ENZYME SPECTRUM OF THE PANCREAS IN CHRONIC STRESS

Sh. K. Kurbanov and Ch. R. Alimhodzhaeva

UDC 616.45-001.1/3-07:616.37-008.931-074

The enzyme spectrum of pancreatic homogenate was studied in acute experiments on male albino rats during adaptation for 30 days to muscular exertion (forced swimming in water at a temperature of 32 ± 1°C), heat (hyperthermia to 40-41°C), and cold (cooling to 3-4°C) for 3 h. The initial periods of adaptation to these factors (second-twelfth day) were shown to be characterized by a considerable decrease in activity of all the enzymes studied, but later, with adaptation of the animals to these factors, enzyme activity was restored to its original level (18th-24th day), and remained more or less constant until the end of the experiment (30th day). It is suggested that changes in the enzyme spectrum of the pancreas are brought about through the participation of the hypothalamo-hypophyseo-adrenal system in accordance with the principle of the general adaptation syndrome.

KEY WORDS: α-amylase; total protease; lipase; stress.

The writers showed previously that a single performance of muscular work and exposure to heat or cold for a varied duration has a significant effect on the enzyme-forming and enzyme-secreting function of the pancreas, and also on the state of the enzyme spectrum of the small intestine in rats [2, 3, 6, 7]. However, the question of the functional state of the pancreatic enzyme systems during chronic stress has not yet been adequately studied despite its great importance for the solution to some theoretical and practical problems in the physiology and pathology of the digestive organs.

The object of the present investigation was to study the enzyme activity of pancreatic homogenate from rats in response to prolonged muscular exertion or exposure to heat or cold.

EXPERIMENTAL METHOD

Experiments were carried out on 144 noninbred male albino rats weighing 140-160 g, kept on a standard diet. The animals were divided into three experimental and one control group, with 36 animals in each group. The rats of group 1 were forced to swim daily for 3 h in water at a temperature of 32 ± 1°C, rats of group 2 were exposed to hyperthermia (40-41°C) for 3 h, and the rats of group 3 were exposed to cooling (3-4°C) also for 3 h. The control group was kept under analogous conditions but not subjected to any of these factors. Later, rats from each group were killed six at a time, after 2, 5, 12, 18, 24, and 30 days, laparotomy was quickly performed, the pancreas was separated from the duodenum and adjacent adipose tissue, and homogenized, with the addition of cold Ringer's solution, pH 7.4, in the proportion of 1 ml to 100 mg tissue. The α-amylolytic, total proteolytic [8], and lipolytic [4] activity were determined in the homogenate. The α-amylolytic activity was expressed in milligrams starch hydrolyzed per minute, total proteolytic activity in micrograms lysine formed per minute, and lipolytic activity in microequivalents per hour of incubation per gram wet weight of pancreas.

EXPERIMENTAL RESULTS

In the rats of the control group the α-amylolytic activity usually varied between 21,900 ± 537 and 24,500 ± 740 mg starch hydrolyzed per minute, the total proteolytic activity between 12,860 ± 670 and 16,880 ± 870 μg glycine formed per minute, and lipolytic activity between 20,100 ± 700 and 39,400 ± 1300 μeq of fatty acids formed. The experiments showed that all

Laboratory of the Physiology of Digestion and Laboratory of General Physiology, Institute of Physiology, Academy of Sciences of the Uzbek SSR, Tashkent. (Presented by Academician of the Academy of Sciences of the USSR O. G. Gazenko.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 87, No. 5, pp. 412-414, May, 1979. Original article submitted June 15, 1978.

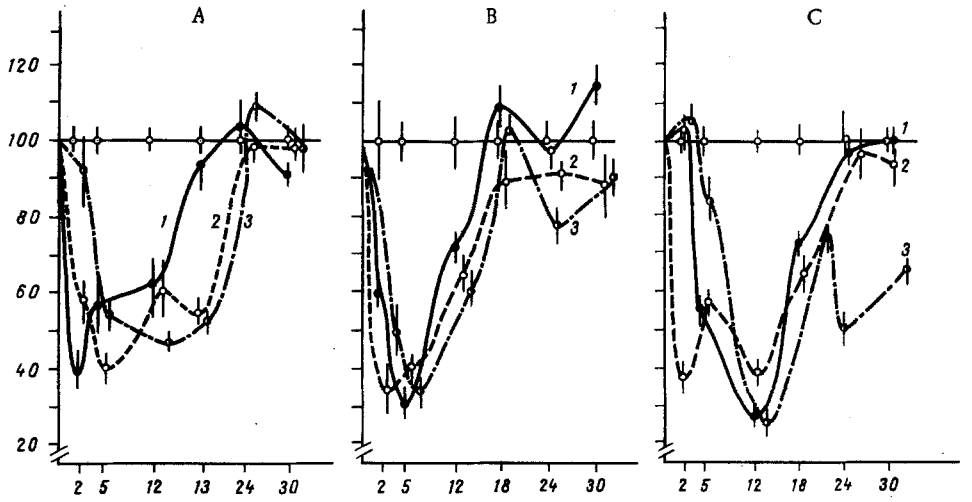

Fig. 1. Changes in α-amylolytic (A), total proteolytic (B), and lipolytic (C) activity of pancreatic homogenate of rats after muscular exertion (1) and exposure to heat (2) and cold (3) daily for 3 h. Abscissa, duration of experiment (in days); ordinate, activity of enzyme (in percent relative to corresponding control values taken as 100). Mean results of six determinations (M) and standard error of means (m) are given.

three types of chronic stress factors led to profound changes in pancreatic enzyme activity (Fig. 1). As Fig. 1 shows, α-amylolytic activity fell sharply on the second day of the experiment during muscular exertion and remained below the control level until the 12th day, after which it was gradually restored to the 18th day and varied within the control limits until the end of the observations. A similar decrease in enzyme activity at the beginning of the experiment also was observed during exposure to heat and cold. However, in the last cases restoration of the activity of this enzyme to the control level occurred on the 24th day of the experiment. Total proteolytic activity, on the other hand, fell sharply during all these procedures on the 2nd and 5th days, returned to normal on the 18th day, and remained within normal limits until the 30th day inclusive.

As regards lipolytic activity, its greatest decrease both during muscular exertion and during exposure to heat and cold was found on the 12th day. Later, with repeated exposure to the stressors, it gradually returned to the control level by the 24th day in the case of muscular exertion and exposure to heat. In the experiments with exposure to cold, a fresh decrease was observed at this time.

Muscular exertion or exposure to heat or cold for 30 days thus significantly changes the enzyme spectrum of the pancreatic homogenate. The character and direction of the changes observed differ during adaptation to repeated loads. The initial periods of observation (2nd-12th day) are characterized by marked decrease in the activity of all enzymes studied, but later, as the animals become adapted to these factors, enzyme activity is restored to its original levels (18-24th days) and remains more or less constant until the end of the experiment (30th day). Since the changes described above are similar for all three types of procedure, it can be concluded that changes in the pancreatic enzyme spectrum under these experimental conditions involve the participation of the hypothalamo-hypophyseo-adrenal system in accordance with the principle of the general nonspecific adaptation syndrome.

After intensive muscular work or acute exposure to a high or low ambient temperature, and also after exposure to many other stress factors, activation of the sympathico-adrenal and hypothalamo-adrenocortical systems is known to take place, and the liberation of catecholamines, ACTH, corticosteroids, and other hormones distinguished by a broad spectrum of action on various enzyme systems of the body, is increased [1, 5, 9. 10]. All these changes are evidently reflected in the metabolic level in the body and in the rates of formation of the

various digestive enzymes, including pancreatic enzymes. The results now obtained indicate that these disturbances under the conditions of chronic stress inhibit the formation and possibly also the activity of the principal pancreatic enzymes. However, this inhibition is observed only at certain times of adaptation and it gradually disappears during repetition of the action of the stressors, suggesting the increasing efficiency of the mechanisms responsible for maintenance of the neurohormonal, metabolic, and enzymic status of the body in situations of stress. The results described in this paper suggest that when scientifically based diets suitable for use during chronic exposure to stress factors are drawn up the state of the pancreatic enzyme spectrum in the different stages of adaptation must be taken into account.

LITERATURE CITED

1. A. A. Viru, Functions of the Adrenal Cortex during Muscular Activity [in Russian], Moscow (1977).
2. Sh. K. Kurbanov, Uzbek. Biol. Zh., No. 6, 57 (1975).
3. Sh. K. Kurbanov, Byull. Éksp. Biol. Med., No. 11, 1335 (1976).
4. A. A. Lazdynysh and M. R. Lazdynya, Izv. Akad. Nauk Latv. SSR, No. 1, 68 (1975).
5. É. Sh. Matlina and G. N. Kassil', Usp. Fiziol. Nauk, No. 2, 13 (1976).
6. Z. T. Tursunov et al., Uzbek Biol. Zh., No. 4, 27 (1975).
7. Z. T. Tursunov et al., Biol. Nauki, No. 2, 35 (1977).
8. A. M. Ugolev et al., Investigation of the Digestive Apparatus in Man [in Russian], Leningrad (1969).
9. I. A. Iskin, Principles of Physiology of the Endocrine Glands [in Russian], Moscow (1975).
10. N. A. Yudaev, in: Chemical Bases of Processes of Vital Activity [in Russian], Moscow (1962), p. 235.

LIPID PEROXIDES AND THROMBOSIS

A. A. Kubatiev and S. V. Andreev

UDC 616-005.6-092:547.915.5

On incubation of rabbit platelets with ADP, adrenalin, serotonin, and thrombin, the level of lipid hydroperoxides in the platelets, determined as malonic dialdehyde, increases parallel with the increased aggregative power. An even higher concentration of malonic dialdehyde is observed in the platelets of animals with pulmonary thrombosis. Dynamic studies showed that the accumulation of malonic dialdehydes in the platelets reflects the initial stage of development of thrombosis; this can be used for the diagnosis of the early stages of intravascular thrombosis.

KEY WORDS: aggregation of platelets; malonic dialdehyde; lipid peroxides; pulmonary thrombosis.

The role of peroxidation products of lipids in the mechanisms of thrombosis has received insufficient study. It is stated [12, 13] that lipid peroxidation products formed by the oxidative destruction of unsaturated fatty acids may injure the platelet membrane and induce irreversible aggregation.

Since platelet aggregation is the trigger mechanism of development of thrombosis, it has been suggested that the accumulation of lipid hydroperoxide in the platelets must be a reliable sign of commencing thrombosis.

To test this hypothesis, the writers studied the dynamics of accumulation of lipid peroxidation products in the platelets of rabbits with experimental pulmonary thrombosis of immune etiology.

Laboratory of Pathophysiology, Institute of Human Morphology, Academy of Medical Sciences of the USSR, Moscow.

(Presented by Academician of the Academy of Medical Sciences of the USSR A. P. Avtsyn.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 87, No. 5, pp. 414-417, May, 1979. Original article submitted July 13, 1978.

0007-4888/79/8705-0423$07.50