Oral health in HIV+ population

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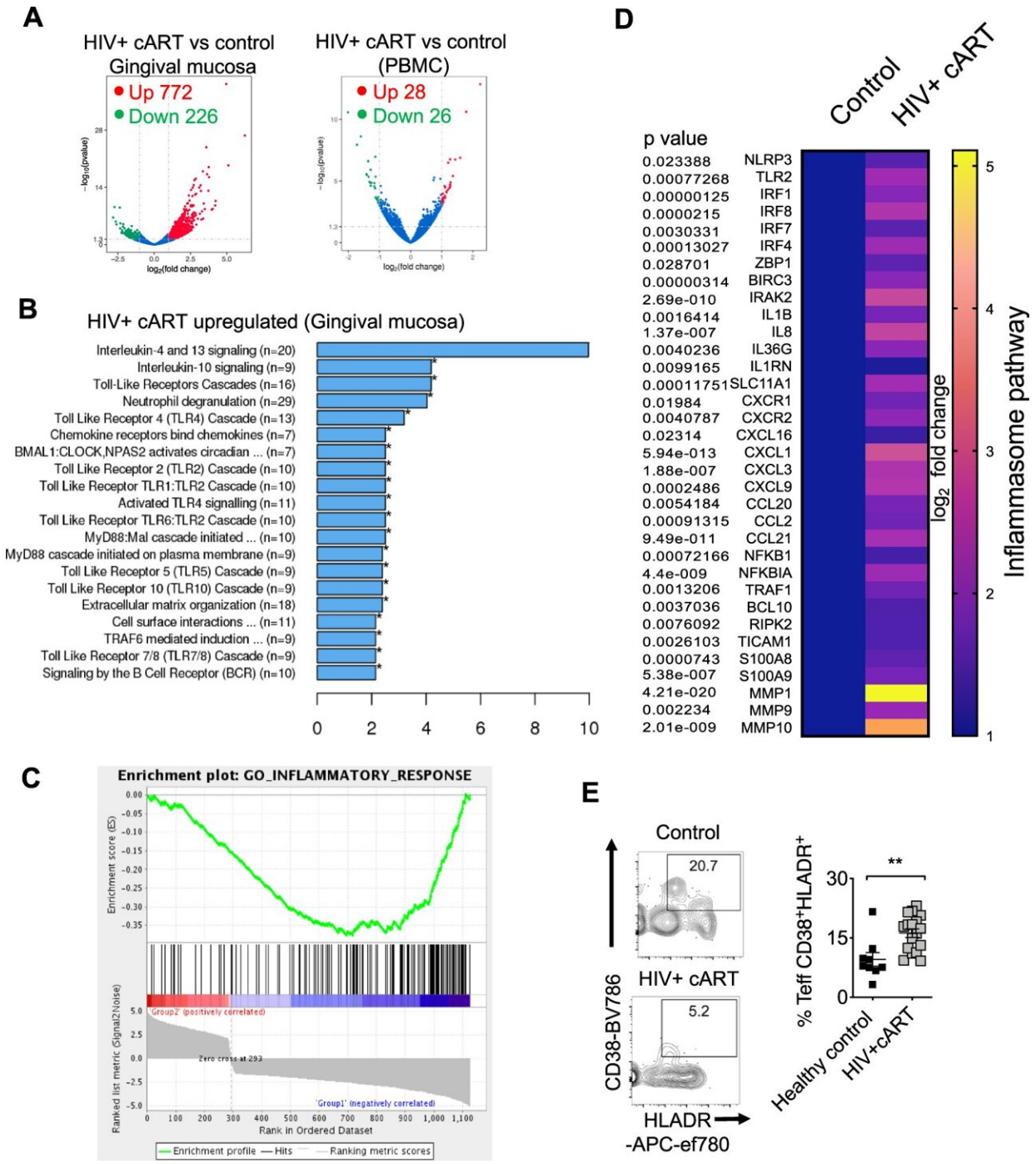

Fig. 1: Transcriptomic profiling and flow cytometric analysis of oral mucosa in HIV+ patients. Forty-six HIV+ patients on cART treatment and 32 uninfected healthy controls were recruited (Supplementary Table 1). RNA sequencing was performed in gingival tissues and PBMCs collected from six randomly chosen age-matched participants; healthy uninfected control (n = 3) and HIV+ cART (n = 3), 2 males and 1 female in each group. Gingival cells were enriched for immune cells by reducing the epithelial cells through gradient centrifugation before transcriptome analyses. Volcano plots showing differential RNA expression in HIV+ cART versus healthy uninfected control groups in gingival mucosa (A, left) and PBMCs (A, right). B REACTOME pathway analysis of the genes upregulated in HIV+ cART gingival mucosa. C Gene set enrich analysis (GSEA) was performed using the GSEA software (Broad Institute; http://www.broad.mit.edu/GSEA) employing the entire gene list generated from transcriptome analyses. This whole gene list was pre-ranked based on T Score and then uploaded to GSEA software. Inflammatory response signature genes were defined based on the gene sets in MSigDB. D Heatmaps showing upregulation of inflammasome signature genes that were defined based on the published literature. Human oral intraepithelial and lamina propria leukocytes (HOILs) from gingival biopsies were processed for flow cytometry. E Effector CD4 cells were gated as shown in Supplementary Fig. 1B and further on FOXP3-negative population. Contour plots (left) and statistics (right) showing the percentage of activated (CD38+ and HLADR+) effector CD4+ cells (n = 20); mean value ± SEM are plotted. (**P = 0.0029; two-tailed; Mann–Whitney test). Source data are provided as a Source data file. Credit: DOI: 10.1038/s41467-021-25340-w

Pushpa Pandiyan, associate professor of biological sciences in the School of Dental Medicine, and a team of researchers have been working to discover the cause behind residual systemic inflammation and dysfunction of the oral cavity in people living with HIV. Their study, "Oral immune dysfunction is associated with the expansion of

FOXP3+PD-1+Amphiregulin+ T cells during HIV infection" was recently published in *Nature Communications*.

The team of researchers found that part of the dysfunction of the oral cavity in HIV-positive individuals who are taking antiretroviral drugs—medications that prevent HIV from spreading throughout the body—is due to the fact that certain cells of the immune system called regulatory T cells (Tregs), that normally dampen inflammation, are dysfunctional. As a result, the Tregs are not reducing inflammation and through the persistent dysfunction are causing these individuals to become more vulnerable to other diseases (such as periodontal disease) and possibly viral and fungal infections.

Through these studies, Pandiyan is hoping to find answers on what causes this condition—whether it is a result of chronic HIV and/or the continual use of the antiretroviral drugs.

More information: N. Bhaskaran et al, Oral immune dysfunction is associated with the expansion of FOXP3+PD-1+Amphiregulin+ T cells during HIV infection, *Nature Communications* (2021). DOI: 10.1038/s41467-021-25340-w

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