THE STICKLEBACK (*GASTEROSTEUS ACULEATUS* L.)
AND PKD IN SALMONIDS - CULPRIT OR INNOCENT BYSTANDER?

BY S.W. FEIST

Since the recognition of PKD emerging as a serious threat to salmonid aquaculture during the 1960s and 1970s, aspects of the disease pathogenesis have been investigated (Ferguson & Needham, 1978; Hoffmann & Lommel, 1984; Clifton-Hadley, *et al*., 1987). The lack of knowledge of the source and true nature of the causative agent, the PKX cell, has severely hampered the search for suitable control measures. The parasite was originally considered as an amoeba (Plehn, 1924) but more recently as a member of the haplosporidia (phylum Ascontospora) (seagrave, *et al*., 1980). However, subsequent studies have indicated the presence of more advanced developmental stages within the renal tubules of convalescing fish and this has suggested the correct taxonomic placement may be within the phylum Myxozoa (Kent & Hedrick, 1985). Consequently, the myxosporean nature of PKX has become accepted. Although mature spores (necessary for specific identification) have not been reported, the developmental stages of the parasite exhibit features similar to the Sphaerosporidae, a myxosporean family typically infecting the kidneys and urinary bladders of marine and freshwater fish; and of the family Parvicapsulidae, which likewise infect kidneys and urinary bladders of both marine and anadromous fish, but possibly not freshwater fish (Lom & Noble, 1984).

It has been postulated that salmonids, especially rainbow trout, are aberrant hosts for the PKX parasite (Seagrave *et al*., 1980). The marked pathological response to the presence of the PKX cell and apparent failure of the parasite to produce mature spores are factors supporting this view. The normal host for PKX (in which sporogony is completed) and possible source of PKD in salmonids remain unidentified.

It is generally accepted that myxosporean infections are transmitted directly fish-to-fish by susceptible hosts ingesting spores which release the infective sporoplasm in the digestive tract and ultimately give rise to spore production at various sites within the host, dependent on the parasite species involved. Recently, this long-standing view has been challenged. Wolf & Markiw (1984) claimed that, in the case of *Myxobolus cerebralis*, an intimate involvement with a secondary host (a tubificid worm) was essential for infection in fish to occur. However, unless other researchers can substantiate this claim, or demonstrate the involvement of secondary hosts for other myxosporeans, the most accepted view of transmission remains the direct one between susceptible fish.

Consequently, in the search for possible vectors for salmonid PKD, attention has been directed toward the examination of native fish species found in PKD-enzootic waters and which harbour Sphaerosporan parasites, especially within the kidney (Bucke, *et al*., in press). Species currently under study include the eel (*Anguilla anguilla* L.) and the three-spined stickleback (*Gasterosteus aculeatus* L.). Investigations at this laboratory have now focused on the possible role of the three-spined stickleback through recognition of several features which appear relevant to the occurrence of PKD in salmonids. Firstly, the stickleback is a known host of a renal sphaerosporan, *Sphaerospora elegans*, Thelohan 1892 (Fig. 1) and is frequently co-infected with *Myxobolus gasterostei*, Parisi 1912. Secondly, it is cosmopolitan essentially coastal over all three northern continents, and likely to be...
DEAR EDITOR:

BY J.L. LARSEN & N.J. Jensen

Dear Editor

We are concerned that in their note, Vestergaard Jørgensen & Olesen (1987) emphasized that we considered or claimed the rhabdovirus to be a new one or a new member of the rhabdovirus group. In fact we never did consider or claim this to be the case. In our discussion (Jensen et al. 1979) we concluded: "but the present study does not include any physico-chemical or serotypical investigations which might allow any definite conclusion as to its relationship to the five rhabdoviruses infecting fish described by Hill et al. (1975). However, on a morphological basis the virus can be characterized as a rhabdovirus isolated from cods with the ulcus-syndrome"; and furthermore we stated: "the two viruses cannot be systematically isolated from cods with the ulcus-syndrome".

In our succeeding work we could not reproduce any pathology with the rhabdovirus, and we therefore found this virus uninteresting. Our final conclusion of this work was: "Thus the present results describe only part of the pathogenic function of the icosahedral virus and Vibrio anguillarum, but they do not fulfill Koch's postulate" (Jensen & Larsen 1982).

Our conclusion was that the iridovirus was the causative agent of the ulcus-syndrome.

In their note, Vestergaard Jørgensen & Olesen (1987) stress the importance of making "new pathogens" available to reference laboratories. We naturally agree with the authors in the use of reference laboratories, as far as concerns both viruses and bacteria. But as we have never suggested the rhabdovirus to be a new one, or a pathogen, but merely an isolate from cod, we did not consider that further studies would be relevant.

We think that this confusion concerns the interpretation of the words isolate vs. strain.

References


present in most water courses with the exception of fast-flowing streams. Thirdly, and of most interest, a mucoid kidney secretion from the male is utilised in the nest construction during the spawning period. This generally occurs between March and August with most activity during the earlier months, a period similar to, if slightly in advance of, the typical 'PKD season' in this country (Clifton-Hadley et al., 1987). It is postulated that the kidney secretion could provide an ideal vehicle for the release into the environment of *S. elegans* (and possibly *M. gasterostei* if present), this occurring at a time when young sticklebacks are possibly most susceptible to infection soon after hatching. From the parasite's view point, transmission to, and subsequent infection, of salmonids (giving rise to PKD) would be entirely coincidental!

Preliminary transmission experiments with sticklebacks and rainbow trout conducted at this laboratory have so far proved inconclusive, but are continuing. However, the observation of 'PKX-like' cells in stickleback kidneys by Hedrick, et al. (1986) has been confirmed in our studies, although the relationship between these, the luminal sphaerospores and PKX remains unclear.

The evidence presented here is purely circumstantial, and the argument entirely speculative, but, in the author's view, suggests the stickleback to be the most likely natural reservoir for the PKX parasite so far examined. It would be of interest to hear of any situations where severe PKD outbreaks can be correlated with the presence of large stickleback populations in other countries or, alternatively, where PKD occurs with sticklebacks absent.

**Summary**

The source of infection giving rise to PKD in salmonids remains unidentified. It is postulated that the three-spined stickleback (*Gasterosteus aculeatus* L.) may be a vector for the disease. Factors supporting this view are given.

**References**


Figure Legend

Figure 1 Stickleback kidney. Tubule lumen packed with spores and developmental stages of *Sphaerospora elegans* Theoharan, 1892.
Normarski interference contrast. Bar = 10 um.

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