Glutamine and ketone-body metabolism in the small intestine of starved peak-lactating rats

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Summary — 1. The effect of starvation on the metabolism of gut glutamine and ketone-bodies of peak lactating, non-lactating and virgin rats was investigated.
   2. The arterial blood ketone-body concentration was increased by ~ 7-, 6- and 13-fold in 48 h-starved virgin, non-lactating and lactating rats, respectively.
   3. The arterial blood glutamine concentration was decreased by ~ 32% in 48 h-starved lactating rats (p < 0.001).
   4. The maximal activity of phosphate-dependent glutaminase was increased or decreased in the small intestine of fed or 48 h-starved peak-lactating rats, respectively.
   5. Portal drained viscera blood flow increased by ~ 25% in peak-lactating rats.
   6. Arteriovenous difference measurements for ketone-bodies across the gut of 48 h-starved rats showed an increase in net uptake of ketone-bodies by ~ 10-, 17- and 29-fold in virgin, non-lactating and lactating rats, respectively.
   7. Glutamine was extracted by the gut of peak-lactating rats at a rate of 487 nmol/100 g of body wt. which was greater by ~ 33% (p < 0.001) than that of virgin or non-lactating animals. In peak lactating rats, 48 h-starvation resulted in marked decreases in the rates of glutamine removal from the circulation (p < 0.001) which was accompanied by decreased rates of release of glutamate, alanine and ammonia.
   8. It is concluded that, although the arterial blood concentration of glutamine was decreased in 48 h-starved peak-lactating rats and was accompanied by diminished rates of uptake of glutamine across the gut, such decreases were partially compensated for by an increase in bloodflow and in the extraction rates of ketone-bodies.

glutamine / ketone-bodies / metabolism / glutaminase / peak-lactation / small intestine / starvation

Introduction

In the rat, lactation is associated with hypertrophy and hyperplasia of various tissues of the gastrointestinal tract [1–5]. These investigators demonstrated that the intestinal mucosal changes during lactation are characterized by an increase in the number of proliferating mucosal epithelial cells resulting in the elongation of the crypts together with an increase in the villus height.

Under normal physiological conditions, glutamine is considered to be a major respiratory fuel for the absorptive epithelial cells of the small intestine (for reviews, see [6, 7]). Moreover, during starvation, ketone-bodies provide an important source of energy for the intestinal mucosa together with that of glutamine [7]. Recently, it has been reported that, during peak-lactation in the rat, there was a marked increase in the maximal activity of phosphate-dependent glutaminase (EC 3.5.1.2) (hereafter referred to as glutaminase) in the small intestine [8]. In addition, in vivo and in vitro studies on the intestinal mucosa of peak-lactating rats showed increased utilization and metabolism of glut-