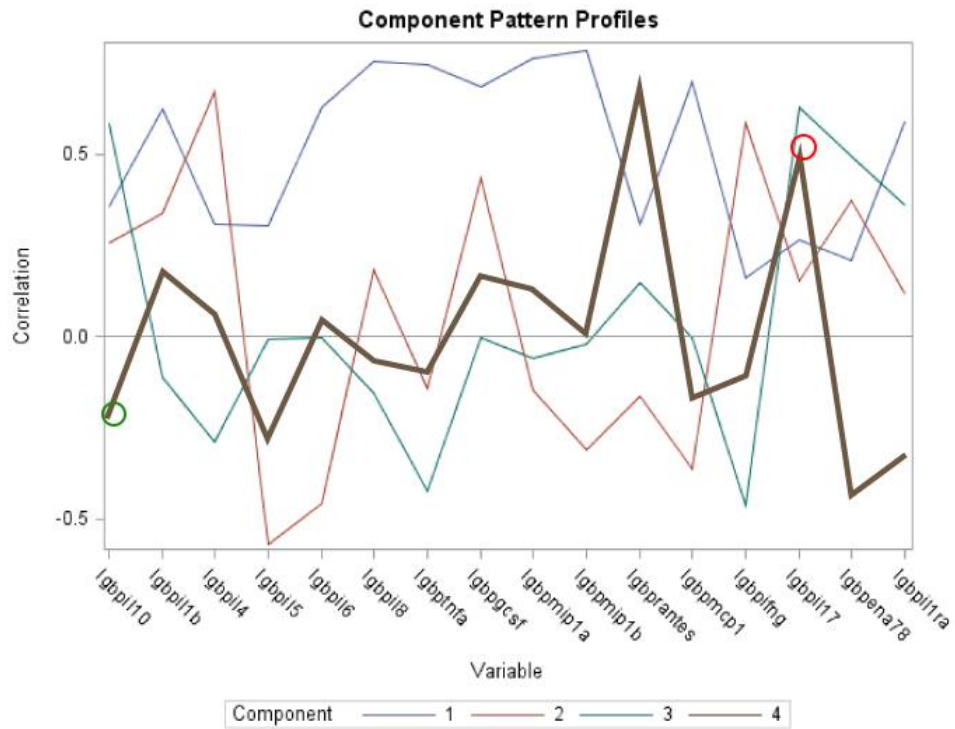
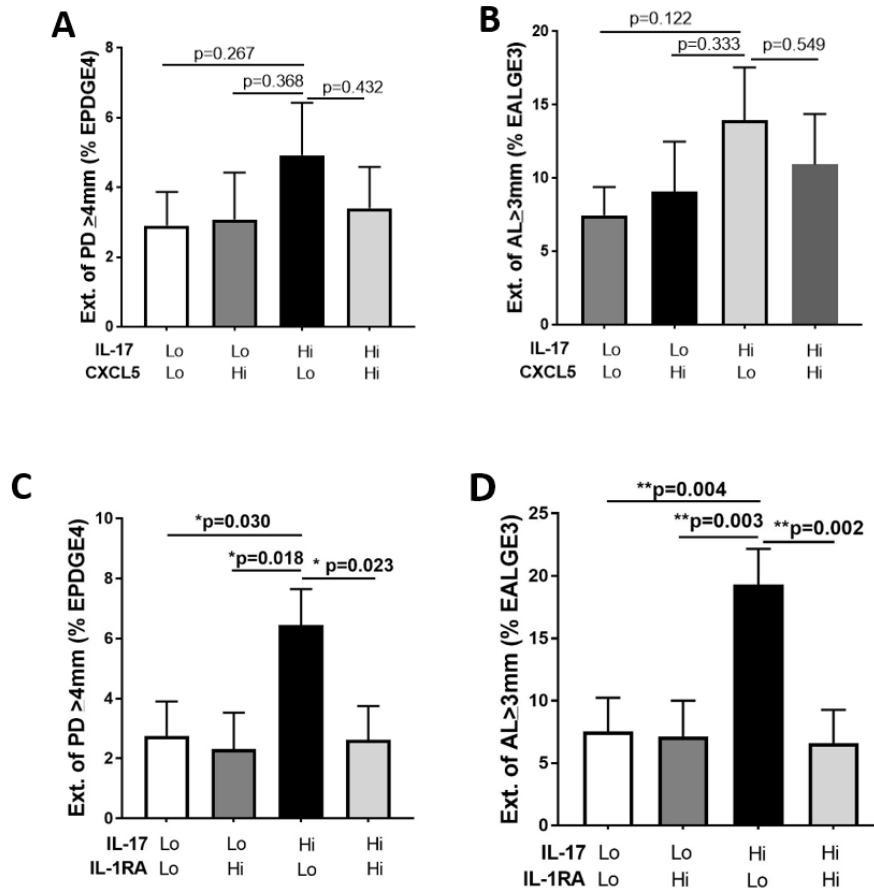


## Supplemental Figure 1



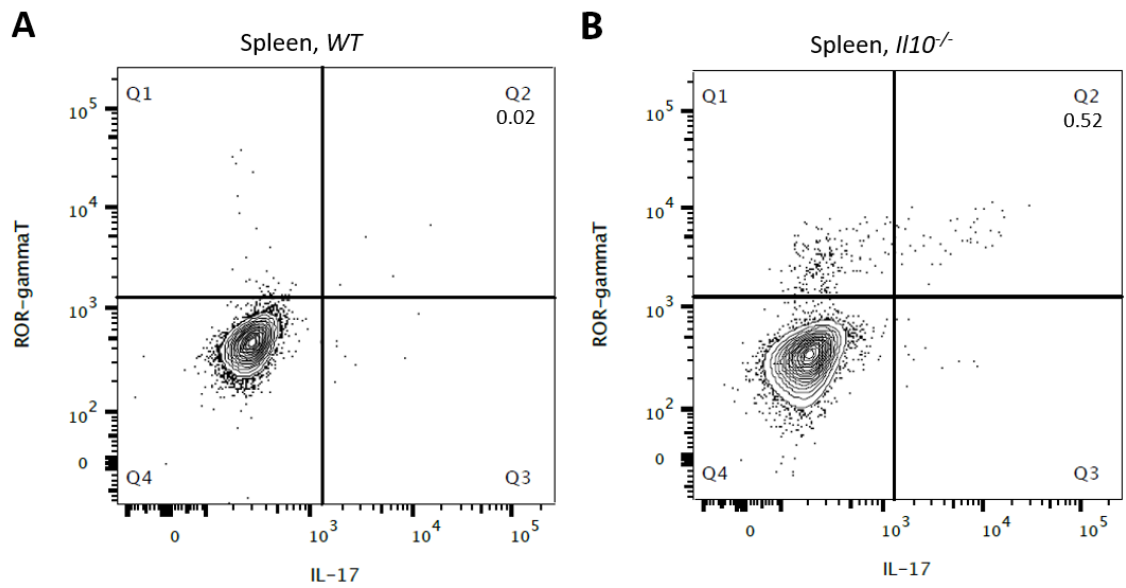
**Supplemental Fig 1. The correlations of Principal Component Trait (PCT) pattern profiles with 16 constitutive GCF inflammatory mediators. Correlations of GCF IL-17 and IL-10 within component 4 ('PIT4', bold) were circled in red and green, respectively.**

## Supplemental Figure 2



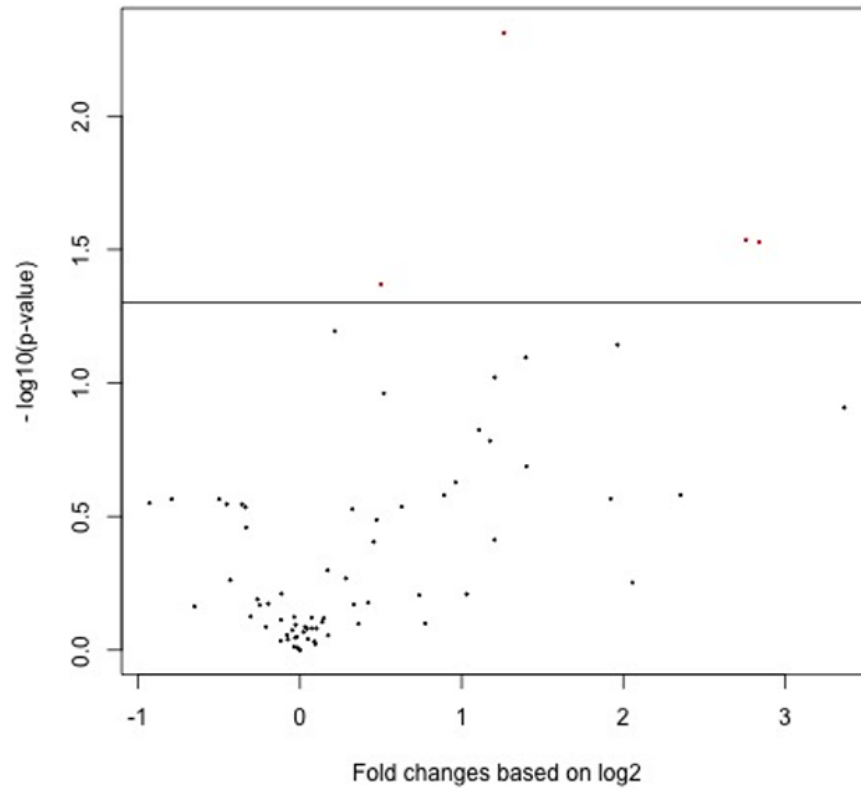
Supplemental Fig 2. Interactions between GCF IL-17 and other mediators in determining periodontal disease severity. Clinical disease activity reflected by extent of probing depth ( $\geq 4$ mm) (A, C) or attachment loss level at the interproximal sites ( $\geq 3$ mm) (B,D) was compared among groups defined by the interactions between GCF IL-17 and GCF CXCL5 (A,B), or IL-1RA (C,D) levels that were dichotomized to “high” or “low”. Box plots show mean + SE. “\*” indicates  $p < 0.05$ ; “\*\*\*” indicates  $p < 0.01$ .

### Supplemental Figure 3



Supplemental Fig 3. Flow cytometry plots show IL-17 secreting T cells (Q2, ROR- $\gamma$ <sup>+</sup>IL-17<sup>+</sup>), which were previously gated on CD45<sup>+</sup>CD3<sup>+</sup>, in the spleen tissues of WT mice (A) and *Il10*<sup>-/-</sup> mice (B).

## Supplemental Figure 4



Supplemental Fig 4. Volcano plot of the NanoString mRNA expression data from the ligature-induced alveolar bone loss model. Fold changes and p values of a customized IL-17 pathway panel were calculated in the ligature site of gingiva from *Il10* KO mice in relation to WT control mice. Red dots above the horizontal line indicate significance ( $p < 0.05$ ).