

P007 The role of DcytB in intestinal iron absorption
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Absorption of dietary iron is a highly regulated process and is the major determinant of body iron stores. Ferric reduction is essential for transport of the metal via intestinal DMT1 which transports only ferrous iron. Duodenal DcytB mRNA and protein levels are highly upregulated by iron deficiency and in iron overload diseases, where iron absorption is increased. We set out to fully characterise the impact of loss of DcytB in DcytB knock-out mice on intestinal iron absorption. Reductase activity was measured in DcytB KO mice after treatment with hypoxia or iron deficiency to increase the body iron demand. In contrast to wild type mice which show three to four fold increases in all cases, there is no change in duodenal reductase activity in the KO mice ($p < 0.0005$, $n = 4$ to 9), suggesting DcytB is the primary iron regulated ferric reductase in the duodenum. The rate of iron absorption was measured *in vivo* in iron-deficient or hypoxic DcytB KO mice using radioactive ^{59}Fe as ferric NTA complex. DcytB KO mice showed significantly lower mucosal retention (measures how much ^{59}Fe is retained in the duodenum) but a higher percentage of mucosal transfer (measures how much ^{59}Fe is exported out of the duodenum into the body). Overall this data suggests that iron uptake into the mucosa from the diet may be lower in DcytB KO mice and that there are compensatory changes in transfer of iron via Ireg1 as a result of changes in body iron stores.