microba

WHITE PAPER

Microba's Oral Species Index

What it is

The Oral Species Index (OSI) is a summary marker that captures the overall presence and abundance of oral-origin microbes detected in a stool metagenome. Oral-origin species are defined as species predominantly found in the oral cavity, and are informed by the Human Oral Microbiome Database (HOMD)1,2. The OSI is calculated as the sum of the square root transformed relative abundances of species classified as oral.

Why square-root? This transformation adjusts the contribution of individual species to the index so that it reflects both the diversity and abundance of oral species. It reduces the dominance of highly abundant species while giving more weight to those with lower abundance. Compared to untransformed abundances, the index places more emphasis on evenness, whilst remaining more sensitive to highly abundant species compared to standard diversity metrics. This transformation also helps deal with issues of compositionality that arise when analysing relative abundance data.

Why we developed it

An increasing body of research links a higher burden of oral species in the gut with intestinal inflammation. By consolidating these signals into a single metric, the OSI helps clinicians quickly assess whether oral–gut translocation may be contributing to a patient's pro-inflammatory microbiome picture. The OSI is reported within the Intestinal Inflammation category.

Evidence linking oral species with intestinal inflammation

A growing literature shows that oral-origin microbes appear more often in the gut microbiomes of people with intestinal inflammatory conditions than in healthy controls. In both inflammatory bowel disease and colorectal cancer cohorts, studies consistently report higher detection and relative abundance of oral taxa in stool metagenomes3,4. This signal also tracks with measures of objective inflammation: in a population study of more than a thousand participants with paired metagenomics and biomarkers, faecal calprotectin levels were positively correlated with the relative abundance of oral species5. Notably, this association persisted even after excluding proton pump inhibitor (PPI) users—a key confounder because PPIs facilitate oral–gut microbial translocation—supporting a link between intestinal inflammation and oral species beyond medication effects.

Our internal analyses mirror these findings. Across Microba's dataset, the OSI was significantly higher in participants reporting inflammatory bowel disease, recent gastrointestinal infection, diverticulitis, coeliac disease, or cancer compared to healthy controls (Figure 1A). These effects were robust to adjustment for common confounders, including age, gender, BMI, and stool form (Bristol Stool Scale), indicating that elevated OSI aligns with disease status rather than basic demographic or stool consistency differences. We also observed a statistically significant association with the faecal biomarker calprotectin; individuals with faecal calprotectin >100 µg/g (the conventional "high" threshold) had higher OSI values than those at or below 100 µg/g (Figure 2B).

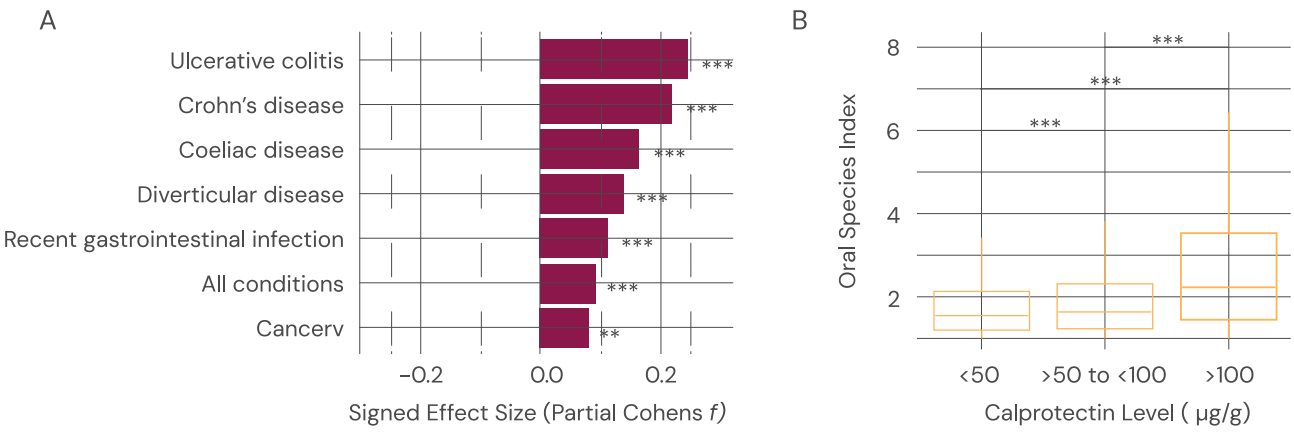

Figure 1. The association of the Oral Species Index with disease (A) and faecal calprotectin levels (B), using internal data from Microba. A) Signed effect sizes (Cohen's f) for associations between the Oral Species Index and disease. Each bar represents the effect size from an ANOVA comparing individuals with each disease (cancer n = 825, coeliac disease n = 113, Crohn's disease n = 113, diverticular disease n = 157, recent gastrointestinal infection n = 713, ulcerative colitis n = 149, all conditions n = 1897) to healthy controls (n = 537), adjusting for age, gender, BMI and stool form. B) The association between the Oral Species Index and faecal calprotectin levels (n ≤50 = 6546, n >50 to ≤100 = 1886, n >100 = 1156), tested using Wilcoxon's rank sum. * = FDR < 0.05, ** = FDR < 0.01, *** = FDR < 0.001.

Mechanistic context (association vs causation)

An elevated presence of oral species in stool is likely a consequence of an inflamed or otherwise permissive intestinal environment. Inflammation can impair intestinal barrier integrity, alter luminal oxygen gradients, and change bile acid profiles6–8—conditions that may collectively make it easier for oral bacteria to survive gastric transit and establish at least transiently in the gut. Importantly, oral species are not uniformly pro- or anti-inflammatory. For example, one of the most common oral species detected in the gut, Streptococcus salivarius, has demonstrated the capacity to inhibit inflammatory responses in cellular and animal models9,10. In contrast, another oral species Streptococcus anginosus, can promote inflammatory responses11. These contrasting behaviours suggest that oral species function more as markers and potential amplifiers of an existing inflammatory state rather than as a singular initiating cause. Clinically, this means a higher OSI should prompt evaluation of inflammatory drivers and barrier function, rather than being interpreted as a standalone causal agent.

Why oral species rise in some people

Several common clinical factors can elevate the OSI in ways that intersect with, but are not limited to, intestinal inflammation. Proton-pump inhibitors reduce gastric acidity and weaken a key gatekeeper against oral-to-gut microbial passage; multiple studies have shown that PPI exposure increases oral bacterial transmission and shifts gut community structure12–15. Oral health also matters: periodontal disease and high levels of dental plaque have been associated with increased detection of oral taxa in stool16,17. When OSI is elevated, it is therefore useful to review medication history—particularly PPI use—and oral health alongside standard assessments of intestinal inflammation.

References

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