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Activity Of Enteral Lactase And Lactobacilli In Maternal And Hereditary Toxic Anemia

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ABSTRACT

In this paper, the activity of lactobacilli in the small intestine and lactobacilli in the colon, which are involved in the digestion of milk sugar during lactotrophy using phenylhydrazine hydrochloride, studied the effects of toxic anemia on mother and child.

Erythrocyte counts, hemoglobin levels, lactase enzyme activity in the small intestine, and lactobacilli activity in the colon were determined in 12- and 24-day-old rats born to mother anemic rats with toxic anemia under the influence of phenylhydrazine hydrochloride, and in growing rats after mother and offspring toxic anemia.

Experiments in white rats have shown that in rats growing in experimental toxic anemia of mother and offspring, there is an increase in lactase activity in the small intestine and a decrease in lactobacilli activity depending on the degree of intoxication in the colon.

KEYWORDS

Rat, phenylhydrazine hydrochloride, small intestine, colon, lactase, lactobacilli.

INTRODUCTION

Today, with the development of industry and agriculture in the world, there is an increase in environmental impacts and an increase in diseases among the population due to environmental pollution.

It is known that anemia is a common pathology among women and children of reproductive age around the world. In Central Asia, anemia accounts for 70% of women of childbearing age and more than 60% of

children under three years of age [1,7]. In ecologically polluted regions, such as the Aral Sea region and the industrial areas of Tashkent region, various forms of anemia occur in almost all children of preschool age [6, 8].

Our many years of experimental observations have shown that the younger the organism, the stronger the influence of various factors on the formation of its functional systems [5]. The presence of negative changes in blood counts, behavior, immune and respiratory systems [2, 4, 6, 11] of children fed with women with anemia is confirmed by the results of many experimental and clinical observations. However, there is almost no data on the effect of maternal anemia on the activity of the digestive process in the growing organism.

Therefore, in this study, we aimed to study the effect of maternal and maternal-child toxic anemia on the activity of lactase in the small intestine and lactobacilli in the colon, which are involved in the digestion of milk sugar during lactotrophy.

MATERIALS AND METHODS

The experiments were performed on pedigree rats bred at the National University of Uzbekistan. The animals were divided into three experimental groups and one control group.

The mother rat in the first experimental group (experiment 1) was given phenylhydrazine hydrochloride at a dose of 80 mg / kg on the third day of lactation. In rats in the second experimental group (experiment 2), the drug

was administered on the third day of lactation and postnatal life in accordance with both maternal and developing rats. Intact rats served as controls.

Biochemical analyzes were performed in 12- and 24-day-old rats. The amount of hemoglobin in the blood (g / l) and the number of erythrocytes (T / l) were determined using the Sali apparatus, Goryaev counting chamber, respectively.

To prepare the enzymatically active drug of the small intestine, the intestine was separated from the mesentery, its cavity was washed with 10 ml of cooled saline solution, and its mass was measured. The intestine was mixed with a 1/10-chilled saline solution and homogenized at 300 rpm using a Teflon homogenizer for 1.0–1.5 minutes. The activity of lactase in intestinal homogenate is glucose oxidaseA. Determined by the Dahlqvist (1984) method.

All processes were performed in a cold environment. The enzyme activity was expressed in $\mu\text{mol} / \text{min} / \text{g}$ of tissue.

The activity of lactobacilli in the colon was studied by titration of bacterial strains in a skimmed milk medium. Bacterial activity was expressed in Turner degrees ($^{\circ}\text{T}$).

The results were analyzed by the student using the Exel program.

Results obtained and their analysis

The results showed that children fed a mother with toxic anemia also developed symptoms of anemia (Table 1).

Table 1

Effect of maternal and hereditary toxic anemia on the amount of hemoglobin and erythrocytes in the blood ($M \pm m$; $n = 6$)

Indicator	Animal groups		
	Control	Experiment 1	Experiment 2
12 days			
Amount of hemoglobin, g / l R	106,4 \pm 3,2	87,6 \pm 3,1 <0,002	80,0 \pm 1,6 <0,001
Erythrocyte count, T / l R	3,78 \pm 0,28	2,90 \pm 0,15 <0,02	2,43 \pm 0,10 <0,001
24 days			
Amount of hemoglobin, g / l R	110,0 \pm 1,6	93,2 \pm 4,4 <0,01	73,8 \pm 1,3 <0,001
Erythrocyte count, T / l R	4,55 \pm 0,12	3,05 \pm 0,21 <0,001	2,50 \pm 0,06 <0,001

In 12-day-old rats with an anemic mother (experiment 1), the hemoglobin content was 17.7% and the erythrocyte count was 23.3%; In 24-day-old rats, hemoglobin levels decreased by 15.3% and erythrocyte counts decreased by 32.9% compared to controls.

When experimental toxic anemia was present in both mother and offspring (Experiment 2), the hemoglobin content in 12-day-old rats increased by 24.8% and the erythrocyte count by 35.7%; In 24-day-old rats, the hemoglobin decreased by 32.9% and the number of erythrocytes by 45.0%.

This means that toxic anemia in a nursing mother also affects the development of anemia in the child. Anemia is also noted when the rat switches to independent feeding. If the toxicant (phenylhydrazine) is exposed not only to the mother but also to the mother and child at the same time, the symptoms of anemia will be exacerbated in the growing organism.

In the next phase of our study, we studied the development of lactase enzyme activity in both mother and offspring and in breeding rats (Figure 1).

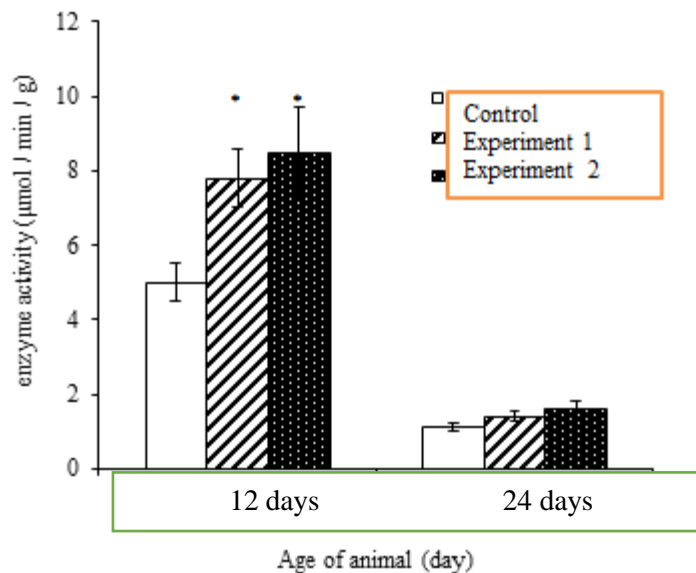


Figure 1. Effect of maternal and hereditary toxic anemia on lactase enzyme activity in the small intestine ($M \pm m$, $n = 6$)

* - statistically reliable relative to the control

As can be seen from the figure, lactase activity was significantly higher in 12-day-old rats in the control group, recorded at $5.0 \pm 0.4 \mu\text{mol} / \text{min} / \text{g}$. In 24-day-old rats that switched to an independent diet, i.e. lactose-lowering carbohydrates in the diet, the enzyme activity decreased sharply to $1.1 \pm 0.1 \mu\text{mol} / \text{min} / \text{g}$.

In 12-day-old rats fed with an anemic mother (experiment 1), lactase activity was recorded at $7.5 \pm 0.3 \mu\text{mol} / \text{min} / \text{g}$. That is, the severity of lactose hydrolysis was increased by 56% compared to the control. In 24-day-old rats, the activity of this enzyme was recorded at the same level in the control groups.

When toxic anemia was present in both mother and child (Experiment 2), lactase activity was $8.5 \pm 0.4 \mu\text{mol} / \text{min} / \text{g}$ in 12-day-old rats; In 24-day-old rats, it was $1.4 \pm 0.2 \mu\text{mol} / \text{min} / \text{g}$. That is, lactase activity was increased by 70% in 12-day-old rats compared to controls, and their activity was not different from controls in 24-day, i.e., rats transitioning to an independent diet.

Along with the activity of the enzyme lactase in the small intestine, the activity of lactobacilli in the colon of maternal and maternal-child anemia was also determined (Fig. 2).

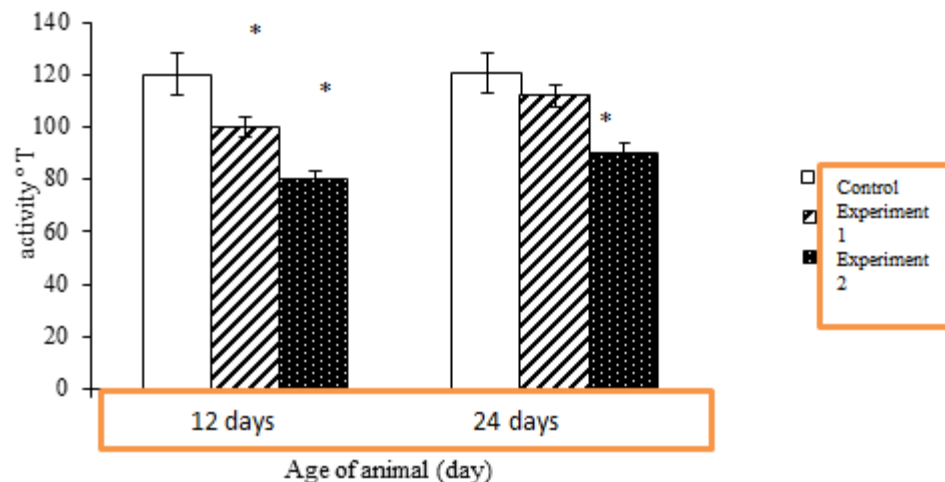


Figure 2. Effect of maternal and hereditary toxic anemia on lactobacilli activity in the colon ($M \pm m$; $n = 5-6$)

* -statistical reliability relative to control

It can be seen that the activity of lactobacilli in control rats was $120.0 \pm 3.20^{\circ}T$ and in 24-day-old rats was $120.7 \pm 0.4^{\circ}T$. In the first experimental group, ie 12-day-old rats fed with anemic mothers, the activity of colon lactobacilli was $100.0 \pm 3.2^{\circ}T$, and in 24-day-old rats it was $112.0 \pm 2.7^{\circ}T$. That is, lactobacilli activity in the colon decreased by 16.6% compared to controls in 12-day-old rats and by 7.2% in 24-day rats.

In the second experimental group, i.e., toxic anemia in both mother and child, colonic lactobacilli activity was $80.0 \pm 3.20^{\circ}T$ in 12-day-old rats and $90.0 \pm 3.70^{\circ}T$ in 24-day-old rats, respectively, at 33.3% and 25, respectively. , Decreased by 4%. Hence, there is an inversely proportional relationship between lactase activity in the small intestine and lactobacilli activity in the colon when maternal-child anemia is cited. In toxic anemia, increased activity of lactase in the small intestine leads to a decrease in the activity of lactobacilli in the colon.

DISCUSSION

Our results show that maternal anemia affects the activity of small intestinal lactase and lactobacilli in the colon associated with the

lactose substrate in the offspring. Maternal and child toxic anemia leads to increased lactase activity, decreased activity of lactobacilli. The biological significance of this phenomenon is reflected in the preservation of the viability of the offspring during lactotrophic feeding. As a result of an increase in the intensity of lactose digestion in the small intestine, the activity of lactobacilli naturally decreases due to a decrease in the passage of the substrate into the colon. When toxic anemia occurs in both mother and child, the induction of lactase activity is further increased, while the activity of lactobacilli is further reduced.

One of the main hormones that control the activity of the enzyme lactase is thyroxine. Typically, an increase in lactase activity in hypothyroidism and a decrease in hyperthyroidism [3]. Gökdeniz E. et al. (2010) noted thyroid insufficiency in anemia. This evidence suggests that increased lactase activity in anemia is associated with functional thyroid insufficiency.

The concentration of the hormone thyroxine gradually increases in postnatal ontogenesis in mammals, correspondingly reducing the

synthesis of the enzyme lactase in the intestine and increasing the activity of enzymes such as α -amylase, maltase, sucrose [5]. As a result, the hydrolytic capacity of the growing organism in the intestine of a single carbohydrate in milk compared to lactose decreases, and the assimilation of carbohydrates such as starch, maltose, sucrose in adult food increases. In toxic anemia, too, high levels of lactase activity due to hypothyroidism indicate a slowing of biological development hours.

In addition, a decrease in the activity of lactobacilli in the colon reduces their colonial stability, creating an environment for the proliferation of pathogenic bacteria in the intestinal mucosa [9].

Thus, maternal anemia slows down the natural repression of the lactase enzyme in the offspring, reduces the activity of lactobacilli, and slows the maturation periods of the hydrolytic system in the offspring.

CONCLUSION

- 1) In maternal and maternal-child toxic anemia, the activity of lactase in the small intestine in the offspring increases.
- 2) In experimental anemia of mother and child, the activity of lactobacilli in the colon is reduced.

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