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SCHERING-PLOUGH AQUACULTURE
Aquaculture Centre, 24-26 Gold Street
Saffron Walden, Essex CB10 1EJ, UK
Telephone 44 (0) 1799 528167
Facsimile 44 (0) 1799 525546

SCHERING-PLOUGH
ANIMAL HEALTH CORPORATION
PO Box 3182, Union
New Jersey 07083-1982, USA
Telephone +1 908 629 3344
Facsimile +1 908 629 3365

www. spaquaculture.com

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Ph 0064 9 533 4336 Fax 064 9 533 4337
email keith@aquaculturehealth.com
www.aquaculturehealth.com

EDITORIAL DIRECTOR:
Dr Scott Peddie

PUBLISHER: Keith Ingram

MANAGER: Vivienne Ingram

ASSISTANT EDITOR: Mark Barratt-Boyes

CONTRIBUTORS:
Dr Scott Peddie
Dr Spencer J Greenwood
Prof Richard J Cawthorn
Dr Giorgos Koumoundouros
Dr Mark Sheppard
Dr Ben Diggles
Dr Eugene Burreson
Robin Wardle

DESIGNER: Rachel Walker

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THE CASE FOR WIDENING **THE FISH WELFARE DEBATE**

DR SCOTT PEDDIE, EDITORIAL DIRECTOR

The welfare of farmed fish has become an increasingly important topic of discussion among scientists, veterinarians, fish farmers, animal welfare groups and consumers across the globe.

This is reflected in an increase in funding for basic research, enabling scientists to better understand the complexities of measuring and improving welfare in farmed aquatic animals. Perhaps nowhere is this push for knowledge more evident than in Europe.

Economists have recently entered into the animal welfare debate, providing an alternative, or perhaps more correctly, a complementary insight into how welfare can be measured, assessed and ultimately managed. The common misperception is that the economist's approach simply puts dollar, pound or euro signs against changes in production practices driven by welfare considerations.

Although this is an important facet of the economists' interest in fish welfare dynamics, it is only a small piece in the jigsaw. An economic perspective on animal welfare incorporates a plethora of information from consumer perceptions, purchasing behaviour, product pricing and production management.

The key message of the approach, as explained by one of its key proponents, Professor John McInerney of the University of Exeter in the United Kingdom, is that animal welfare is essentially an extension of human welfare and values. Put more simply, the economist's position is based on the premise that people specify standards in food production, therefore it is a matter of human perception as to what constitutes animal welfare. Controversial? Perhaps, but it certainly provides a different perspective to the scientist's "fish centred" approach, and it provides the backdrop against which economists approach the issue.

Given the growing interest of policy makers in fish welfare, it is disappointing that the debate lacks a comprehensive input from economists. The situation in terrestrial agriculture is somewhat different. Animal health economists have attempted to develop and refine a framework to better inform and expand the welfare debate.

Granted, they have been working with much more diverse, comprehensive and widely available data on consumer perceptions, end-product price sensitivity and the like. Nevertheless, such information can also be gathered for food products produced by aquaculture, provided the funding is made available and the interest is there to carry out the work. Perhaps we could start by attempting an in-depth assessment of societal (and by extension, consumer) attitudes towards welfare in farmed fish?

To my mind, exploring the economist's approach to welfare would complement the excellent work currently taking place in the scientific arena, as well as that occurring in the field of human ethics.

As a final point, it is perhaps worth bearing in mind that in the final analysis, consumers' purchasing behaviour will drive the evolution of the fish welfare debate and ultimately fish farm management practices. It is therefore important that we scientists are cognisant of this.

CRUSTACEANS: THE IMPACT AND CONTROL OF VIBRIOSIS IN SHRIMP CULTURE WORLDWIDE

BY DR SCOTT PEDDIE (PATTERSON PEDDIE CONSULTING, UK) AND ROBIN WARDLE (SCHERING-PLOUGH AQUACULTURE, UK)

This article is the first in the Vibriosis series, produced in conjunction with Schering-Plough Aquaculture. Subsequent articles will consider the impact of the condition and means of prevention and control in sparids (sea bass and sea bream), salmonids (rainbow trout and Atlantic salmon) and "cold water" species such as Atlantic cod.

Vibriosis is the term used to refer to a multitude of infections caused by bacteria belonging to the genus *Vibrio*. Infections commonly known as black shell disease, tail rot, septic hepatopancreatic necrosis, brown gill disease, swollen hindgut syndrome and luminous bacterial disease are some of the many conditions in shrimp caused by this group of pathogens.

The direct economic implications of vibriosis can be devastating, particularly when acute outbreaks occur. Losses reaching almost 100 percent have been recorded in the hatchery phase of production. The situation in the grow-out phase is somewhat more nuanced, with chronic losses occurring either directly, or following immuno-suppression and consequential secondary infections with white spot syndrome virus, yellow head virus and the Taura syndrome virus, among others. As well as enhanced mortality rates, reduced feed conversion efficiencies and growth rates in surviving individuals have a negative impact on the financial efficiency of the business.

WHAT CAUSES THE DISEASE?

There is a wide range of *Vibrio* species associated with disease in cultured shrimp throughout the world. Pathogenicity varies according to both species and strain encountered. Some of the most economically important species are:

- *V harveyi*
- *V penaeicida*
- *V parahaemolyticus*
- *V vulnificus*
- *V alginolyticus*
- *V splendidus*

It is important to note that *Vibrio* spp are ubiquitous in the marine environment and are commonly found on the external surfaces and internal organs of clinically healthy individuals. In most cases, predisposing factors such as post-optimal stocking density and poor water quality are required to precipitate a clinical outbreak of disease.

DISTRIBUTION AND SUSCEPTIBILITY

Vibriosis has a worldwide distribution. Outbreaks have been documented in black tiger shrimp *(Penaeus monodon)* and giant prawn *(Macrobranchium rosenbergii)* throughout continental Asia, white shrimp *(Penaeus chinensis)* in China, Indian white shrimp *(Penaeus indicus)* in India, Kuruma prawns *(Penaeus japonicus)* in Japan and Korea, and white shrimp *(Litopenaues vanamaii* and *Litopenaeus stylirostris)* in the Americas and the Pacific Islands.

TRANSMISSION AND EPIDEMIOLOGY

Some *Vibrio* species have very high growth rates under optimal conditions (doubling in number every 15 minutes or so in some cases). Disease transmission can therefore occur rapidly in confined culture units. Transmission is either via water or as a result of ingestion of infective material, although there is some evidence that wounds can also provide a means of entry.

Background levels of bacteria are generally kept under control by the host's immune system. However, when an individual is immuno-suppressed or otherwise physiologically "stressed", the pathogen is able to out-compete the host for iron, thus triggering a growth phase characterised by rapid multiplication.

SHRIMP GROWOUT PONDS

Thereafter the pathogen releases exotoxins that effectively break down the wall of the gastrointestinal tract and destroy the host's immune cells. Death can occur rapidly thereafter.

CLINICAL SIGNS

In the hatchery phase, a cessation in feeding activity can signal the onset of vibriosis. Microscopic examination of affected individuals typically reveals a discoloured and necrotic hepatopancreas, together with the presence of "clumping" (aggregations of hepatopancreatic digestive cells) in the lower digestive tract. In South America, the latter phenomenon is colloquially known as bolitas.

During the grow-out phase, affected individuals may appear lethargic, congregate in shallow water and exhibit little interest in feed when offered. Closer examination of moribund individuals may reveal a reddish discolouration of the body caused by the breakdown of the hepatopancreas and subsequent release of carotenoid pigments into the haemolymph.

Other symptoms can include a yellowing of the gill tissue, white patches in the abdominal muscle, melanisation of the appendage tips and luminescence. The latter phenomenon is associated with the so-called "luminescent" *Vibrio* species, *V harveyi* and *V splendidus*. Light is produced from these pathogens following the oxidation of the substrate luciferin by the enzyme luciferase.

Wet mounts of haemolymph extracted from moribund shrimp can be used to look for evidence of bacterial populations. Histological manifestations of *Vibrio* infection include necrosis and inflammation of the lymphoid organ, gills and heart. In particularly heavy infections, rod-shaped bacteria are observable in these and other tissues.

DIAGNOSIS

Vibrio spp can either be isolated from watercourses or directly from moribund animals. Vibrio selective agar, (also known as TCBS agar) is the typical isolation medium, with marine blood agar used to determine the haemolytic ability of the resultant bacteria. In addition, molecular probes can be used to distinguish between different strains and serovars.

As *Vibrio* spp are common "secondary invaders", it is important that clinical signs of disease are evident in conjunction with the isolation of bacteria before a definitive diagnosis can be made.

CONTROLLING VIBRIOSIS

Several preventative approaches can be adopted to reduce the likelihood of vibriosis occurring in farmed stock. The most important of these is good husbandry. In the hatchery phase for example, water should be well filtered and sterilised (via UV light and/or ozone treatment).

Water exchange is also an important feature of maintaining optimum rearing conditions, particularly from the Zoea 3/Mysis 1 lifecycle stages. Feed quality and general levels of sanitation are also important prerequisites for healthy stock.

During the grow-out phase, water quality should be monitored regularly, maintaining a stable phytoplankton bloom and paying particular attention to the temperature profile, salinity, dissolved oxygen, ammonia levels and other key parameters. Handling should be minimised and stocking densities should be maintained at appropriate levels to reduce "stress" in the cultured stock.

PREVENTION BETTER THAN CURE

Vaccination has long been available for preventing vibriosis in farmed fish. These vaccines were developed in the mid-1970s for trout. Relatively little was known at the time about the immune system of fish and how it responded to vaccines and vaccination. However, with the urgent need in the developing fish farming industry, farmers and researchers accepted the fact that the products worked and were useful tools in reducing the impact of disease.

FIGURE 1

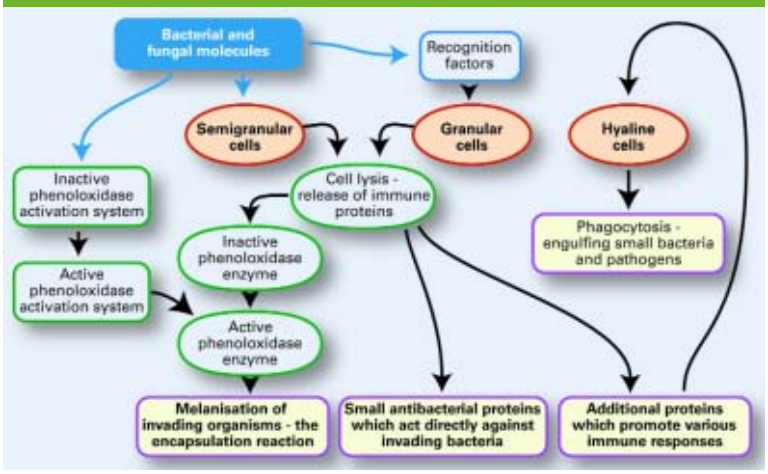

FIGURE 1: A SCHEMATIC REPRESENTATION OF THE IMMUNE SYSTEM IN SHRIMP AND THE INTERACTIONS THAT MAY BE RESPONSIBLE FOR THE "PROTECTION" INDUCED BY VACCINATION . COURTESY OF DR C HOUGHTON, UNIVERSITY OF ST ANDREWS

Since that time a huge international and multifaceted research effort has gone into better understanding fish and shellfish immune systems.

The situation in shrimp is analogous to that of fish 30 years ago. Although there is some primitive understanding of the immune system, there is an urgent need for preventing vibriosis in farmed shrimp. Commercially available products such as Schering-Plough's AquaVac Vibromax have been shown to enhance productivity, reduce mortality and increase the health of farmed shrimp. Nevertheless, there is still only a limited understanding of the mechanisms and mode of action of the vaccine.

Aquaculture pharmaceutical companies, including Schering-Plough Aquaculture, have invested heavily in recent years in product development and product assessment. Pure immunology studies have also been funded in order to better elucidate the functioning of the shrimp immune system.

Indeed, Dr C Houghton, a comparative immunologist at St Andrews University in Scotland, has recently produced a schematic representation of the immune system in shrimp and the interactions that may be responsible for the "protection" induced by vaccination. (See Figure 1)

Immunostimulation can be achieved by contact of the antigen (stimulant) with three classes of cells within the haemolymph. This can either directly result in phagocytosis, or it initiates a cascade of subsequent immune processes. These are analogous in many ways to the immune system in fish, although it is much less differentiated. Commercially available vaccine preparations are likely to have a direct effect upon the cells, therefore potentially triggering the cascade of responses. Further research will elucidate the pathways and specificity of the responses.

Today's generation of vaccines have been developed to include a wide variety of strains to give the broadest spectrum of stimulation. They have moved a step beyond early prototype vaccines by using a protective coating system or antigen delivery vehicle to enhance uptake and protect the antigens during their passage into the shrimp larvae.

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CRUSTACEANS: BUMPER CAR DISEASE IN THE AMERICAN LOBSTER

BY DR SPENCER J GREENWOOD AND PROF RICHARD J CAWTHORN
(AVC LOBSTER SCIENCE CENTRE, UNIVERSITY OF PRINCE EDWARD ISLAND, CANADA)

Bumper car disease is caused by the ciliated protozoan parasite *Anophryoides haemophila*. It was first recognised as a significant pathogen associated with the cold water (<5°C) storage of American lobsters (*Homarus americanus*) in the early 1970s.

Although the disease contributes to the annual 10 to 15 percent losses associated with lobster-holding facilities in northeastern North America, the direct impact of bumper car disease to the industry (wild and held lobsters) is unknown.

The most confounding aspect in determining the importance of bumper car disease is that the lobster industry typically only reports outbreaks anecdotally. If we are to gain a clear picture of the importance of this disease in terms of its occurrence, prevention and management, we need more open and productive collaboration between researchers and industry players.

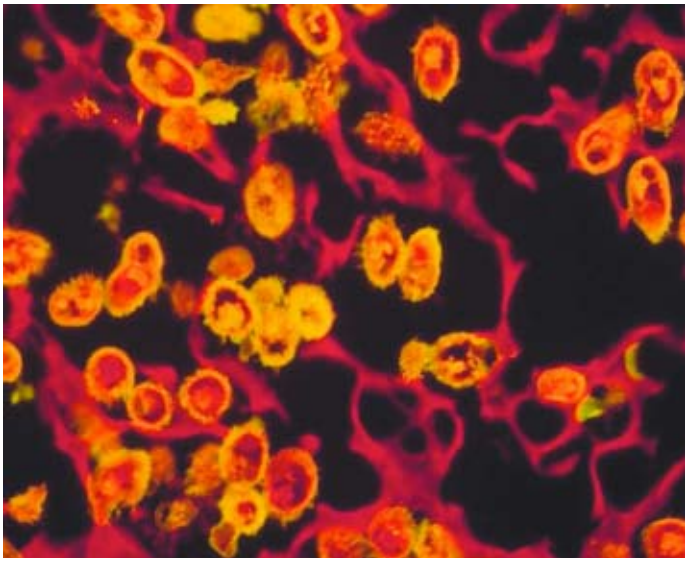
HOW DOES THE CILIATE CAUSE DISEASE

The exact portal of entry for the parasite into the lobster is unknown, although breaks in the carapace caused by previous disease (eg shell disease), fighting, rough handling and poor transportation practices are probably important.

The parasite may also breach the thin cuticular surface of the gill or epipodite during or around moulting when the lobster's carapace, its main external defence, is weakest. Once inside the lobster, the parasite undergoes rapid multiplication by binary fission within the local haemolymph sinuses of the gill.

In the early stages of disease there are no external cues that the lobster is infected. The parasite appears to "consume nutrients" within the haemolymph and there is also an accompanying decrease in the number of circulating haemocytes.

The exact mechanism of how the parasite causes disease in the lobster is unknown. After the haemocytes have been depleted the parasite spreads throughout the lobster's body. Systemically infected lobsters may appear weak and do not respond to external stimuli. Typically, lobsters die as a result of systemic infections within approximately four to nine weeks, depending on the size of the initial parasite challenge.



IMMUNOFLUORESCENT ANTIBODY STAINED ANOPHRYOIDES HAEMOPHILA WITHIN LOBSTER CONNECTIVE TISSUE

EIPDEMIOLOGY

The bumper car ciliate, *Anophryoides haemophila*, is known to exist endemically in wild lobster populations. It has been reported at levels of almost 20 percent in some populations on the eastern coast of North America, but a recent survey estimated the overall prevalence of *A haemophila* in the waters around Prince Edward Island at only 0.39 percent.

This low prevalence may reflect the temporal and spatial variation that can exist for a free-living organism that is opportunistically pathogenic. There may be some value therefore in yearly health monitoring of lobsters "dockside" to acquire long-term indices of prevalence in natural populations. This could be complemented and correlated with comparative data from holding facilities to monitor the annual impact of natural infections to the losses encountered within lobster pounds.

CLINICAL SIGNS AND HISTOPATHOLOGY

As with many internal diseases of crustaceans, there are few, if any, external clinical signs of disease, and lobsters may therefore only show general signs of weakness or reduced response to stimuli.

The general features of infections of lobsters with *A haemophila* parallel the non-specific response of crustaceans to other ciliate diseases. Evidence from both natural outbreaks and experimental infections of *A haemophila* has revealed two recognisable disease phases.

During the first phase, within the first few weeks of infection, there are no clinical signs, and ciliates multiply locally in the gill vascular spaces and associated tissues. Little pathology appears to be associated with the early phase of ciliate invasion and replication.

The second phase occurs as the ciliates disseminate systemically via the haemolymph to ramify throughout the connective tissue of other organs. The most consistently noticeable histopathological change is the appearance of localised haemocyte granulomas and encapsulated parasites in the associated connective tissue of major organs (especially the gills), and the presence of large numbers of ciliates "free" in the haemolymph spaces. Additionally, localised necrosis of the intestinal tract and hepatopancreas may lead to the secondary systemic spread of a mixed-rod shaped bacterial population. The second phase of disease is concomitant with marked haemocytopenia and the disappearance of reserve inclusion cells. The time course of the disease varies, with the time to death decreasing with an increased initial number of parasites.

It has been reported at levels of almost 20 percent in some populations on the eastern coast of North America,

DIAGNOSIS

Since diseased lobsters initially show no external clinical signs, the diagnosis of bumper car disease requires aseptic sampling of haemolymph (eg via the ventral sinus) and observation of ciliates within haemolymph wet mounts under direct light microscopy. Ciliates can also be identified further by axenic culture in the laboratory in defined media.

The parasites can then be fixed in Bouin's fluid prior to protargol staining and examination with light microscopy for species confirmation. The presumptive light microscopy diagnosis can be confirmed through small, sub-unit rDNA gene (SSU rDNA) PCR and sequencing. Diagnosis can also be made on cultured/isolated ciliates and from lobster tissue sections by using an indirect fluorescent antibody stain.

DISEASE CONTROL

Chemotherapy

Published laboratory studies have demonstrated the susceptibility of *A haemophila* to a number of commercial therapeutants approved for use in other food-producing animals, including monensin, lasalocid and pyrimethamine-sulphaquinoxaline.

However, definitive experiments to determine efficacy and withdrawal times have yet to be undertaken. This research will be required before the industry will be able to embrace the routine use of these therapeutic agents.

Management

The practice of preventing bumper car disease within holding facilities begins with the selection of lobsters for storage. As discussed above, lobsters in the early stages of infection do not show any clinical signs. Therefore, routine practice within holding facilities is for the early removal of dead and non-responsive animals, maintaining appropriate water quality (eg oxygen and ammonia levels) in balance with proper stocking densities.

The appropriate cleaning/disinfection protocols are essential to maintaining healthy lobsters. One caveat to disinfection is that lobsters appear to be highly susceptible to some of the routinely used chemicals. The challenge is delivering appropriate amounts of disinfectants to tidal pounds or refrigerated holding facilities that have high rates of turnover.

CURRENT RESEARCH

Current research has concentrated on gaining more information about the parasite and on the host:pathogen interactions between lobsters and *A haemophila*.

Molecular characterisation

A recent outbreak of bumper car disease from a lobster-holding facility in Nova Scotia provided the opportunity to initiate the first comparative genetic evaluation of the temporal and spatial relationships between *A haemophila* isolates.

The SSU rDNA sequence and randomly amplified polymorphic DNA fingerprinting analyses have confirmed that this Nova Scotia isolate was identical to the original isolate identified from a Maine lobster pound in 1993. The relevance of this finding is that, as for many other marine protozoa, we may be dealing with the same population and the same genotype over wide

ANOPHRYOIDES HAEMOPHILA, THE BUMPER CAR CILIATE. DIFFERENTIAL INTERFERENCE CONTRAST ILLUMINATION

geographic areas. This may help simplify our approaches to understanding the pathogenesis of bumper car disease, as there may be a homogeneous population.

An *A haemophila* cDNA library from which almost 10,000 expressed sequence tags were sequenced has revealed approximately 1000 recognised genes. Not unexpectedly, about 40 percent of the *A haemophila* ESTs have shown high similarity to genes found within the genomes of other protozoa, especially the ciliate *Tetrahymena*.

Sequence comparisons have identified specific groups of genes in the bumper car parasite that provide clues as to how the parasite attaches to the host (eg trichocyst matrix proteins), invades the tissues (eg chitinases) and compromises the host's immune response to establish disease systemically and eventually kill the lobster (eg cysteine proteases).

These results are very encouraging, and provide us with some molecular evidence towards the mechanism of pathogenesis and our understanding of the interactions between the parasite and the lobster.

Host-pathogen interactions

Research on host-pathogen interactions is attempting to determine indicators of lobster health by monitoring changes in the lobster's innate immune response during infection. Controlled infection experiment models have been developed with various pathogens, including *A haemophila*, to measure variations in the lobster's inflammatory response (acute phase proteins) and antimicrobial peptides. Concurrent research focuses on evaluating changes in the lobster haemolymph biochemical profile (enzyme changes indicative of tissue damage) using non-lethal sampling techniques.

The benefits of studying both sides of the host-pathogen interaction equation should provide a more comprehensive view of the disease. The true value of this increased knowledge to the lobster industry will be reflected in how this new information alters current management practices in an attempt to lessen the impact of bumper car disease.

ACKNOWLEDGEMENTS

Funding for the Canadian Lobster Health Research Project delivered by the AVC Lobster Science Centre represents a consortium of private sector companies, fishermen's organisations, First Nations and provincial and federal government agencies, including the Atlantic Innovation Fund through the Atlantic Canada Opportunities Agency.

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FINFISH: SKELETAL DEFORMITIES IN MEDITERRANEAN AQUACULTURE

BY DR GIORGOS KOUMOUNDOUROS (UNIVERSITY OF PATRAS, GREECE)

Mediterranean aquaculture is a relatively young industry and is focused on the production of marine finfish. The majority of the on-growing phase is performed in sea cages, while juveniles are produced in land-based hatcheries. Current commercially exploited species are gilthead sea bream *(Sparus aurata L)* and European sea bass *(Dicentrarchus labrax L)*.

In 2004, approximately 63,000 tonnes of sea bass and 100,300 tonnes of sea bream were produced (data from www.feap.info), representing a total increase in production of some 300 percent from 1996. Given that product prices followed an opposite trend over the same time period, the growth and sustainability of the industry is largely based on improvements in financial and biological management centred on marketing, production costs, product quality and species diversification, among others.

AN OVERVIEW OF SKELETAL DEFORMITIES

Skeletal deformities are not an exclusive characteristic of aquaculture. In 1553, Belon provided the first report in wild fish populations. This was followed shortly thereafter by another report by Guillaume Rondelet in 1555. Since then, a variety of studies have been published on the incidence and severity of deformities in wild fish populations.

The development of skeletal deformities in reared fish is a problem in the Mediterranean region with negative effects on product image, animal welfare, biological performance (growth, survival, feed conversion efficiency) and costs of production.

The latter is influenced as a result of the requirement to cull deformed fish (typically 10 to 90 percent observed frequency in commercial hatcheries), as well as increased grading pressure and the resultant manual removal of affected individuals. The problem is not confined to Mediterranean aquaculture, however.

Skeletal deformities also develop in other reared fish, including salmon, trout and cod. However, it is the marketing characteristics (whole, unprocessed fresh fish) that bring skeletal deformities to the consumer's attention, and therefore constitute a significant quality problem for the industry.

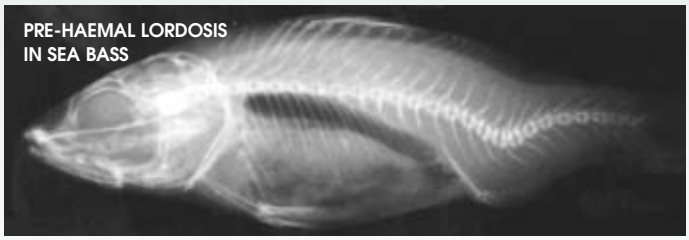
TYPES OF SKELETAL DEFORMITIES

Skeletal abnormalities are classified as vertebral, cranial or fin deformities, depending on the affected anatomical region of the fish.

Vertebral deformities

Vertebral deformations are divided macroscopically into lordosis, kyphosis, scoliosis and compression. They have been reported to develop either in isolation or in combination, with varying degrees of severity. Vertebral deformities are attributed to dislocation, fusion, shortening or deformation of the affected vertebrae. Lordosis and kyphosis are the most extensively studied of all of the vertebral deformities.

In Mediterranean marine fish culture, vertebral lordosis was initially the most severe and frequent abnormality. In general terms, lordosis can affect the pre-haemal or haemal parts of the vertebral column. In all species, pre-haemal lordosis is induced by non-inflation of the swim bladder. This is thought to be a consequence of an abnormal oblique swimming orientation.



PRE-HAEMAL LORDOSIS IN SEA BASS

JAW DEFORMITIES IN SEA BASS

Haemal lordosis develops in fish with inflated swim bladders, but under conditions of intense swimming effort precipitated by high-velocity water currents in weaning tanks. Haemal lordosis can affect up to 70 percent of individuals in a rearing unit, and is especially prevalent in sea bass.

Pre-haemal kyphosis develops in both sea bream and sea bass. In the latter species, kyphosis develops during the weaning phase, primarily on the fifth and sixth vertebrae. Kyphotic specimens initially exhibit lethargic swimming behaviour.

Cranial deformities

Skull abnormalities are diverse, but are typically found on the gill cover, jaws and hyoid arch.

Abnormalities of the gill cover are the most commonly observed and can affect a large proportion (up to 80 percent) of reared populations. To date, various types of deformities have been reported in sea bream and sea bass, of which shortened opercula is the most important. Although the causative factors remain unknown, it was recently shown that the incidence of opercular deformities in milkfish *(Chanos chanos)* was significantly reduced when larvae were fed on live food enriched with EFA, DHA and vitamin C.

Pugheadness, cross-bite and lower jaw reduction/elongation are the main oral deformities noted in sea bass and sea bream. Pugheadness refers to the antero-posterior compression of the snout area, while in cross-bite the lower jaw is skewed off-centre or displaced laterally. The reduction of the lower jaw is almost identical to the intense cross-bite, and is anatomically attributed to the ventro-lateral distortion of the jaw elements.

Although deformities are not species specific, there is a clear sensitivity of sea bream to pugheadness, and of sea bass to cross-bite and lower jaw deformities. To date, no single factor has been clearly demonstrated to induce jaw deformities in Mediterranean fish.

Fin deformities

Partial fin development is one of the most severe deformities of the dorsal or anal fin and occurs during the early larval stage.

Lateral bending, duplication, partial lack and stricture of the caudal fin are severe osteological deformities, both in terms of their incidence (up to 100 percent) and their effects on overall body shape.

Abnormalities of the caudal fin are the developmental result of posterior notochord deformities, first detected at the yolk-sac larval stage. Other fin problems include internal abnormalities of the fin supporting elements and curvatures of the rays and spines, although thankfully these developmental issues have a negligible effect on external morphology.

PREVENTION AND CONTROL

In general terms, identifying the optimum nutritional and environmental characteristics during the ontogeny of each species is a prerequisite for reducing both the incidence and the severity of skeletal deformities. In parallel, research can directly elucidate the most significant causal factors, although experience has shown that this is more difficult in practice than in theory.

The lack of specific guidelines for producing "deformity free" juveniles is also a key issue. As causative factors may have overlapping symptomatology, producing such guidelines is particularly difficult. Nevertheless, the study of the ontogeny of each deformity is very important, as it can focus research on the target developmental phase and identify the key environmental factors that influence it.

As a second step to be carried out in conjunction with an improvement in rearing conditions, research has to focus on finding methods that identify deformed fish, therefore reducing production costs and guaranteeing product quality. In this respect, skeletal deformities have to be viewed not simply as a problem of "abnormal bones", but as a problem of "abnormal fish shape". Based on precise scales of quality, deformities have to be assessed in relation to their effects on the external appearance and biological performance. Finally, the establishment of quality control procedures is of major importance in the fight against skeletal deformities.

Under conditions of optimum production, the routine control of larvae and juveniles will guarantee product quality and "best practice" with respect to hatchery procedures. In addition, it will allow the early detection and correction of any potential divergence from optimum conditions and quality, well before the problem escalates and the economic consequences become serious.

The nature of the problem requires an integrated research approach, focused not only on the practical aspects but also understanding the basic mechanisms involved. This will facilitate not only identifing key causative factors, but also contribute to understanding the relative mechanisms in other economically important species.

Nevertheless, researchers and producers have to treat each species according to its specific biological characteristics. For example, although egg quality has been considered crucial for the development of skeletal deformities, it does not have the same significance in sea bass or sea bream as it does in salmonids. The importance of egg quality to fish development is much more important in salmon, due to the significantly larger egg size, and the more advanced developmental stage at hatching or yolk consumption.

AND FINALLY...

The Mediterranean aquaculture sector continues to be a healthy industry. To cope with the decrease in prices, production and research have to enhance the quality not only of the final product, but also of production methodology. Nowadays the industry copes with the problem of skeletal deformities mainly by applying removal procedures at high cost (ie manual sorting of deformed fish), and therefore low quality products rarely reach the marketplace.

However, in the years ahead, efforts should be directed towards increasing cost efficiency by following the strategy of improved rearing conditions, quality assessment and mass selection. ■

RESEARCH **SNIPPETS**

Iceland: Research links UV water treatment with cataract formation in cod

Research carried out at the Marine Research Institute in Iceland and published in the journal Aquaculture demonstrated a link between UV-treated seawater and cataract formation in juvenile cod.

The greatest effect was observed in the tank located closest to the UV unit, while it was lowest in the one farthest away. It is thought that ozone, or other photoproducts formed by UV exposure, may be involved in cataract formation. That the effect is exacerbated by increased stocking density also suggests that physical damage is an important co-factor in cataract development in juvenile cod.

For further information, consult Bjornsson B (2004). Can UV-treated seawater cause cataract formation in juvenile cod *(Gadus morhua L)*. Aquaculture 240. pp 187-199

Spain: Efficacy of furunculosis vaccines in turbot varies according to the mode of delivery

Researchers from the Universidad de Santiago and Stolt Sea Farms found that the commercial furunculosis vaccine Aquavac Furovac 5 and an autogenous (water-based) alternative both induced protection against furunculosis when administered by injection.

However, when administered via the immersion route, the vaccines failed to result in significant levels of protection. Interestingly, field trials suggested that giving turbot an oral boost following the primary vaccination had no additional effect on the degree of protection against furunculosis.

For further information, consult Santos Y, Garcia-Marquez Pereira PG, Pazos F, Riaza A, Silva R, El Morabit A and Ubeira FM (2005). Efficacy of furunculosis vaccines in turbot, *Scophthalmus maximus (L):* evaluation of immersion, oral and injection delivery. Journal of Fish Diseases 28. pp 165-172

Israel: Selective breeding enhances resistance to koi herpes virus

Yechiam Shapira and colleagues recently published a paper in the journal Aquaculture describing a series of experiments looking at the resistance of various crossbreeds of domestic and wild strains of carp *(Cyprinus carpio)* to KHV challenge.

In laboratory trials, a domestic/wild crossbreed resulted in substantial resistance to viral challenge (60.7 percent survival). At the other end of the spectrum, one particular strain of domestic crosses was particularly susceptible to KHV, resulting in a post-challenge survival rate of only eight percent. Such results highlight the potential of selective breeding to reduce the impact of KHV in both ornamental and koi carp.

For further information, consult Shapira Y, Magen Y, Zak T, Kotler M, Hulata G and Levavi-Sivan B (2005). Differential resistance to koi herpes virus (KHV)/carp interstitial nephritis and gill necrosis virus (CNGV) among common carp *(Cyprinus carpio L)* strains and crossbreds. Aquaculture 245. pp 1-11

FINFISH: LACTOCOCCOSIS – COMPLEX BUT MANAGEABLE

BY DR MARK SHEPPARD (SAKANA VETERINARY SERVICES LTD, CANADA)

Lactococcosis, or Rensa (chains) as it is named in Japanese, is a lingering bacterial infection of warm-water fish, particularly in yellowtail and other species of the *Seriola* genus.

It is a common disease that tends to become increasingly problematic in one to two-year-old fish, and the disease becomes most devastating as fish populations approach harvest size of 3.5kg to 4kg (Photograph 1). Lactococcosis alone produces moderate, ongoing mortality rates and the cumulative loss of fish can become a significant problem to farmers. Lactococcal infections frequently arise in combination with other concurrent infections, or poor environmental conditions, and the situation easily exacerbates the mortality rates.

Lactococcus bacteria are often discovered incidentally upon examining sick fish exhibiting signs of more obvious diseases, such as jaundice syndrome or blood fluke infestations.

1. FLOATING FISH CARCASSES IN CAGES. *LACTOCOCCUS*-RELATED MORTALITY RATES CAN INCREASE IN SUMMER IF MONITORING IS NEGLECTED

CAUSATIVE AGENT

The bacterial history of this disease is a bit confusing. Lactococcosis was initially considered to be caused by a *Streptococcus* bacteria, then by an *Enterococcus* species. Taxonomists and microbiologists now tend to agree that the disease is a result of a systemic invasion by *Lactococcus garvieae*. However, confusion still arises when a grossly similar disease to Lactococcosis is diagnosed in the laboratory, and found to be a result of *Streptococcus dysgalactiae* infection.

TRANSMISSION AND EPIDEMIOLOGY

The initial exposure of fry to *Lactococcus* is largely unknown. However, it is speculated that fry that consume uncooked fish tissues, whether live, raw or frozen, are at greater risk of exposure to the bacteria.

The use of raw, low-quality trash fish should be avoided when rearing any type of fish. The infection develops silently as the bacteria multiply slowly inside its new fish host over weeks or months. The bacteria are thought to progressively invade, dwell and multiply inside numerous types of fish host cells, including white blood cells of the immune system.

The duration of the infection is long-term, resulting in mortality rates that gradually increase in the summer and autumn as water temperatures increase and dissolved oxygen concentrations decrease. Due to the chronic and systemic nature of this bacterial infection, sick fish may become anaemic. With an overall reduction of red blood cells the fish are more susceptible to environmental challenges and other infections.

The cohabitation of infected or diseased fish with healthy fish leads to the horizontal transmission of *Lactococcus* from one fish to the next. So proximity and crowding seem to be contributing factors of this disease. Research indicates that healthy yellowtail sharing tank space with sick juveniles previously injected with *Lactococcus* readily become infected by the bacteria, yet the fish may not succumb to the disease provided the level of dissolved oxygen remains high.

In marine finfish culture, lactococcosis and mortality rates progress more quickly during the summer months after water temperatures reach 24˚C and available oxygen levels decline.

CLINICAL SIGNS AND GROSS PATHOLOGY

The visual symptoms of this disease vary somewhat (See photographs 2, 3, 4 and 5). Moribund, lethargic fish are evident ▶

2. BULGING, RED AND CLOUDY EYES WITH LACTOCOCCOSIS DISEASE. THE IMMUNE REACTION TO BACTERIAL ACCUMULATIONS BEHIND THE EYES PUSH THE GLOBES OUTWARD

3. RED JAWS AND GILL COVERS ARE TYPICAL SYMPTOMS OF LACTOCOCCAL INFECTIONS

Please visit the Intervet booth (D-345) at AQUA NOR 2005

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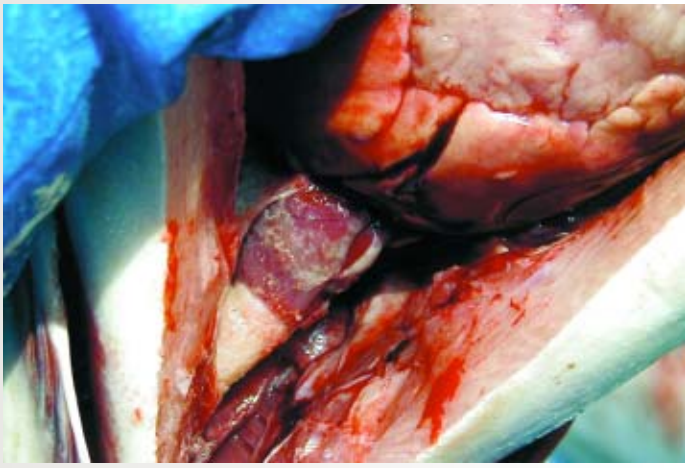
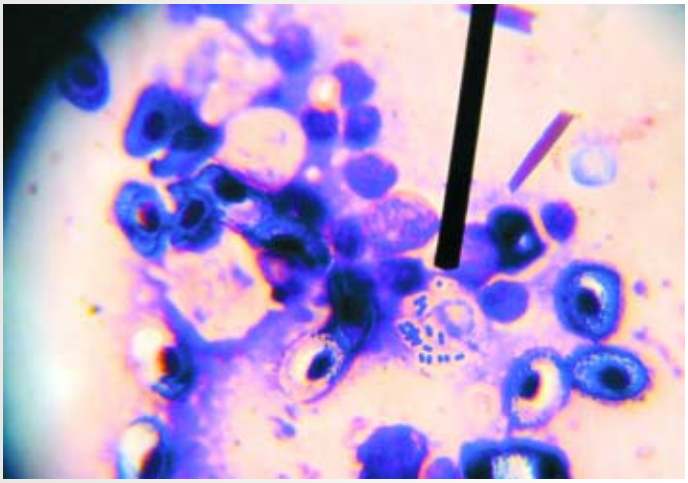


5. INTERNAL LESIONS ASSOCIATED WITH LACTOCOCCAL INFECTIONS ARE NOT OBVIOUS. SOME DRYNESS AND MINOR REDNESS IS EVIDENT WITHIN THE BODY CAVITY, BUT THE HEART CAN BE COVERED WITH A WHITISH MEMBRANE (FIBRINOUS EPICARDITIS)



6. IMPRINTS OF KIDNEY TISSUE OR FLUID FROM AN ABSCESS WILL REVEAL GRAM-POSITIVE ROUND BACTERIA, IN PAIRS OR IN SMALL CHAINS, WITHIN WHITE BLOOD CELLS (1000X)

among the affected populations since the disease is chronic.

The typical external lesions are seen in relatively robust fish. Signs include bulging, cloudy eyes with red iris or red fleshy tissue protruding around the eyeball, pale gills, ulcerated tail abscesses, a red, tattered tail fin, a red lower jaw and red inner gill cover with yellow abscesses or pus at its base.

If the internal body organs appear normal, the brain is often red, indicating a local infection and inflammation of the meninges. The meningitis syndrome is clinically evident as fish swim in erratic or circular patterns on the surface. The kidney is usually enlarged and grey, and the heart may have a soft, white outer membrane. Mixed bacterial infections are common with Lactococcosis, in which case additional pathology develops.

MICROBIOLOGY

The laboratory isolation of *Lactococcus* or *Streptococcus* is not difficult. A hot, sterile loop or sterile swab is used to collect a sample from the kidney. Once the sample is transferred to a bacterial agar plate (ie brain-heart infusion agar, blood agar or triptych soy agar), the incubation time is normally 24 to 48 hours at 18 to 22˚C. Drug sensitivity tests in the laboratory must be performed before contemplating the use of an oral antibiotic to control the disease.

DIAGNOSIS AND PRIMARY ON-SITE TESTS

A thorough visual examination of the fish will reveal usually pale gills and redness on the inner side of the operculum. Kidney, brain and heart imprints or "stamps" onto glass slides can be collected, dried and stained using Gram's stain.

With a compound microscope, one can read the slide to look for Gram-positive (purple) round bacteria arranged in pairs or short chains (Photograph 6). Five millimetre sections of the same tissues preserved in 10 percent buffered formalin are helpful if a histological diagnostic service is available.

MANAGEMENT AND CONTROL

The best prevention and control of Lactococcosis is through husbandry and good management practices such as strict diving hygiene, disinfection of hands and equipment and stringent bio-containment measures.

Minimising fish stress is beneficial to prevent or control the spread of infections. Avoid the use of uncooked live, raw or frozen fish feeds when rearing fish of any age or type. Feed only dry, cooked feed. Reduce the shellfish fouling (ie barnacles and mussels) on nearby floats and ropes of finfish cages whenever possible.

Vaccination against *Lactococcus* is another common approach. Both oral and injectable vaccines are now used effectively to minimise this disease. The *Lactococcus* vaccine, however, does not prevent the disease caused by *Streptococcus dysgalactiae*.

Both these diseases can also be controlled somewhat through the judicious use of prescribed, in-feed, antibiotic therapy. In general, the oral application of an appropriate anti-microbial chemical for 10 to 14 days (and repeated as prescribed) usually helps to minimise losses, particularly if the infection is detected early.

The monitoring of carcasses and the early detection of silent infections among juvenile live fish is the key. The use of drugs to control bacterial infections in semi-open culture systems is controversial. Environmentally, consumers are not in favour of drug treatments and, from a fish production viewpoint, it is very difficult to ensure that fish will consume sufficient volumes of medicated feed to achieve a therapeutic daily dose.

When medicating sick fish, the fish group tends to reduce their daily food intake, which slows their weight gain, thus creating additional costs to the farmer. As usual, the problems are challenging but manageable.

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SHELLFISH: MSX DISEASE STILL GOING STRONG

BY DR EUGENE BURRESON (VIRGINIA INSTITUTE OF MARINE SCIENCE, USA)

MSX disease was first documented in 1957 in Delaware Bay, where it caused massive mortalities of the eastern oyster, *Crassostrea virginica*. In 1959 it was found in the lower Chesapeake Bay, where it also caused massive mortalities of the eastern oyster. At that time the disease agent was given the acronym MSX, for multinucleated sphere X (unknown) because of the morphology of the plasmodial stage.

In the 1960s the parasite spread north and south from the middle Atlantic region and was found in the coastal bays of North Carolina, Virginia, Maryland, Delaware, New Jersey, Connecticut and New York, but associated oyster mortalities occurred primarily in Chesapeake and Delaware Bays.

Identification of the spore stage of the parasite revealed that it was a member of the phylum Haplosporidia, and led to it being formally named *Minchinia nelsoni* in 1966. The parasite was transferred to the genus *Haplosporidium* in 1980.

In the 1980s, an apparent range extension occurred as the parasite was reported as far north as Maine and as far south as Florida, and epizootics occurred in Long Island Sound and in Maine in the 1990s. In 2002, *H nelsoni* caused mortality of *C virginica* in Bras d'Or Lakes, Nova Scotia, Canada and there is anecdotal evidence that the pathogen was introduced into that area in ballast water. The pathogen has not been reported from the Gulf of Mexico, even though there are large populations of susceptible oysters in that region.

INTRODUCED PATHOGEN

Molecular diagnostic techniques have greatly increased our understanding of *H nelsoni*, and we now know that the pathogen was introduced to the eastern coast of the United States from the Pacific Ocean. A morphologically similar parasite in the Pacific oyster, *Crassostrea gigas*, in Korea, Japan and along the western United States was determined to be *H nelsoni* by DNA sequence analysis.

The parasite is rare in *C gigas* and does not cause noticeable mortality in that host. Neither the exact mechanism nor the timing of the introduction is known, but it is often inferred that the parasite was introduced in deliberate, documented introductions

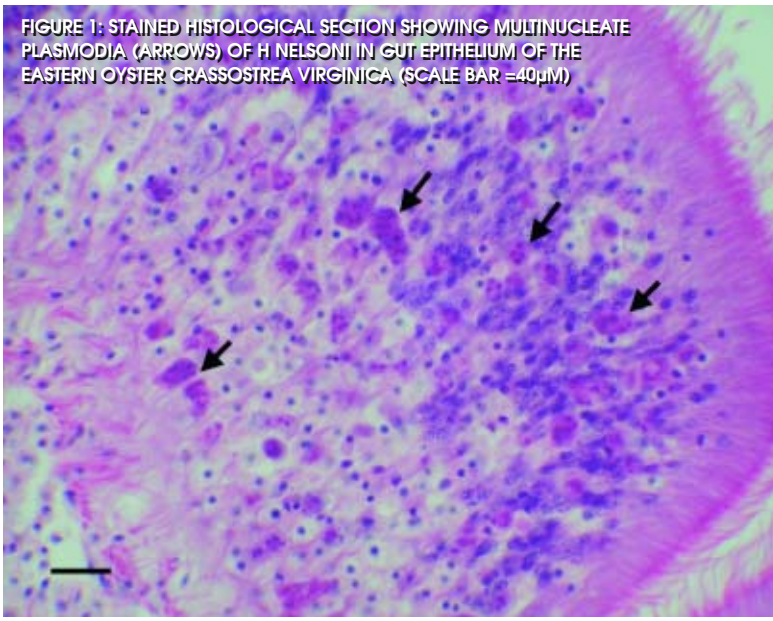

FIGURE 1: STAINED HISTOLOGICAL SECTION SHOWING MULTINUCLEATE PLASMODIA (ARROWS) OF H NELSONI IN GUT EPITHELIUM OF THE EASTERN OYSTER CRASSOSTREA VIRGINICA (SCALE BAR =40µM)

of *C gigas* made by oyster growers and scientists.

However, other possibilities include infected *C gigas* attached to ship's hulls or the release of infective stages in ballast water, as likely occurred in Nova Scotia. The parasite also occurs in *C gigas* in France, where it was likely introduced with shipments of *C gigas* from Japan.

MORPHOLOGY AND BIOLOGY

The predominant stage of the organism in the oyster is a spherical multinucleated plasmodium ranging in diameter from 5 to 100µm (Figure 1) and in which the nuclei have a peripheral nucleolus. The production of spores (sporulation) (Figure 2) is rare in adult oysters, but has been observed at prevalences as high as 40 percent in young oyster spat.

Sporulation occurs from late June to early July and over a more prolonged period in the autumn. Spores are about 8µm long ▶

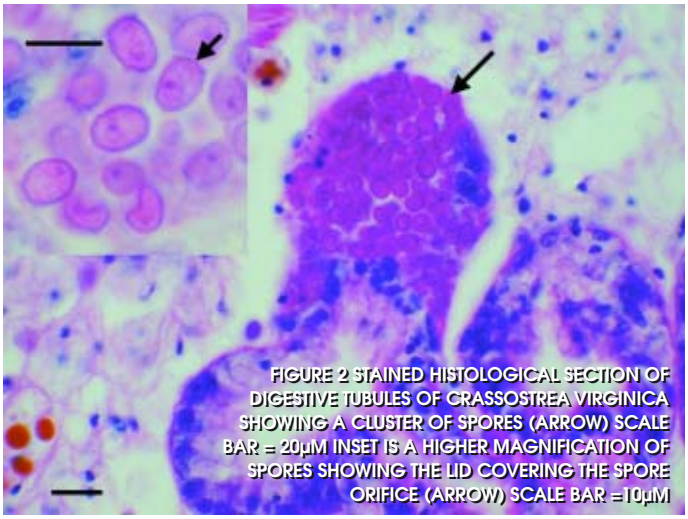

FIGURE 2 STAINED HISTOLOGICAL SECTION OF DIGESTIVE TUBULES OF CRASSOSTREA VIRGINICA SHOWING A CLUSTER OF SPORES (ARROW) SCALE BAR = 20µM INSET IS A HIGHER MAGNIFICATION OF SPORES SHOWING THE LID COVERING THE SPORE ORIFICE (ARROW) SCALE BAR =10µM

and have an opening at one end that is covered with an overhanging lid.

Spores of *H nelsoni* occur only in the epithelium of the digestive tubules and are presumed to be released in faeces or upon the death of the host. The fate of spores in the environment is unknown.

Spores are not directly infective to oysters and it is presumed that they are ingested by an (as yet) unidentified intermediate host. The complete life cycle and the source of the infective stage to oysters remains unknown.

In Chesapeake Bay, oysters become infected from mid-May through October. However, infection pressure during late summer and autumn is variable from year to year. Infections develop rapidly in susceptible oysters, resulting in mortalities from July through October.

Surviving oysters may maintain a high prevalence of the disease through the winter and a second period of mortality may occur in spring. Oysters acquiring infections in late autumn may harbour low-level infections which intensify the following summer. These infections often proliferate as temperatures warm in June, causing early summer mortalities. The disease can affect all ages of oysters, from spat to adult.

Temperature and salinity play an important role in regulating *H nelsoni*. Temperature is responsible for the annual cycle of infection, disease development and remission. Infections are acquired at temperatures above about 20°C.

Three critical temperatures have been proposed for oyster-*H nelsoni* interactions. Both parasite and oyster are inactive at temperatures less than 5°C. At 5-20°C the parasite proliferates rapidly. Above 20°C, selectively bred resistant oysters can overcome the parasite while susceptible oysters are killed.

Salinity is important in determining the distribution of the disease within an estuary. A salinity of 15psu is required for infection, and 20psu is required for rapid parasite proliferation. Salinities of 10psu or below result in expulsion of the parasite from oysters within 10 days at temperatures above 20°C.

Therefore, annual variability in climate can have a dramatic impact on the distribution and abundance of the pathogen. During drought periods, when salinities increase within an estuary, *H nelsoni* can spread rapidly up an estuary. Conversely, during unusually wet years, the parasite may be eliminated from oysters in many areas.

DIAGNOSIS AND CONTROL

Histological examination using light microscopy of paraffin-embedded tissue sections is the standard diagnostic technique for *H nelsoni*. DNA-based molecular diagnostic tools have also been developed for *H nelsoni* and include specific PCR primers and a DNA probe for use in hybridisation in situ.

There is no known effective chemical treatment for *H nelsoni* infections. Low salinity treatment may effectively remove infections at temperatures above 20°C, but is impractical on a large scale. The most effective control measure is to use oysters selectively bred for tolerance to *H nelsoni*.

Selective breeding programmes are underway at the Rutgers University Haskin Shellfish Research Laboratory and the Virginia Institute of Marine Science. These programmes have produced strains of oysters that show significantly reduced mortality from *H nelsoni* compared with susceptible strains, and they are currently being used by the shellfish industry.

RECENT TRENDS

After nearly 50 years of infection pressure, there is some evidence that native oysters in Delaware Bay and lower Chesapeake Bay are developing a natural resistance to *H nelsoni*.

Whereas late-summer *H nelsoni* prevalence in Delaware Bay ranged between 50 and 90 percent before 1989, prevalence has rarely exceeded 30 percent since that time, even though environmental conditions have been optimal for disease development. A similar observation has been made in lower Chesapeake Bay.

Nonetheless, *H nelsoni* continues to be a serious threat to oyster populations in many areas along the eastern coast of North America. During four consecutive drought years from 1999 through to 2002, *H nelsoni* spread further up Chesapeake Bay and its major tributaries than any time before, contributing to the highest percentage of disease-related oyster mortality recorded in the last decade.

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FINFISH: TUNA HEALTH – THE FINAL FRONTIER?

BY DR BEN DIGGLES (DIGSFISH SERVICES PTY LTD, AUSTRALIA)

The aquaculture of southern bluefin tuna *(Thunnus maccoyii)* (hereafter referred to as SBT) in South Australia has been one of the more interesting stories in Australian finfish aquaculture. Major declines in the SBT wild fishery due to overfishing of this relatively slow-growing, long-lived tuna species resulted in the implementation of a global quota in the early 1980s.

However, this did little to halt plummeting SBT populations, and the quotas were gradually reduced throughout the 1980s, including a 50 percent cut in quota in 1988 to a level that remains virtually unchanged today.

In the early days of the fishery a large proportion of the catch was captured by pole and line fishing, and the product was processed into canned tuna which demanded relatively low market prices. However, following the quota cuts, increasing demand from lucrative Asian markets opened up opportunities for entrepreneurial fishermen to add value to their catch by fattening wild-caught tuna in holding pens.

The Australian town of Port Lincoln has historically been the major centre of exploitation of SBT. It was therefore fitting that SBT aquaculture was first initiated there in 1990 through a collaborative research and development programme involving the Tuna Boat Owners Association of Australia, the Japanese Overseas Fishery Corporation Foundation and the South Australian government.

The first experimental tuna-fattening farm was set up in Port Lincoln in the early 1990s. Since then, the fishers have gradually developed and refined a system of growing out tuna that can increase their value in a relatively short time.

Juvenile fish, mainly two to four years old, are purse-seined from the Great Australian Bight and towed to the waters off Port Lincoln, where they are fed a diet consisting largely of imported pilchards (*Sardinops* spp) and mackerel (*Scomber* spp).

They are ready for harvest after three to six months of fattening, depending on market price movements. The tuna are then prepared for the sashimi market and exported as whole dressed-out fish, mostly to Japan. Around 75 percent goes as fresh product by air, and the balance as frozen product by sea.

TECHNICAL DIFFICULTIES

While the basics of tuna farming seem simple, the logistics of holding thousands of tonnes of tuna captive in the high seas means there were many significant technical and operational hurdles to overcome.

The economic success of these ventures today says volumes for the ingenuity and persistence of the industry and is reflected in the profitability of the aquaculture companies growing tuna.

The industry now employs more than 2000 people and generates around A$250 million in exports. However, in the face of increasing overseas market competition the SBT aquaculture subprogramme run by the South Australian government predicts that the future growth of the industry will depend on research and development successfully addressing a range of issues.

These include:

- access to additional quota
- new farm sites and sources of bait fish
- the availability of manufactured feeds
- a knowledgeable workforce

THE RELIANCE ON IMPORTED BAITFISH POSES A NUMBER OF FISH HEALTH AND PERFORMANCE ISSUES

- the longer-term holding of wild-caught SBT
- improved husbandry practices that ensure a healthy farm environment
- control of fish health issues, and
- the marketing of a high-quality product.

If the SBT industry in Australia is to continue to grow, given that access to additional quota is not a viable option (at least until wild spawning stocks recover to a safe level), the research emphasis shifts towards closing the lifecycle to provide a supply of tuna fingerlings.

However, even though the life cycle of the closely related Pacific bluefin tuna *(Thynnus orientalis)* has been completed in experimental culture (See www.blackwell-synergy.com/doi/abs/10.1111/j.1365-2109.2005.01222.x;jsessionid=hmlpMOcTOEV9?journalCode=are), progress with SBT has been slow.

In any case, the logistics of ongrowing juvenile tuna for years are mind-boggling. In the short term the status quo is likely to remain with respect to the industry's source of fish, hence to increase production the emphasis shifts to improved husbandry, holding the fish for longer periods to increase their growth, and reducing mortalities while fish are held in sea cages.

Which brings us to the increasingly important question of tuna health. Compared to other aquaculture industries, the SBT farming industry has experienced relatively few health problems, probably because the fish are held only for relatively short periods. However, fish health has been identified as a key restraint likely to prevent fishers from holding SBT for longer periods.

TUNA HEALTH

The importance of fish health was bought home to the industry for the first time in 1996 after a large-scale tuna kill occurred in aquaculture operations in Boston Bay, the calm waters closest to Port Lincoln. The disease event resulted in the loss of approximately 1700 tonnes (or A$40 million worth) of tuna.

The South Australian government report that collated and synthesised the information on this event concluded that the cause of the fish kill was asphyxiation caused by sediment stirred up by a storm.

However, there is evidence that the presence of toxic micro-algae was also implicated (Munday & Hallegraeff 1998), and some ▶

FINFISH: TUNA HEALTH – THE FINAL FRONTIER?

THE SOUTHERN BLUEFIN TUNA FATTENING INDUSTRY HAS BEEN A SUCCESS STORY OF AUSTRALIAN FINFISH AQUACULTURE

speculation that nutrient enrichment associated with tuna farming in semi enclosed waters may have initiated an algal bloom which precipitated the disease outbreak.

A lack of data before and at the time of the event makes it difficult to be conclusive about the cause. Since that time the industry has moved cages offshore and now regularly undertakes routine environmental monitoring. To date the environmental monitoring programme has shown the impact of tuna cages on water quality in their offshore locations is low, and that there is also little impact on sediments directly below the farms.

The industry has become increasingly active in recent years implementing biologically focussed fish health research programmes that have investigated a variety of important topics. SBT are warm-blooded, high-performance fish, and research has been undertaken to determine how this characteristic influences their physiological and nutritional status during their confinement.

Surveys of the parasites and other potential disease agents have also been undertaken. SBT can suffer from fatal encephalitis called "swimmer syndrome", due to infection of the nervous tissue by the ciliate *Uronema nigricans*, while other disease agents such as sanguinicolid blood flukes *Cardicola forsteri* and an unidentified capsalid monogenean gill fluke have also been identified as priority targets for research.

A recent parasitological investigation (Deveney et al, 2005) revealed a diverse parasite community on cultured tuna, including

- *C forsteri* (prevalence 31 percent)
- didymozoid monogeneans at prevalences of up to 93 percent
- two species of copepods (*Pseudocycnus appendiculatus* and *Euryphorus brachypterus*) at 44 and 32 percent prevalence, respectively
- an isopod *Ceratothoa* sp (0.006 percent prevalence)
- a polyopisthocotylean monogenan (*Hexostoma* sp 24 percent prevalence)
- cestode larvae (four percent prevalence), and
- *U nigricans* at a prevalence of 58 percent in dead fish and 100 percent prevalence in fish clinically affected by swimmer syndrome.

Interestingly, the capsalid monogenean was not recorded in this survey. Research into the epidemiology of these parasites and their effects on tuna health continues.

One controversial fish health issue that remains is tuna feed. A significant amount of research has been conducted into the nutritional requirements of SBT, including research into the development of a palatable and artificial pellet which gives improved food conversion ration and growth. However, to date the industry still relies on baitfish caught in the wild.

Until recently over 50,000 tonnes of whole (head on, ungutted) frozen baitfish, mainly pilchards and mackerel, were imported into South Australia each year for use as tuna food. Recent increases in the quota allowed to be taken from the local pilchard fishery has resulted in increased use of locally caught pilchards, however a shortfall remains and around 25,000 tonnes of feed must still be imported.

Some observers consider this practice to be one of Australia's most significant marine biosecurity risks. This is because a significant proportion of these baitfish are imported from areas such as California, where fish diseases exotic to Australia, such as viral haemorrhagic septicaemia (VHS), are known to be endemic.

Though the importation of whole baitfish was suspended for a time after an outbreak of VHS in California, risk assessments done since then suggest that the likelihood of introduction of exotic diseases via this route is very low, and the practice is permitted provided the fish are wild-caught and free of visible lesions. See www.daff.gov.au/content/publications.cfm?objectid=c3e4a1f9-93df-4e34-ad05cd99e2582d7e.

Opponents to this decision point out that in two of the world's largest single species fish kill events, both associated with novel herpesvirus strains which infected Australia's native pilchard populations, the initial source of both epizootics in 1995 and 1998 was within 250km of Port Lincoln. See www.dx.doi.org/10.1006%2Fjmsc.2001.1077). Cause or coincidence? The debate remains, but given that both epizootics extended over 7700km, the statistical likelihood of them both randomly originating within the same 250km stretch is 0.001 (Gaughan 2002).

When the factors surrounding this thorny issue are considered, most objective observers would conclude that reducing the reliance of the industry on imported frozen baitfish through development of a cost-effective artificial pelleted diet would be a major step forward towards improving both the sustainability of the tuna farming industry in Australia, and its fish health status with respect to both tuna and native fish populations.

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INTERVET DEVELOPS INNOVATIVE CD

Intervet Norbio Singapore and Intervet International have developed an innovative educational and training CD on fish vaccination and health management, with a focus on Asia.

The content is divided into various sections, including chapters about Intervet, aquaculture, health management and fish vaccination. Media used includes presentation slides, video clips, animations, PDF downloads and website links.

The CD was first shown at Intervet's booth at the recent World Aquaculture 2005 congress in Bali. All the participants at Intervet's workshop, fish vaccination in Asia, held during the congress also received a copy. According to Intervet, the feedback on the first limited edition was extremely positive.

If you are interested in receiving a copy of the CD, contact info.aquaINS@intervet.com giving your full name, organisation, postal address, mail address and phone number.

NEW DISINFECTANT LISTING SCHEME

The use of safe, effective disinfectants is important for implementing effective biosecurity in intensive aquaculture, as it is for other farming operations.

Important uses include treating effluent and equipment such as tanks, nets and transportation containers, and limiting the spread of disease within and between sites.

Disinfectants are also required for decontaminating premises where there have been outbreaks of notifiable diseases such as infectious salmon anaemia, spring viraemia of carp, or viral haemorrhagic septicaemia. They are also commonly used to sanitise the eggs of cultured species, particularly salmonids.

In the United Kingdom, under the provisions of the Animal Health Act 1981, the Department for Environment, Food and Rural Affairs, or DEFRA, maintains a list of disinfectants approved for use in controlling notifiable diseases of terrestrial animals and birds. This system of approval does not extend to the pathogens of fish and shellfish.

DEFRA is developing a new scheme that will give disinfectant manufacturers the opportunity to prove that their products are effective against relevant aquaculture pathogens, as well as terrestrial diseases. Products that satisfy DEFRA criteria will be placed on a list which will be freely accessible to farmers and aquaculture health professionals, aiding them in the selection of effective products.

The standards used to evaluate a disinfectant's effectiveness are being developed in consultation with relevant experts from across the United Kingdom and Norway.

For further information, contact David Verner-Jeffreys at CEFAS, Weymouth Laboratory, Barrack Road, The Nothe, Weymouth DT4 8UB. Email d.verner-jeffreys@cefas.co.uk or phone 44 (0) 1305 206725.

BOOK REVIEW:
A Photographic Guide to Diseases of Yellowtail (*Seriola*) Fish

There is an ever-growing range of specialist fish health management and pathology texts aimed at fish health professionals. What is commonly lacking, however, are easily accessible, practical guides suitable for a wider audience.

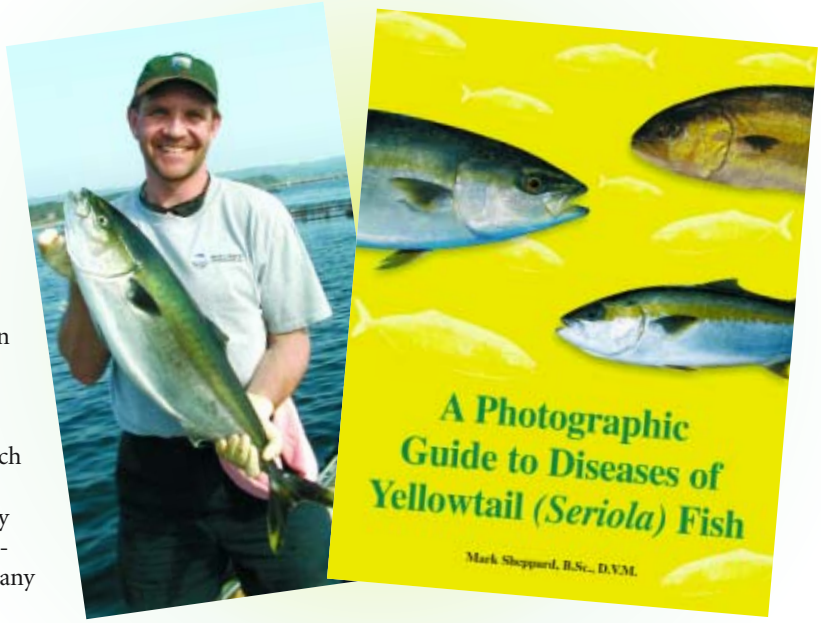
Those at the "coal face" of fish culture, including fish husbandry operators, laboratory personnel and fish health managers, require photographic reference guides to aid "on-farm" disease identification.

Mark Sheppard's new book, which is available in English and Japanese versions, fulfills such a role for those involved in the culture of *Seriola* fish (yellowtails, amberjacks and kingfish). Dr Sheppard is a Canadian fish veterinarian with wide-ranging practical experience of Seriola health management in Australia, New Zealand and Japan, and as such is well placed to write such a publication.

The book is divided into five main sections. The first briefly discusses general topics, including biosecurity and those non-specific symptoms and associated pathologies common to many diseases. Thereafter the author focuses on specific disease conditions in the sections entitled bacterial diseases, parasitic diseases, viral diseases and finally, other syndromes.

For each disease/syndrome, Dr Sheppard gives a brief introduction to the condition and its key characteristics before outlining symptomology, the range of on-site tests available to the fish health manager and the sampling options for laboratory diagnostics. Colour photographs of gross symptoms and histopathology accompany the text where it is necessary to highlight key points and to aid identification. Each section is rounded off with an overview of the prevention and control options available to the farmer.

In keeping with the book's purpose as an easily accessible guide, a short glossary of diagnostic tests is appended to help demistify the jargon and technical phraseology for the non-specialist reader.



Mark Sheppard has clearly succeeded in providing a well-written, concise and richly illustrated photographic guide to disease in Seriola. Not only is this book a must for those interested in Seriola, it surely has a wider appeal, given that many of the conditions described are not restricted to this genus alone.

The book can be ordered via the Sakana Veterinary Services web page. See www.oberon.ark.com/~svs. Alternatively, contact Dr Sheppard, email svs@oberon.ark.com

Mark Sheppard
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4 Prince Regent Drive, Half Moon Bay, Auckland 1706, New Zealand
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