GLUTAMINASE ACTIVITY OF KIDNEY TISSUE OF RATS WITH EXPERIMENTAL NEPHRITIS

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Investigation of the glutaminase activity in the cortical, medullary, and papillary layers of the kidney tissue of rats with experimental nephritis of different duration showed an increase in the acute phase with restoration of the normal level by the fourth to fifth month of the disease. Changes in glutaminase activity were combined with activation of acid and ammonia production and they are, evidently, adaptive in character.

The activity of phosphate-activated glutaminase (3.5.1.2-1) was studied in the kidney tissue of rats with experimental nephritis. This enzyme participates in the regulation of water and electrolyte metabolism and acid-base balance, disturbance of which is one of the chief manifestations of nephritis. Virtually no investigations of this type have been described in the literature.

EXPERIMENTAL METHOD

Experimental nephritis was induced in albino rats weighing 180-200 g by injection of nephrotic rabbit serum (titer 1:1000) by Mazuga's method. The presence of nephritis was confirmed histologically and by clinical and biochemical tests.

Glutaminase activity was determined in the cortical, medullary, and papillary layers of the kidneys seven and 14 days, and two, four, and five months after the end of immunization. To determine glutaminase activity, a medium containing 0.5 ml 0.1% L-glutamine solution, 0.5 ml 0.067 M phosphate buffer, pH 8.0, and 0.5 ml homogenate (2-6 mg protein) was used. Samples were incubated for 1 h at 37°C, and the ammonia thus formed was estimated by microdiffusion in Conway dishes [3]. The results were expressed in milligrams nitrogen per gram protein per hour. Protein was determined by the Kjeldahl method in kidney tissue homogenates.

EXPERIMENTAL RESULTS

On histological examination of the kidneys of the rats at various times after the induction of experimental nephritis, changes characteristic of the picture of proliferative membranous glomerulonephritis were found.

In the first week of the disease, the glutaminase activity of the rats was significantly increased in the cortical, medullary, and papillary layers of the kidneys. The increase in glutaminase activity continued in the rats with nephritis of two months' duration, but in the chronic stage of experimental nephritis (four to five months) the glutaminase activity returned to normal (Table 1).

In rats with experimental nephritis the excretion of acid radicals (titratable acids + ammonia) was increased in the first week of the disease and returned to normal by the fourth to fifth month.

The increase in glutaminase activity of the kidneys was thus combined with increased excretion of titratable acids and ammonia. The changes in glutaminase activity are evidently adaptive in character and

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TABLE 1. Glutaminase Activity of Kidney Tissue of Rats with Experimental Nephritis (in mg nitrogen/g protein/h)

Layer of kidney	Control	Time after immunization			
		7 days	14 days	2 months	4–5 months
Cortical	7,46±0,39 (11)	11,12±0,69 (25)	8,74±0,50 (9)	10,74±0,52 (10)	8,12±0,46 (11)
Medullary	8,03±0,38 (11)	11,3±0,60 (25)	13,62±0,82 (9)	13,65±0,53 (10)	9,44±0,55 (11)
Papillary	11,0±0,72 (11)	20,6±1,02 (21)	13,01±1,07 (9)	12,22±0,43 (10)	12,56±0,77 (11)

Note. Number of animals given in parentheses.

reflect the intensity of acid and ammonia production by the kidney tissue of rats with experimental nephritis.

However, no correlation was found between the degree of increase in the excretion of acid radicals and the increase in glutaminase activity of the kidney tissue. Ammonia production possibly is increased also because of the increase in gluconeogenesis, leading to the removal of glutamate, which inhibits the glutaminase reaction. The possibility cannot be ruled out that the increased ammonia production may also be due to an increase in the activity of glutaminase activated by α-ketoacids [1, 2, 4, 5].

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