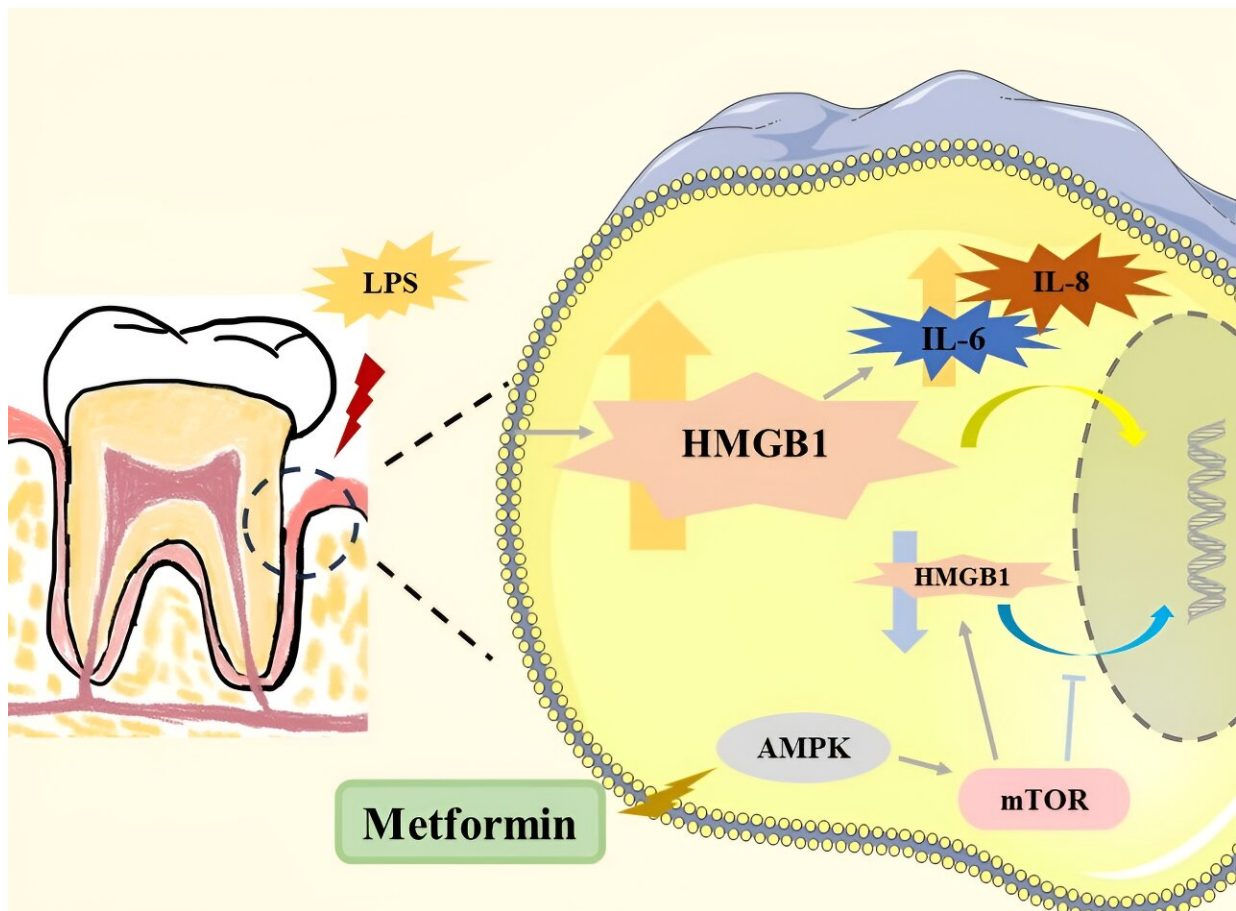


# New study explores the role of metformin in ameliorating oxidative stress in periodontitis

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Periodontitis increase the expression of proinflammatory cytokine HMGB1. HMGB1 affects the expression of inflammation-related cytokines (IL-6, IL-8) and aggravates periodontal tissue destruction and oxidative stress. Metformin activates autophagy through mTOR pathway, regulate the expression, translocation and release of HMGB1, and then reduce the oxidative stress and tissue destruction of periodontal tissue. Credit: *Genes & Diseases* (2021). DOI:

Periodontitis, a prevalent global health concern, results in the gradual destruction of tooth-supporting tissues and is often exacerbated by oxidative stress conditions and bacterial changes. Current treatment methodologies include mechanical debridement, anti-inflammatory drugs, and regenerative surgery.

Researchers conducted detailed examinations on [cellular interactions](#) with metformin, investigating its role in relation to HMGB1 during the progression of [periodontitis](#). It was observed that metformin could inhibit [oxidative stress](#), and activate autophagy via the AMPK/mTOR pathway. Experimental periodontitis was induced in a murine model, and Metformin was found to attenuate alveolar bone resorption, a major hallmark of the disease.

A notable discovery from this study is that metformin not only decreases oxidative stress in periodontal ligament cells but also activates autophagy, a cellular waste removal and recycling process, through the AMPK/mTOR pathway. This pathway has been shown to inhibit HMGB1-mediated oxidative stress in periodontal tissues.

The study concludes that metformin can mitigate periodontal tissue damage through its anti-inflammatory effects, which involve reducing the expression and translocation of HMGB1, a key pro-inflammatory factor. These results align with previous studies demonstrating metformin's capabilities in reducing oxidative stress. The research is published in the journal *Genes & Diseases*.

HMGB1's pivotal role in inducing oxidative stress in periodontal cells adds further credence to this protein being a potential target for

periodontal intervention. This opens up a promising avenue for future research and the development of drugs aimed at HMGB1.

"While our study presents compelling evidence on the protective role of metformin in periodontitis, further research is needed," the research team stated. "We are optimistic that our findings will pave the way for more comprehensive studies on the relationship between metformin and HMGB1 in periodontitis, ultimately leading to more effective treatment options."

The potential role of metformin in managing periodontitis could be transformative, especially for patients suffering from both diabetes and periodontitis. The study's results offer hope for an affordable, well-tolerated, and readily available therapeutic option, building on [metformin](#)'s known antidiabetic effects. This aligns with the broader medical goal of developing targeted treatments that manage the disease's symptoms while also addressing its root cause.

**More information:** Boyang Sun et al, Metformin ameliorates HMGB1-mediated oxidative stress through mTOR pathway in experimental periodontitis, *Genes & Diseases* (2021). [DOI: 10.1016/j.gendis.2021.06.003](https://doi.org/10.1016/j.gendis.2021.06.003)

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