SHORT COMMUNICATIONS

CHANGES IN ASCORBIC ACID CONTENT IN VARIOUS ORGANS
AND SERUM OF MICE EXPERIMENTALLY INFECTED
WITH TAENIA CRASSICEPS (ZEDER, 1809) CYSTICERCI

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Abstract. It was demonstrated that helminth infection affects the ascorbic acid content in various tissues of experimental mice. The level of vitamin C in the mouse liver, kidneys, spleen and serum decreases after infection with *T. crassiceps*. Maximum decrease in blood serum was observed on day 7 p.i. at the intensity of infection 15 and 30 cysticerci per mouse. There was a correlation between the intensity of infection and decrease in the ascorbic acid level in the spleen.

It is known that the helminths markedly affect the content of vitamin C in the host body. Katsman (1957) and Bogomaz (1960) demonstrated a decrease in ascorbic acid level in the human blood serum during ascariasis. Jablonowski (1970a) observed a decrease in vitamin C level in the liver, kidneys and suprarenal glands of guinea pigs experimentally infected with *Ascaris lumbricoides*. He reported that in the course of larval ascariloses in rats, the vitamin C level increases in suprarenal glands and simultaneously decreases in the liver. According to Jablonowski (1970b) the ascorbic acid level increases in all studied organs of rats infected with *Hymenolepis nana*. Gameel (1982a, b) reported a decrease in ascorbic acid level in the plasma of sheep infected with *Fasciola hepatica* and in the plasma, liver, kidneys and spleen of mice infected with the same parasite. Ismagilova (1968) (ex Bayandina and Kovchur 1973) observed a decrease in vitamin C level in organs and tissues of rabbits experimentally infected with *Taenia pisiformis*. The results obtained by the above authors are inconsistent, but in all cases the helminth infection produced an effect on the vitamin C content in organs and blood of the host.

The present paper deals with the effect of *T. crassiceps* infection on ascorbic acid content in organs and blood of mice.

MATERIAL AND METHODS

A total of 48 ICR female mice at the age of 1—1.5 month were used. They were divided into 4 groups including 12 mice each. The first group was infected intraperitoneally with 5 cysticerci, the second group with 15 cysticerci, and the third group with 30 cysticerci of *T. crassiceps* per mouse. The fourth group served as a control.

The mice were killed on days 7, 14, 21 and 28 p.i. The level of vitamin C was detected in the kidneys, liver, spleen and serum of mice using the method after Denson and Bowers (1961) with 2,4-dinitrophenylhydrazine. This method is based on the oxidation of ascorbic acid by copper cations (Cu⁺) to form dehydroascorbic acid and diketogulonic acid. These products react with 2,4-dinitrophenylhydrazine to form hydrazones. The hydrazones undergo a rearrangement in the medium of concentrated sulphuric acid to form products which can be easily detected photometrically at 520 nm. The reaction is performed in the presence of thiourea in order to provide a mildly reducing medium which prevents the interference from nonascorbic acid chromogens.

The tissues used for the detection of ascorbic acid were taken from dead mice, washed in physio-
logical saline and dried by filtration paper. Tissue samples were homogenized by MSE knife homogenizer at 8,000—10,000 rpm in 9 ml of ice-cold 5% TCA and centrifuged for 20 min at 25,000 g. 0.5 ml of supernatant was used for the detection of ascorbic acid.

RESULTS

As it is shown in Figs. 1—4, vitamin C level is decreased in all studied organs and blood serum of mice infected with T. crassiceps. Maximum decrease was observed on day 7 p.i. at the intensity of infection 15 and 30 cysticeri per mouse. This level remained almost constant during the following 3 weeks. At the infection with 5 T. cras-

Fig. 1. Changes in vitamin C concentration in the serum of mice infected with T. crassiceps. A — infection with 5 cysticeri, B — infection with 15 cysticeri, C — infection with 30 cysticeri, D — control. Abscissa — days after infection, ordinate — vitamin C level.

Fig. 2. Changes in vitamin C concentration in spleen of mice infected with T. crassiceps. For explanations see Fig. 1.

Fig. 3. Changes in vitamin C concentration in the kidneys of mice infected with T. crassiceps. For explanations see Fig. 1.

Fig. 4. Changes in vitamin C concentration in the liver of mice infected with T. crassiceps. For explanations see Fig. 1.

The decrease in the ascorbic acid level is explained in two ways. Jaklowski et al. (1964) and Jaklowski (1970a, b) assume that a great amount of vitamin C is used for the protective and adaptation reactions of the infected host. However, it is consumed also by the parasite. Our results (unpublished) demonstrated that the ascorbic acid content in the fluid of T. crassiceps cysticeri was 4.1 mg/1000 ml, which is comparable with the content in the host liver. This problem might be elucidated also by the indirect proportion between the intensity of infection with T. crassiceps and ascorbic acid level in host organs, as it was found in our experiments. The ingestion of vitamin C by the parasite is the most probable explanation of the decrease, but it should be ascertained to what extent this vitamin may be synthesized directly by the cestode. This will be dealt with in our further studies.

DISCUSSION

Our results indicate that the helminths induce a decrease in ascorbic acid level in various mouse tissue. Comparing the contradictory results of the authors it may be assumed that these changes are related to the parasite species and, undoubtedly, also to the host species. For example, the guinea pig, in contrast to other rodents, is unable to synthesize the ascorbic acid. Our results are most consistent with those of Ismagilova (1968), who infected rabbits with Taenia pisiformis, and Gameel (1982a, b), who infected rats with Fasciola hepatica.

ИЗМЕНЕНИЯ В СОДЕРЖАНИИ АСКОРБИНОВОЙ КИСЛОТЫ В РАЗНЫХ ОРГАНАХ И СЫВОРОТКЕ МЫШЕЙ, ЭКСПЕРИМЕНТАЛЬНО ЗАРАЖЕННЫХ ЦИСТИЦЕРОМ T. CRASSICEPS (ZEDER, 1860)

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Резюме. Было обнаружено, что заражение гельминтами оказывает влияние на содержание аскорбиновой кислоты в разных тканях подопытных мышей. Уровень витамина С после заражения цестодой T. crassiceps понижался в печени, почках, селезенке и сыворотке.

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