Acta Veterinaria Academiae Scientiarum Hungaricae, Tomus 27 (1—2), pp. 99—113 (1979)

GILL SPHAEROSPOROSIS IN THE COMMON CARP AND GRASSCARP

By

K. MOLNÁR

Veterinary Medical Research Institute, Hungarian Academy of Sciences, Budapest

(Received May 4, 1977)

Certain *Sphaerospora* (*Myxosporida*) species are common parasites of fresh-water fishes.

Among the 17 species described in SCHULMAN's (1966) monograph, only *Sphaerospora carassii* Kudo, 1919 is a gill parasite, the others establish themselves in the kidneys and urinary tract. According to SCHULMAN, apart from *Carassius carassius* the fishes *Carassius auratus gibelio, Cyprinus carpio* and *Rutilus rutilus* serve as hosts of *Sph. carassii*, and the parasite, originally described in Japan, is indigenous in several European habitats, including the catchment area of the river Danube. In Siberia, USSR, RAZMASHKIN and SKRIPTSHENKO (1967) described a new species under the name *Sphaerospora branchialis* in the gill of the common carp, and KASHKOVSKIY et al. (1974) claimed to have found, besides *Sph. branchialis*, the species *Sph. carassii* and *Sph. cyprini* (Fujita, 1912) in the gills of *Cyprinus carpio*, *Carassius auratus gibelio* and *Rutilus rutilus* hosts. In Bulgaria, LOM et al. (1976) found *Sph. carassii* in the common carp and postulated its identity with *Sph. branchialis.*

In Hungary, MOLNÁR (1971) was the first to report the occurrence of *Sphaerospora* parasites in the gills of the grasscarp, and to identify them as *Sph. carassii.* Later HÁMORY and MOLNÁR (1972) found the same parasite species in the gills of common carps in a pond farm, and attracted attention to the hazards of sphaerosporosis in cultured fish populations. According to NOGEROV (1974) and ALIGADZHIYEV et al. (1974), gill sphaerosporosis has been an economically relevant parasitosis also in the pond farms situated in the southern areas of the USSR.

Among the scanty data available on the normal histology of the gills, those published by KOVÁCS-GAYER (1975) on the carp and by MOLNÁR (1972) on the grasscarp were relied upon in the present study, in which we intestigated the incidence, epizootiology, pathogenicity and pathohistology of gill sphaerosporosis caused by *Sph. carassii* in pond farms in Hungary.

Materials and methods

Cultured fishes, above all carp and grasscarp fry taken from the largest pond farms, were regularly examined from 1970 on to assess the frequency of occurrence of sphaerosporosis on a country scale. After this preliminary study, the examinations were limited to those pond farms in which a notable proportion of the population was constantly affected by the disease.

Ten to 15 fishes were taken from the rearing ponds (6000 m^2 surface area each) in the most massively infected farm at weekly or biweekly intervals, for parasitological examination. Fishes from the same pond were used for aquary experiments and histological study. Follow-up of the parasitosis for longer than 4 months was possible in only 3 cases, for managemental reasons. As the introduction of fishes from other units was prohibited, sphaerosporosis-free scaled common carp and grasscarp fry hatched in the same farm was transferred to the infected mirror carp ponds for experimental study, or carp fry hatched under parasite-free conditions was placed in plastic mesh containers and immersed in the infected ponds for the same purpose.

Aerated aquaries, each 40 litre in volume, were used for the laboratory experiments, during which the fishes were fed on a commercial diet (meat meal + crushed maize).

The fishes were starved for 3 days before collection of the spores. These could be most easily recovered by placing the hosts in an aerated conical jar for a few days. After removal of the hosts, the spores sedimented at the bottom of the jar were collected by pipetting. Part of the spores to be used for experimental infection was stored in tap water at room temperature, part was immersed in the pond within a glass jar covered by a dense mesh, and was maintained there from November to March.

Fishes originating from non-infected pond farms and reared under parasite free-conditions were used in the aquary experiments. The spores used for the experimental infection of these hosts were administered either through a gastric tube or mixed in the diet. Water temperature ranged from 20 to 24 °C. Fishes with current parasitosis (dactylogyrosis, trichodinosis, chilodonellosis) occurring in some of the aquary populations during the experiments, were cured by dipping them for 15 min in 2.5% saline.

The gills were fixed for histological study in 4 or 10% formalin, or in Bouin solution. Paraffin sections cut in the longitudinal and sagittal planes were stained with haematoxylin and eosin, Farkas-Mallory or Giemsa stains.

Results

Incidence

Among 7 pond farms regularly surveyed from 1969 to 1976, sphaerosporosis occurred in 5. The carp population was involved in all 5 farms, while the grasscarp population in only 2. Among the carps exclusively the fry was affected, while among the grasscarps also part of the one- and two-summer fish harboured the parasites. The extensity of infection never exceeded 10% in the grasscarp populations, but ranged from 30 to 100% among the carps. The individuals checked within a given population were as a rule either massively infected or free from any indication of sphaerosporosis. A low degree of infection was only exceptionally found.

Clinical observations and diagnostic considerations

The infected hosts did not show any characteristic symptom, nor any notable difference in body weight compared to non-infected pond mates. The most conspicuous change was the pale pink discoloration of the gills and gritty appearance of the gill mucosa and gill lamellae, owing to desquamation of shreds of epithelium packed with spores.

Occasionally, there was also fragmentation of the gill lamellae, but in such cases some complication, *e.g.*, branchiomyces infection, gill necrosis and, still more often, chilodonella or trichodina invasion, was as a rule present. Firm diagnostic evidence was obtained by demonstration of spherical or slightly spheroid spores, $8-13 \times 8-12$ μm in size, in scrapings of gill mucosa. In the early stages of sphaerosporosis, round or elliptic (occasionally amorphous) plasmodia (trophozoites) 12—20 μm in diameter, with a finely granular cytoplasm, were also detected in scrapings. Early diagnosis while the parasite is in the vegetative (trophozoite) stage may be hampered by the presence in the gills of acidophilic cells which perform amoeboid movements and differ from the plasmodia practically only in the coarseness of their cytoplasmic granules.

Field observations

1. In 1974, a newly-established fish pond was stocked with 1 million newly-hatched carps one week after having been filled with water. Three weeks after transfer, 100% of the fry were found to be infested by sphaerospores (on June 20). Subsequently, parasitological check-up was made every week, and all fishes examined until de-stocking on October 1, on a total 140, were found to be infected. Mortality from sphaerosporosis was not demonstrable. The fishes were marketed in the autumn. For experimental purpose, carps and grasscarps, 1.5 months younger than the infected population, had been introduced into the pond while it had been stocked, but we failed to recover any carp of this lot, and only two grasscarps could be recovered. These were both free from sphaerospores. Another experimental approach to follow-up of the infection by study of fish groups immersed into the pond in wire-mesh cages failed for technical reasons.

2. In several ponds of the same farm, sphaerosporosis continued to occur for several subsequent years, but the extensity of the infection never exceeded 50%.

Observations on spores

1. Spores were continuously released from the gills of the infected fishes throughout the 4-month period of observation.

2. Although the majority of the spores became deposited on the bottom of the aquary, floating spores were still found 6 h after removal of the host from the aquary.

3. All spores were found on the bottom of the tube after centrifugation at 100 rpm for 5 min.

Experiments in the aquary

1. Spontaneously infected 1.7—2 cm long mirror carps were transferred from the pond to the aquary on June 20, and 2 specimens each were subsequently killed for study every other week. Massive sphaerosporosis of the gills was found in each case studied until October 2, after which the majority of the fishes died of chilodonellosis. The three survivors were killed on November 3. Two of them harboured only a few spores on the gills, the third was as massively infected as the fishes examined before October 2.

2. Two and a half cm long scaled carps originating from a sphaerospora-free pond farm were placed in the same aquary with similarly long mirror carp fry from the above mentioned infected pond farm, and were kept together for 3 months. Not a single infected specimen was found in the groups of 5 scaled carps killed and examined at the end of each month.

3. Massively infected, starved carp fry passing spores was placed in an aquary. Two days later the fry was removed and the spores were allowed to mature in the aquary for 5 months at 20—22 °C. Subsequently, 5 fishes, reared under parasite-free conditions, were placed in the aquary. All 5 fishes were found to be free from sphaerospora infection when killed after 40 days.

4. Spores stored in water (20 °C) for a few days, or 1, 2, 3 or 4 months, were mixed in the diet of the fishes and fed with them. No sphaerospora infection occurred. Feeding with spores overwintered in the pond either in the diet or through a gastric tube, did not give rise to infection either.

5. Thirty fishes, from a population of which about 50% had been found to be infected by sphaerospores in previous studies, were placed in an aquary. No attempt was made to cure the fishes from concurrent chilodonella and trichodina infections. From day 10 after transfer to the aquary deaths began to occur. A severe degree of gill chilodonellosis developed. Examination of fishes in the terminal stage of disease and of fresh carcases showed that 8 of the 9 fishes studied between days 10 and 27 harboured sphaerospores. Four fishes which died between days 27 and 32, as well as 3 survivors killed on day 33, were free from sphaerospores, carrying only large masses of *Ciliata* on the gills. The remaining 14 carps, which died during the period of the experiment, were unsuitable for examination. The 20 carps taken of the same pond to serve as controls and cured of chilodonellosis by treatment with 2.5% saline all survived. They were killed at the conclusion ot the experiment. Sphaerospora infection was found in 11.

6. Mud samples taken from different parts of a pond massively infested by sphaerospores were placed in an aquary, which was subsequently stocked with 2-week or 4-week-old carp fry free from sphaerospora infection. Hosts killed at regular intervals showed that the fry did not contract sphaeroporosis over a period of 2 months.

7. Twenty 3—3.5 cm long carp fry originating from a sphaerospora-infected population were kept in a closed container (at decreasing O_2 concentration), and the carcases were removed and examined in the sequence of dying. Among the first 10 carcases 7, while among the second lot of 10 carcases only a single host, had harboured sphaerospores.

8. A group of grasscarps from a sphaerospora-infected population was transferred to an aquary on September 1. Sphaerospora infection was found to persist some of the hosts killed on December 10 and in the spring next year.

Histological studies

Gill parasitosis can be best studied in longitudinal sections, because in these the respiratory folds are arranged along the axis of the gill lamellae like the branches of a tree, and the interlamellar stratified epithelium, the stratified epithelial coat surrounding the lamellar tips and margins as well as the epithelium of the gill arches can be easily distinguished.

Parallel use of haematoxylin-eosin and Farkas—Mallory's stain proved to be the technique of choice. The former makes possible the study of fine details in the cell, but the spores are less easily distinguishable from their surroundings whereas the latter is less suitable for studying details, but enables easy differentiation of the parasite's developmental stages, imparting a pale blue shade to developing spores, first bluish-violet, then red hue to developed ones, while the mature stain yellow. Spores are also demonstrable by the Giemsa technique, but without metachromasia.

Microscopic lesions

The microscopic appearance of sphaerosporosis was identical in the grasscarp and carp. The spores and developmental stages localized exclusively in the stratified gill epithelium. The parasites established themselves in similar numbers in the epithelial coats of gill lamellae, gill arches and gill rakers, but did not in the respiratory epithelium of the gills. A spore found in a single case may be attributed to secondary spread. Sphaerospores were not found in any non-epithelial tissue of the gills.

In the normal gills, cells constituting the deep layers of the stratified gill epithelium have irregularly shaped, chromatin-rich nuclei surrounded by a narrow cytoplasm, while the surface layers are composed of more differentiated cells with a clear cytoplasm and nuclei less abundant in chromatin, as well as of goblet cells. Occasionally, a few acidophilic cells, with granular cytoplasm, were seen among the undifferentiated cells of the deep layers of gill epithelium (Fig. 1). In sphaerospora-infected fishes, 70—80% of the total volume of gill epithelium was occupied by spores and developmental

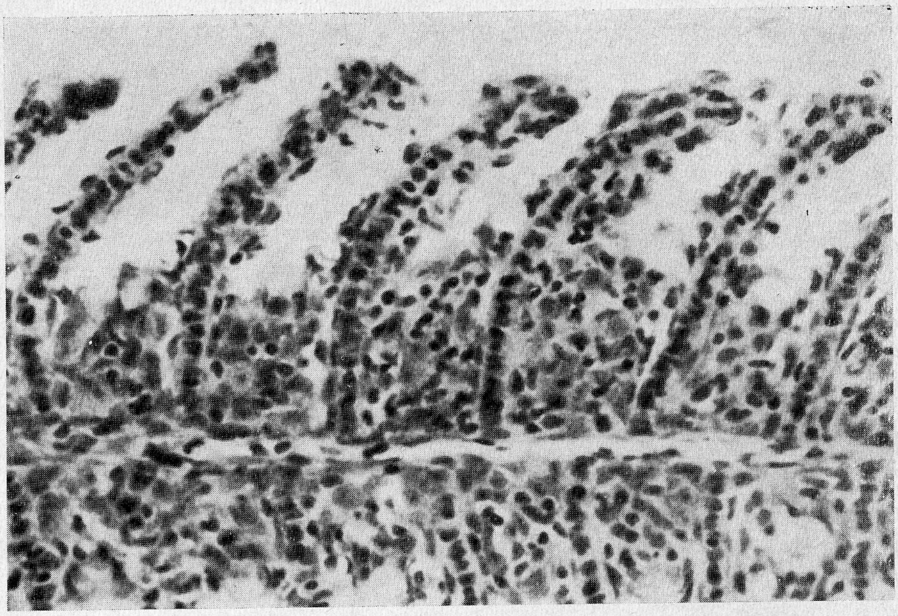

Fig. 1. Gill lamella of a sphaerosporosis-free carp in longitudinal section. H. and E., ×300

stages (Fig. 2). Early stages always established themselves in the deeper layers of the epithelium. It was well-visible, especially in sagittal sections that the youngest stages began to develop between the undifferentiated cells forming the stratum germinativum immediately above the connective tissue cells separating the latter from the cartilaginous tissue. The earliest stages seen in histological preparations were the pansporoblast containing 8 to 14 nuclei (Fig. 3). The small nuclei of the youngest pansporoblasts assumed a homogeneous dark shade due to haematoxylin, while in older sporoblasts certain nuclei, *e.g.* those forming the capsule of the spore, became faded, others, *e.g.* those of the vegetative germ or at the polar capsule became enlarged. Within each pansporoblast, 2 spores arose, which remained attached to each other for a while by the chromatophobic residue of the pansporoblast (Fig. 4). The spores then were gradually lifted near to the epithelial surface, where the more mature spores were localizing. In fishes infected by sphaerospores, the multinucleated pansporoblasts and developing spores accounted for further deformation of the undifferentiated cells, anyway poorly furnished with cytoplasm. Compression by the developmental stages accounted for semilunar, triangular or polygonal deformation of the nucleus, and the extremely thin rim of the cytoplasm formed a supporting net around the sporoblast and spores. Necrosis of the cells wedged between the spores resulted in the formation of spore aggregations each consisting of 15 to 30 spores.

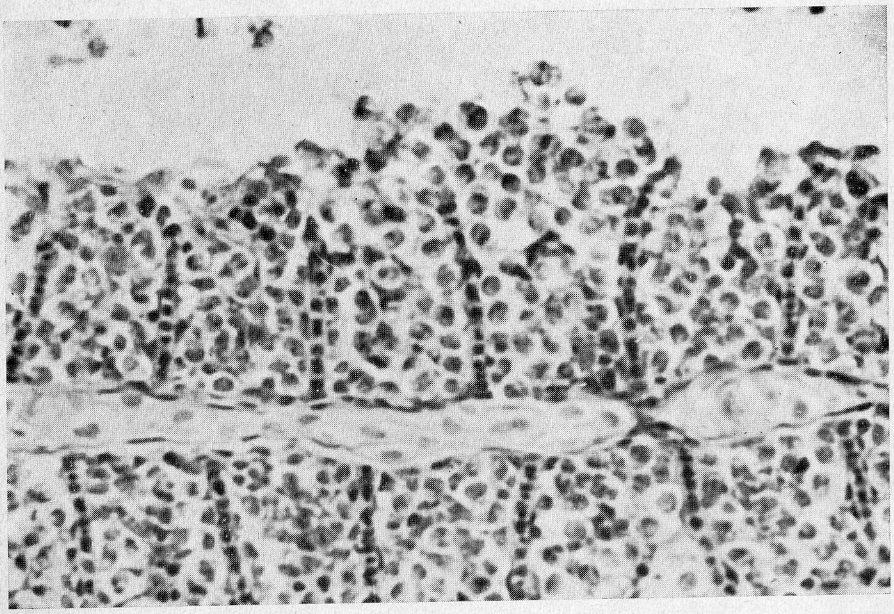

Fig. 2. Gill lamella of a sphaerospora-infected carp in longitudinal section. The interspaces between the gill folds are filled by spores. Note extrusion of spores from the interspaces in centre. H. and E., ×300

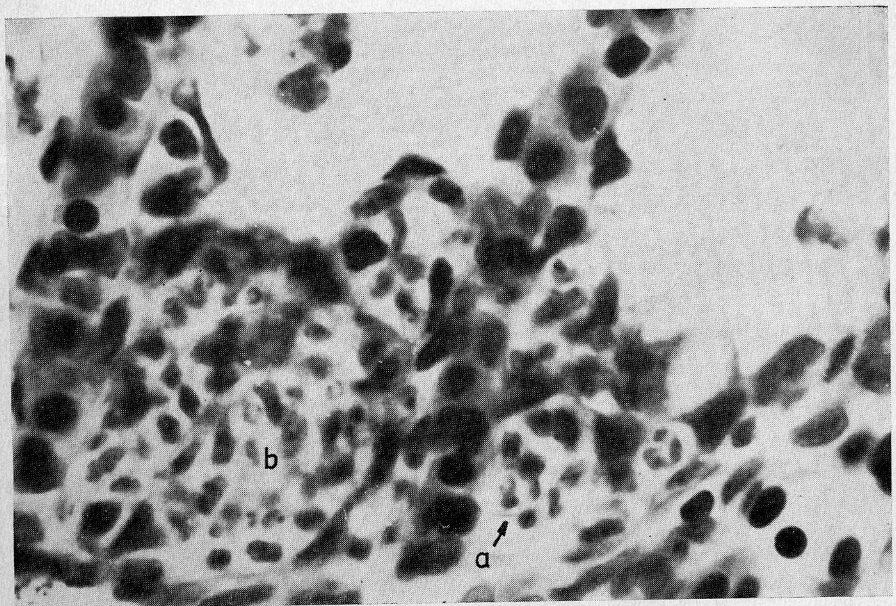

Fig. 3. Early stages of *Sph. carassii* in the gill lamella of a carp. *a*, pansporoblast, *b*, chromatin-rich layer formed by admixture of sphaerospora stages and nuclei of degenerated epithelial cells. H. and E., ×1000

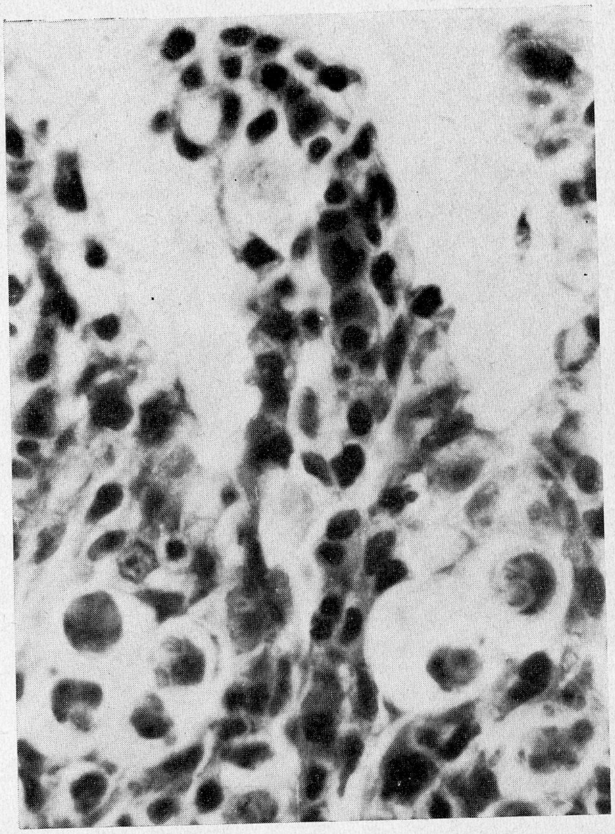

Fig. 4. Spores localizing in pairs inside oval residues of pansporoblast. H. and E., ×1000

Despite compression by the many spores belows, the superficial epithelial cells with round or elliptic nuclei and pale, but abundant, cytoplasm maintained a more or less continuous layer (Fig. 5); through the gaps occurring here and there, spores became extruded to the outside world (Fig. 2).

At the respiratory border of the gill lamellae the spores localized in the epithelium between two adjacent respiratory folds (Fig. 5). The multinucleated pansporoblasts and the deformed nuclei of the undifferentiated cells formed a homogeneous, chromatin-rich mass at the bottom of the interspaces between respiratory folds (Fig. 3). In advanced sphaerosporosis spores also appeared in the deeper layers. In the latter case, the nuclei of the host cells were either sandwiched between spores, or 6 to 8 nuclei formed islet-like aggregations in interspaces between spores. The endothelium of the respiratory folds was not affected by the parasites, either along its interepithelial or its free portion.

The stratified epithelium surrounding the tips and edges of the gill lamellae was evenly filled by spores and developmental stages, between which nuclei of deformed epithelium cells were recognized in places (Fig. 6). One

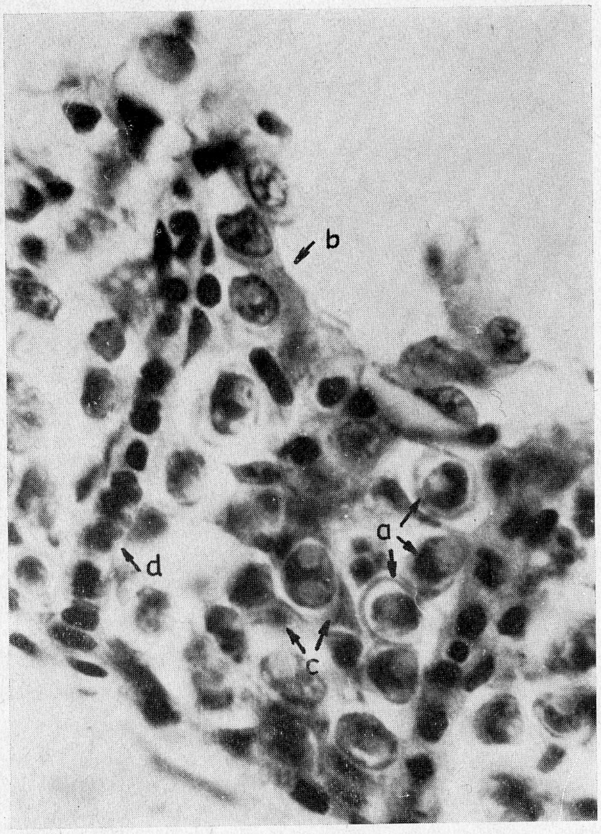

Fig. 5. Interspaces of gill folds packed with sphaerospora stages. *a*, spore; *b*, differentiated epithelial cells; *c*, deformed epithelial cell nuclei; *d*, respiratory fold. H. and E., $\times 1000$

or two layers at the external surface appeared less affected, it showed flattening and even discontinuity in places.

The stratified epithelium of the gill arches and gill rakers was as a rule less massively infected than that of the gill lamellae. The localization of the parasites was the same: the early stages were found in the deeper layers, whereas the spores near the surface. Spores were only exceptionally found in the superficial epithelium, especially in the goblet cells, and few spores occurred near-by the base of gill rakers and sensory buds. Extrusion of the spores usually occurred in the interspaces between sensory buds, where they were released in groups of 8 to 10, after disruption of the surface epithelium (Fig. 7).

Concurrent infections

In the cases in which chilodonellosis or trichodinosis were concurrent with sphaerosporosis, the respiratory epithelium also became involved. The

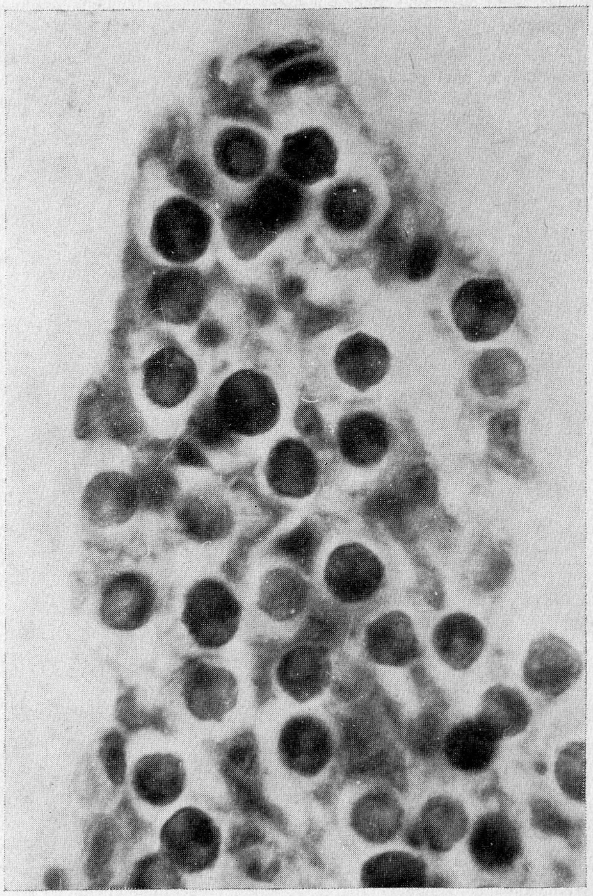

Fig. 6. Sphaerospores at the tip of a gill lamella. Farkas-Mallory's stain, ×1000

histopathological changes caused by *Mucophilus* invasion were, however, practically the same as those due to sphaerosporosis. Simultaneous occurrence of the above complications was also observed in aquary experiments (Fig. 8).

Other common complications were branchiomycosis and gill necrosis, which involved, besides the stratified epithelium, connective tissue, blood vessels and cartilage, accounting occasionally for necrosis and detachment of the entire affected gill lamella. Such phenomena never occur in pure sphaerosporosis.

Proliferation of acidophilic (pseudo-basophilic) cells, which are normal elements of the gill, was often taking place simultaneously with the invasion of sphaerospores. Although the acidophilic cells resemble the vegetative stages of sphaerospores in both shape and size, in stained preparations they can be easily distinguished from the latter by the marked acidophilia of their coarse cytoplasmic granules, while the trophozoites show little affinity

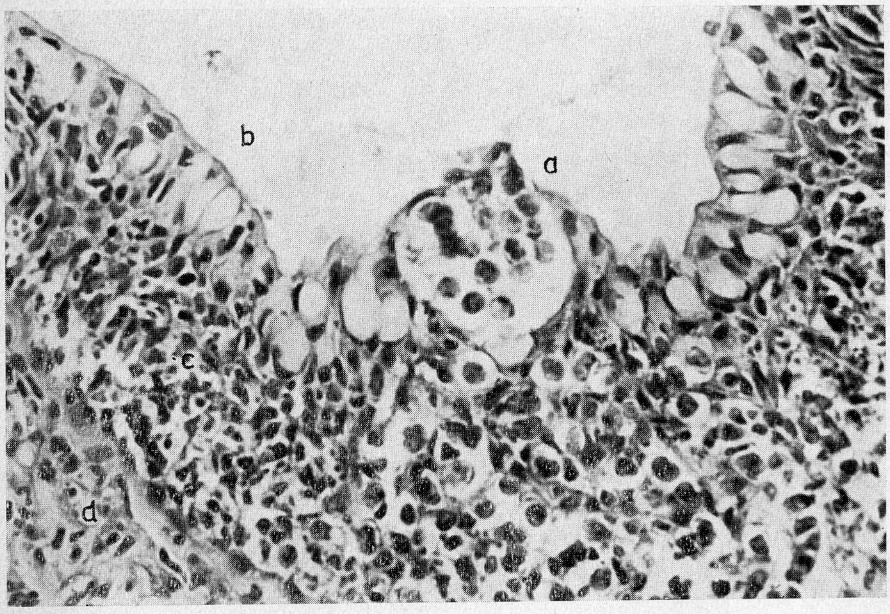

Fig. 7. Sphaerospores in the process of extrusion from the epithelial coat of a gill arch, *a*, spores breaking through the superficial layer; *b*, goblet cells; *c*, developmental stages in the stratified epithelium; *d*, connective tissue layer. H. and E., ×300

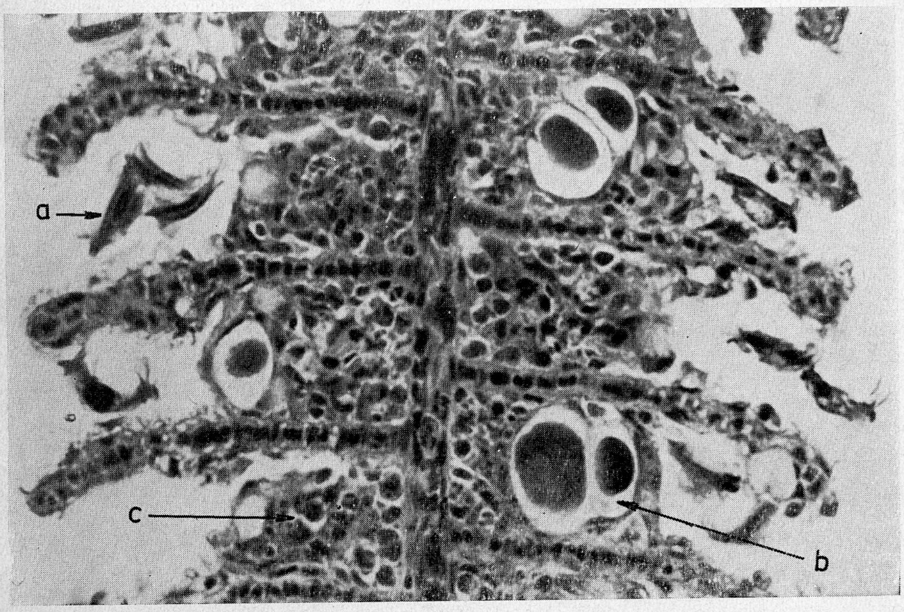

Fig. 8. Concurrent infection by ***Trichodina*** and ***Mucophilus*** species in the gills of a carp diseased in sphaerosporosis. *a*, trichodina stages; *b*, mucophilus stage; *c*, sphaerospora spores inside the epithelium. H. and E., ×300

to dyes. Acidophilic cells are abundantly present in the gills of sphaerospora-free fishes, too.

Apart from *Sph. carassii*, the kidney parasite *Sph. angulata* Fujita, 1912, is another common sphaerospora ot the carp. The two fish sphaerospores differ in both localization and morphological features.

Discussion

Earlier studies of sphaerospores affecting cyprinid fishes (MOLNÁR, 1971; HÁMORY and MOLNÁR, 1972) have led to the conclusion that the criteria established by RAZMASHKIN and SKRIPTSENKO (1967) are not convincing enough for the creation of a new species, *viz. Sph. branchialis.* As a result of further detailed investigations, the parasite responsible for sphaerosporosis in both common carp and grasscarp has been identified as *Sph. carassii* Kudo (1919).

According to our findings, *Sph. carassii* is very widely spread in the pond farms of Hungary. The annual recurrence of sphaerosporosis is a fairly regular phenomenon in certain pond farms, but the extensity of infection varies by units. Usually, only part of the pond population has sphaerosporosis, 100% infectedness occurs rarely. In the infested pond farms the fry is more frequently affected than the older fishes, and generally 30—50% of the population harbours sphaerospores. Sphaerosporosis is less frequent among grasscarps than among common carps.

The intensity of infection is always high. The examined fishes were either massively infected, or free from sphaerospores; mild infection was encountered in only two instances throughout the study. In one instance, two carps killed at the end of the first aquary experiment were found to harbour only a few spores, obviously retained in the tissues after completion of the developmental cycle. The other instance, in which very few spores, but masses of developing pansporoblasts were found at autopsy of a 3.5 -weeks-old young carp, obviously represented a very early stage of sphaerosporosis.

The severity of the sphaerosporosis in the invaded fishes cannot be attributed either to a single massive infection or to lasting exposure to minor invasions. Both would presuppose high concentrations of spores in the pond. A more feasible explanation seems to be that an intake of even a few spores by the host is followed by such an intensive multiplication of the vegetative stages that a massive invasion of the gill epithelium necessarily ensues.

Authors generally agree that the spore-bearing unicellular parasites give rise to infection in fishes after having gained access to the gut on intake from the soil (SCHULMAN, 1966; KOCYLOWSKI and MYACZYNSKI, 1963). It is however, still not known how the spores become infectious. According to Hoffman and Putz (1969), *Myxosoma cerebralis* spores required at least 4 months long maturation in the soil. Our attempts at an experimental infection of fishes under aquary conditions failed, although, tens of thousands of spores were used for this purpose. Observations in the ponds have also indicated that more intricate interactions than generally implied may play a role in the mechanism of infection by sphaerospores. Considering that in the infested ponds the extensity of infection established at the beginning did not rise further during the observation period, we may conclude that the carp fry becomes infected in a very early stage of life. Since the young fry feeds almost exclusively on plankton, the hazard of spore ingestion is considerably less than later, when bentos is additionally consumed. The exclusive responsibility of spores deposited at the bottom of the pond is contradicted by the practical observation that the most massive (100%) infection met in the course of this study occurred in a newly-established pond, into which no parasite could gain access otherwise than in the flood water. It was shown in the present study that the *Sph. carassii* spores sediment very slowly and may therefore be carried to great distances by the inflowing water; nevertheless, the role of aquatic organisms in their spread cannot be excluded.

Evidence that sphaerospora infection can persist in an aqueous habitat for over 4 months has been emerging from the aquary experiment no. 1. During this period, new generations of spores arose in succession in the deep layers of the gill epithelium, and become extruded into the outside world at regular intervals on breaking through the superficial layers.

The observations made on the sphaerosporosis of the grasscarp are less informative, yet, clearly indicate that the infection may persist in a population considerably longer than 4 months and that, in addition to the fry, one-summer and two-summer fishes may become involved.

Since attempts at experimental infection have failed, spontaneous cases were analyzed from epizootiological and histological aspects to obtain more information about the life cycle of *Sph. carassii*. Our tentative conclusions are forthcoming:

The vegetative stages, detectable exclusively in unstained preparations, multiplied intensely between the cells constituting the stratum germinativum of the stratified gill epithelium, and gave rise to pansporoblast containing 8 to 14 nuclei. The pansporoblasts were well-visible in stained slides; two spores developed within each. No evidence could be obtained whether the development in the gills had been preceded by multiplication of some early developmental stage elsewhere.

The study of histological preparations has clearly shown that for *Sph. carassii*, the site of preference is the stratified gill epithelium, irrespective whether associated with the lamellae or arches. Maturation of the spores and the development of young spores in the deep epithelium accounts for a certain degree of injury also in the superficial layers: the nuclei become flat and discontinuities arisen in consequence of cell necrosis promote the extrusion of spores. Not more than 30 spores form the groups extruded from the interspaces between respiratory folds but the large patches of stratified epithelium occasionally shed from the lamellar edges may contain considerable masses of spores. Detached spores and patches of epithelium, along with tissue fluids released from the injured parts, impart to the gills the opaque, gritty appearance characteristic of sphaerosporosis. Owing to the great intensity of the infection, the injury of the stratified epithelium is very severe, but since its main function is to serve as a supporting structure, and the respiratory folds do not become affected by the parasite, *Sph. carassii* is less pathogenic than implied from the intensity of infection. This can account for the surprising observation that in a 100%-infected pond population practically no death occurred from sphaerosporosis. However, in spite of its low pathogenecity, the role of *Sph. carassii* in fish mortality has been interpreted by certain authors (Razmashkin and Skriptshenko, 1967; Hámory and Molnár, 1972; Nogerov, 1974; Aligadzhiev et al., 1974). As shown by the results of experiment no. 5, the parasite accounts for a considerable depression of resistance in the infected host. The infected fishes proved to be less resistant to other parasitic disease or to environmental stress than those free from sphaerosporosis. The tissue injuries caused by the extrusion of spores serve as gates for the invasion of microorganisms. Damage of the undifferentiated epithelium cells by developing stages interferes with the regeneration of the gills, thereby favouring the attack of ciliated or flagellated protozoa on the respiratory folds, which are never invaded by sphaerospores.

It follows that, although in sphaerospora-infected populations the direct cause of mortality is as a rule a concurrent disease, the background of the fatal outcome is the sphaerosporosis. In this light, the losses reported by Hámory and Molnár (1974) may be attributed to sphaerosporosis complicated by gill necrosis. The excessive proliferation of the acidophilic (pseudobasophilic) cells at the onset of sphaerosporosis may well have been a consequence of gill necrosis; Kovács–Gayer (1977) has interpreted the mass occurrence of such cells as an early sign of necrosis.

Our 1st fish-pond experiment supports the view that a well-fed, well-growing, adequately controlled fish population made free of all current diseases except sphaerosporosis can be reared to market size without considerable loss. In the practice, however, it is more likely that mass losses will be caused by a concurrent disease in the population whose resistance has been depressed by sphaerosporosis.

Summary

Gill sphaerosporosis has been shown to be a frequent disease of the carp *(Cyprinus carpio)* and grasscarp *(Ctenopharyngodon idella)* fry in the pond farms of Hungary. The extensity of infection reached even 100% in a rearing pond. The spores and developmental stages of the causal agent, *Sphaerospora carassii* Kudo, 1919, localized exclusively in the stratified epithelium of the gills, where the infection may persist longer than 4 months. During this time spores are being produced and extruded continuously. The intensity of the infection is invariably high — the spores may occupy up to 80% of the stratified epithelium coating the lamellae and arches of the gill. Compression by the growing parasites accounts for polyangular deformation of the cell nuclei and extreme thinning of the cytoplasm, which surrounds the spores and other stages like a net. The superficial layers of epithelium are less affected, but are in places interrupted by discontinuities due to extrusion of spores to the outside world. Since *Sph. carassii* does not involve the respiratory epithelium, it is moderately pathogenic, but depression of resistance by sphaerosporosis may account for the fatal outcome of concurrent parasitic or other infections.

References

Алигаджиев, А. Д., Атаев, М. А., Газимагомедов, А. А. and Хайбулаев, К. Х. (1974): О паразитах рыб прудовых хозяйств Дагестанской АССР. VI. Всес. совещ. по болезням и паразитам рыб. Тезисы. Москва, 7—10.

Hámory, G. and Molnár, K. (1972): Egysejtű paraziták okozta ivadékbetegségek tógazdaságokban. Magyar Állatorvosok Lapja (MÁL) **27**, 358—360.

Hoffman, G. L. and Putz, R. E. (1971): Effect of freezing and aging on the spores of *Myxosoma cerebralis*, the causative agent of salmonid whirling disease. Prog. Fish-Cult. **33**, 95—98.

Кашковский, В. В., Размашкин, Д. А. and Скрипченко, Э. Г. (1974): Болезни и паразиты рыб рыбоводных хозяйств Сибиры и Урала. Свердловск.

Kovács-Gayer, É. (1975): A halak kopoltyújának vizsgálata. I. A ponty *(Cyprinus carpio)* kopoltyújának anatómiája és szövettana. MÁL **30**, 707—712.

Idem (1977): A halak kopoltyújának vizsgálata. II. A ponty kopoltyú-necrosisának vizsgálata. MÁL **32**, 119—124.

Kocylowski, B. and Myaczynski, T. (1963): Halbetegségek. Budapest.

Kudo, R. (1919): Studies on *Myxosporidia*. A synopsis on genera and species of *Myxosporidia*. Biol. Monogr. **5**, 1—265.

Lom, J., Golemansky, V. and Grupcheva, G. (1976): Protozoan parasites of carp (*Cyprinus carpio* L.). A comparative study of their occurrence in Bulgaria and Czechoslovakia, with the description of *Trichodina perforata* sp. n. Folia parasit. (Praha) **23**, 289—300.

Molnár, K. (1971): Protozoan disease of the fry of herbivorous fishes. Acta vet. hung. **XXI**, 1—14.

Molnár, K. (1972): Studies on gill parasitosis of the grasscarp *(Ctenopharyngodon idella)* caused by *Dactylogyrus lamellatus* Achmerov, 1952. IV. Histophathological changes. Acta vet. hung. **XXII**, 9—24.

Ногенов, У. О. (1974): Паразитофауна рыб в прудовых хозяйствах Кабардино—Балкарской АССР. VI. Всес. совещ. по болезням и паразитам рыб. Москва. Тезисы, 167—169.

Размашкин, Д. А. and Скрипченко, Э. Г. (1967): Болезни рыб в прудовых хозяйствах Западной Сибири и Урала. Озерное и прудовсе хозяйства в Сибири и на Урале. Тюмеиь.

Шульман, С. С. (1966): Миксоспоридии фауны СССР. Москва—Ленинград.

Address of the author: Dr. Kálmán Molnár, 1143 Budapest, Hungária krt. 21. Hungary