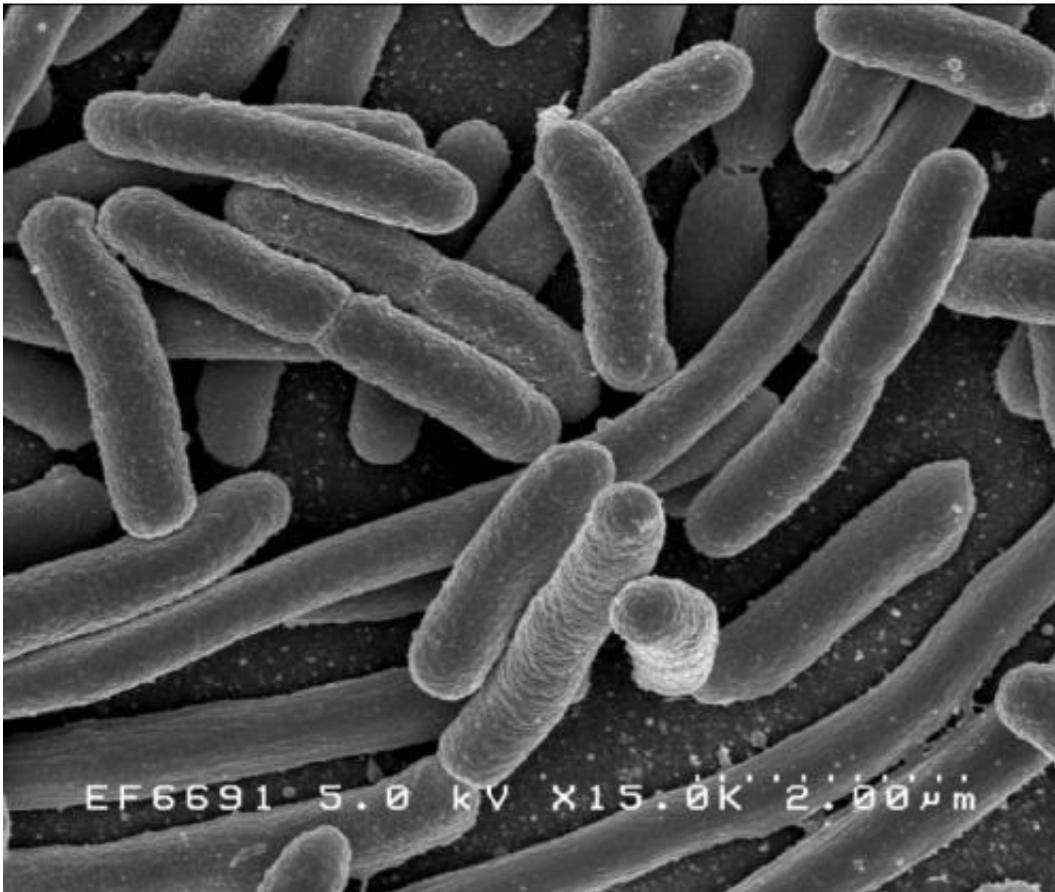


***E. coli* bacterium key for boosting iron absorption, new study shows**

August 23 2018



Escherichia coli. Credit: Rocky Mountain Laboratories, NIAID, NIH

Best known as a pathogen that causes food poisoning or steals nutrients away from its host, the *E. coli* bacterium actually plays a critical role in promoting health by producing a compound that helps cells take up iron,

new University of Colorado Boulder research shows.

The study, published today in the journal *Cell*, sheds new light on the mechanism by which *E. coli* - the most prevalent bacterium in the human gut—benefits its host and could ultimately lead to more effective therapies for [iron](#) deficiency anemia, which impacts more than 1 billion people worldwide.

"In recent years, we have begun to realize that many microorganisms populating the [human gastrointestinal tract](#) are good for us, but we are only beginning to discover exactly what benefits they offer and how," said senior author Min Han, a professor in CU Boulder's Department of Molecular, Cellular and Developmental Biology (MCDB). "This new finding identifies one key role of *E. coli* and that is to help [cells](#) absorb iron."

While some strains of *E. coli* can cause illness, most are harmless and the human gastrointestinal tract is crawling with them.

Because *E. coli* is so prevalent, Han and his colleague Bin Qi, a research associate in his lab, presumed it played beneficial roles and set out to identify which compounds it produces and their functions.

They started with a series of experiments using *C. elegans*, a roundworm naturally rich in *E. coli*. When young worms were fed *E. coli* genetically altered to lack the ability to produce a compound called enterobactin, they grew slowly and their [iron levels](#) were low. When enterobactin was re-introduced into the worms, natural growth resumed and iron levels rose.

Subsequent experiments in worms and human cells showed that supplementing the diet with enterobactin, even without the addition of iron, prominently boosted iron levels in cells.

Scientists have long known that *E. coli* produces enterobactin to scavenge iron for its own survival and propagation. But they presumed that in doing so, it stole iron from its host in what is often referred to as an "iron tug of war."

The new study challenges that paradigm, suggesting that enterobactin also binds to a protein called ATP synthase inside the host's mitochondria, drawing iron into the host's cells too.

"While bacteria make this iron-scavenging compound for their own use, our research suggests that mammals—including humans—have learned over time to hijack it for their own benefit," said Han, an investigator with the Howard Hughes Medical Institute.

Iron deficiency is the most prevalent nutrient-deficiency disorder in the world and a major cause of anemia that impacts one-fourth of the global population, particularly women, children and people in underdeveloped countries.

But Han notes that oral iron supplements, while helpful in some cases, can potentially backfire by suppressing enterobactin production and reducing iron absorption. Oral antibiotics could potentially have the same impact, he says.

Han has filed for a patent for the breakthrough and plans to continue developing new therapeutics that could serve as adjuncts to or replacements for iron supplements.

"Ultimately, we believe this molecule has great potential for addressing [iron deficiency](#) disorders, which are so prevalent," he said. "Studies like ours demonstrate just how host animals are benefiting from the activities of the huge number of microbes in their gut."

More information: *Cell* (2018). [DOI: 10.1016/j.cell.2018.07.032](https://doi.org/10.1016/j.cell.2018.07.032)

Provided by University of Colorado at Boulder

Citation: E. coli bacterium key for boosting iron absorption, new study shows (2018, August 23)
retrieved 19 September 2024 from

<https://phys.org/news/2018-08-coli-bacterium-key-boosting-iron.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.