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Host factors important in sea lice infections

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An overview is presented on the interactive parameters that influence fish susceptibility to infection. In particular, the importance of genetically determined resistance, stress, immunocompetency, and nutrition are discussed in relation to their influence on susceptibility of salmonids to infection with sea lice. It is suggested that these factors should be taken into account, together with other factors that determine infection intensity, such as the source and number of infective stages of sea lice, when devising programs for the management of wild and aquacultured salmonids.

Key words: sea lice, *Lepeophtheirus salmonis*, host susceptibility, genetics, stress, immunity, nutrition, salmonid.

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Introduction

Sea lice, in particular the salmon louse, *Lepeophtheirus salmonis* (Kroyer, 1837) have plagued the salmon aquaculture industry for more than 25 years. These crustaceans cause serious damage to the surface of the fish host and, when present in sufficient numbers, can cause the death of the fish. While a number of treatment and control measures are used on fish farms to reduce infection with this parasite, similar measures cannot be applied to wild fish stocks that also harbour the infection. Increasing concern has been felt for wild salmonids that typically carry lighter infections than are seen on untreated aquacultured fish but which may be exposed to infection when they return to coastal waters. Tully *et al.* (1993) reported heavy infections with *L. salmonis* on migrating sea trout in 1990 and 1991 in western Ireland. They reported extensive pathological symptoms and observed mortality of these fish which was attributed to sea lice infections. Jacobsen *et al.* (1992) also reported a high incidence of chalimus larvae compared with low numbers of adults on wild Atlantic salmon returning to fjords in Norway, which indicated that infection pressure was highest in fjords.

Intensity of infection and subsequent disease symptoms are broadly controlled by two forces. The first is the source and number of infectious stages to which the fish are exposed. Many factors determine the number of copepodid larvae in the vicinity of a fish, including number of gravid adults on previously infected fish, number of eggs produced per female, and environmental parameters such as temperature and water currents. The source of these infective stages has been the topic of considerable debate. The second parameter is the susceptibility of the fish to infection, which will determine the relative numbers of parasites successfully establishing on the fish and the harm caused by these parasites. This latter aspect is the subject of this paper.

Host susceptibility is determined by numerous interactive factors. Different species of salmonid host differ in their susceptibility to infection by the salmon louse. Not only do different species of host harbour different numbers of parasites but these hosts also vary in the degree of damage that ensues as a result of the infection. The various parameters that affect, from the point of view of the fish, host susceptibility, and from the point of view of the parasite, host preference, include host stress level, nutrition and immunocompetency, all of which have a genetically determined component and are highly interactive. Each of these factors will be discussed, specifically for infection with salmon lice where the literature is available, or for other infections in salmonids and teleosts in general.

Genetically-determined host susceptibility

L. salmonis has been recorded on several salmonid species in the genera *Salmo, Oncorhynchus*, and *Salvelinus*. Johnson and Albright (1992a) showed that Atlantic

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salmon, *Salmo salar*, were more susceptible to the salmon louse than were chinook, *Oncorhynchus tshawytscha* and coho, *Oncorhynchus kisutch*. Coho salmon were shown to produce a cell-based reaction, which killed many attached chalimus larvae, making these fish relatively more resistant to infection than the other two host species examined. Jones *et al.* (1990) and Jonsdottier *et al.* (1992) also found that Atlantic salmon showed hyperplastic and inflammatory reactions to infection with *L. salmonis*. Nagasawa and Takami (1993) found that pink salmon, *Oncorhynchus gorbuscha*, harboured heavier infections (96% of all *L. salmonis* recovered) than masu salmon, *Oncorhynchus masou* (4%).

Differences in infection intensity have also been recorded for hosts of a related sea louse, *Caligus elongatus* Nordmann, 1832, which infects arctic charr, *Salvelinus alpinus*, more heavily than Atlantic salmon when these two host species are held in the same sea-pen. This difference in infection intensity may be due in part to increased susceptibility of charr as a result of osmoregulatory stress in salt water. Charr showed increased mucous production and some loss of intercellular contact between epithelial cells. These changes may have created a more ''preferable'' environment on the charr (Mustafa, 1997). Different host species can also have an effect on the development of the copepod. Johnson (1993) found *L. salmonis* to develop faster and produce approximately twice as many eggs on Atlantic salmon than on chinook salmon, and suggested that this difference may be due to differences in nutritional status of the host or to non-specific defence mechanisms. Hosts harbouring quickly developing parasites will contribute, over the long term, more generations of infective stages.

Genetically-selected strains of Atlantic salmon also show intraspecific differences with respect to infection with copepods and other infectious organisms. MacKinnon *et al.* (1995) have shown that differences in infection intensity with the sea louse *C. elongatus* on different full sibling families of Atlantic salmon have an heritability index of 0.2 indicating that susceptibility to infection has a moderate genetic basis. Future breeding programs may be able to select for relatively more resistant families of Atlantic salmon.

Several studies indicate genetic variation in disease resistance in Atlantic salmon (Roed *et al.*, 1993). Fevolden *et al.* (1993) showed that in Atlantic salmon selected for high- or low-stress responses, mortality increased in the high-stress group when these were challenged with bacterial pathogens. The high-stress group of fish also showed lowered immunocompetency compared with the low-stress response fish. Although this study involved bacterial pathogens, genetic selection on the basis of high- or low-stress response is an important consideration in salmon susceptibility to sea lice.

Different populations of the same host species have differing effects on the development of *L. salmonis*. Tully and Whelan (1993) reported that wild Atlantic salmon harbour lice that are larger and have twice as many eggs than lice on aquacultured Atlantic salmon. This has important implications for the concept of disease transfer between and among wild and cultured fish. Although individual adult sea lice from wild fish produce more eggs than individuals on aquacultured fish, their contribution to a reservoir of infective stages at critical locations such as estuaries is unlikely to be correspondingly high. Since there are more aquacultured salmon located in coastal cages year-round, these probably contribute a greater number of lice to the estuary than do wild salmon. Tully and Whelan (1993) have estimated that 95% of nauplii originate from farmed vs. wild salmon, although this estimation is based on assumptions as well as hard data.

Influence of host stress on infection

It is well known that chronically stressed fish are more susceptible to infection with pathogens. Stress is any stimulus that impairs the performance of fish, the reaction being the stress response (Mazeaud and Mazeaud, 1981; Pickering, 1981). Numerous investigations indicate the correlation between elevated levels of plasma cortisol due to stress or due to implanted cortisol, and altered immune system parameters, as reviewed by Ellis (1981).

With respect to sea lice, Johnson and Albright (1992b) showed that coho salmon implanted with cortisol, by intraperitoneal injection in an oil-based pellet, have a decreased inflammatory response and less epithelial hyperplasia when infected with *L. salmonis*, and increased susceptibility to the parasite. Mustafa (1997) showed that Atlantic salmon given cortisol implants acquired heavier infections with *C. elongatus*. While cortisol implantation experimentally stimulates some of the secondary effects of stress, it has often been observed on fish farms, and even in wild fish, that fish compromised by high water temperatures, other diseases, poor nutrition etc. acquire heavier sea lice infections.

The causes of stress, and therefore of increased susceptibility to sea lice infections, are numerous and compounding in their effects. Natural events in the life of a salmon, such as smoltification, migration and sexual maturation raise the stress level of fish. Harsh climatic conditions (high or low temperatures, strong winds) and infection with bacteria, fungi, viruses and parasites can affect both wild and cultured salmon. Cultured salmon are also stressed by handling, crowding, even by feeding. Salonius and Iwama (1993) reported that early rearing conditions in wild vs. hatchery reared salmon have an effect on stress and immune function. Plasma cortisol

levels in wild coho salmon were higher, and the number of antibody-producing cells lower, after handling stress than in coho that had been reared entirely in a hatchery. Aquacultured salmon are more likely to tolerate anthropogenic stress better than wild salmon since many generations have been bred in captivity and those that tolerate stress well have been either intentionally or inadvertently selected.

Pollution also seriously compromises salmon through toxic effects and by increasing stress. Weeks *et al.* (1992) listed numerous instances where exposure to low-level toxicants resulted in depressed immune function in fish, including reduced macrophage phagocytosis, chemotaxis and chemiluminescence, and a reduction in antibody-producing cells. Khan and Thulin (1991) indicated that pollutants may both reduce host immunocompetency and indirectly and directly affect the survival of the parasite.

Parasitism itself causes stress. Under aquaculture conditions, the stress level in salmon is related to the interaction of rearing conditions and infection. Urawa (1995) showed that crowding and low water supply cause growth reduction and stress-induced mortality when salmon infected with *Ichthyobodo necator* are subsequently given a seawater challenge. Crowded but uninfected fish did not show high mortality levels when similarly tested.

Susceptibility of salmonids to sea lice infections may vary throughout the year, even in individual fish. Maule *et al.* (1996) showed that freshwater migration increases cortisol levels in chinook salmon. Maule *et al.* (1993) indicated that the degree of physiological response to given levels of cortisol showed seasonal differences in juvenile coho salmon. In general immune function declined during smoltification. During this study, *in vitro* incubation of anterior kidney leucocytes in cortisol altered glucocorticoid receptors during culturing between March and September but not during culturing between November and February. Muono and Soivio (1992) also showed that lysozyme activity, total blood leucocyte, and lymphocyte number decline during smoltification. Thus wild or aquacultured salmon may be more stressed and more susceptible to infections when they are smoltifying.

Effect of nutritional status on infection

Providing optimal nutrient requirements for fish is an important factor in regulating resistance to disease (Blazer, 1992; Lall and Olivier, 1993). A nutritional deficiency that impairs, for example, protein synthesis, could affect the functioning of both the humoral and cellular, specific and non-specific components of the immune system. Likewise, excess nutrients can cause toxicity. The nutritional status of a fish is dependent on several interactive parameters including food nutrient content and bioavailability of micronutrients, environmental stress, disease, changing physiological needs, and anorexia resulting from drugs or infection.

Little research has been conducted on the effect of the nutritional status of the fish host on sea lice infection intensity and subsequent pathological effects. Mustafa and MacKinnon (1993) showed that iodine supplementation, either in the diet or in the holding tank water, of smoltifying Atlantic salmon, reduced plasma cortisol levels and in some cases reduced infection with *C. elongatus*. During times of insufficient thyroid hormones, fish produce cortisol as a means of controlling metabolic conversions that would otherwise have been handled by thyroid hormones. Providing extra iodine allows for increased production of T3 and T4, which then reduces the need for high cortisol levels.

Blazer (1992) and Lall and Olivier (1993) have reviewed the importance of macro- and micronutrients in disease resistance in fish. They indicated that diet can have a significant effect on immunocompetency and disease resistance but more research needs to be conducted on specific host/pathogen systems.

Regulation of sea lice infection by the immune system

As evident from the foregoing sections, the degree to which the immune response affects infection with sea lice varies with the species and general health of the fish, being particularly influenced by various stressors. Johnson and Albright (1992a) found that in coho salmon, a non-specific response to chalimus stages of *L. salmonis* effectively rid the host of many attached larvae. A less effective response is seen in other species of salmon. Grayson *et al.* (1991) demonstrated that naturally infected Atlantic salmon have specific antibodies to *L. salmonis*. However, this antibody response appears not to be protective since Atlantic salmon continue to acquire new infections. The extent to which bioengineered antigens are effective in stimulating a protective response has yet to be fully established. Antibodies developed against these antigens are specific for the intestinal lining of the salmon louse (Roper *et al.*, 1995), and have an effect on numbers of gravid females and on fecundity (Grayson *et al.*, 1995) but do not sufficiently prevent infection with all stages.

The fish immune response (for review see Ellis, 1982; Lall and Olivier, 1993) to parasitic infection can be influenced by a variety of factors including stress, pollutants, hormone levels, season, diet, concomitant infections and temperature (Ellis, 1981; Fletcher, 1986; Maule *et al.*, 1987, 1993, 1996; Weeks *et al.*, 1992; Pulsford *et al.*, 1995). Ellis (1982) considered temperature to be the most significant factor affecting immune

function. Low temperatures prevent or delay an immune response. The warmer the temperature, within the normal temperature range for any given species, generally the better the immune response. The effect of temperature is highly interactive with seasonal changes in pathogen infection dynamics and in fish hormone levels. For example, although warm water temperature enhances the immune response, it also enhances the development rate of sea lice. Warming temperatures also coincide with certain maturation events such as smoltification, which raise cortisol levels in salmon that in turn can be immunosuppressive.

The immune response may also be stimulated by immunostimulants such as vitamin C or A- beta- 1,3 glucan (Vita Stim-Taito), which showed promise as immunostimulants when injected into juvenile coho salmon with formalin-killed *Aeromonas salmonicida* (Nikl *et al.*, 1991). The use of immunostimulants has not been investigated for *L. salmonis* infections.

Conclusions

The susceptibility of salmonids to infection with sea lice is different for different species of fish and varies, even in one individual fish, with different stages of its life cycle and its overall state of health. Given the same exposure to infection, certain hosts may acquire heavier infections than others or may show relatively more disease even when harbouring similar intensities of infection. Thus the consequences of infection with the salmon louse to any fish are not only determined by the relative availability of infective stages but also by the susceptibility of the fish to infection.

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