

Cells pumping iron to prevent anemia

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Kyoto University researchers identify Regnase-1, a gene previously thought to prevent autoimmune disease, as a key regulator in iron metabolism. Credit: Kyoto University

Maintaining a good balance of iron in the body is necessary for health: too little can lead to anemia, but too much can cause debilitating disorders such as *hemochromatosis*.

The uptake and metabolism of [iron](#) in the body is one of the most tightly-controlled systems in mammals. Iron 'regulation' occurs at multiple levels, from controlling gene expression to degrees of [protein synthesis](#), but some key factors have yet to be uncovered.

As described recently in *Cell Reports*, Kyoto University researchers have now identified a specific gene—initially known to prevent autoimmune diseases—as a key regulator in iron uptake.

"We found previously that when mice lack the gene *Regnase-1* they suffer from severe autoimmune diseases and anemia," explains first author Masanori Yoshinaga.

"At first, we assumed that anemia was a secondary effect, but after detailed analysis we found that the two symptoms develop independently."

Continued study of mice with a *Regnase-1* mutation revealed a functional defect in the principle site for iron absorption in the body, the *duodenum*, which is the first section of the small intestine, coming directly after the stomach.

"The next step was to find the role of *Regnase-1* in iron-uptake maintenance. We started by looking at the most important iron-uptake gene, Transferrin Receptor 1, or *TfR1*," continues Yoshinaga.

"Our results showed that *Regnase-1* degrades the mRNA of *TfR1*, thereby inhibiting the synthesis of the *TfR1* protein, and additionally that it likely regulates other important iron-controlling [genes](#)."

"Further analysis of *Regnase-1* in iron-related homeostasis," concludes team leader Osamu Takeuchi, "may provide insight into the mechanisms causing [anemia](#) and other iron-related disorders, perhaps eventually

leading to new methods of treatment."

More information: Masanori Yoshinaga et al, Regnase-1 Maintains Iron Homeostasis via the Degradation of Transferrin Receptor 1 and Prolyl-Hydroxylase-Domain-Containing Protein 3 mRNAs, *Cell Reports* (2017). [DOI: 10.1016/j.celrep.2017.05.009](https://doi.org/10.1016/j.celrep.2017.05.009)

Provided by Kyoto University

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