Supplemental Figure Legend

FigureS1. Oral *P. gingivalis* quantity analysis during B cells adaptive transfer to *P. gingivalis*–associated ligature-induced periodontitis mouse model. In *P. gingivalis*–associated ligature-induced periodontitis mouse model, at day 0 (pre-ligation), day 2 (before transfer) and day 14 (termination), oral swabs from each animal were resolved in 200ul PBS containing protease inhibitor cocktail (Roche).DNA was extracted from each sample and subjected to real-time PCR amplification of DNA fragment using *P. gingivalis* 16S rRNA gene-specific primers 5'-GAGTTTGATYMTGGCTCAG-3' and 5'-TCAGTCGCAGTATGGCAA-3' and universal bacterial 16S rRNA gene primer 5'-GAGTTTGATYMTGGCTCAG and 5'-AAGGAGGTGWTCCARCC-3'. The quantity of P. gingivalis DNA or total bacterial DNA from each sample was extrapolated from the DNA standard curve derived from serial dilution of bacteria with known concentrations. The percentages of *P. gingivalis* DNA quantity of total bacterial DNA was calculated by division and compared between groups at same time point. There was none detectable (ND) *P. gingivalis* at day 0, and non-significance (NS) between any two groups at day 2 and day 14 (mean±SE, n=4).

FigureS2. Adoptive transfer of $CD1d^{hi}CD5^{+}$ B cells from TLR2 KO mice inhibited *P. gingivalis* – associated ligature-induced periodontal bone loss. *P. gingivalis*-soaked ligatures were placed subgingivally around the maxillary second molars on Day 1 and were retained for 2 weeks in C57/BL6 mice to establish experimental periodontitis model. $CD1d^{hi}CD5^{+}$ B cells and $CD1d^{lo}CD5^{-}$ B cells were sorted by flow cytometry from splenic B cells of separated from *P. gingivalis*-immunized C57/BL6 Wild tpe mice or TLR2 KO mice and then transferred ($1x10^6$ cells in 100 μl PBS per mice) into recipient mice through tail vein on Day 2, same amount of PBS were injected to control group without ligation and ligation no transfer group. Maxilla was collected on day 14 and the palatal alveolar bone resorption area around maxillary second molars was measured. Data were presented as bone resorption area/mm^2 at the magnification of 30X. (mean±SE, n=6, $^{**}p<0.01$, NS, no significant difference).

FigureS3. Adoptive transfer of CD1d^{hi}CD5^{+} B cells from TLR2 KO mice suppressed gingival expressions of RANKL/OPG, TNFa and IL-1b but increased IL-10 expression. Gingival tissues were isolated under a surgical microscope from mice of no transfer control group, CD1d^{lo}CD5^{-} B cells (TLR2 KO mice) transfer group and CD1d^{hi}CD5^{+} B cells (TLR2 KO mice) transfer groups at day 14 after ligation. The gingival mRNA expressions of (**a**) RANKL/OPG, (**b**) IL-10, (**c**) TNFα and (**d**) IL-1β were detected and analyzed by RT-qPCR as indicated in duplicates and the relative levels were normalized by GAPDH. (mean±SE, n=3, *p<0.05, **p<0.01).