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EFFECT OF COLD STRESS ON POSTNATAL FORMATION OF MECHANISMS OF CARBOHYDRATE HYDROLYSIS AND TRANSPORT IN THE RAT SMALL INTESTINE

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Acute experiments using the accumulating mucosal preparation (AMP) method showed that cold adaptation (exposure for 2 h daily to a temperature of 6-7°C) of young rats during the first week after birth has a lasting inhibitory action on postnatal formation of the maltase transport and γ-amylase-transport digestion-assimilating systems but does not change the dynamics of development of the intrinsic transport systems. The same adaptation at the age of 10 to 17 days after birth, on the other hand, appreciably quickened the rates of formation of the hydrolytic-transport conveyor on the outer surface of the cell membranes.

KEY WORDS: cold; small intestine; accumulation of glucose; maltose, starch.

Previous investigations showed that in rats exposed to cold from the 1st to the 7th days after birth there is very persistent inhibition of the rates of development of the enzyme systems concerned in the initial and final stages of intestinal carbohydrate hydrolysis. Meanwhile, similar exposure between the 10th and 17th days after birth stimulated the formation of mechanisms of membrane hydrolysis of starch and sucrose in rats [2-4].

The investigation described below was aimed at a further study of the effect of stressors on the formation of the intestinal function in the growing organism, and in particular, to characterize possible modifications at the stage of interaction between systems of carbohydrate hydrolysis and transport after cold stress to which the animals were exposed at different times of early postnatal development.

EXPERIMENTAL METHOD

Rats born in the laboratory animal house and kept in groups of eight to each lactating mother were divided into two groups. The rats of one group were exposed for 2 h daily to cold (6-7°C) from the 1st until the 7th day after birth, the animals of the other group were similarly exposed from the 10th until the 17th day. The rats of the control groups, like those of the experimental groups also, were taken from their mothers for 2 h daily and kept in an incubator at a thermoneutral temperature (33-35°C).

To obtain quantitative data, the animals were killed six at a time both from the experimental and from the control groups on the day of ending of exposure to cold and 1, 2, 3, and 7 weeks later. After sacrifice,

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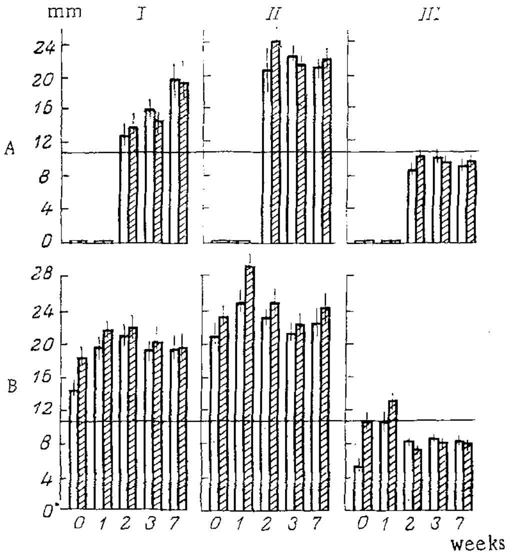

Fig. 1

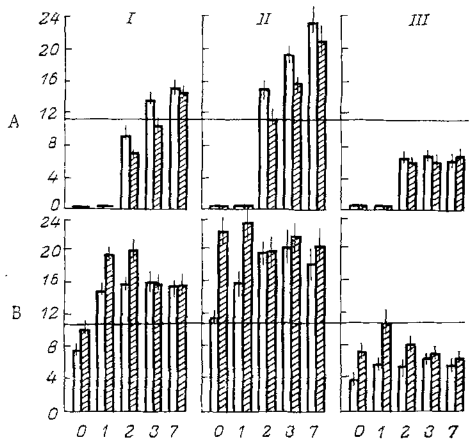

Fig. 2

Fig. 1. Accumulation of glucose in AMP of proximal (I), middle (II), and distal (III) portions of small intestine during incubation in glucose solution in growing rats under normal conditions (unshaded columns) and at various times after exposure to cold (shaded columns), namely from 1st until 7th days (A) and from 10th to 17th days (B) after birth. Abscissa, time after exposure (in weeks); ordinate, accumulation of glucose (in millimoles). Straight line marks substrate concentration.

Fig. 2. Glucose accumulation in AMP of proximal (I), middle (II), and distal (III) portions of small intestine during incubation in maltose solution in growing rats under normal conditions (unshaded columns) and at various times after exposure to cold (shaded columns) from the 1st to the 7th days (A) and from the 10th to the 17th days (B) after birth. Legend as to Fig. 1.

laparotomy was performed quickly and the small intestine (without the duodenum) separated from the mesentery, immersed in cold Ringer's solution (pH 7.4), and carefully washed. The intestine was then gently dried on filter paper, weighed, placed on a glass plate cooled with ice, and divided into three equal portions, conventionally described as proximal, middle, and distal parts. From each part of the intestine three segments were cut, turned inside out, and tied at both ends with ligatures. The resulting accumulating mucosal preparations (AMP) [7] were incubated in 11.1 mM saline solutions of glucose, maltose, or starch for 60 min at 37-38°C, with constant oxygenation. The quantity of glucose accumulating in the AMP during incubation was determined by an arsenic-molybdenum method [8] and expressed in millimoles glucose per 100 mg tissue. Accumulation of glucose in the AMP from glucose solution characterized the initial stages of transport, whereas accumulation of glucose from a solution of maltose and starch characterized the functional state of the maltase-transport and γ-amylase-transport enzyme-carrier systems respectively.

EXPERIMENTAL RESULTS

The experiments showed that the rates of development of the carbohydrate transport mechanisms in the small intestine were considerably modified in rats exposed repeatedly to cold. The character, depth, and persistence of these changes depended on the region of the small intestine studied, the degree of polymerization of the assimilated carbohydrate, the age period when the rat was exposed to the stressor, and so on. These data will be examined below initially for the intrinsic transport function (transmembrane transport of free glucose, or G-glucose), then for transport of glucose liberated after hydrolysis of maltose (transmembrane transport of maltose glucose, or M-glucose) and, finally, for the function of the γ-amylase transport assemblage, responsible for transmembrane transport of glucose set free after hydrolysis of starch (transmembrane transport of starch glucose, or S-glucose).

It will be clear from Fig. 1 that in rats exposed to cold from the 1st until the 7th days after birth, development of transmembrane transport of G-glucose was virtually indistinguishable in all parts of the small intestine from that in the control animals, whereas exposure to cold from the 10th until the 17th days led to

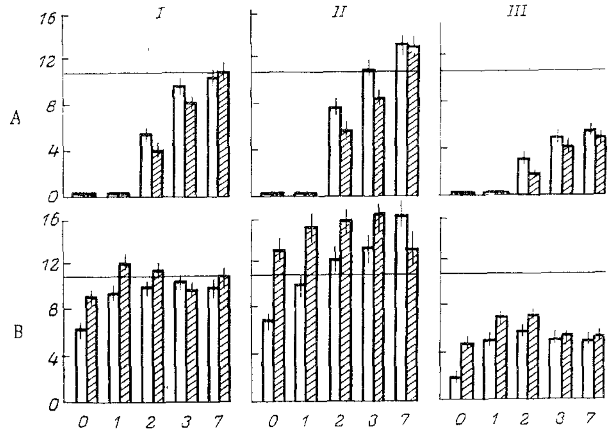

Fig. 3. Glucose accumulation in AMP of proximal (I), middle (II), and distal (III) portions of small intestine during incubation in starch solution in growing rats under normal conditions (unshaded columns) and at various times after exposure to cold (shaded columns), from the 1st until the 7th days (A) and from the 10th until the 17th days (B) after birth. Legend as to Fig. 1.

some stimulation of the transmembrane transport function of the small intestine. This effect of cold was particularly marked on the middle portion, which normally exhibits maximal transport activity, and it was not found in the proximal and distal portions of the small intestine. The stimulation effect was significant only at the end of the first week after the end of exposure to cold, and thereafter it quickly disappeared.

As Fig. 2 shows, functional development of the maltase transport system in animals exposed to cold during the first week of life was considerably retarded compared with animals of the control group in the proximal and middle portions of the small intestine. Meanwhile exposure to cold from the 10th until the 17th days after birth of the rats had the opposite effect and led to a marked increase in the rates of transmembrane transport of glucose added to the incubation medium in the form of maltose. This increase, as regards all three parts of the small intestine, was found as soon as exposure ceased and it continued throughout the next 2 weeks in the proximal and distal portions and 1 week in the middle portion.

It will be clear from Fig. 3 that, depending on the animals' age, exposure to cold had different effects also on transmembrane transport of S-glucose. In rats exposed to cold during the first week of life delay in the development of the hydrolysis and transport systems was observed in all three portions of the small intestine; this delay persisted during the next 3 weeks after the animals had returned to normal conditions of keeping. Exposure of the animals to cold from the 10th until the 17th days after birth, on the other hand, stimulated transport of S-glucose, and this process continued for 2 or 3 weeks depending on the part of the small intestine tested.

The data described above show that exposure to cold at different periods of postnatal development significantly modifies the rates of formation not only of the digestive system proper of the small intestine, as the writers' previous investigations [2-4] showed, but also the rate of formation of systems which couple digestive and transport processes on the outer surface of the cell membranes. There is much evidence in the literature to show that cold adaptation in early ontogeny has an inducing effect on the rate of development of growing animals and that this effect is connected with the more rapid functional maturation of the pituitary, thyroid, and adrenal glands [9].

It can be tentatively suggested that under the experimental conditions used exposure to cold led to quickening of the rates of formation of the hypothalamic-hypophyseo-adrenal and thyroid systems which, through their participation in the development of mechanisms of intestinal digestion and transport [1, 6, 10, 11], induced and controlled the rates of formation of the hydrolytic-transport conveyor, functioning on the outer surface of the epithelial cells [5, 6]. However, the results of the present experiments emphasize that the stimulating effect of exposure to cold on the hydrolytic-transport function of the small intestine, and also perhaps on the other functional systems, is found when adaptation to cold is carried out not at the very beginning of postnatal life (from the 1st until the 7th days after birth of rats), but somewhat later (from the 10th until the 17th days). Early exposure to cold in the present experiments led to the opposite effect: a persistent delay in the formation of the digestive transport conveyor. It is important to note that the development of the intrinsic transport system responsible for transmembrane transport of "free" glucose under the present experimental conditions, underwent no appreciable change. This result suggests that the mechanisms of development of enzyme-transport systems are more sensitive to stress than the mechanisms of development of intrinsic transport systems. This is in agreement with the abundant data showing that in certain pathological states selective impairment of enzyme systems may be observed while the intestinal cells maintain normal transport functions [5, 6].

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ROLE OF β-ADRENORECEPTION IN REALIZATION OF GLYCOGENOLYTIC AND LIPOLYTIC EFFECTS OF AN EXCESS OF THYROID HORMONES

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Experiments on rats showed that chronic administration of the β-adrenoblocker propranolol in doses blocking glycogenolytic and lipolytic effects of exogenous adrenalin, does not prevent the fall in the glycogen level in the liver and myocardium and the rise in the serum free fatty acid level and in the lipolytic activity of adipose tissue in vitro arising under the influence of large doses of thyroxine.

KEY WORDS: thyrotoxicosis; adrenergic mechanisms; glycogenolysis; lipolysis.

Previous investigations [1] showed the catecholamine-independent action of thyroid hormones on function and metabolism of the myocardium. However, the role of adrenergic mechanisms in the genesis of the disturbances of lipid and carbohydrate metabolism arising during thyrotoxicosis has been inadequately studied. It has been shown that β-adrenoblockers prevent manifestation of the activating effect of thyroxine on heart

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