

coincides with a reduction in the heterogeneity of the muscle fibers under these conditions as revealed histochemically [3]. The decrease in the frequency of MEPP is also evidence of a disturbance of spontaneous acetylcholine secretion by the nerve endings.

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STRUCTURAL CHANGES IN ENTEROCYTE MEMBRANES IN ACHOLIA WITH SPECIAL REFERENCE TO MICROTOPOGRAPHICALLY DIFFERENT CARBOHYDRASES

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Activity of surface and intracellular carbohydrases was compared in rats with chronic total loss of bile. The function of adsorbed amylase, a glycocalyx marker, is disturbed by a much greater degree than the function of invertase, a marker of the plasma membrane proper.

KEY WORDS: small intestine; hydrolysis of carbohydrates; bile.

The physiological role of bile is not yet fully understood. The results of membrane studies point to a possible role of the components of bile in the regulation of the metabolic activity of the enterocyte and, in particular, in the metabolism of its membrane structures.

The properties of the plasma membrane proper and of the glycocalyx were assessed in rats with acholia with special reference to the corresponding marker enzymes: invertase [2] and adsorbed amylase.

EXPERIMENTAL METHOD

Albino rats (37 animals) were fed in the ordinary way on a mixed diet. A fistula of the bile duct was formed as described by Shlygin and Vasilevskaya [3]. Fasting rats were decapitated on the 4th and 7th days after the operation.

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TABLE 1. Hydrolysis of Starch and Sucrose by Everted Pieces from Various Segments of Rat Small Intestine on 4th and 7th Days of Chronic Loss of Bile (in μ moles hexoses/cm; $M \pm m$)

Segment of small intestine	Control (n = 7)		On 4th day (n = 5)			On 7th day (n = 5)		
	μ moles/cm	%	μ moles/cm	$\Delta\%$	P	μ moles/cm	$\Delta\%$	P
Hydrolysis of starch								
I	21 \pm 1	100	16 \pm 1	-24	>0,05	4,4 \pm 0,3	-80	<0,001
II	22 \pm 2	100	14 \pm 1	-36	>0,05	5,9 \pm 0,3	-73	<0,001
III	28 \pm 2	100	14 \pm 1	-50	<0,001	6,4 \pm 0,2	-77	<0,001
IV	35 \pm 3	100	15 \pm 1	-57	<0,001	7,4 \pm 0,5	-79	<0,001
V	33 \pm 2	100	9 \pm 1	-73	<0,001	11,7 \pm 0,8	-65	<0,001
VI	20 \pm 2	100	7 \pm 1	-63	<0,01	9,5 \pm 0,7	-53	<0,01
Total	159 \pm 12	100	75 \pm 7	-53	<0,001	45 \pm 3	-71	<0,001
Hydrolysis of sucrose								
I	4,8 \pm 0,2	100	3,1 \pm 0,2	-35	<0,001	2,5 \pm 0,1	-48	<0,001
II	5,9 \pm 0,3	100	3,2 \pm 0,1	-46	<0,001	2,7 \pm 0,2	-54	<0,001
III	5,7 \pm 0,2	100	2,0 \pm 0,2	-65	<0,001	2,5 \pm 0,3	-56	<0,001
IV	5,5 \pm 0,6	100	3,3 \pm 0,2	-40	<0,05	2,7 \pm 0,1	-51	<0,01
V	4,5 \pm 0,1	100	2,8 \pm 0,3	-38	<0,01	1,9 \pm 0,1	-58	<0,001
VI	2,7 \pm 0,1	100	1,7 \pm 0,2	-33	<0,01	1,3 \pm 0,1	-52	<0,001
Total	29 \pm 2	100	17 \pm 1	-40	<0,01	14 \pm 1	-52	<0,01

TABLE 2. Hydrolysis of Starch and Sucrose by Homogenates of Mucous Membrane of Various Segments of Rat Small Intestine on 4th and 7th Days of Chronic Loss of Bile (in μ moles hexoses/100 mg; $M \pm m$)

Segment of small intestine	Control (n = 8)		On 4th day (n = 6)			On 7th day (n = 6)		
	μ moles/mg	%	μ moles/mg	$\Delta\%$	P	μ moles/mg	$\Delta\%$	P
Hydrolysis of starch								
I	24 \pm 2	100	23 \pm 2	-4	>0,05	28 \pm 2	+16	>0,05
II	26 \pm 3	100	24 \pm 3	-8	>0,05	30 \pm 3	+15	>0,05
III	38 \pm 4	100	31 \pm 1	-18	>0,05	25 \pm 2	-34	<0,02
IV	41 \pm 4	100	30 \pm 2	-27	<0,02	27 \pm 3	-34	<0,01
V	40 \pm 1	100	28 \pm 2	-30	<0,001	22 \pm 2	-45	<0,001
VI	32 \pm 3	100	16 \pm 2	-50	<0,01	17 \pm 2	-47	<0,01
Total	202 \pm 18	100	152 \pm 12	-25	<0,05	149 \pm 13	-26	<0,05
Hydrolysis of sucrose								
I	15,8 \pm 1,2	100	10,8 \pm 0,4	-32	<0,01	7,4 \pm 0,4	-53	<0,001
II	14,5 \pm 0,9	100	11,3 \pm 0,8	-22	<0,05	7,9 \pm 0,9	-46	<0,01
III	14,3 \pm 1,2	100	11,3 \pm 0,9	-21	<0,05	7,5 \pm 0,8	-48	<0,01
IV	10,2 \pm 0,5	100	9,6 \pm 0,7	-6	>0,2	7,7 \pm 0,7	-25	<0,05
V	8,7 \pm 0,7	100	7,1 \pm 0,7	-18	>0,05	6,1 \pm 0,5	-30	<0,02
VI	7,3 \pm 0,8	100	4,8 \pm 0,4	-34	<0,05	4,4 \pm 0,3	-40	<0,02
Total	71 \pm 6	100	55 \pm 4	-23	<0,05	45 \pm 4	-37	<0,02

The activity of surface and intracellular carbohydrases was compared in six segments of small intestine. Hydrolysis of sucrose and starch was determined in everted pieces or homogenates of mucous membrane [1].

EXPERIMENTAL RESULTS AND DISCUSSION

Loss of bile is accompanied by a sharp decrease in the rate of starch hydrolysis together with a much less marked reduction in the rate of sucrose hydrolysis by everted pieces of intestine (Table 1). From the 4th to the 7th day carbohydrase activity in the surface of the mucous membrane decreased in most segments of intestine, and the function of adsorbed amylase was disturbed particularly sharply.

Conversely, in homogenates of mucous membrane (Table 2) the levels of amylolytic and invertase activity fell similarly (on average by 25%). From the 4th to the 7th days the activity of six segments remained unchanged as regards hydrolysis of starch, although there was a tendency for hydrolysis of sucrose to take place more slowly (on average by 15%).

In acholia the function of the surface carbohydrases, located on the outer membranous structures of the enterocyte, is thus disturbed more sharply than the function of the intracellular enzymes. Among the surface carbohydrases, there was a particularly marked decrease in the function of the enzyme associated with the glycocalyx (adsorbed amylase) compared with the change in activity of the marker enzyme of the plasma membrane proper (invertase).

These results, together with data in the literature [1], suggest that chronic loss of bile leads to a significant disturbance of the structure and function of the outer membranes of cells of the intestinal epithelium.

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EFFECT OF OXYGEN AND HYPERGRAVITATION ON ALVEOLAR SURFACTANT

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The effect of prolonged (up to 66 h) exposures to pure oxygen and of brief (5 min) inhalation of oxygen combined with gravitational overloads produced by longitudinal acceleration (5g) on the surface tension and surface potential of the alveolar washings from the lungs of albino rats was studied. Under both experimental conditions atelectasis of the lungs was formed, with a decrease in the surface activity of the surfactant. The mechanisms of the changes in surfactant activity during hyperoxia, alone and combined with hypergravitation, are discussed.

KEY WORDS: hyperoxia; acceleration; atelectasis; surfactant.

It is now generally accepted that prolonged normobaric hyperoxia, lasting several hours, causes disorders of respiration (the Lorrain-Smith syndrome) and atelectases of the lungs as a result of the toxic action of oxygen on lung tissue [1, 3, 5]. It is also known that similar respiratory changes may arise following brief (for a few minutes) inhalation of pure oxygen, but combined with hypergravitation caused by exposure to longitudinal (+G_Z) or transverse (+G_X) acceleration [6, 8]. Atelectases of the lungs are considered to develop under these conditions on account of the increased regional inequality of ventilation of the lungs, their deformation, and the rapid absorption of oxygen by blood in the pulmonary capillaries from the unventilated alveoli [8, 14].

At the same time, absence or inactivation of the surface-active substance of the lungs (surfactant) is known to facilitate the formation of atelectases [4, 13, 15]. In particular, a decrease in alveolar surfactant activity has been found after prolonged exposures to oxygen [7, 10, 12].

Since both prolonged and brief exposures to oxygen, if combined with acceleration, lead ultimately to the development of atelectases of the lungs, changes in the surface-active properties of the surfactant are to be expected in both experimental situations. In accordance with this hypothesis the state of the surfactant was studied in the lungs of rats exposed for different times to oxygen and also in rats exposed to acceleration combined with inhalation of air and of pure oxygen.

EXPERIMENTAL METHOD

Experiments were carried out on 53 male albino rats weighing 140-170 g. In the experiments of series I (17 rats in the experimental and five in the control group) the animals were kept for between 6 and 66 h in a chamber through which pure oxygen was passed at the rate of 0.5 liter/min per rat. Carbon dioxide was absorbed by the KhPI absorber.

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