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The effect of quercetin on intestinal iron transporter expression

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Iron (Fe) is an essential metal required by all organisms. It is crucial in processes such as oxygen binding in haemoglobin and various enzymatic reactions. However, when present in excess, Fe can also catalyse highly toxic-free radical production; hence, there is a great need to control body Fe levels. Human subjects have no physiological mechanism for excreting excess Fe and thus Fe homeostasis is maintained primarily by regulating the absorption of dietary Fe by the proximal small intestine[1].

Flavonols are part of a larger group of phenolic compounds collectively known as flavonoids. They are found extensively in the diet and widely used as food additives thus their interaction with Fe is of considerable interest. Quercetin, the most abundant flavonol in the diet, is an effective Fe-chelator and may therefore affect non-haem Fe bioavailability and its subsequent metabolism. The present study has investigated the effects of varying concentrations of quercetin on the expression of genes involved in intestinal Fe transport (*DMT1; Dcytb; ferroportin* and *hephaestin*). Studies were carried out using fully differentiated Caco-2 cells, which were treated with quercetin (0, 0.1, 0.3, 1, 3 and 10 μM) for 24 h. Changes in mRNA expression were measured by quantitative PCR.

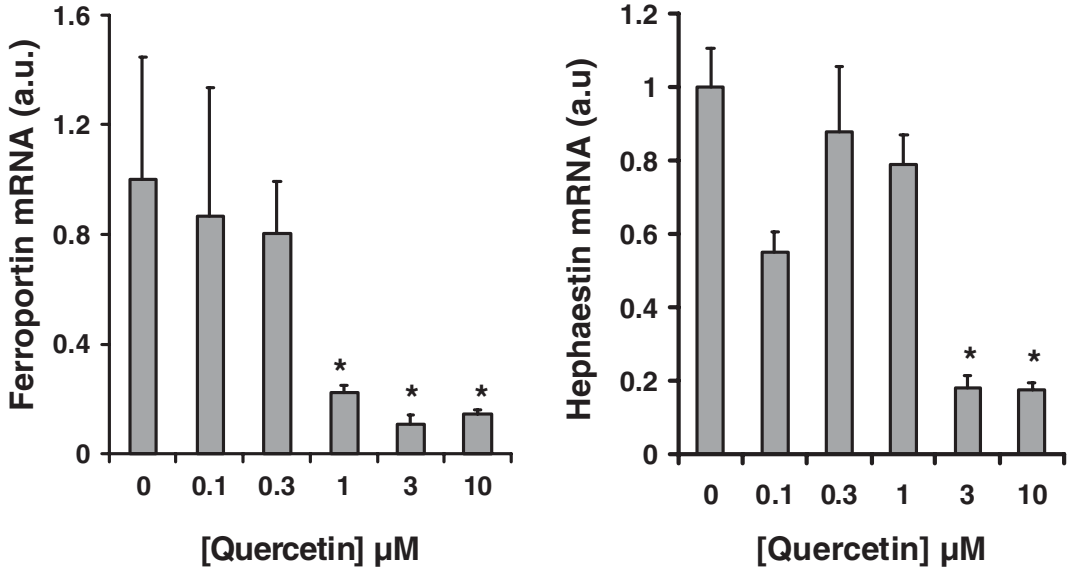

Fig. 1. Effects of quercetin on ferroportin and hephaestin mRNA expression in Caco-2 cells.
*$P<0.05$. Data (mean (SEM)) were analysed by one-way ANOVA and Tukey's post-hoc test's. N 6 in each group

Exposure to quercetin significantly decreased ferroportin and hephaestin mRNA expression. In contrast, the expression of Dcytb and DMT1 was not significantly altered by quercetin. Ferroportin and hephaestin are both expressed at the basolateral membrane of enterocytes[1]. Our findings are consistent with recent data showing that polyphenols inhibit intestinal Fe transport by targeting the basolateral Fe efflux pathway[2].

1. Sharp P & Srai SK (2007) *World J Gastroenterol* **13**, 4716–4724.
2. Kim EY, Ham SK, Shigenaga MK *et al.* (2008) *J Nutr* **138**, 1647–1651.