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Vitamin A modulates the expression of genes involved in iron bioavailability

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Iron bioavailability seems to be regulated by vitamin A (VA) but the molecular events involved in this mechanism are not well understood. It is also known that retinoids mediate most of their function via interaction with retinoid receptors, which act as ligand-activated transcription factors controlling the expression of a number of target genes. Here, we evaluated the VA effects on the modulation of the levels of mRNA encoding proteins involved in the iron bioavailability, whether in the intestinal absorption process or in the liver iron metabolism. The expression of genes involved in iron intestinal absorption (divalent metal transporter 1, duodenal cytochrome B, ferroportin 1 FPN1, and ferritin) were evaluated in vitro by treating Caco-2 cells with retinoic acid or *in vivo* by observing the effects of vitamin A deficiency (VAD) in BALB/C mice. Liver hepcidin and ferritin mRNA levels were upregulated by VAD; however, this condition did not promote any change on the expression of those genes that participate in the iron absorption. Moreover, data from the *in vitro* analysis showed thatVA induced FPN1 gene expression by a hepcidin independent manner. Therefore, the in vivo results support the idea that VAD may not affect iron absorption but would rather affect iron mobilization mechanisms. On the other hand, our results using Caco-2 cells raises the possibility that VA addition to intestinal epithelium may improve iron absorption through the induction of FPN1 gene expression.

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