

THE IMPACT OF HIV ON THE ORAL MICROBIOME OF CHILDREN AND ITS RELATION TO TOOTH DECAY

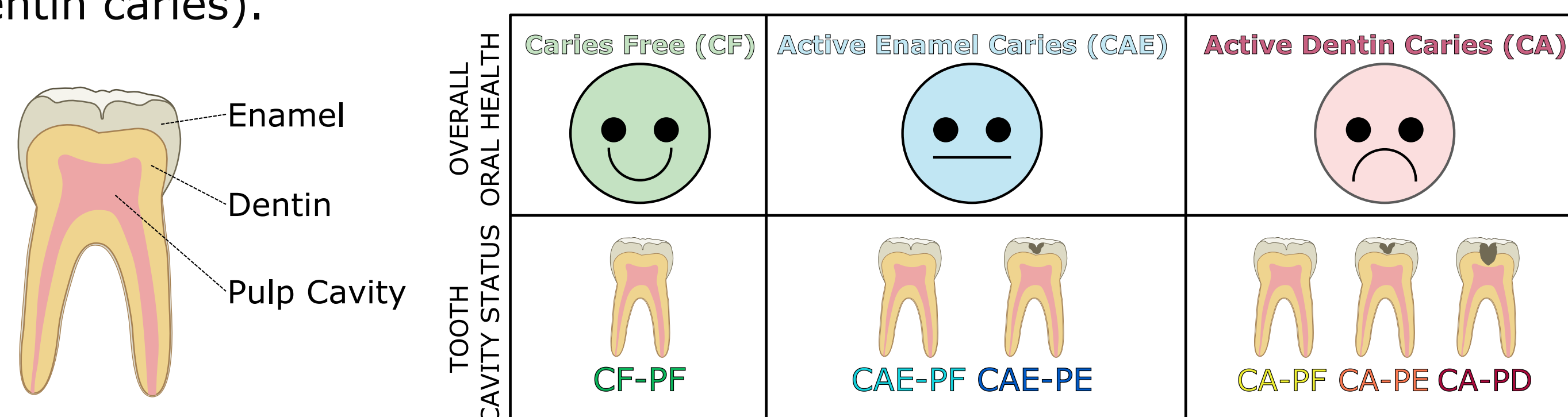
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INTRODUCTION

- Tooth decay (dental caries) is the most common chronic oral disease, affecting ~2 billion people worldwide, 520 million of which are children.
- Leads to poor nutrition & educational outcomes, delayed speech development, overall lower quality of life.
- Etiology of caries typically involves proliferation of acidogenic/acidophilic bacteria (e.g., *Streptococcus mutans*).
- Children living with HIV (CLWH) have a high prevalence of oral diseases including tooth decay (cavities), the mechanisms for which are not well understood

METHODS

- Supragingival plaque collected from 484 children representing 748 individual teeth
- Children living with HIV (HI: n=295); Exposed but uninfected children (HEU: n=224); Unexposed and uninfected children (HUU: n=230)
- Samples categorized by **tooth cavity status** (PF: Caries free tooth; PE: Active enamel lesion; PD: Active dentin lesion) nested in three **overall oral health categories** (CF: Caries free; CAE: Active enamel caries; CA: Active dentin caries).



- Each sample sequenced with primers targeting the bacterial *rpoC* gene

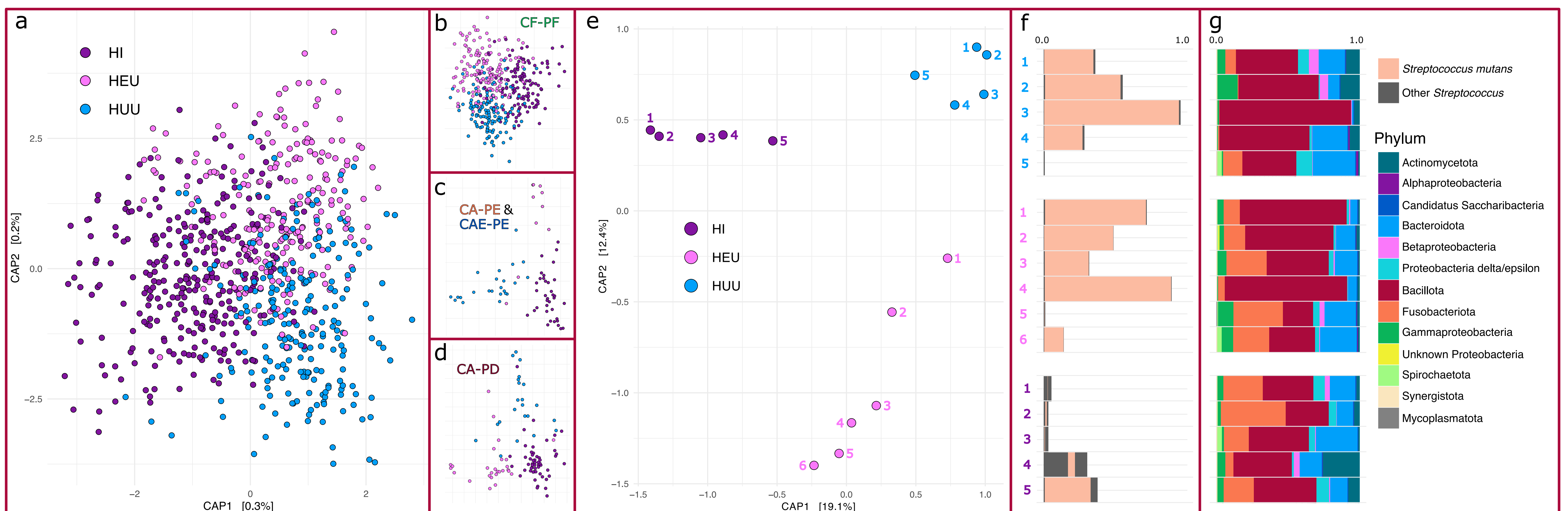
QUESTIONS

(1) Is oral microbiome of children living with HIV (CLWH) different from exposed or uninfected children?

(2) How does tooth health influence impact of HIV?

(3) Is oral microbiome of CLWH more cariogenic?

RESULTS



(a) The oral community of CLWH (HI) is distinct from children exposed to the virus (HEU) and unexposed children (HUU) and (b) this difference is more pronounced in later stage caries (CA-PE, CAE-PE, CA-PD) as opposed to healthy teeth (CF-PF). (e) Intra-individual beta diversity of multiple supragingival plaque samples collected from CA-PD teeth from three individuals (one of each HIV category, HI: n=5, HEU: n=6, HUU: n=5). Community diversity between teeth of the same health category collected from the same individual can vary substantially, largely driven by the proportion of *Streptococcus mutans* (f & g). Contrary to our expectations, CA-PD teeth sampled from CLWH tend to have a lower proportion of both those bacterial groups typically classified as commensals as well as those associated with caries development (e.g., *S. mutans*). Barcharts represented in panels f and g correspond to numbered samples in CAP plot (e)

DISCUSSION

The oral microbiome of CLWH is distinct as compared to HEU or HUU children, and this distinction is more pronounced in diseased teeth. We did not detect an increase in the acidophilic bacteria associated with cariogenesis in CA-PD teeth sampled from CLWH. Instead, CA-PD teeth from CLWH tend to be depleted of both common oral symbionts and those associated with the progression of tooth decay. However, we also document substantial inter- and intraindividual variation in the oral community independent of HIV infection status. Because plaque samples collected at a single time point represent only a brief ecological snapshot of the oral community, it is possible that the frequency of *S. mutans* on a single tooth is a function of a shifting microbial ecology during caries intensification wherein the population of *S. mutans* rapidly expands, lowering the surrounding pH until only itself and other acidophilic bacteria can thrive followed by ecological collapse of the microbial community which is then repopulated. Our results highlight the taxonomic and spatial diversity of the oral community in health and disease and suggest that small-scale ecological (and potentially temporal) changes are responsible for the pathogenesis of caries in CLWH. Given the co-morbidity of HIV and tooth decay, a better understanding of the mechanisms underpinning microbial community dynamics may provide targeted avenues of intervention for caries progression in CLWH.