Hydrogen Peroxide Keeps Gut Bacteria Away From the Colon Lining

Neuroscience News 1 day ago

Summary: *An enzyme in the colon lining releases hydrogen peroxide to help protect the body from gut microbes.*

Source: *UC Davis*

Scientists at UC Davis Health have discovered that an enzyme in the colon lining releases hydrogen peroxide(H_2O_2) – a known disinfecting compound- to protect the body from gut microbes. Their study, published Dec. 9 in the journal *Cell Host and Microbe*, sheds light on the way microorganisms are spatially organized in the colon. It also calls for a new approach to treating gut inflammation.

Most microbes reside in the large intestine, a naturally low-oxygen environment. They form a community called the gut microbiota.

the study.

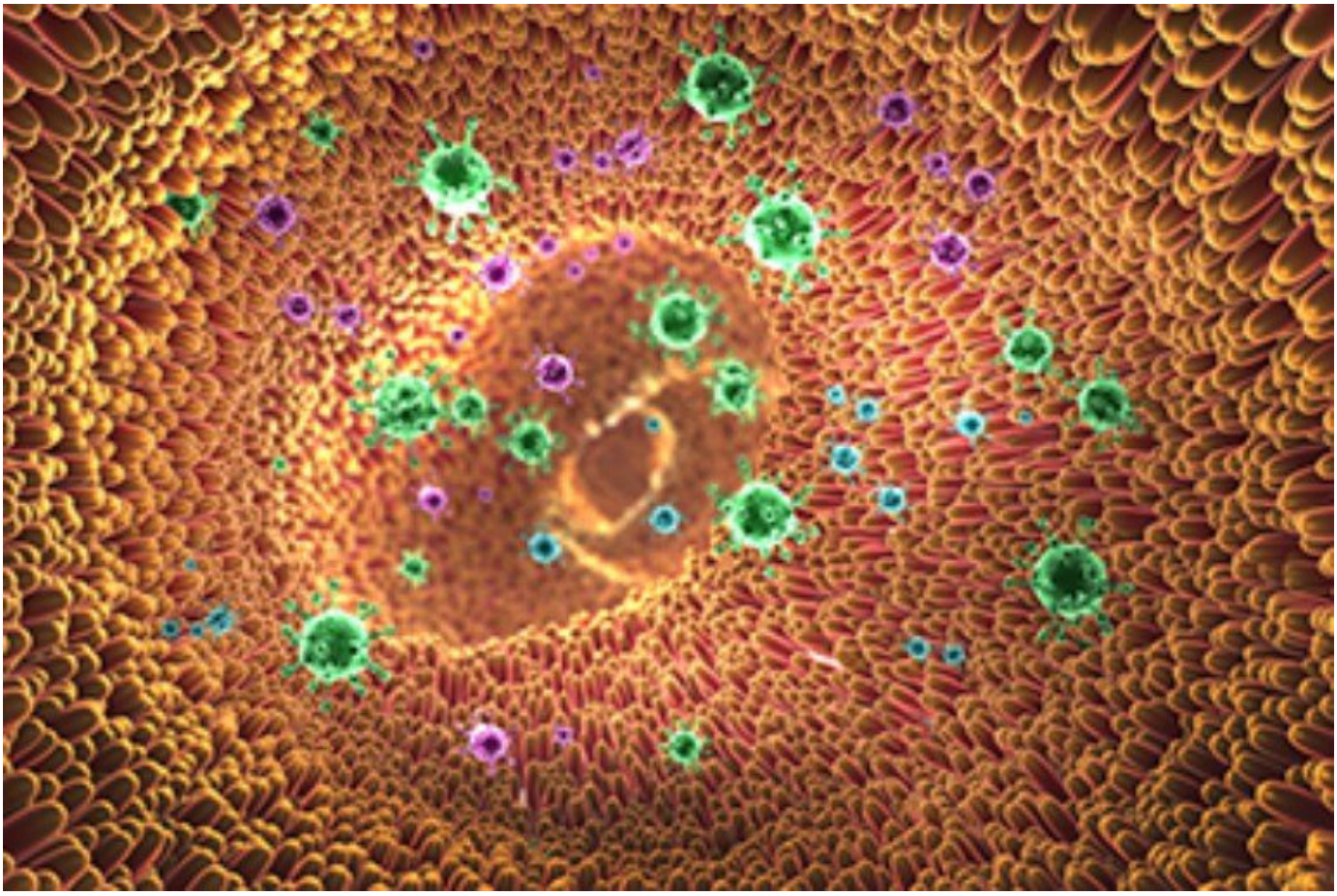
The gut microbiota is kept away from the colon's surface. This separation is essential to avoid inflammation caused by unnecessary immune responses to gut microbes. Scientists believed the spatial separation is maintained by oxygen released by cells to prevent microbes from coming too close to the intestinal lining. This study upends that theory.

"We looked at the spatial relationships between the bacteria in the gut and its host, the colon," Bäumler said. "We found that cells in the colon's lining release hydrogen peroxide- not oxygen- to limit microbial growth."

NOX1, an enzyme found in the intestinal lining, provides a significant source of H_2O_2 in the colon. The naturally generated H_2O_2 serves as a filter regulating the location of the microbiota in the colon. Pathogens that utilize hydrogen peroxide can only do so when they are directly attached to the intestinal lining. This finding suggests that the body uses the disinfectant to protect the mucosal surface. Meanwhile, the microbial communities at a distance from the colon surface remain unharmed.

Treating gut inflammation with natural filter restoration, not antibiotics

When the body experiences an imbalance in the gut microbial community, it suffers from dysbiosis, a gastrointestinal condition. Dysbiosis may cause inflammation and symptoms such as nausea, upset stomach and bloating. Traditional treatments of dysbiosis rely mainly on



The colon lining releases H2O2 to protect itself from gut microbes. Credit: UC Davis

Findings from the new study indicate the need for a different approach to treating gut inflammation and dysbiosis. They pointed to the opportunity of restoring host functions instead of eliminating microbes.

“We need to shift the focus of gut inflammation treatments from targeting bacteria to fixing habitat filters of the host and restoring their functionality,” Bäumler said.

Co-authors on this study are Brittany M. Miller, Megan J. Liou, Lillian F. Zhang, Henry Nguyen, Yael Litvak, Eva-Magdalena Schorr, Kyung Ku Jang, Connor R. Tiffany and Brian P. Butler.

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Source: UC Davis

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"Anaerobic Respiration of NOX1-Derived Hydrogen Peroxide Licenses Bacterial Growth at the Colonic Surface" by Miller et al. *Cell Host and Microbe*

Abstract

Anaerobic Respiration of NOX1-Derived Hydrogen Peroxide Licenses Bacterial Growth at the Colonic Surface

- •Hydrogen peroxide drives *Citrobacter* growth in the non-inflamed gut
- •*Citrobacter* requires intimate epithelial attachment to respire hydrogen peroxide
- •Anaerobic hydrogen peroxide respiration by *Citrobacter* is*NOX1*-dependent
- •Epithelial –derived hydrogen peroxide shapes the epithelial surface environment

Summary

The colonic microbiota exhibits cross-sectional heterogeneity, but the mechanisms that govern its spatial organization remain incompletely understood. Here we used *Citrobacter rodentium*, a pathogen that colonizes the colonic surface, to identify microbial traits that license growth and survival in this spatial niche. Previous work showed that during colonic crypt hyperplasia, type III secretion system (T3SS)-mediated intimate epithelial attachment provides *C. rodentium* with oxygen for aerobic respiration. However, we find that prior to the development of colonic crypt hyperplasia, T3SS-mediated intimate attachment is not required for aerobic respiration but for hydrogen peroxide (H_2O_2) respiration using cytochrome *c* peroxidase (Ccp). The epithelial NADPH oxidase NOX1 is the primary source of luminal H_2O_2 early after *C. rodentium* infection and is required for Ccp-dependent growth. Our results suggest that NOX1-derived H_2O_2 is a resource that governs bacterial growth and survival in close proximity to the mucosal surface during gut homeostasis.

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