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Ferritin Intestinal Absorption

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Ferritin as a source of iron was considered. A good iron absorption rate appears in normal rats with an *in vivo* absorption technique. The same absorption appears in iron-deficient animals. The iron stored in intestinal wall is lower in anemic rats than in normal ones, suggesting a higher draw of iron from lumen to blood.

Iron is ordinarily given by mouth as specifically remedial in iron deficiency anemia. Most iron salts are astringents in the alimentary tract producing gastrointestinal irritation (2). Ferritin is an iron containing protein with an important physiological role in the absorption, transport, and storage of iron (3), a role that will not be considered in this paper.

The pourpose here is to test the ferritin as a source of iron. As a protein of high molecular weight (750,000) and iron content up to 20-24 % (1, 9, 10), it would be a good iron source because of two facts: *a*) The liberation of the iron would be gradual, following the lysis of the protein, enhancing absorption due to the low sustained concentration in the lumen (14). *b*) The dammage to mucosal cells by iron at elevate concentration would not be produced, or at least diminished.

Material and Methods

The SOLS-PONZ *in vivo* absorption technique (11) was used in adult male 230-250 g Wistar rats. The animals were anesthetized with ethyl-urethan i.p. The intestinal loops were aproximately 25 cm long with a filling presure of 6-8 cm water and the animal temperature kept physiologically constant. Only one absorption period, lasting 60 minutes, was studied in each animal. The solutions used in the intestinal lumen were: 0.9 % NaCl to previous washing, 1/1,000 ferritin solution in buffer phosphate pH 6 to study the absorption rate, EDTA 0.01 M to wash the loop after the absorption period.

The ferritin was obtained from horse spleen, being the iron proportion 20 mg Fe/ml.

Two lots of rats were used, one with normal rats, the other with anemic rats, considering iron deficiency as a tool to change the absorption rate. The anemia

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Table I. *Amount of iron absorbed and stored in the intestinal wall according to iron deficiency.*
Number of animals per group, 15.

RATS	Iron absorbed γ/cm	Iron stored in wall γ/cm	Sideremia γ/ml	Hematocrit %
Normal	2.329±0.405 *	1.11 ±0.398	157±14	44.6±3.1
Iron-deficient	2.51 ±0.190	0.507±0.300	86± 8	21.8±5.2

* $\pm\sigma$.

was produced by bleeding (1 ml of blood daily during six days). In both groups the absorbed iron and the iron stored in the intestinal wall were determined.

The iron was measured by hydroxylamin-o-phenanthroline method, both in the solution introduced in the intestinal loop, and in the intestinal wall. Because the iron is bounded to protein, a previous treatment with ClH 50 % for solubilization and further pH adjustment with NH_4OH was required. Sideremia was determined by RAMSAY method (7), and the hematocrit through a micro type technique.

Results

Table shows the amount of iron absorbed, iron stored in the intestinal wall, sideremia, and hematocrit, both in normal and iron deficient rats.

Discussion

It is now known that ferrous iron and ferric iron are absorbed equally provided that iron remains in the ionized form (4, 8). Moreover ferric iron is evidently reduced in the intestinal lumen of rats. This explains why these animals utilize oral ferrous and ferric iron equally well (12, 13).

FORTH *et al.* (4) found an absorption of ferrous and ferric iron by rats *in vivo* of 17.9 and 16.7 % amount absorbed in relation to the total dosis. Our results, considering the intestinal loops of an average length of 25 cm, and the volume of ferritin solution (1/1,000) of 5 ml, mean an absorpition of aproximately 50 %. Perhaps the difference could be explained because most of the salts employed, generally at high dosis, produce easily secondary intolerance phenomena impairing absorption. But may be also to the fact that hydroxides are formed dependig on pH, being hydroxides a form insuitable for absorption. In ferritin solution those phenomena could not take place enhancing absorption.

In our results non significant differences in absorption rate appear between normal and iron deficient rats, data in contradiction with those found by WHEBY *et al.* (14). Perhaps the high absorption rate we found could account for the non increased rate when the rat need more iron supply.

Respect the iron stored in the intestinal wall — iron that sooner or later enters the blood —, it may be seen that the kinetics is different for normal or iron-deficient rats. As these latter draw iron into blood at higher speed, the iron in the intestinal wall is lowered. We have no explanation for this phenomena because the mechanism of iron intestinal absorption continues to be disputed. HAHN *et al.* (6) and GRANICK (5) proposed the «mucosal block» theory in which the endogenous syntetized ferritin content of the mucosa regulates iron uptake. In contrast WHEBY and CROSBY (15) have suggested regulation occuring between mucosal cell and portal blood. Also several authors

claim to have identified iron carriers (appear to be proteins).

This study merely suggests that ferritin may provide iron which is easily absorbed, without visible damage to the intestinal tract.

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