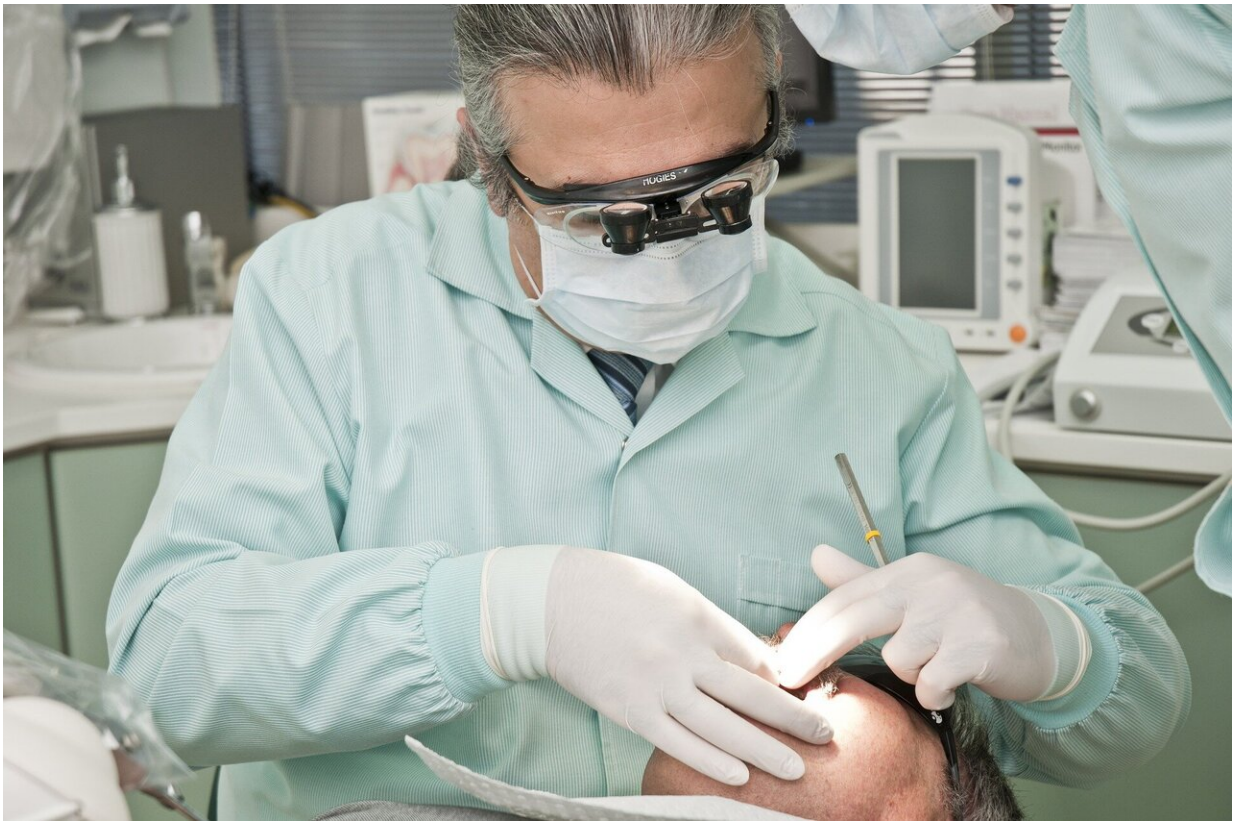


How gum disease aggravates chronic obstructive pulmonary disease

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Severe gum disease has been linked to the progression of chronic obstructive pulmonary disease, or COPD, but an understanding of how the connection plays out in the immune system remains unclear. This

week in *mSystems*, a [new study](#) identifies immune system cells that play a critical role in the microbial link between COPD and gum disease.

Researchers from Sichuan University, in China, report that bacteria associated with the [gum disease](#) promote COPD through the activation of two types of cells, $\gamma\delta$ T cells and M2 macrophages, that are important to the [immune system](#). Focusing on this mechanism might offer new, practical strategies for COPD prevention or control, said the researchers, from the West China Hospital of Stomatology at Sichuan University.

"By enhancing periodontal therapy and targeting the inhibition of $\gamma\delta$ T cells and M2 macrophages [we] may be able to help control the progression of COPD," said microbiologist Boyu Tang, Ph.D., who led the study with microbiologist Yan Li, Ph.D.

According to the World Health Organization, COPD is the sixth-leading cause of death worldwide. It is not curable. In higher-income countries, tobacco smoking is the leading cause of COPD; in low- and [middle-income countries](#), both [tobacco smoking](#) and household air pollution are significant risk factors.

Periodontitis is a gum disease that results from the untreated buildup of plaque, a sticky film made primarily of bacteria. Over time, the plaque can harden into tartar and cause irritation and inflammation of gum tissue, and then produce deep gaps between the teeth and gums where bacteria flourish and may lead to bone loss. Periodontitis is a chronic infectious disease, and previous studies have found that it's a risk factor for a raft of diseases, including diabetes, hypertension, some cancers, cardiovascular disease and COPD.

Previous studies, including some led by Li and Tang, have established that the oral bacteria *Porphyromonas gingivalis* plays an important role in gum disease. For the new work, Li, Tang and their colleagues used

mouse models to show how those bacteria could aggravate progression of COPD. In one experiment, they showed that mice infected with both periodontitis and COPD had worse progression of COPD than mice infected with COPD alone.

In another experiment, they found that in mice orally infected with *P. gingivalis*, the bacteria migrated to and infected lung tissue, leading to a significant, observable change in the lung microbiota. Further observations using flow cytometry and immunofluorescence revealed that periodontitis promoted the expansion of the immune cells in the lung tissue.

Finally, in experiments using mouse [lung tissue](#), the group connected the dots by showing that *P. gingivalis* could activate the immune cells, promoting their ability to produce cytokines associated with worsening COPD.

The researchers noted that the decrease in lung function and increase in immune cells was more modest than they'd predicted, but that could be an artifact of the experimental setup. The team created COPD animal models using exposure to cigarette smoke.

"If the [cigarette smoke](#) exposure could be extended for a longer period of time, these changes might be more pronounced," Li said. In future studies, Li said that the group plans to investigate how increases in smoke exposure might affect the immune response.

"We'll further carry out additional studies on human subjects to confirm the mechanism," Li said. They plan to recruit patients with both conditions and offer periodontitis treatment, then compare lung function and immune cell counts before and after. "Our finding could lead to a potential new strategy for treating COPD."

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