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Comments on the host, organ and tissue specificity of fish myxosporeans and on the types of their intrapiscine development

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Abstract: Different fish-parasitic myxosporeans are characterized by a varying degree of host specificity. While certain species have very strict specificity and can colonize only a single fish species, most species of Myxosporea share several closely related fish species as hosts. Myxosporeans are tissue-specific parasites and always develop in a specific host tissue. The author assumes that the development of all myxosporean species includes an intracellular stage. Organ specificity depends on the tissue types that occur in the given organ. The author distinguishes three main types of development: *Myxobolus*-type, *Hoferellus*-type and *Sphaerospora*-type development. *Myxobolus*-type myxosporeans develop in large plasmodia (or, according to an incorrect but generally used term: cysts). Some species of them showing muscle or nervous tissue specificity usually complete their development intracellularly and form spores within the cell. Host cells of small size, however, are destroyed during the growth of the parasite: the developmental stages released from them usually become surrounded by cells of the same type, and the parasite continues its development within the capsule formed by these cells. From plasmodia developing on the surface or in organs having efferent ducts the spores are usually excreted directly into the outworld; while the spores of species developing in the inner organs are transported by the blood to organs suitable for spore dejection. Coelozoic (*Hoferellus*-type) development means that the early development of myxosporeans takes place in the cells lining the efferent ducts while the second stage occurs in the lumen of the said ducts. During *Sphaerospora*-type development a short intracellular stage, one or two blood stages characterized by a series of internal cleavage, and a coelozoic or intercellular sporogonic stage can be distinguished.

Key words: Myxosporea, host specificity, tissue specificity, organ specificity, types of intrapiscine development

INTRODUCTION

Fish-parasitic myxosporeans comprise an extraordinarily large number of species. The genus *Myxobolus* alone, which is the best studied genus of Myxosporea, was reported to contain 444 species (Landsberg and Lom 1991). Accurate determination of the number of species is rendered difficult by the fact that taxonomic classification is based on the morphology of spores showing only slight structural differences, and that the development and the species, organ and tissue specificity of the parasites are little studied.

Reliable information on the extrapiscine development of myxosporeans has existed only since the studies of Markiw and Wolf (1983) who demonstrated the involvement of Oligochaeta alternative hosts in the development of *Myxobolus cerebralis*. The stages formerly called Actinosporea develop in these oligochaetes. The studies of Markiw (1989) revealed that infection by myxosporeans can take place also via pathways other than the alimentary route, and that *Myxobolus cerebralis* infection may occur also in the manner observed by Daniels et al. (1976), i.e. through the percutaneous entry of sporoplasms released from triactinomyxons. El-Matbouli et al. (accepted for publication) demonstrated that after some divisions the sporoplasms that have entered the epithelial cells of the skin, gills and fins actively travel along the nerve tracts to the final site of colonization specific of the given species. However, there still are only few species whose life cycle is known in every detail. Precisely the inadequate knowledge and poor reproducibility of the life cycle of myxosporeans account for the scarcity of data available on the species, tissue and organ specificity of these parasites.

DISCUSSION

Relying on my own studies spanning a period of 25 years and on data of the literature, in this work I will attempt to determine the species-specificity of different myxosporeans, to demonstrate their strict tissue specificity and their occasional organ specificity arising from the former. The same studies enable me to describe the three basic models of the intrapiscine development of myxosporeans. The task undertaken is very difficult, as numerous excellent books have been published on the subject, which contain a detailed description of the complex intrapiscine developmental processes of these parasites. At the same time, these sources still contain some erroneous statements while failing to mention some general characteristics of myxosporeans. Of the latter, here I would like to point out the following:

1. The host specificity of different myxosporean species is different; however, it is always restricted to a well-definable circle of related hosts.
2. Myxosporeans are characterized by a very pronounced tissue specificity.
3. The organ specificity of myxosporeans depends primarily on whether the cell type necessary for the development of the given myxosporean species is available in the organ concerned.

4. Myxosporeans equally include intracellular, intercellular and coelozoic parasites; however, the development of all species starts in intracellular location.
5. Internal cleavage is a process highly typical of myxosporean development. The formation of pansporoblasts and the emergence of the "enveloped" condition also take place by such internal cleavage, as opposed to the view that the latter condition develops by the fusion of two cells.
6. The "blind alleys" observed during the development of certain species of Myxosporea are based on an erroneous assumption.
7. The relationship of breeders and fingerlings seems to have decisive importance in the development of myxosporean infections, and the myxosporean infection of fry are ensured primarily by spores that get into the outworld during spawning.

The host specificity of myxosporeans

Myxosporeans equally include species with a relatively broad host range and those showing strict specificity. *Myxobolus cerebralis* can colonize numerous salmonid species (Hoffman and Putz 1969), while *Myxobolus pavlovskii* can infect only the two closely related *Hypophthalmichthys* species (Molnár 1979). Some *Thelohanellus* species possess even stricter specificity. Achmerow (1955, 1960) described 4 *Thelohanellus* species which occur exclusively in common carp. The uncertainty existing in connection with host specificity is mainly due to the fact that some specialists less than perfectly versed in this subject tend to identify spores found in different hosts with spores of the morphologically most similar species. At the same time, in the absence of holotypes and based upon inaccurate original drawings other specialists often describe as new species parasites of an already known species but derived from closely related fish hosts. This is how it could occur that some of the species recorded by Shulman (1966, 1984) have been reported from more than 40 fish species, while others have been described in a single host only. The actual situation is obviously between these two extremes: this means that the majority of these species parasitize a few closely related fish hosts, and that the currently recorded species include numerous synonyms and many new species not yet distinguished. Species like *Myxobolus exiguus*, whose hosts reportedly include numerous cyprinids as well as *Mugil* and *Silurus* spp., are obviously collective species that comprise several morphologically similar myxosporeans. Although theoretically feasible, cross-infections suitable for the exact determination of host specificity yet seem to be a remote possibility. The advances made in the PCR technique, a method suitable for detecting a specific DNA sequence, are more likely to bring a simple and rapid solution. Until that is accomplished, the identification of typical hosts serving as a subject of description and the accurate determination of location within the host remain to be tasks of fundamental importance.

The tissue and organ specificity of myxosporeans

Tissue and organ specificity has remained a neglected aspect of myxosporean research to this very day. Although it has long been known that *M. cerebralis* typically forms plasmodia in the cartilaginous tissue of the skull or that *M. musculi* forms its pseudocysts in the muscle cells of fish, in most cases the spores' momentary site of occurrence is given as their location, irrespective of the organ or tissue in which they have developed. For these reasons, several technical books (Shulman 1966, Bauer et al. 1981) describe *M. cyprini*, a very common parasite of the common carp, as a species that forms spores in small plasmodia in different organs. In contrast, Molnár and Kovács-Gayer (1985) demonstrated that the given species is a typical muscle parasite, and the formations mistaken for small plasmodia are actually the parasite's macrophage-engulfed spores disseminated all over the body. Therefore, the location shall never be determined merely on the basis of the spores' site of detection. The developmental stages, but at least the plasmodia often referred to as cysts, shall be detected in each case.

For species developing in large plasmodia the determination of location usually does not pose a problem if the examination is thorough enough. At the same time, for species whose specific developmental stages take place in different organs the location shall be determined separately for the individual stages.

The tissue specificity of myxosporeans

The species belonging to Myxosporea are highly tissue-specific parasites. Their development is consistently restricted to a single specific cell type. Myxosporea include species specialized in parasitizing epithelial, muscle, nervous, cartilaginous, bone and connective tissue; moreover, certain species begin to develop, within the basic tissue type, only in a single specific cell type, e.g. gill epithelium, liver cells, compact connective tissue, or perichondrial cells. Although among the myxosporeans Shulman (1966) distinguished species of intracellular, intercellular and coelozoic development, my studies conducted so far allow me to assume that, upon gaining entry into the organism, the Actinosporea sporoplasms reach the site of final colonization and invariably continue their development in an intracellular location. Whether spore formation takes place within the cell or is completed in the intercellular or coelozoic space upon disruption of the host cell seems to depend exclusively on the size of the attacked specific host cell. Species having a specificity for muscle and nerve cells usually have a prolonged intracellular phase of development, as the relatively large host cells provide ample space for the growth of their pseudocysts. These parasites form their spores within the cell, and the spores are released into the intercellular space only after destruction of the host cell (Molnár and Kovács-Gayer 1985, Ferguson et al. 1985).

The development of species showing a specificity for endothelial, epithelial, cartilaginous and connective tissue cells differs from the way of development described above. These small cells are destroyed by the growing parasites. However, the trophozoite released from the destroyed cell consistently becomes surrounded by cells of the

same type, and the plasmodium continues its development in that capsule formed by the host cells. This is how *Myxobolus cerebralis* develops in the cartilaginous tissue of the head (Halliday 1973), *Thelohanellus nikolskii* in a location surrounded by the perichondrial cells of the fin rays (Molnár 1982), *Myxidium giardi* and *Henneguya psorospermica* in the endothelium of capillaries (Dyková et al. 1987, Dyková and Lom 1978), and *Myxobolus kotlani* and *Thelohanellus hovorkai* in the connective tissue (Molnár et al. 1986, Molnár and Kovács-Gayer 1986a).

As a result of the parasite's perfect mimicry, the cells attacked by the parasite nourish the developing plasmodium and protect it against external influences, and a host response against the parasite is mounted only in the period of spore formation (Molnár 1982, Dyková and Lom 1988). A typical instance of mimicry and of the protection conferred by host cells is the development of *Thelohanellus nikolskii* whose large plasmodia could easily fall out of the thin-walled fins if they were not protected by another connective tissue layer formed around the cartilaginous capsule.

The organ specificity of myxosporeans

The species developing in the muscle cells, endothelium and loose connective tissue may occur at different sites of the body, in various organs: therefore, in their case no organ specificity can be spoken of. The skeletal muscle parasites (e.g. *Myxobolus cyprini*, *M. musculi* and *M. pseudodispar*) can be found in all parts of the fish body (Molnár and Kovács-Gayer 1985, Baska 1987). The endothelial parasite *Myxidium giardi* may also occur in different organs such as the gills, the liver and the kidney. A species occurring in such variable locations may easily be mistaken for a new species even by specialists (Hine 1975, Komourdjian et al. 1977), especially if the morphology of its spores also shows substantial variability.

At the same time, for species parasitizing the epithelium and the cartilaginous tissue, as well as for some myxosporeans showing an affinity for connective tissue an organ specificity is clearly demonstrable. Organ specificity is also determined by the tissue specificity of the given myxosporean. In such cases, the parasite can develop exclusively in a distinct type of epithelial, cartilaginous or connective tissue even within the basic tissue type. An example of such organ specificity is *Myxobolus pavlovskii* which forms cysts exclusively in the stratified epithelium among the secondary gill lamellae, or *Thelohanellus nikolskii* which starts to develop in the perichondrial cells of the fin rays. Two parasites characterized by an affinity for connective tissue form cysts on the gills of the common carp. Of them, *Myxobolus dispar* parasitizes the compact connective tissue of the gill filaments and possibly that of the tunica adventitia of the blood vessels, while *M. basilamellaris* forms its cysts partly at the base of the gill filaments, partly within the gill arch.

Types of the intrapiscine development of myxosporeans

The intrapiscine development of myxosporeans is highly diversified. The works of Shulman (1966), Mitchell (1977), Uspenskaya (1984), Lom and Dyková (1986,

1992) and Garden (1992) contain a multitude of data on the variations of their development and on the cytologic processes. Relying on recent knowledge obtained on *Sphaerospora* and *Hoferellus* species, Lom and Dyková (1986) discussed in detail the majority of problems addressed also by myself, including e.g. the "enveloped condition", pansporoblast formation, the behaviour of generative cells, the extrasporogonic stages, and intracellular development. Despite the complexity of developmental processes outlined by the above authors, the simplified development models of myxosporeans can be divided into three major types, i.e. *Myxobolus*-type development, *Hoferellus*-type development, and *Sphaerospora*-type development.

Myxobolus-type development

In this type of development, the form infecting the fish is probably identical with the sporoplasm released from the Actinosporea, which penetrates the epithelium that covers the body surface of the fish, undergoes a limited number of divisions there, and finally travels to the final site of colonization along the nerve tracts or via the blood stream. The infective form starts to develop in a specific cell typical of the given species, and forms a relatively large plasmodium. Within the plasmodium, vegetative nuclei and generative cells appear: the latter develop into pansporoblasts in most cases. Many researchers including Lom and Dyková (1986) conceive pansporoblast formation as the association of two generative cells, one of which surrounds the other and becomes a pericyte, while the surrounded sporocyte forms sporoblasts then a spore. My own observations are more consistent with the view of Shulman (1966) and Uspenskaya (1984), i.e. that the pansporoblast develops through internal cleavage of a single generative cell. As a result, the primary cell gives rise to a pansporoblast while the secondary cell to a sporoblast. If the sporoblast does not divide further, monosporoblastic development shall be spoken of; its division into two parts is designated bisporoblastic development, while its division into several parts is termed polysporoblastic development. The multinucleated spore, which is usually composed of 6 nuclei, develops by division of the sporoblast. The majority of known Myxosporea species also develop in this way. Besides *Myxobolus* spp., some *Thelohanellus*, *Henneguya* and *Kudoa* spp. also follow that form of development. The further fate of the plasmodium greatly depends on the type of the attacked cell and its location within the body; therefore, numerous variations may exist within that type of development (Figs 1 and 2).

In simpler cases of *Myxobolus*-type development the plasmodia develop near the body surface (in the skin, on the fins and gills) and, thus, the spores are released directly into the outside world after the cyst opens up. The majority of species parasitic in organs having efferent ducts (intestine, liver, kidney) leave the fish organism in a similar, rather easy way (Fig. 1). However, from plasmodia developing in the inner organs (spleen, liver and renal parenchyma), heart, brain, muscles and on serous membranes the spores may get into the outworld only via the lymph and blood circulation. In such cases the lymph and blood flow transports the numerous spores released from the disrupted cysts into the intercellular space to the gills, skin, kidney and intestine where they cause capillary obstruction and minor necroses, and from

where they are expulsed into the outworld with the help of macrophages (Fig. 2). The majority of spores, however, get stuck in the macrophage centres of the spleen, liver

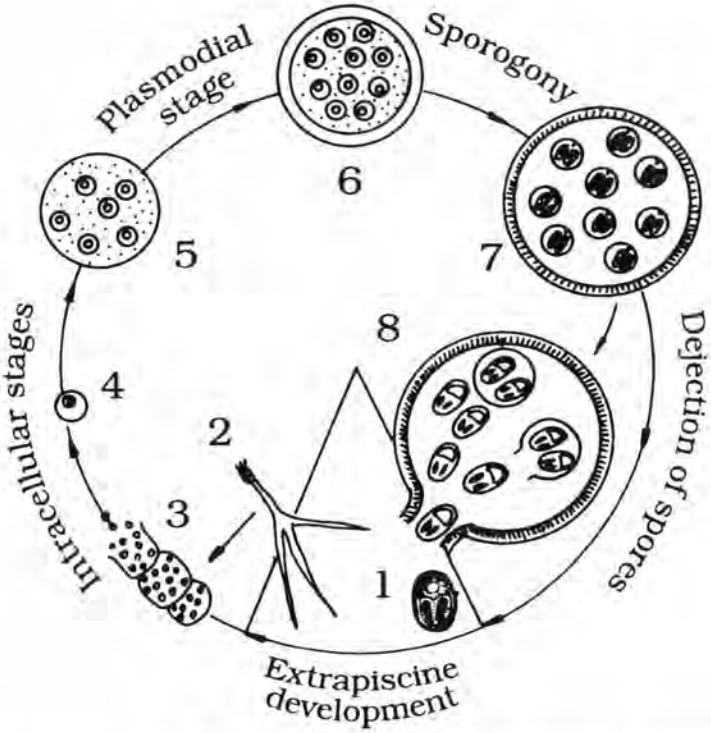


Fig. 1. *Myxobolus*-type development. Extrapiscine development is followed by two intracellular stages, plasmodium formation and then sporogony and spore dejection.

(1) During extrapiscine development, stages of Actinosporea develop in Oligochaeta intermediate hosts. (2) Actinosporeans floating in the water infect the fish through the skin. (3) The sporoplasms released from actinosporeans colonize the epithelial cells, undergo a limited number of divisions, then migrate, probably along the nerve tracts, to a site specific of plasmodium formation. (4) Sporoplasms (trophozoites) that have invaded a host cell specific of the development of the given species start to grow by multiple endogenous divisions and form a plasmodium. (5) In large cells the plasmodium develops intracellularly, while small cells become disrupted and the plasmodium continues its development extracellularly. As a result of serial divisions, vegetative nuclei and generative cells appear within the plasmodium. (6) The plasmodium is surrounded by a cell layer, so-called "cyst wall", formed of host cells. These cells are of the same type as those in which the parasite commenced its development. Internal cleavage of the generative cells gives rise to pansporoblasts. (7) The number of sporoblasts (1–2 or more) developing in the pansporoblast is characteristic of the given species. From each sporoblast a spore develops. (8) After the formation of the spore the pansporoblasts undergo necrosis, the cyst wall bursts, and from the plasmodia developing near the surface the spores get into the outworld and continue their development in oligochaetes.

and kidney, where they undergo gradual destruction. Inexperienced researchers often regard the large numbers of spores (sometimes numbering several hundred), located in the macrophage centres, as if they were formed on the spot, and erroneously indicate the organ concerned as the site of development of the given parasite.

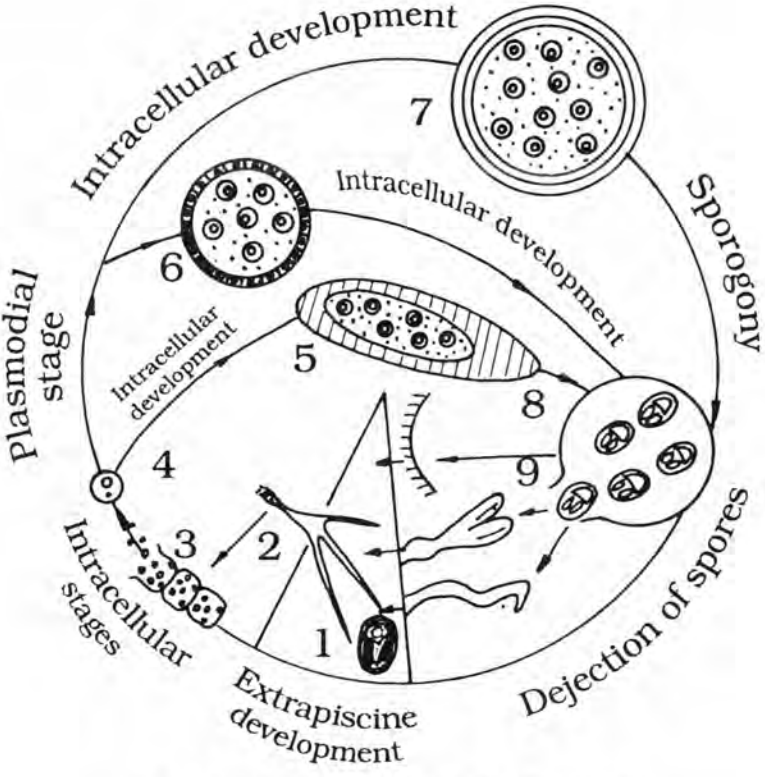


Fig. 2. Variations in *Myxobolus*-type development.

(1) Extrapiscine development. (2) Actinosporeans floating in water. (3) Sporoplasms released from actinosporeans infect the epithelial cells of fish. (4) Trophozoites formed from sporoplasms continue their development at the site of final colonization, in cells specific of the parasite species. (5) If the attacked host cell is large (muscle cell, nerve cell), the plasmodium will develop intracellularly until the spores emerge. (6) If the plasmodium commences its development in smaller cells, the attacked cell will die but the plasmodium will become surrounded by cells of the same type as those in which development started. (7) Primarily plasmodia specific of connective tissue are surrounded by a connective tissue capsule. However, an external connective tissue envelope may encapsulate also plasmodia covered by epithelial or cartilaginous tissue. (8) After the disruption of plasmodia that develop in organs not communicating with the outworld or lacking an efferent duct, the lymph and blood circulation transports the spores to organs that have direct communication with the external world. (9) From these latter organs (gills, kidney, intestine, skin) the spores are dejected into the outworld.

Hoferellus-type development

This type of development (Fig. 3) is characteristic of *Hoferellus* and *Myxobilatus* species, though certain *Myxidium* and *Chloromyxum* species also develop in that way. In the case of *Hoferellus*-type development no "cyst" formation can be spoken of. The early stages of *Hoferellus* and *Myxobilatus* species start to develop in the epithelial cells of organs having efferent ducts, primarily the kidney. The trophozoites infect new epithelial cells, then form secondary, tertiary and quaternary individuals within the primary parasite cell by multiple internal cleavage (Kovács-Gayer et al. 1987, Molnár

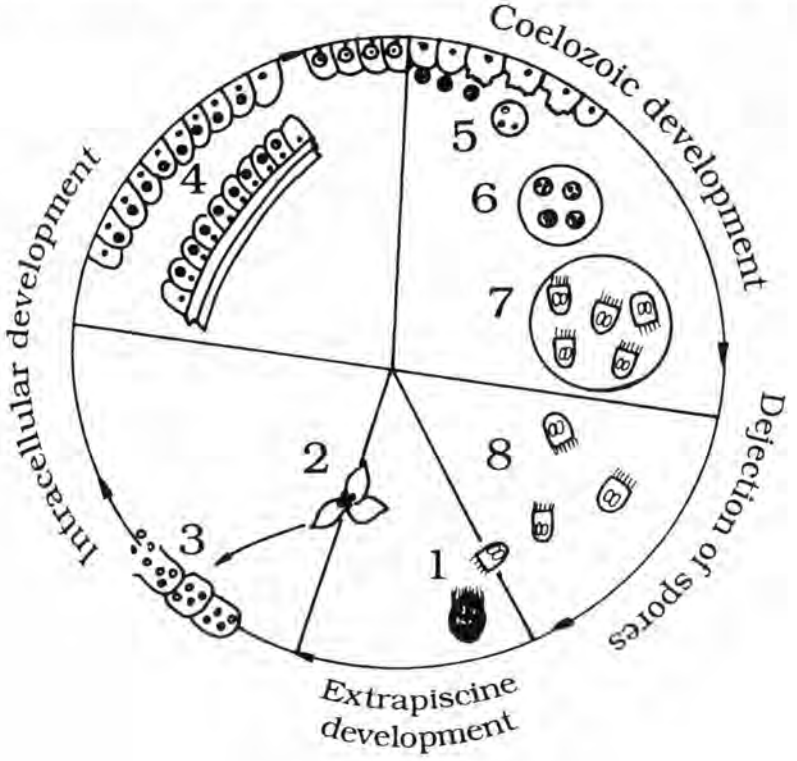


Fig. 3. *Hoferellus*-type development. In this type of development, the extrapiscine stage is followed by a relatively long intracellular stage which then continues in a coelozoic development.

(1) Extrapiscine development. (2) Actinosporeans floating in water. (3) Sporoplasms released from actinosporeans infect the epithelial cells of fish. (4) The trophozoites continue their development in cells specific of the parasite species, in the epithelium of efferent ducts. In the primary parasite cell located within the attacked epithelial cell secondary, tertiary and even quaternary cells are formed by internal cleavage. (5) After disruption of the host cell, secondary cells change into plasmodia in the lumen of the efferent ducts (ureter, bile duct). (6) From the tertiary cells sporoblasts are formed in the plasmodia. (7) Spores within the plasmodia may develop already in the lumen of the ducts; however, often they develop only in the bladders (gallbladder, urinary bladder). (8) The spores dejected from disrupted plasmodia get to the outworld with the excretions.

and Kovács-Gayer 1986b, Yokoyama et al. 1990). The second phase of the development of parasites belonging to this type takes place in the ureter and urinary bladder in a coelozoic manner. The secondary cells, which in most cases already contain tertiary and quaternary cells at that stage, leave the epithelial cells, change into small plasmodia and form 2–10 spores. In the plasmodium the tertiary cells represent the sporoblasts while the quaternary cells correspond to the nuclei of future spores. The tissue specificity of *Hoferellus*-type myxosporeans is determined by the cell type in which intracellular development takes place. Usually they can be considered organ-specific parasites, although certain *Chloromyxum* and *Myxidium* species are likely to be capable of developing in both the kidney and the liver (Dyková et al. 1987).

The development of *Myxidium lieberkuehni* as outlined by Lom et al. (1989) can be considered a special form of coelozoic development, in the same way as that of *Chloromyxum inexpectatum* described by Baska (1990, 1993) from the renal glomeruli where the plasmodia form a xenoma with the glomerular epithelium.

Sphaerospora-type development

This type of development (Fig. 4) is characteristic of *Leptotheca* and *Ceratomyxa* species, in addition to the numerous representatives of the genus *Sphaerospora*. For a long time, *Sphaerospora*-type development was known to consist of only two blood stages and one renal, or occasionally epithelial, stage (Csaba 1976, Lom et al. 1983, Csaba et al. 1984, Molnár and Kovács-Gayer 1986c, Baska and Molnár 1988). Recently, Voronin (1993) has demonstrated that the development of also these parasites includes an intracellular stage which precedes the blood stages. Thus, in the same way as with other myxosporeans, development continues intracellularly after the invading Actinosporea sporoplasms have gotten to the species-determined site of colonization. The first developmental stage of *Sphaerospora renicola* establishes itself in the endothelium. By internal cleavage, the trophozoite getting into the blood (C-protozoan, UBO) gives rise to a secondary cell which then divides into eight other secondary cells. Subsequently, tertiary cells develop within the secondary cells. After the disruption of the primary cell, the secondary cells, together with the tertiary cells included by them, may initiate another cycle of 8. After a certain time, from the tertiary cells a second blood stage (K-protozoan) develops, in which about 40 secondary and 2–2 quaternary cells arise by internal cleavage. The nucleus of secondary cells transported by the blood stream to the renal glomeruli and getting, through the latter, into the convoluted tubules corresponds to the nucleus of pseudoplasmodia. The tertiary cells develop into a spore each. In my opinion the pseudoplasmodium is a form analogous with the pansporoblast. The development of the PKX organism, which is probably a parasite of *Sphaerospora* nature and the causative agent of proliferative kidney disease of salmonids, is somewhat different but similar in its tendency. The second blood stages (K-protozoans) that accompany the circulating primary blood stages and get jammed in the swimbladder capillaries, in the choroid of the eye and in the renal parenchyma are described by many researchers studying *Sphaerospora*-type development as swimbladder etc. stages, while actually they are blood stages occurring in the capillaries of

the given organs. Therefore, for *Sphaerospora*-type myxosporeans organ specificity can be spoken of only in the phase of spore formation, when the site of dejection, the kidney, the ducts of sex organs and the gill epithelium can be indicated as locations. If we regard the blood path as an organ, the endothelium and the blood vessels can also be given as a location of the early *Sphaerospora* stages.

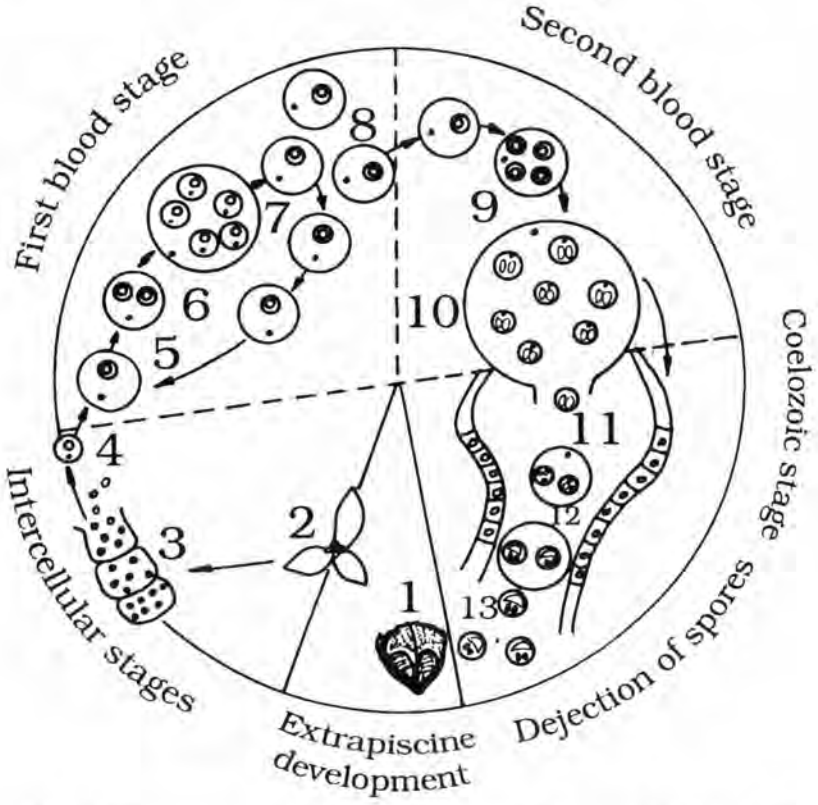


Fig. 4. *Sphaerospora*-type development. The extrapiscine stage is followed by two intracellular stages, two blood stages and a coelozoic stage.

(1) Extrapiscine development. (2) Actinosporeans floating in water. (3) Sporoplasms released from actinosporeans infect the epithelial cells of fish. (4) The trophozoites continue their development in the endothelial cells of capillaries. (5) In the parasite released from the endothelial cell a secondary cell develops by internal cleavage. (6) Division of the secondary cell gives rise to further cells. (7) In the secondary cells tertiary cells develop by internal cleavage. The majority of *Sphaerospora* species have a total of 8 secondary cells in one primary cell. (8) After disruption of the primary cell, the secondary cell and the tertiary cell contained by it may commence a new cycle of 8, but it also may start the second blood stage. (9) During the second blood stage numerous secondary cells are formed in the primary cell. (10) In the secondary cells usually two tertiary cells develop, which are identical with the future sporoblast. (11) After disruption of the primary cell the secondary cells get into the renal glomeruli and from there into the urinary passages. (12) In the urinary passages, the sporoblasts change into spores that leave the fish through the urinary bladder. (13) Spores.

The sporogonic stage of *Sphaerospora* species forming spores in the kidney invariably takes place in coelozoic manner, although Lom and Dyková (1985) assumed that certain *Sphaerospora* stages undergo abortive intracellular development in the tubular epithelium. However, these forms are undoubtedly identical with the developmental stages of *Hoferellus cyprini* and are unrelated to the concurrent *Sphaerospora* infection. Similarly, we can rule out that the forms occasionally seen in the wall of renal tubules by Odening et al. (1989) and probably representing macrophages would be identical with sphaerospores. According to Odening et al. (1988), the population dynamics of *Sphaerospora renicola* shows a distinct seasonality. In my experience, that seasonality is shared by all *Sphaerospora* species and manifests itself in the phenomenon that spore formation and dejection take place during spawning in older fish (primarily brood-stock) and at the age of 2–3 months in fry. Although occasionally large masses of spores are produced in fry, not these fish but the egg-producing breeders act as the source of the subsequent year's infection. Probably a similar process takes place in the case of proliferative kidney disease which is now unambiguously considered a sphaero-sporosis. During that disease, the so-called PKX organism (a developmental stage occurring in all organs but mainly in the renal parenchyma) is detectable along with *Sphaerospora* developmental stages present in the renal tubules (Kent and Hedrick 1985). As the sporogonic forms found in affected salmonid fry never reach complete sporogony in the renal tubules, Kent and Hedrick (1986) suggested that this infection is caused by a *Sphaerospora* species for which the salmonids are not suitable hosts. In my opinion, this "blind alley" hypothesis is also built upon an error. Blind alleys are rare in nature, while unsolved problems are much more common. My supposition, supported by the observation of Kent et al. (1993), is that proliferative kidney disease is caused by spores excreted by older fish after the appropriate Actinosporea stage, in the same way as is the swimbladder inflammation of common carp fry.

Naturally, the existence of developmental models other than the three basic types outlined above (*Myxobolus*, *Hoferellus* and *Sphaerospora*) cannot be ruled out either. It seems probable that after acquiring a more in-depth knowledge of the intrapiscine development of different parasites numerous other types of development will be identified.

The development of different myxosporean species in alternative hosts also poses numerous problems to be solved. From the work of Janiszewska (1957) it is known that Actinosporea also include species developing in the gut epithelium or in the body cavities. At the same time, the specificity of myxosporeans (actinosporeans) for alternative Oligochaeta hosts is unknown. Should it prove true that myxosporeans show a strict specificity also for oligochaetes, monitoring of the entire developmental cycle could become extremely difficult.

Molnár K.: Megjegyzések a halakban élő myxosporeák gazda-, szerv- és szövetspecificitásáról és halon belüli fejlődésük variációiról

Az egyes halélősködő Myxosporea-fajok gazdák iránti specificitása eltérő. Egyes fajaik rendkívül szigorú specificitással rendelkeznek, s csupán egyetlen halfajban képesek megtelepedni. A *Myxosporea*-fajok többsége közel rokon halfajok közös élősködője. A myxosporeák szövetspecifikus élősködők, fejlődésük mindig egy meghatározott gazdaszövetben megy végbe. A szerző feltételezi, hogy valamennyi *Myxosporea*-faj fejlődésében van egy intracelluláris szakasz. A szervspecificitás az illető szervben előforduló szövettípusok függvénye. A szerző 3 fő fejlődési típust különít el: a *Myxobolus*-típusú, *Hoferellus*-típusú és *Sphaerospora*-típusú fejlődést. A nagy plasmodiumokban (helytelen, de általánosan elfogadott terminus szerint cisztákban) u. n. *Myxobolus*-típus szerint fejlődő myxosporeák közül az izom- és idegszövet-specifikus fajok fejlődésüket rendszerint intracellulárisan fejezik be, s a sejten belül képeznek spórákat. A kisebb gazdasejtek a parazita növekedése során elpusztulnak, a belőlük kiszabadult parazita fejlődési alakokat rendszerint azonos típusú sejtek veszik körül, s a parazita az általuk képezett tokon belül folytatja fejlődését. A felületen, vagy kivezető járatokkal rendelkező szervekben fejlődő plasmodiumokból a spórák rendszerint közvetlenül a külvilágra ürülnek, a belső szervekben fejlődő fajok spórái viszont a vérkeringés segítségével jutnak el a kiürülésre alkalmas szervekbe. Coelozoicus (*Hoferellus*-típusú) fejlődés esetén a myxosporeák korai fejlődése a kivezető csatornákat bélelő sejtekben zajlik le, a fejlődés második szakasza az illető csatornák lumenében történik. A *Sphaerospora*-típusú fejlődés során egy rövid intracelluláris fázis, egy vagy két, endogén sarjadzások sorozatával jellemzett vérfázis és egy coelozoicus vagy intercelluláris spóraképző szakasz különíthető el.

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