Study suggests causative pathway between gum disease and rheumatoid arthritis

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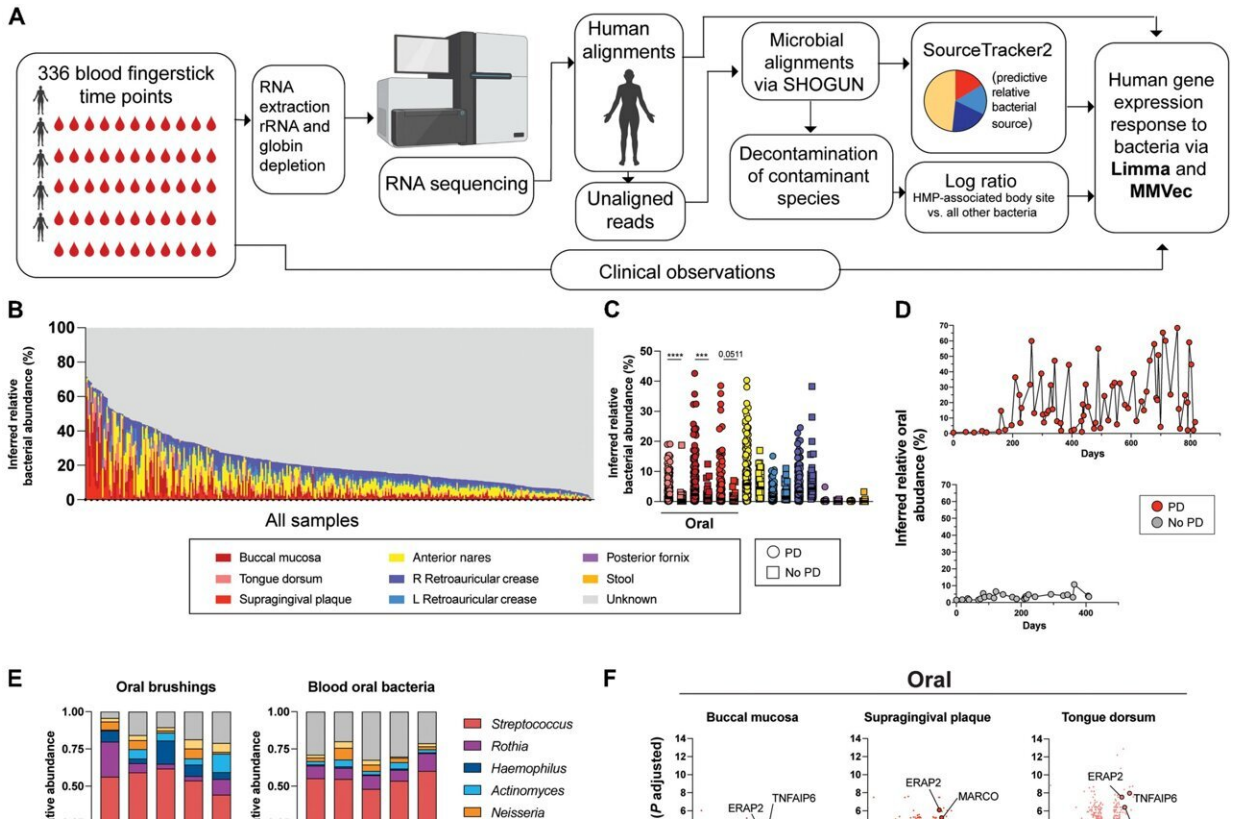

Oral mucosal breaks trigger systemic inflammatory responses.(**A**) Experimental workflow. (**B**) Inferred relative bacterial abundances from eight HMP body sites (n = 336). (**C**) Body site–inferred relative abundances for time points from RA patients with and without periodontal disease, median. (**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_{10}-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-γ, interferon-γ. (**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 $ISG15^+$ monocytes in $CD14^+$ monocytes incubated with PBS, oral bacteria, and anti-FcγR2a 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 ± SD. *P