AMMONIA POISONING IN CARP

by

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The microbiological processes that take place in flooded soils may cause serious trouble not only in plant cultivation but also in fish breeding. The harmful effect of gases produced in the mud of fish ponds has been known for a long time. A. L. Stänzl de Cronfels (7) writes in his work *„Piscinarium oder Teichordnung"* published in 1680 that during periods of great temperature rise in summer the gases that form in the mud may penetrate into the water and cause the destruction of fish. Until quite recently the study of the formation of gases in the mud and its effects was a rather neglected field of limnology despite the fact that even from a practical point of view detailed analysis of this problem complex was considered important. Until recently it was rather the oxygen supply of the fish ponds that was studied and the destruction of fish was mostly ascribed to suffocation through lack of oxygen. (6, 8). So it is understandable that algae were held to be responsible to a great extent for the destruction. In connection with this theory we have more than once observed in the ponds of Fehértó near Szeged that before sunrise thousands of carp gasped for breath, owing to want of oxygen. This phenomenon lasted only till sunrise because after this, when the algae started to produce oxygen, the animals soon recovered and disappeared. However, the algae may, under certain circumstances, become oxygen-consuming or even cause oxygen deficiency. On examination of the circumstances of such damages it was found that the oxygen deficiency is really a serious danger only when poisonous gases are also present. The gases (NH_3, H_2S) are the products of microbiological activity, or rather of the reactions of the compounds (SO_2), that form and accumulate depending on the qualities of the water and soil and especially the pH value and mechanical composition of the soil and also the temperature. The toxic effects of these gases are well-known. The two main factors of their formation and toxic effects are the soil and the weather.

Hydrogen sulphide causes damage chiefly in fish ponds on acidic, peat soils and degraded forest soils, while ammonia poisoning is more likely to occur in ponds with alkaline water.

There is a difference also in the point of time when the damaging influence of the hydrogen sulphide and the ammonia takes effect. To wit, the mass poisoning of fish through ammonia occurs chiefly in the early period of summer, especially when the dog-days heat sets in as early as the end of June or the beginning of July, whereas hydrogen sulphide poisoning is more apt to occur in the second half of summer, but it is not infrequent even in winter in the frozen ponds. Such damage occurred in the pond of Grébics near Tata in the winter of 1961—62.

The poisoning effect of ammonia on the fauna of the water of rice fields has been studied by Megyeri (2). He demonstrated that by using ammonium sulphate the animal pests of the rice plant, crustaceans of lower order, mosquito and midget larvae can be destroyed.

In the course of our investigations we have repeatedly demonstrated the ammonia, hydrogen sulphide, phosphorous hydrogen, ferrous and manganese ions formed as products of connected microbiological processes, especially bacterial activity, but we had no data at

our disposal that could have informed us as to the poisoning effects of the different compounds said to be poisonous. Besides, the question remained open as to what microbiological processes cause the accumulation of the various poisonous compounds in the water.

Since ammonia causes frequently recurring and considerable damages in fish-ponds on alkali soils, we have made investigations in order to determine its poisoning power, i. e. its toxic limit value the results of which are given below.

Material and Method

The experiments were carried out in a concrete basin of 10 m^3 capacity that could be filled up to a desired depth and a quantity of 2500—3000 l from a nearby artesian well and could also be easily drained. For the experiments the water was brought to an adequate pH value by the help of sodium hydroxide and sodium carbonate. The ammonia-containing compound used was chemically pure ammonium sulphate. The determination of the ammonia was carried out photometrically by means of NESSLER's reagent and the standard curve. Carp weighing 60—80 dg were used in the experiments.

Experimental

The ammonium-ion (NH_4) is no immediate danger to the fish. (3, 4, 5.) Its damaging toxic effect appears only when with the rising of the pH value of the water the ammonium ion is transformed into undissociated ammonia.

$$NH_4^+ + OH^- \rightleftharpoons NH_3 \cdot H_2O$$

The intensity of the formation of so-called free ammonia (NH_3) depends on the quantity of ammonium ions dissolved in water, the pH value of water, and the temperature. WOKER (10) has made studies on this subject and his results are shown in Table 1. High temperature and the increase of the pH value promotes the transformation of ammonium into ammonia. As can be seen from the table, in water with a pH value of 9,3 half of all the ammonium-ions were already transformed into ammonia. In fact, this pH value frequently occurs in the water of our fish-ponds on limy or alkali soils.

1st experiment. Before studying the effect of ammonia we thought it necessary to find out through our own experiments, too, how the fish behave in alkaline medium. These experiments bore out the statement of SCHÄPERCLAUS (5) that waters with pH values of 9,0—9,5 have in themselves no harmful effect.

2nd experiment. 10 carp, each weighing 60—80 dg, were placed into water containing 6 mg of NH_4 ions per litre. The pH value of the water was brought to 7,8. Its temperature was 18° C. The behaviour of the animals was constantly observed and since no change could be noted within 18 hours we decided that the above concentration represented no immediate danger to the carp under the given circumstances. According to the WOKER's curves only 7% of the ammonium could have transformed itself into ammonia and therefore there must have been 0,24 mg of ammonia per liter of water. So in the case of the carp this quantity does not reach the lethal dosis. The result of this experiment then bears out DONÁSZY's (1) statement that the quantity of free ammonia is tolerable up to a concentration of 0,2 mg/l.

3rd experiment. This experiment was also carried out with 10 carp in a water with a pH value of 8,2. The temperature of the water was 25° C. So much ammonium sulphate was dissolved in the water that it contained 8,43

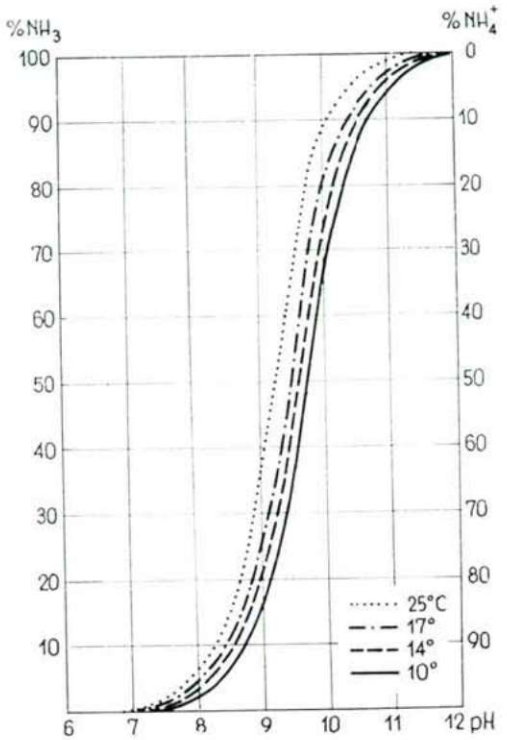

Table 1.

mg of ammonium ions, or, according to the WOKER's curves 0,67 mg of ammonia per litre. In this experiment we could observe gasping for breath at the surface which is a sign of respiratory insufficiency and the equilibrium disturbance which is a sign of disturbance of the nervous system after 17 minutes. At the 25th minute of the experiment we observed the first signs of liveliness indicating the end and which consisted in that the animal which had lost its equilibrium jumped out of the water once or twice slashing about, or swam sometimes on its back, sometimes rising out of the water in perpendicular position. After this they sank to the bottom. When an hour had passed there remained only one animal swimming in the water, but when this animal was forced to move more intensely, spinning set in in this case too, and this animal also sank to the bottom of the basin. (Fig. 1., 2., 3.)

15 minutes after sinking all the animals were taken out of the basin and placed into fresh water of 14° C temperature. Here all of them revived and, judging by outward appearance, quite recovered. From this fact the conclusion could be drawn that the toxic effect of ammonia, the change it causes, is, within a certain period, reversible.

4th experiment. The former experiment was repeated with a quantity of 0,52 mg NH_3/1 at 22° C. The result differed from the above only in that the effect became evident after 45 minutes and within 30 minutes after this all the animals sank to the bottom. After being placed in fresh water these animals, too, revived. By means of these experiments we found that 0,5 mg NH_3/litre has already a toxic effect on carp.

Fig. 1.

5th experiment. In order to find out how a higher temperature and a higher pH value influence the period of manifestation, we carried out the following experiment. We brought the pH value of water of 26° C to 8,8 and strewed so much ammonium sulphate into the water that it contained 8,5 mg of NH_4 i. e. 2,3 mg ammonia per litre. Within 4 minutes from the introduction of ammonia all the fishes rose to the surface so that their backs were sticking out of the water and at the same time they began to gasp for breath at the surface intensely. By the 13th minute of the experiment all the animals sank to the bottom after circling fast several times at the surface, slashing about and jumping out.

6th experiment. In this experiment we compared the behaviour of carp with abdominal dropsy and that of healthy carp in water that contained 0,6 mg of ammonia per litre. We found that the ammonia effect manifested istself sooner in the specimens suffering from abdominal dropsy than in healthy carp.

We could observe that the time of manifestation is proportional to the health condition of the fish.

7th experiment. With the specimens revived in fresh water we made further experiments. 12 hours after the shock these fish were again placed into water with 0,7 mg of ammonia per litre, i. e. with a value above the toxic limit. At the same time freshly caught control specimens were also put into the water. We found that the ammonia effect manifested itself already within 20 minutes

Fig. 2.

in the control animals, while the animals, which had already undergone the toxic effect, developed the symptoms only 60 to 85 minutes later. Presumably, in the case of the specimens that had formerly undergone the ammonia shock the over-compensation of the organism may prove protective against further poisoning effects.

8th experiment. In order to examine how the ammonia effect is modified when the permeability of the cell membrane is influenced, i. e. changed, the following experiment was carried out. 8 marked carp were each perorally given half a pill of *Suprastin* which reduces the permeability of the cell membrane and which contained 12,5 mg of N-dimethyl-aminoaethyl-N-p-chlorbenzyl-α-aminopyridin hydrochlor. After 30 minutes these fish were put, together with 8

untreated control specimens, into water containing 0,72 mg of ammonia per litre. In the course of this experiment of orientative nature it could be observed, that the control specimens sank to the bottom between the 17th and the 30th minute of the experiment after having developed the characteristic symptoms, while the treated and marked specimens did not sink to the bottom although they showed serious disturbances of equilibrium.

Fig. 3.

As to the origin of the ammonia we established that the destruction of algae and water plants following the bloom of the water are the biological factors that increase the ammonia content of the water. We think that one of the direct reasons for the perishing of water plants and algae is the hydrogen sulphide that becomes liberated from the mud VÁMOS (9).

Summary

The author carried out experiments in order to examine the toxic limit, the poisoning power of free ammonia and the symptoms of ammonia poisoning.

It has been stated that the toxic limit of ammonia is 0,5 mg/l. After the shock effect the sunken specimens can be revived in fresh water. In these specimens repeated poisoning with the same degree of ammonia concentration produces symptoms only after a longer time of manifestation. It was also observed that drugs (Suprastin) reducing the permeability of the cell membrane reduce the poisoning effect to a certain extent.

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