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ROLE OF HYPERPHAGIA IN STRUCTURAL CHANGES OF SMALL INTESTINE DURING LACTATION

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Abstract: The present study was conducted to investigate the cause of the structural changes of small intestine during lactation in albino rats. Anatomical measurements (total length, total wet weight and total dry weight) and histological studies of small intestine were undertaken in virgin control rats, lactating control rats, lactating rats with restricted food intake and lactating rats with restricted litter size. Restriction of food intake prevented the growth of small intestine during lactation, while restriction of litter size had no effect. Results indicate that the structural changes in small intestine are due to work hypertrophy secondary to hyperphagia and not due to any hormonal factors.

Key words: food intake, small intestine, hypertrophy, lactation

INTRODUCTION

The small intestine in rats undergoes marked morphological and functional changes during pregnancy and lactation (1, 2, 3, 4, 5). These changes are very prominent particularly during lactation. There is hypertrophy and hyperplasia of the intestinal mucosal epithelium and the muscle tissues show marked hypertrophy (1). Intestinal absorptive functions have been found to be jeopardised during lactation (2, 4, 6). However, the cause of these structural and functional changes is not clearly known as yet. Fell (1) hypothesized that the hypertrophy and hyperplasia of the small intestine were a consequence of increase in food intake. Datta et al (5) indicated that prolactin might be responsible for functional changes, but the structural changes were not due to the effect of the hormone. In the present investigation attempt has been made to elucidate the mechanism of the structural changes in small intestine during lactation in albino rats.

METHODS

The study was carried out on adult virgin and lactating rats of Charles – Foster strain weighing between 180-220 g. They were divided into four groups – virgin control (group I), lactating control (group II), lactating with restricted food intake (group III) and lactating with restricted litter size (group IV). Lactating animals were sacrificed on day 21 of lactation. The animals were sacrificed on day 21 of lactation. The animals were maintained on Hindustan Lever Rat diet and had free access to water. All the lactating animals were nursing the first litter. For measuring the length, wet weight and dry weight of small intestine, the animal was fasted for 24 hrs prior to the experimentation but had free access to water. The animal was killed by cervical dislocation and the abdomen was opened by a midline incision. The small intestine was stripped off its mesentery and removed from the abdomen. Total length was measured by the method of Barry et al (7). The small intestine was then flushed with warm normal saline to clear of its contents

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and weighed to obtain wet weight. Dry weight of the tissue was determined after drying it in a hot air oven at $110^0 \pm 5^0$C for 3 hrs.

Before restricting the food intake in group III rats, diet consumption was studied in the control and the lactating rats. For this, each animal belonging to either group was given 30 g of diet per day initially. However, the amount of the diet per lactating animal was doubled (60 mg), as the lactating animals are known to consume more amount of diet (2). The dietary consumption was measured by deducting the weight of the diet left behind each day from the weight of the diet given the previous day. Near the weaning period, one pup was taken out at random from each mother and killed for examining any intake of solid food. Pups were separated from the mother on 21st day of lactation. As the average diet consumption was found to vary from 10.4 g to 12.7 g in control rats (Table I), food intake was restricted to 15 g in group III rats throughout the period of lactation.

TABLE I : Food consumption during lactation (Mean ± SEM, n in parentheses).

Group of animal	*Total food consumption (g/day)*		
	Initial consumption (1st day)	*Maximum consumption (19th day)*	*Consumption on last day (21st day)*
Control	12.40±0.74 (6)	11.40±0.36 (6)	11.20±0.46 (6)
Experimental (lactation)	12.60±1.09 (6)	46.80±3.08* (6)	46.10±2.12* (6)

*P < 0.001

To study the histology of the small intestine in different groups of animals, one slice of jejunum was dissected out and collected in 10% neutral formal saline solution from which blocks were prepared and sections were stained by haematoxylin and eosin. The jejunum was chosen because it is the site of maximum hypertrophy and hyperplasia during lactation (1) and having thicker mucosa and longer villi, it has some natural advantages over duodenum and ileum.

Statistical calculations were done following the Students' 't' test.

RESULTS

Results of the anatomical measurements are shown in Table II. It is found that there was significant increase in total length, total wet and dry weight of small intestine in the lactating control (group II) and lactating with restricted litter size group (group IV). But the changes found in lactating with restricted litter size group were not significantly different from the lactating control group. On the other hand, there was no significant change in the anatomical measurements in lactating with restricted food intake group (group III) compared with the virgin control group (group I).

TABLE-II : Anatomical measurements on small intestine in virgin control and different groups of lactating rats (mean ± SEM, n in parentheses).

Group	*Total length (cm)*	*Total wet wt. (mg)*	*Total dry wt. (mg)*
Virgin control (I)	109.7±1.4 (6)	5720±167 (6)	1197±61 (6)
Lactating control (II) Food *ad libitum*, Litter size 8	140.8±1.9* (6)	10508±536* (6)	2031±155* (6)
Lactating with restricted food intake (III) Litter size 8	109.3±1.1 (6)	5399±188 (6)	1143±52 (6)
Lactating with restricted Litter size (IV) Food *ad libitum*, Litter size 4	137.5±0.9* (6)	10403±236* (6)	2105±143* (6)

Histological findings of the jejunum in the lactating control (group II) and lactating with restricted food intake group (group III) are shown in Fig. 1 and 2 respectively. It is seen that there

is remarkable hypertrophy and increase in the height and thickness of the villi in the jejunum in group II rats (Fig. 1), while these changes are absent in group II rats (Fig. 2).

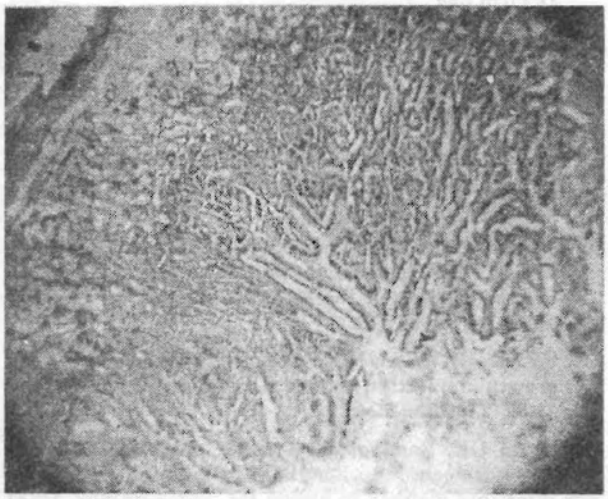

Fig. 1 : Showing hypertrophy of the mucosa during lcactation in photomicrograph of a section of rat jejunum (Haematoxylin and Eosin, X 400).

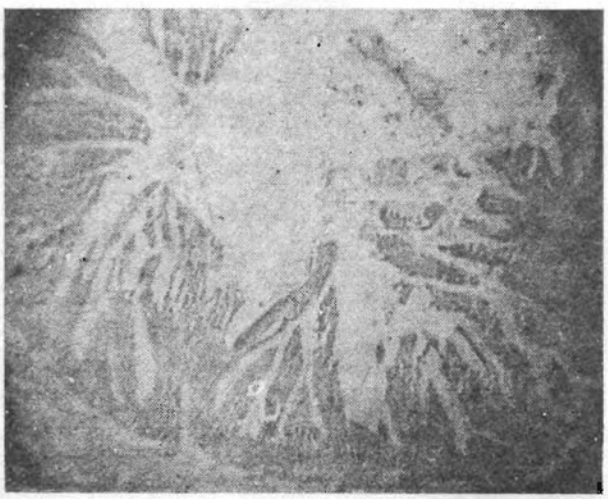

Fig. 2 : Showing no hypertrophy of the mucosa during lactation following restricted food intake in photomicrograph of a section of rat jejunum (Haematoxylin and Eosin, X 400).

DISCUSSION

These results indicate that the hypertrophic changes in the small intestine during lactation is secondary to the increased food intake, as food restriction in the lactating animals prevented and growth of small intestine as evidenced by no significant changes in total length, wet weight, dry weight and histology in these rats. Our results corroborate with the earlier suggestion that gut hypertrophy is secondary to increased food intake and not due to hormonal factors (1, 5, 8).

Prolactin level in the blood rises steadily until parturition and then prolactin secretion is maintained by nursing (9). The secretion of oxytocin is increased as well during parturition and then suckling causes oxytoxin secretion at the same time that it causes prolactin secretion (9). When litter size is restricted with an idea to restrict nursing and lactation and thus to reduce the influence of hormonal factors like prolactin and oxytocin, the small intestine showed hypertrophy as usual. Thus it is evident that prolactin as well as oxytocin may not be responsible for the structural changes. This agrees with the earlier observation that induced hyperprolactinaemia by pituitary grafting and chronic prolactin treatment in virgin female rats failed to produce any structural change in small intestine (5). Absence of any structural changes in lactating with restricted food intake group in otherwise normal rats in experimental undernutrition (10). Profound alterations in the morphology of the gastrointestinal tract are also found in experimentally induced chronic diabetes mellitus in rats (11, 12), which is associated with hyperphagia. Thus it is concluded that the structural changes in small intestine during lactation occurs as a compensatory mechanism secondary to increased food intake to meet the increased demand of energy during lactation (1).

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