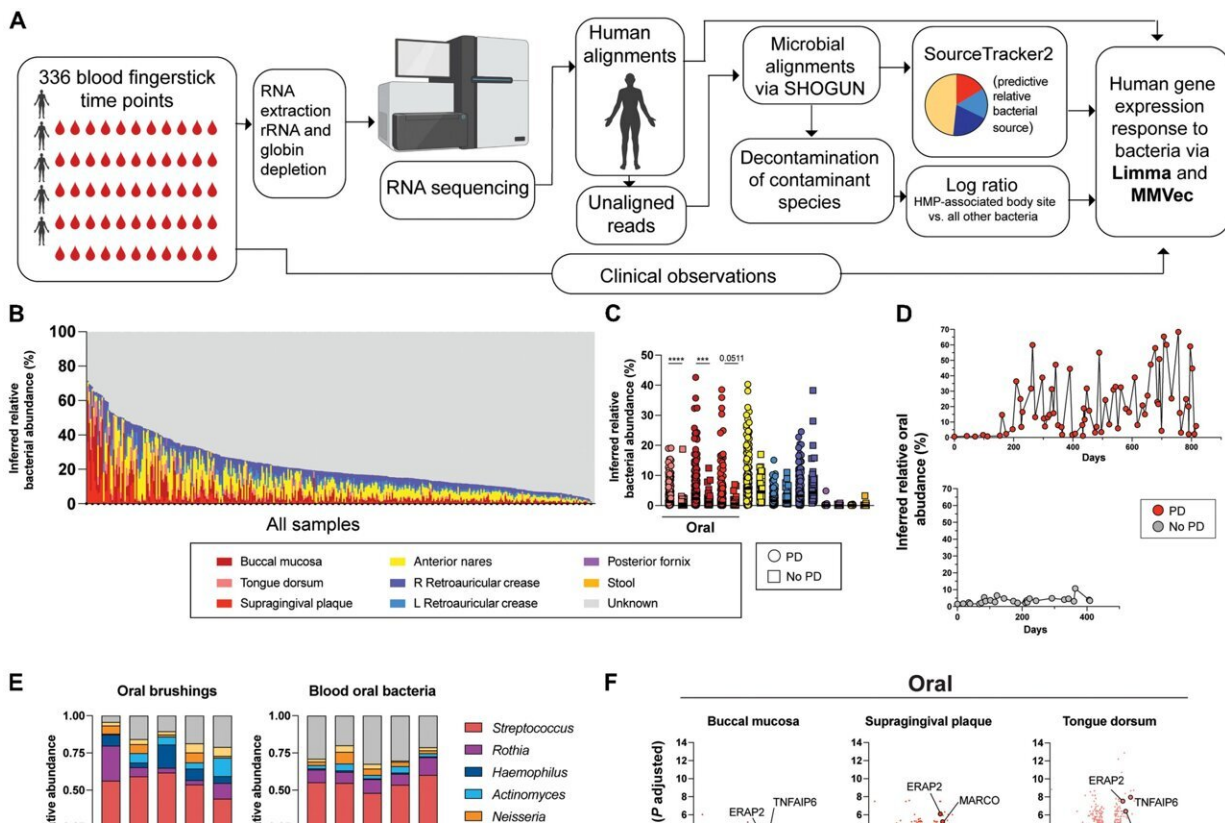


# Study suggests causative pathway between gum disease and rheumatoid arthritis

February 23 2023, by Justin Jackson



Oral mucosal breaks trigger systemic inflammatory responses. (A) Experimental workflow. (B) Inferred relative bacterial abundances from eight HMP body sites (n = 336). (C) Body site-inferred relative abundances for time points from RA patients with and without periodontal disease, median. (D) Inferred relative oral abundances in blood for one RA donor with and one without periodontal disease. (E) Relative abundances of bacteria genera from oral brushings (left) and blood (right). (F) Log<sub>10</sub>-adjusted P values versus log fold changes of human gene expression relative to bacterial abundances of the three oral body sites. (G)

Enriched GO pathways in differentially expressed human genes from (F) (adjusted *P* values). IFN- $\gamma$ , interferon- $\gamma$ . (H) Percentage of monocytes in blood cell counts compared with inferred relative abundances of oral bacteria, Pearson's correlation. (I and J) RT-qPCRs of mRNA of inflammatory genes in (I) whole blood, (J) granulocytes, monocytes, and lymphocytes ( $n = 4$  to 6) stimulated with oral bacteria versus unstimulated control. (K) Flow cytometry data showing proportion of ISG15<sup>+</sup> monocytes in CD14<sup>+</sup> monocytes incubated with PBS, oral bacteria, and anti-Fc $\gamma$ R2a or isotype ( $n = 8$ ). (B, H, and I) Two-tailed Kruskal-Wallis test, Dunnett-corrected multiple comparisons. (C) Mann-Whitney *U* test. (K) Within-subjects analysis of variance (ANOVA), Tukey-corrected multiple comparisons. Mean  $\pm$  SD. \**P*

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