

# Abstract

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## **Prolactin is an important regulator of intestinal calcium transport.**

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**BACKGROUND:** Prolactin has been shown to stimulate intestinal calcium absorption, increase bone turnover, and reduce renal calcium excretion. The small intestine, which is the sole organ supplying new calcium to the body, intensely expresses mRNAs and proteins of prolactin receptors, especially in the duodenum and jejunum, indicating the intestine as a target tissue of prolactin.

**DISCUSSION:** A number of investigations show that prolactin is able to stimulate the intestinal calcium transport both in vitro and in vivo, whereas bromocriptine, which inhibits pituitary prolactin secretion, antagonizes its actions. In female rats, acute and long-term exposure to high prolactin levels significantly enhances the (i) transcellular active, (ii) solvent drag-induced, and (iii) passive calcium transport occurring in the small intestine. These effects are seen not only in pregnant and lactating animals, but are also observed in non-pregnant and non-lactating animals. Interestingly, young animals are more responsive to prolactin than adults. Prolactin-enhanced calcium absorption gradually diminishes with age, thus suggesting it has an age-dependent mode of action. Although prolactin's effects on calcium absorption are not directly vitamin D-dependent; a certain level of circulating vitamin D may be required for the basal expression of genes related to calcium transport.

**CONCLUSION:** The aforementioned body of evidence supports the hypothesis that prolactin acts as a regulator of calcium homeostasis by controlling the intestinal calcium absorption. Cellular and molecular signal transductions of prolactin in the enterocytes are largely unknown, however, and still require investigation.

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