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New findings shed light on body's iron-absorption process

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Iron is a key mineral for human health. Too much of it in your body — or too little — can lead to major health problems.

University of Florida researchers have discovered that an iron-absorption process thought to happen only within cell membranes can also happen within the interior of iron-deficient rodents' intestinal cells. The finding suggests there is at least one unidentified protein involved in iron absorption, and that it may help maintain proper iron levels in the blood by mediating iron extraction from the diet.

The scientists, led by Institute of Food and Agricultural Sciences faculty member James F. Collins, an associate professor in food science and human nutrition who specializes in molecular and biochemical aspects of nutrition, recently published their findings online in the journal Proceedings of the National Academy of Sciences.

Iron absorption in humans is still a somewhat mysterious process, but Collins said the team hopes its findings can someday be used to alleviate suffering from iron-related health problems.

"Understanding exactly how the process works could reveal therapeutic targets where one could modulate the process of iron absorption," he said. "You could potentially give somebody iron and they would absorb it better or you could block the process in conditions of iron-overload."

From the time you sit down for a nice steak or a big spinach salad, your

body's process for handling and dispersing that iron is a complicated one, to say the least.

Once iron reaches the intestines, it must go through a reduction process so that it can be absorbed by intestinal cells. Then it must be modified again, through a process called oxidation, so that it can bind to a protein in the blood plasma called transferrin, which delivers the iron to cells throughout the body.

A protein called hephaestin that is contained in intestinal cell membranes is part of the oxidation process that allows iron to be transported into the circulatory system.

But the mutant mice the scientists studied didn't have hephaestin, yet they were able to survive and had only mild iron deficiency indicating that hephaestin was important but not absolutely necessary for iron absorption.

"So we hypothesized that there's something else going on there. And that's what we've done in this paper, we've described an activity – we can't ascribe it to a single protein yet – so we've described another potentially important player in this process," Collins said.

Greg Anderson of the Queensland Institute of Medical Research and a widely known expert in iron metabolism, was not part of the study, although one of his graduate students is one of the paper's co-authors.

"The implications of this work are considerable," he wrote in an email. "The Collins work helps us to understand the main regulatory step in iron absorption, the movement of iron from the cells lining the small intestine and into the circulatory system. If we can understand this process in detail, we may be able to design therapeutic interventions that can alter how much iron you absorb, by either decreasing it or increasing it."

Collins said his lab will continue the National Institutes of Health-funded study, by working to separate and identify proteins in intestinal cells, track their activity, and pinpoint the one that is doing the iron-absorption work in mice lacking hephaestin.

The research team included Perungavur Ranganathan, a biochemist and IFAS research assistant scientist (whom Collins credited as making the observation that led to their initial hypothesis); Yan Lu, a UF doctoral student in nutritional sciences, and Brie Fuqua, a doctoral student at the Queensland Institute of Medical Research in Brisbane, Australia.

Provided by University of Florida