# MONOAMINE OXIDASE ACTIVITY OF RAT ORGANS DURING EXPERIMENTAL PNEUMOCOCCUS INFECTION

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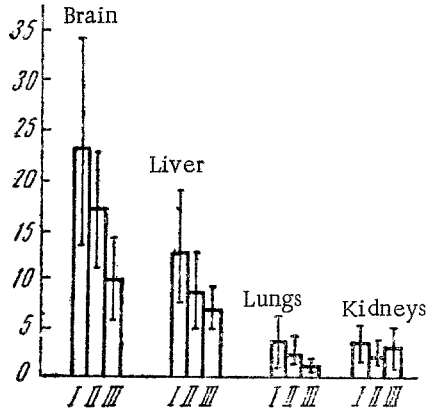
In our previous studies it was indicated that the content of serotonin in the blood and organs of experimental animals undergoes phase changes during Salmonella intoxication [1] and Pneumococcus infection [2]; in these cases the substantial rise in the level of the biogenic amine is observed at the height of the disease. There are no data the literature on a change in the monoamine oxidase activity – the enzyme that breaks down serotonin in the organism during bacterial infections and intoxications. However, there is information on a decrease in the monoamine oxidase activity in the organs during radiation sickness [5] and on a disturbance of the serotonin metabolism during allergic disease, complicated by infection [3]. In view of this, it was of interest to determine where the cause of the increased serotonin level in the organism during experimental Pneumococcus infection lies, and whether it is due to a disturbance of its biotransformation in the organism.




Variation of the monoamine oxidase activity of rat organs during experimental Pneumococcus infection. I) Normal rats; II) 1 day after infection by Pneumococcus; III) 2 days after Pneumococcus infection. Vertical lines) confidence limits of the average values. Along Y-axis) 5-hydroxyindolylacetic acid (in μg per 100 g of dry tissue).

In this work we studied the activity of monoamine oxidase in the organ and the excretion of 5-hydroxyindolylacetic acid – the chief metabolite of serotonin – with the rat urine during Pneumococcus infection.

## EXPERIMENTAL METHODS

The work was conducted on male white rats, weighing 180-200 g. The animals were infected intracutaneously wit' Pneumococcus in a dose of 0.1 ml of an 18-hour culture, diluted 1:100. The controls were intact rats. The monoamine oxidase activity was determined in homogenates of the brains, liver, lungs, kidneys, and small intestines of the animals 24 and 48 h after the injection of Pneumococcus. The enumerated organs were rapidly removed from the decapitated rats and freed of excess blood. The liver and lungs were homogenized in 2 volumes of water, the brain, kidneys, and small intestine in 1 volume of water. Serotonin (5-hydroxytryptamine, creatinine sulfate) produced by L. Light Co., Ltd. (England) was used as the substrate. The monoamine oxidase activity was judged by the formation of 5-hydroxyindolylacetic acid from the added serotonin in homogenates of the organs, converted to 100 mg of dry tissue. For the rest, the method described in [8] was used to determine the monoamine oxidase activity, and the method cited in another work [10] was used to determine the amount of 5-hydroxyindolyl-

Excretion of 5-Hydroxyindolylacetic Acid with the Urine of Rats during Experimental Pneumococcus Infection (μg per 100 g of Body Weight)

| Group of rats | Norm | Days after infection with Pneumococcus | | | |
|---|---|---|---|---|---|
| | | 1st | 2nd | 3rd | 4th |
| Infected with Pneumococcus | 4,67 (3,32 — 6,02) | 5,9 (4,31 — 7,49) | 7,86 (5,52 — 10,2) | 7,36 (5,16 — 9,56) | 5,91 (2,36 — 9,46) |
| Control | 5,77 (3,14 — 8,4) | 6,88 (5,1 — 8,66) | 8,43 (4,64 — 12,22) | 6,62 (5,15 — 8,09) | 8,95 (5,89 — 12,01) |

acetic acid, formed under the action of the homogenate or excreted with the urine (see below). The standard was 5-hydroxyindolylacetic acid produced by Sandoz Ltd. (Switzerland).

In the experiments studying the excretion of 5-hydroxyindolylacetic acid with the urine, the animals were kept on the usual ration of food and water. The 24-hour urine from the rats — control animals and animals infected with Pneumococcus — was collected before infection, and then daily for 4 days after infection with Pneumococcus. A quantitative determination of 5-hydroxyindolylacetic acid was performed in 6 ml of urine, collected from 6 rats of each group. The total of 11 experiments were conducted. The results obtained were subjected to statistical treatment and are presented in the form of average values and their confidence limits in the figure and in the table.

## EXPERIMENTAL RESULTS

A comparative study of the monoamine oxidase activity of the organs of normal rats indicated that the greatest monoamine oxidase activity is characteristic of brain tissue, the least of the tissue of small intestines of the intact rats. If we assume the monoamine oxidase activity of the brain homogenate as 100, then the activity of this enzyme comprises 55% in the liver, approximately 15% in the lungs and kidneys. A homogenate of the rat's small intestine evidently is characterized by negligible monoamine oxidase activity, which does not yield to determination by the method described above. These results coincide with the literature data [6].

In rats infected with Pneuomococcus, the monoamine oxidase activity in brain, liver, and lung homogenates is reduced, and this decrease progresses as the infection develops (see figure). Thus, one day after Pneumococcus infection, the activity of this enzyme was reduced by 28% in the brain, by 33% in the liver, and by 37% in the lungs. Two days after the injection of Pneumococcus, the monoamine oxidase activity in the brain was 69% lower, in the liver 46% lower, and in the lungs 74% lower than the activity of this enzyme in a homogenate of the corresponding organs of the control rats. Statistical treatment established reliability of the results on the second day of Pneumococcus infection. The monoamine oxidase activity of a kidney homogenate was also somewhat reduced (by 45% on the first day and 31% on the second day of the Pneumococcus infection) with respect to the monoamine oxidase activity of a kidney homogenate of control rats. However, statistical analysis demonstrated unreliability of this difference.

The table presents the amount of 5-hydroxyindolylacetic acid excreted in 24 h, calculated on the basis of 100 g of body weight of the animal. As a result of a comparison of the amounts of this acid, excreted by the rats during experimental Pneumococcus infection, the impression is created of a tendency for increased excretion of this metabolite with the urine as the infectious process develops (first to third days after infection with Pneumococcus). On the fourth day, when the severity of the infection is somewhat reduced, the excretion of a 5-hydroxyindolylacetic acid decreases. However, a statistical treatment of the results and comparison of the excretion of acid by Pneumococcus infected rats with simultaneous excretion of the indicated acid by control animals kept under the same conditions, show that the increased excretion of 5-hydroxyindolylacetic acid with the urine of rats infected with Pneuomococcus is statistically unreliable and should be considered as a normal physiological fluctuation of the excretion of this metabolite with the urine. Moreover, the content of 5-hydroxyindolylacetic acid obtained does not exceed the limits of the amounts of it determined in the urine of normal rats by other authors [4, 7, 9].

Hence, the observed decrease in the monoamine oxidase activity in the homogenates of organs and the absence of an increased excretion of 5-hydroxyindolylacetic acid with the urine of the rats during Pneumococcus infection permit us to assume that in experimental Pneumococcus infection, the monoamine oxidase activity is changed

in the bodies of these animals, decreasing during the culmination of the disease; this is one of the causes of the increased serotonin level in the blood and organs during the indicated period of the disease [2].

## SUMMARY

Rats infected with Pneumococcus were noted to have a reduction of monoaminoxidase in the brain, liver, and lung homogenates, which progressed with the development of the infection. No significant reduction in the activity of this enzyme in the kidneys was noted. The secretion of 5-oxyindolacetic acid with the urine in pneumococcus-infected rats did not differ from that seen in the control animals.

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**All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.**

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