Sonderdruck aus Journal of Applied Ichthyology
Bd. 2 (1986), H. 2, S. 86–94
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# Experimental induction of *Sphaerospora renicola* (Myxosporea) infection in common carp *(Cyprinus carpio)* by transmission of SB-protozoans

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*Received for publication January 18, 1985*
*Accepted Sept. 23, 1985*

**Summary**

The authors have experimental evidence that the protozoa causing the swimbladder inflammation (SBI) of the common carp *(Cyprinus carpio)* are indentical with presporogonic stages of *Sphaerospora renicola* Dyková et Lom, 1982 parasitizing the renal tubules. Homogenates prepared from the thickened and inflamed swimbladder of naturally infected common carp, when injected into the abdominal cavity of fish, produced renal sphaerosporosis in the infection-free common carp if the homogenates contained the parasites described by Kovács-Gayer et al. (8). By intraperitoneal injection, the Unidentified Blood Organisms (UBOs) living in the blood of the common carp were transmissible to common carp, from the blood of which they were demonstrable for a long time. However, they were not transformed into *Sphaerospora*. To other cyprinids (gibel carp, silver carp, grass carp, tench, roach) neither the blood stages nor the swimbladder stages were transmissible from the common carp.

**Zusammenfassung**

*Experimentelle Sphaerospora renicola (Myxosporea) Infektion beim Karpfen (Cyprinus carpio) durch Übertragung von SB-Protozoen*

Intraabdominale Injektion von Homogenaten aus entzündeten und verdickten Schwimmblasen der Karpfen (mit Präsenz der von Kovács-Gayer et al. (1982) beschriebenen Parasiten) verursachten Nieren-Sphaerospore. Die im Blut der Karpfen vorkommenden UBO (unidentified blood organisms) waren zwar experimentell auf gesunde Karpfen übertragbar, eine Umwandlung in Sphaerosporen konnte aber nicht beobachtet werden.

Weder die im Blut noch die in Schwimmblasen der Karpfen existierenden Parasiten konnten auf andere Cypriniden (Silberkarauschen, Graskarpfen, Silberkarpfen, Schleie, Plötzen) übertragen werden.

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J. Appl. Ichthyol. 2 (1986) 86–94

ISSN 0175–8659

Die Ergebnisse dieser Arbeit zeigen, daß die Schwimmblasenentzündung der Karpfen durch *Sphaerospora renicola* (Dyková und Lom 1982) induziert werden kann.

**Résumé**

*Introduction expérimentale de l'infection Sphaerospora renicola (Myxosporea) chez la carpe commune (Cyprinus carpio) par transmission de SB-protozoaires*

Les auteurs ont démontré experimentalement que les protozoaires (SBP) causant l'inflammation de la vessie natatoire de la carpe commune *(Cyprinus carpio)* sont identiques aux stades présporogoniques de Sphaerospora renicola Dyková et Lom, 1982, parasite vivant dans les tubulures rénales. Des préparations homogènes faites à partir de la vessie natatoire enflammée et ayant augmenté de volume de la carpe infectée par voie naturelle ont causé, lorsque injectés dans la cavité abdominale du poisson, une shaerosporose rénale chez la carpe non infectée si les préparations contenaient les parasites décrits par Kovács-Gayer et al. Les U.B.O., organismes du sang non identifiés, vivant dans le sang de la carpe commune, ont pu être transmis à la carpe commune par injection dans le péritoine et ont pu être longtemps constatés dans le sang de celle-ci. Ils ne se sont cependant pas transformés en Sphaerospora. Ni les stades du sang, ni ceux de la vessie natatoire de la carpe commune n'ont pu être transmis à d'autres cyprinidés (Carassius auratus gibelio, Ctenopharyngodon idella, Hypophthalmichthys molitrix, Tinca tinca, Rutilus rutilus).

## Introduction

In a short communication Molnár (15) reported that the protozoan developmental stages parasitizing the swimbladder of the common carp *(Cyprinus carpio)* and described by Kovács-Gayer et al. (8), Körting (9) and Csaba et al. (3) can be transmitted experimentally into infection-free fish, in the renal tubules of which the protozoans develop into the spores of *Sphaerospora renicola* Dyková et Lom, 1982.

In the present paper the results of those repeated and expanded experiments are reported which have produced many-sided evidence on the identity of SB-protozoans causing swimbladder inflammation (SBI) of the common carp with an early developmental stage of *Sphaerospora renicola,* and which seem to contradict the postulated *Sphaerospora renicola*-nature of blood protozoans described by Csaba (2) and designated by Lom and Dyková (11) as UBO (Unidentified Blood Organism).

## Materials and methods

The experiments were conducted in 1982, 1983 and 1984 from the beginning of July up to mid-September, in the period when swimbladder inflammation usually occurs. In 1982, common carp fry obtained from an SBI-free stock, while in 1983 and 1984 common carp artificially hatched in 1983 and reared under parasite-free conditions were used in the experiments. When hatched from the eggs, the fry were fed on brine shrimp, then on a dried, synthetic food, and were kept in good-quality drinking water. These fish remained parasite-free throughout and contained no specific pathogenic germs either.

Three different types of experiments were conducted. In the first type experiments (1, 2, 3, 4, 5, 7, 8, 9, 10) common carp fry diseased in acute SBI and obtained from a pond farm were used as donors. The affected swimbladders of the fish were divided into three parts: one part was fixed for histological examination; from the second part impression smears were prepared, fixed with methanol and stained according to Giemsa; tissues of the third part of the swimbladder were lacerated with a dissecting needle in 0.65 % NaCl solution and filtered through a fine sieve; 0.1–0.3 ml of the obtained filtrate was injected with a syringe into the abdominal cavity of the experimental common carp fry of 1–4 g body mass before being checked for positivity.

In the second type of experiments (experiments 11) homogenates were prepared in 0.65 % NaCl solution from the kidneys of fish showing lesions characteristic of more

chronic SBI, already free from SB-protozoans but containing sporogonic developmental stages of *Sphaerospora renicola* in their renal tubules, and these homogenates were injected into the abdominal cavity of experimental fish.

In the third type of experiments (experiment 6, 12) 0.1–0.2 ml blood of fish free from SB-protozoans and renal sphaerosporosis but containing UBOs was injected into the abdominal cavity of infection-free common carp.

In 1984 our experiments were extended, in addition to the common carp, to other carps (gibel carp, grass carp, silver carp, tench, roach); also these were injected intraperitoneally with parasite-containing blood and swimbladder and kidney homogenates of common carp origin.

The fish kept at 20° C in aquarium and were killed, depending on the type of experiment, on post-infection days 2–42. After the native examination (squash preparation, blood drop examination) impression smears, histological preparations and blood smears were prepared. From fish inoculated only with blood parasites (UBOs) blood samples were taken regularly also before the extermination of the fish.

## Results

### Observations

Csaba et al. (3) have reported that swimbladder protozoans (SBP) can always be demonstrated in common carp fry diseased in acute SBI. In the present experiments the disease was found to occur also in two-summer common carp, in the swimbladder of which SB protozoans occasionally appeared already in the end of June. In fry, infection was in correlation with the age of the fish. In fish hatched earlier, infection appeared already in the beginning of July, in stocks hatched in May the disease was demonstrated mostly in the end of July and in the beginning of August. In the end of August and in September acute SBI occurred only in stocks that had been hatched late.

Within a stock, infection passed off synchronously, and 3 weeks after the appearance of the first symptoms SB-protozoans were found only occasionally. Because of this fact, for the experiments performed at different times donors had to be selected always from new fish stocks. In donor fish with acute SBI lesions most of the SB-protozoans consisted of primary and secondary cells; however, in fish exhibiting pronounced symptoms, triple forms corresponding to tertiary cells were abundant.

### Experiments

In 1982 only one experiment was carried out.

*Experiment 1*

The swimbladder homogenate of a common carp affected with SBI was injected into the abdominal cavity of four common carp fry, each weighing 10–12 g, derived from an SBI-free stock. Twenty-six fish served as control. Large numbers of SB-protozoans were found in the impression smears prepared of the swimbladder of the donor fish. One inoculated fish died during the experiment. Of the remaining three fish one proved to be infection-free, whereas in the renal tubules of the remaining two *S. renicola* spores and developmental stages were found. (It should be mentioned here that both in the present and in further experiments the developmental stages found in the renal tubules were exclusively in the sporogonic stage, in accordance with what is seen in natural cases, i.e. one pansporoblast contained two sporoblasts representing different degrees of development). No *Sphaerospora* was demonstrated in the controls; however, in part of the fish the blood contained UBOs.

In 1983 five experiments were conducted in which positively parasite-free fish were used.

*Experiment 2*

On August 4, 3 fish, each weighing 3 g, were inoculated into the abdominal cavity with swimbladder homogenate. In the swimbladder impression smears and in the histological preparation of the donor fish the parasite stages described by KOVÁCS-GAYER et al. (8) were demonstrated. The fish were killed on August 15. The kidneys of two fish contained numerous *S. renicola* spores and developmental stages (One of these fish had numerous UBOs in the blood). The kidneys of the third fish contained only developmental stages.

*Experiment 3*

This experiment was started on August 17. Swimbladder homogenates of 8 fish showing the symptoms of SBI were used for infection. Of the donors showing a slightly more chronic form of SBI, the swimbladder impression smear of only one fish contained SBPs. No histological samples were taken. On the other hand, UBOs were demonstrable in the blood of all donors. The homogenate was injected into the abdominal cavity of 14 fish, each weighing 1–4 g. These fish were killed at different times. In the fish killed on August 25 (the 8th day of the experiment) numerous *Sphaerospora* developmental stages were present in the kidney. In the kidney of fish killed on August 26 some spores had also appeared in addition to the developmental stages, and in both fish killed on August 29 numerous spores and developmental stages were present. Infection was less severe in fish killed on September 2, and its severity had decreased further in fish examined on September 12. It should be mentioned that at both of the latter examinations the number of spores and developmental stages was nearly the same, and at the last examination (September 12) numerous UBOs were demonstrable in the blood of the fish. The remaining 4 fish were killed on September 19. At that time the kidneys of two fish were already negative, while in the other two low-degree *Sphaerospora* infection was observed. In the latter fish there still always were sporogonic developmental stages in addition to the spores. Large numbers of UBOs were present in the blood of both the *Sphaerospora*-negative and the *Sphaerospora*-positive fish. During the experiment slight opacification of the swimbladder was observed only one occasion; however, parasites other than UBOs were found in the swimbladder neither in that case.

*Experiment 4*

This experiment was started on August 18. At this time, swimbladder homogenate from 8 fish showing slightly more chronic SBI was inoculated into the abdominal cavity of 12 parasite-free fish. No parasites were demonstrable in impression smears prepared from the swimbladder of donor fish and the swimbladders were negative also histologically. The fish were killed parallel with those of Experiment 3. Their organs proved to be negative in all cases.

*Experiment 5*

Five infection-free fish were inoculated with swimbladder homogenate on August 31. No SBP were found in impression smears made of the swimbladder of donor fish. The fish, killed on post-infection (PI) days 11 and 20, proved to be free from infection.

*Experiment 6*

During the last experiment conducted in the autumn of 1983, 10 parasite-free common carp fry were inoculated into the abdominal cavity with blood from donor fish which contained 1–4 UBOs per visual field when examined in 400-fold magnification by microscopy. The inoculated fish were killed, whenever possible, at 3-day intervals. No parasites were found in the fish up to the 12th day postinfection. On the 14th day PI a few UBOs were seen in the

blood smears and in native preparations of kidney and swimbladder capillaries. Infection by UBOs aggravated up to the 30th day when 4–6 parasites per visual field were found in the blood. The experiment was concluded at that time. During the experiment no parasites other than UBOs were found in the kidney and swimbladder. (For the sake of completeness it should be mentioned that we had already conducted similar experiments with similar results several times. However, the interpretation of these experiments was hindered by the lack of undoubtedly parasite-free fish, although appropriate controls were available.)

In 1984, experiments with SB-parasites were performed in July–August, when acute cases still occurred.

In this year, the swimbladders of all donors used in the experiment proved to be infected according to the subsequent examination of impression smears and histological preparations (Fig. 1).

*Experiment 7*

Swimbladder homogenate from two common carp fry showing an early stage of SBI was inoculated into 2 infection-free common carp fry and one grass carp *(Ctenopharyngodon idella).* In the donors mostly early SBP stages occurred. The kidneys of both common carp killed on day 11 contained numerous *Sphaerospora* spores and developmental stages; however, their swimbladder and blood proved to be negative. The grasscarp remained free from *Sphaerospora* infection.

*Experiment 8*

Swimbladder homogenate of 2 twosummer common carp were used for infection. The swimbladders, as in the further experiments, contained the triple forms of SBP in large numbers. The homogenate was inoculated into the abdominal cavity of 7 common carp. In the kidneys of the two fish killed on PI day 5 young sporogonic *Sphaerospora* stages were demonstrated, whereas in those of fish killed on PI days 9, 11 and 14 both *Sphaerospora* developmental stages and spores were found. No developmental stages were seen in the renal tubules of fish killed on PI days 33 and 36, and spores occurred only occasionally. UBOs were observed only in fish killed on PI day 36.

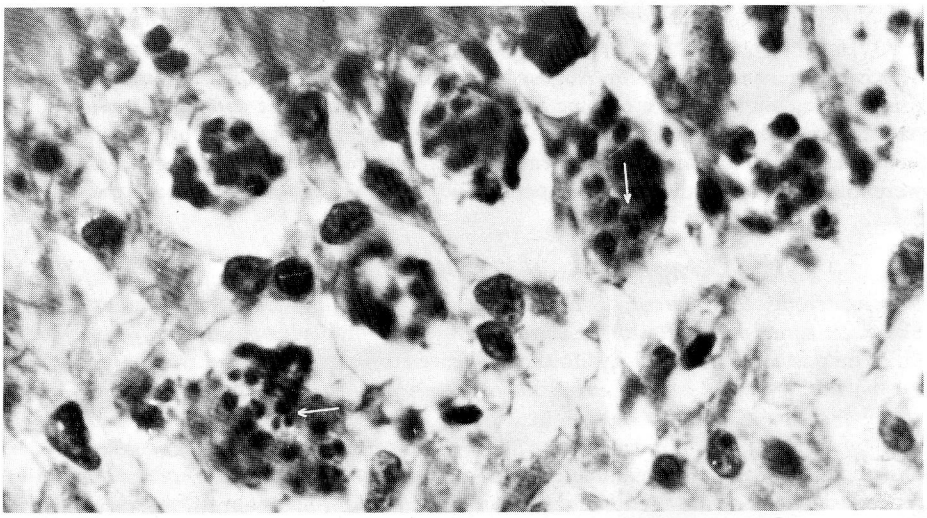

*Fig. 1.* SBP developmental stages in a histological section made from the swimbladder wall of a donor common carp. In the parasite mass representing one primary cell each, infective tertiary forms (arrow) are also seen. H. and E., × 800

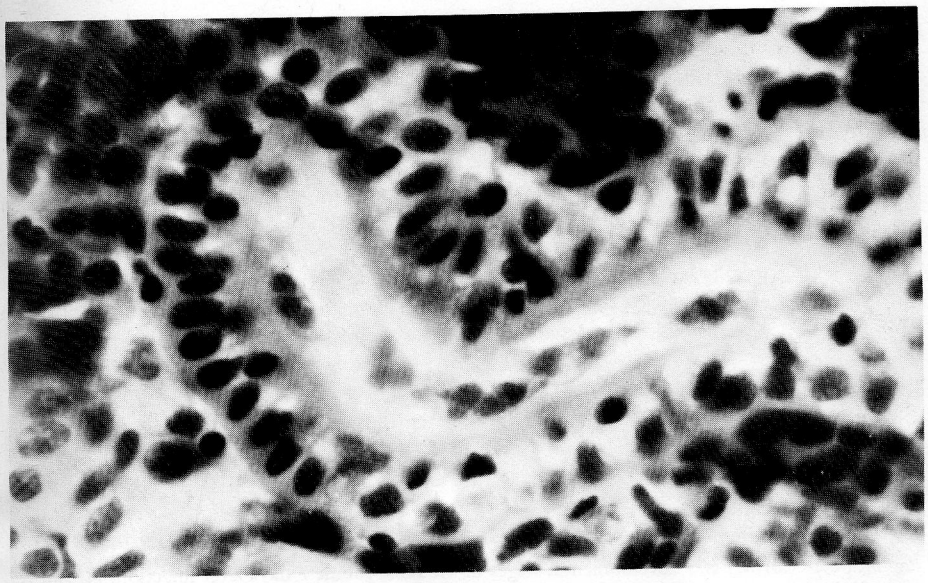

*Fig. 2* Young *Sphaerospora renicola* stages in the renal tubule 3 days after infection. H. and E., × 800

*Experiment 9*

Four gibel carp *(Carassius auratus gibelio),* and one silver carp *(Hypophthalmichthys molitrix),* roach *(Rutilus rutilus)* and common carp were inoculated with swimbladder homogenate of an SBP-infected donor. Of the fish killed on PI day 11 only the common carp had *Sphaerospora* stages in the kidney.

*Experiment 10*

Twelve common carp and two grasscarp, silver carp, and gibel carp each were inoculated into the abdominal cavity with swimbladder homogenate from an SBP-infected donor. The common carp were killed at one-day intervals. The first *Sphaerospora* developmental stages were observed on PI day 3 (Fig. 2), and the first spores on PI day 7 (Fig. 3). Between PI days 4 and 12 the kidneys of all common carp but two proved to be *Sphaerospora*-positive. The kidney of the fish surviving up to day 36 was negative; however, the blood of this fish contained UBOs. The silver carp, grass carp and gibel carp killed on PI day 13 were negative.

*Experiment 11*

Ten common carp and 2 gibel carp were inoculated with kidney homogenate from common carp infected with *Sphaerospora renicola.* The kidneys of fish killed on PI day 11 were free from *Sphaerospora.*

*Experiment 12*

Five common carp, grasscarp, silver carp, gibel carp and tenches *(Tinca tinca)* each were inoculated into the abdominal cavity with UBO-containing blood from severely infected common carp; the blood was diluted in Alsever's solution. During the 42-day experimental period the blood of the fish was examined at two-day intervals. The blood of all common carp became infected. First UBOs appeared on day 10, and infection was demonstrable even on day 42. However, no UBOs appeared in the blood of grass carp, silver carp, gibel carp and tenches. In the kidney and swimbladder of common carp killed between day 14 and 42 no *Sphaerospora* stages were seen.

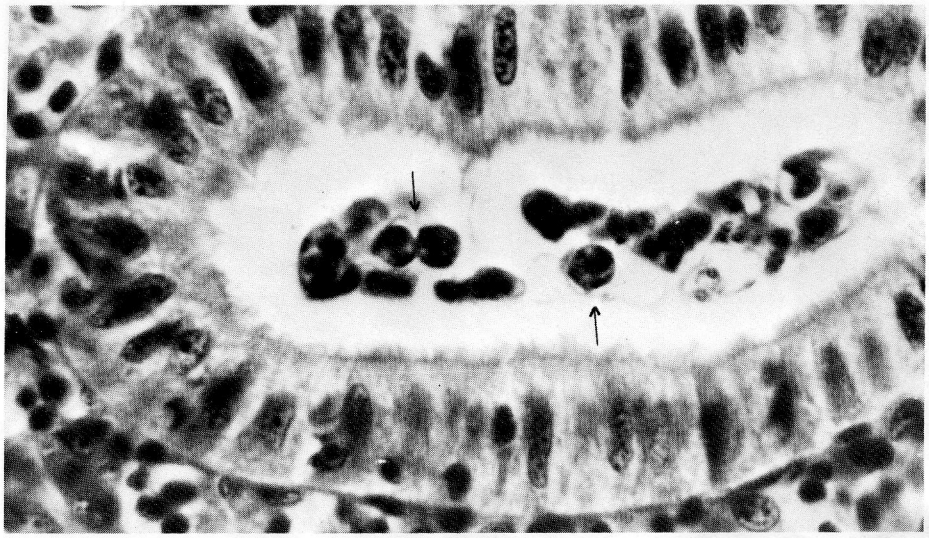

*Fig. 3* Spores (arrow) and developmental stages in the renal tubule 7 days after infection. H. and E., × 800

## Discussion

The present results support the findings of KOVÁCS-GAYER et al. (8), KÖRTING (9) and CSABA et al (3), and furnish evidence that the Myxozoa-stages (SBP) developing in the swimbladder wall of the common carp correspond to the early, presporogonic developmental stages of *S. renicola* parasitizing in the lumen of renal tubules. In all cases when the parasites were demonstrated in the impressions mears or histological preparations, the swimbladder homogenate inoculated into the abdominal cavity of infection-free carp resulted in the development of *Sphaerospora* stages in the kidney. *Sphaerospora* developmental stages causing SBI of the common carp are first demonstrable simultaneously with the onset of clinical symptoms. The parasites are present in the swimbladder for about 2–3 weeks; thus, under the climatic conditions of Hungary, they are expected to appear in one-summer fish in the end of June and in the beginning of July, while in 4–6 weeks old fry, disregarding the rarely occurring late infections, in July and in the beginning of August. In the light of the above facts, the partial failure of the experiments conducted in 1983 is fully understandable; this failure was corrected in 1984. In cases when SBP were present in the swimbladder, the final, so-called triple, forms of the parasites inoculated into the abdominal cavity developed into sporogonic stages of *S. renicola* in the renal tubules; infection developed also if the donors contained mostly young SBP stages. On the other hand, in cases when the parasites had already left the swimbladder and only chronic forms of SBI were diagnosed (see Experiments 4 and 5), for the lack of infective stages the swimbladder homogenate inoculated into the abdominal cavity produced no sphaerosporosis.

In our experiments, renal sphaerosporosis was demonstrable already 3 days after infection, and by PI days 7–9 the first spores had appeared. In the subsequent period, the spore/pansporoblast ratio kept increasing; however, no periodicity could be observed in their development since developmental stages were demonstrable, together with spores, even a month later. At the same time, the degree of infection lessened gradually from the 2nd week, and by 5 weeks PI infection practically ceased to exist.

The asynchronous formation of spores, i. e. the prolongation of the practically 8-day

period of sporogony to 4 to 5 weeks can probably be explained by the fact that the developmental stages inoculated into the abdominal cavity reached the kidney at different times.

However, the above experiments failed to elucidate the mode by which the kidney became infected. It is not clear yet how the developmental stages injected into the abdominal cavity managed to get into the renal tubules. It seemed an obvious hypothesis that swimbladder parasites were somehow transformed into motile UBOs which were transported to the renal tubules by the blood stream. In fish inoculated with swimbladder homogenate UBOs did actually appear; however, their role in producing renal sphaerosporosis has not been confirmed by experiments 6 and 12. UBOs can be transmitted easily into uninfected common carp with the infected blood, but in the latter they cause neither renal sphaerosporosis nor SBI. A possible explanation for the fact that UBOs still appeared in the blood of fish inoculated with swimbladder homogenate is that these parasites had been present in blood vessels of the swimbladder of donor fish.

As regards the mode of infection of renal tubules, the most probable possibility is that the triple SBP forms that developed in the swimbladder are transported by the blood stream to the renal glomeruli and from there to the renal tubules, where they are transformed into pansporoblasts and produce two *S. renicola* spores each. We consider impossible the hypothesis of Lom and Dyková (10) and Dyková and Lom (5) according to which *S. renicola* might have intracellular developmental stages in the epithelium of the renal tubules. The parasite forms observed by them were obviously developmental stages of *Mitraspora cyprini* or *Hoferellus cyprini,* occurring as co-infecting agents.

As regard the host-specificity, no final conclusions can be drawn from the experiments performed. The results obtained so far indicate that both the *S. renicola* stages and the UBOs are specific common carp parasites which could not be transmitted even to the genetically closely related gibel carp.

Apart from proving the aetiological relationship of SBI and renal sphaerosporosis, the above experiments have also theoretical significance. In the literature (13, 16, 17) it is an established fact that the germinative sporoplasm released from the spore multiplies and produces spores at the site of its colonization in the host. The present results have confirmed our earlier observation (14), differing from reports known from the literature, and agree also with the similar opinion of Lom et al. (12) that only the sporogonic stage of the development of *S. renicola* takes place in the renal tubules, while intensive multiplication occurs in the swimbladder. This means that in certain Myxozoa the well-defined developmental periods of the given parasite take place in host organs differing in respect of location and structure.

Experiments similar to ours were conducted by Clifton-Hadley et al. (1) and D'Silva et al. (4) who succeeded in transmitting, by intraperitoneal inoculation, the causative agent of proliferative kidney disease (PKD), the so-called PKX organism, into uninfected trouts in which PKD developed. However, these authors did not know that the PKX organism was probably a Myxosporea protozoon. Fergusson (6) and Kent and Hedrick (7) arrived at the conclusion that the PKX organisms living in the renal interstitium of salmons were the developmental stages of some Myxosporea (presumably a *Sphaerospora* or *Mitraspora* species) parasitizing the renal tubules. The vegetative and sporogonic stages of these parasites develop also in different locations even if in this case the different location means the interstitial and tubular part of the very same organ. However, the experiments conducted by Clifton-Hadley et al. (1) and D'Silva et al. (4) cannot be considered fully analogous with those performed by us. The above-cited authors produced the same phase of PKD by transmitting the causative agent, while we were unable to produce SBI, only the subsequently developing renal sphaerosporosis. Also, we failed in transmitting the sporogonic stages present in the renal tubules. However, this fact is not at all surprising since the coelozoic forms are unable to migrate. The role of the blood-parasite UBOs is still doubtful; however, based upon the present experiments, despite their undoubted similarity to SBP, they cannot be regarded as developmental stages of *S. renicola.*

## Acknowledgements

The authors express their thanks to Dr. László Békési, Dr. György Csaba and Dr. József Szakolczai (Central Veterinary Institute) for the diagnosis of swimbladder inflammation and for critical discussions, and Dr. S. S. Desser for his comments on the manuscript.

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