005	0.02
Proteobacteria Pseudomonas	0.65	1.73	0.012	0.03
Firmicutes Ruminococcaceae	0.55	2.4	0.005	0.02
Firmicutes Oscillospira	0.28	1.59	0.016	0.033
Firmicutes Staphylococcus	0.24	0.04	0.009	0.027
Firmicutes Ruminococcus	0.0024	0.0084	0.007	0.026

Table S1. Oral bacteria from db/db diabetic and db/+ normoglycemic controls related to Figure 1C. Bacteria were analyzed by 16S rRNA gene tag sequencing from bacteria obtained from 9 diabetic and 8 normoglycemic mice. The most abundant bacteria in the diabetic group that were signficantly different from the normoglycemic are shown with the nominal p-value and the p-value following adjustment for false discrovery rate (FDR).

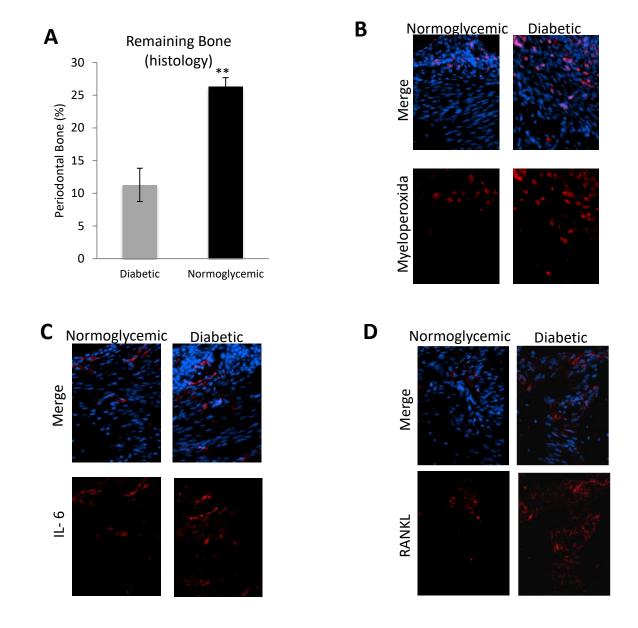
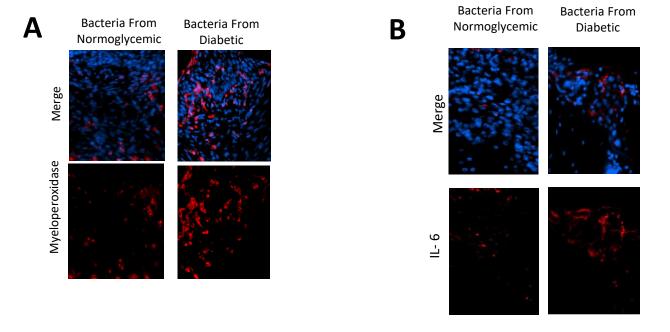


Figure S1 related to Figures 1E, 1J and 1K. Periodontal bone loss and detection of myeloperoxidase, IL-6 and RANKL in normoglycemic and diabetic mice. A: Percent periodontal bone remaining in 6 normoglycemic and 6 diabetic mice measured by histomorphometric analysis of histologic sections. The analysis was performed twice with similar results. B-D: Immunofluorescence was carried out with antibody specific for myeloperoxidase to identify neutrophils (B); IL-6 (C); and RANKL (D). Representative images of the gingival connective tissue are shown. All sections were counterstained with DAPI to detect nuclei (merged image). Immunofluorescence with control antibody was negative (not shown). Original magnification of fluorescent images 400x.



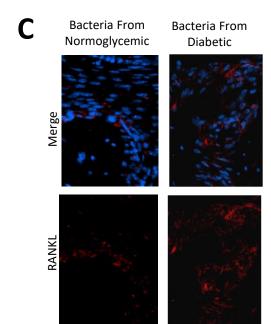
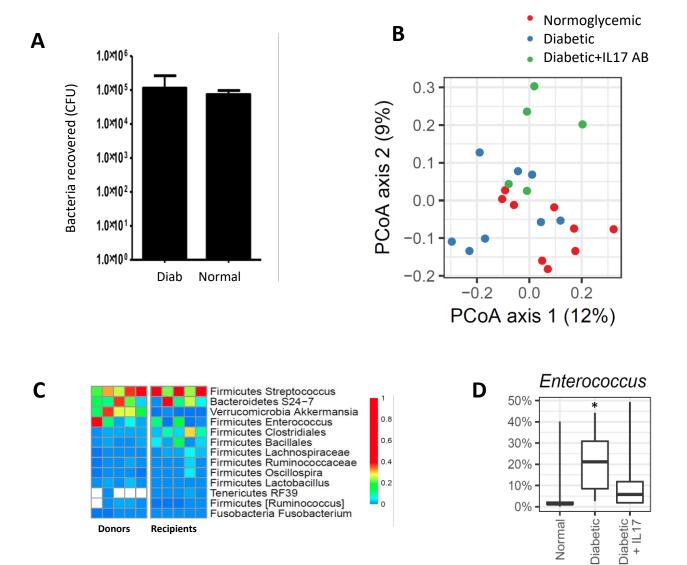


Figure S2 related to Figures 2E-G. Detection of myeloperoxidase, IL-6 and RANKL in germ free recipient mice after transfer of bacteria from normoglycemic and diabetic mice. Bacteria were collected from donor mice and pooled for a given group and transferred to germ free recipients. Immuno-fluorescence was carried out with antibody specific for myeloperoxidase (A); IL-6 (B); and RANKL (C). Representative images of the gingival connective tissue are shown. All sections were counterstained with DAPI to detect nuclei (merged image). Immunofluorescence with control antibody was negative (not shown). Original magnification of fluorescent images 400x.



**Figure S3 related to Figures 2A and 3A. Transfer of bacteria from donor mice to germ free recipients. A.** Colony forming units (CFU) collected from oral swabs of 9 diabetic and 8 normoglycemic mice were assessed by real-time PCR by comparison with a standard curve. The analysis was performed twice with similar results. **B:** Principal coordinates analysis of recipient mice (5 mice per group) following transfer of microbiome from normal, diabetic or diabetic treated with IL-17 antibody. **C:** Heat map of bacteria from the oral cavity of diabetic antibody treated mice (Donors) and bacteria from the oral cavity of corresponding germ-free mice one week after transfer (Recipients). **D:** Relative abundance of *Enterococcus* in recipient mice (5 mice per group) after transfer of oral microbiome from normoglycemic (normal), diabetic or IL-17 antibody treated diabetic mice. (\*p<0.05) when compared to germ-free recipient that received bacteria from normoglycemic donor mice.