

# SLC22A17 binds LCN2:2,5DHBA:Fe<sup>3+</sup>

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## Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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## Literature references

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Reactome database release: 88

This document contains 1 reaction ([see Table of Contents](#))

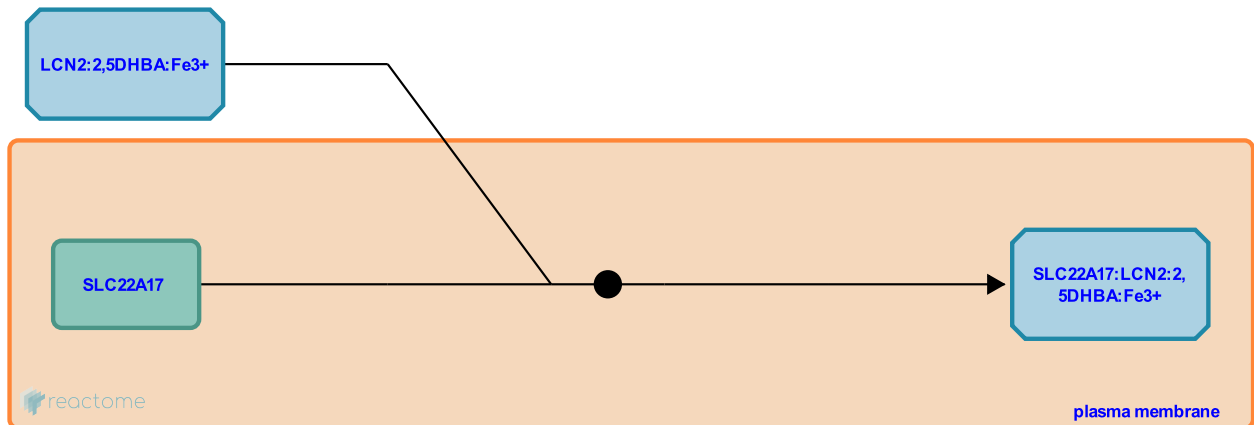
## SLC22A17 binds LCN2:2,5DHBA:Fe3+ ↗

**Stable identifier:** R-HSA-5246444

**Type:** binding

**Compartments:** plasma membrane, extracellular region, cytosol

**Inferred from:** [Slc22a17 binds Lcn2, internalising it, releasing Fe3+ \(Mus musculus\)](#)



Neutrophil gelatinase-associated lipocalin (LCN2, NGAL) is a member of the lipocalin superfamily that is involved in iron trafficking both in and out of cells (Goetz et al. 2002). LCN2 binds iron through association with 2,5-dihydroxybenzoic acid (2,5DHBA), a siderophore that shares structural similarities with bacterial enterobactin, and delivers or removes iron from the cell, depending on the context. The iron-bound form of LCN2 (holo-LCN2) is internalised following binding to the solute carrier family 22 member 17 (SLC22A17) receptor, leading to release of iron which increases intracellular iron concentration and subsequent inhibition of apoptosis. This step is inferred from experiments using the highly homologous 24p3 mouse lipocalin and 24p3R mouse cell surface receptor (Devireddy et al. 2005). During infection, bacteria scavenge iron from the host cell and transfer it to the pathogen cell. Upon encountering invading bacteria, Toll-like receptors on immune cells can stimulate the transcription, translation and secretion of LCN2. LCN2 can then limit bacterial growth by sequestering the iron-laden siderophore so this event is pivotal in the innate immune response to bacterial infection (Flo et al. 2004).

### Literature references

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### Editions

2014-01-21	Authored, Edited	Jassal, B.
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