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Factors affecting the susceptibility of sea lice infection in Atlantic salmon

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ABSTRACT. Parasitic copepods of the family Caligidae, otherwise known as sea lice (*Lepeophtheirus salmonis* and *Caligus elongatus*), are responsible for serious disease problems in salmon aquaculture. This mini-review examines the host factors that are affecting the susceptibility of sea lice infection in salmon; in particular, host species, environment, host stress levels, and host immunocompetency. In order to develop more effective methods to control sea lice infection, these factors should be taken into account, along with other factors such as sea lice life cycle and fecundity as well as salmon husbandry methods.

Key words: Atlantic salmon, *Caligus elongatus*, infection susceptibility, *Lepeophtheirus salmonis*, *Salmo salar*, sea lice.

Sea lice are parasitic crustaceans representing the subclass Copepoda. There are two major species of sea lice affecting Atlantic salmon: *Lepeophtheirus salmonis* Krøyer, 1837 [1] and *Caligus elongatus* von Nordmann, 1832 [2]. The former species, known also as salmon louse, is much bigger, more host specific to salmon, and definitely much more pathogenic. Therefore the present paper will focus mainly on *L. salmonis*. Sea lice are responsible for serious disease problems in salmon aquaculture. These ectoparasites feed on salmon mucus, skin, and blood [3, 4], and cause osmotic damages and even death in extreme cases [5, 6]. High intensities of sea lice are common on farmed salmon, probably as a result of stress and increased availability of the infective stages under crowded conditions of a sea pen. Financial losses due to sea lice infection on salmon farms are therefore, considerable due to reduced fish growth, reduced feed conversion rate, market downgrading, secondary infection, mortality, and treatment [7]. A number of treatment and control methods are currently being used on fish farms to reduce infections with these parasites, but these methods have inherent limitations of availability, costs, fish loss, withdrawal periods, and resistance [7]. Development of more effective methods to control sea lice infections is therefore highly desirable and to develop these effective methods a better understanding of host factors should be examined. Intensity of infection and susceptibility of fish host to diseases are controlled by many interactive factors. These factors include host species, host stress levels, immunocompetency, and environment. The aim of this mini-review is to overview these factors.

Host species differences in susceptibility to sea lice infection have been reported several times [8, 9]. Johnson and Albright [8] observed that Atlantic salmon, *Salmo salar*, were more susceptible to sea lice than were coho- and Chinook salmon. Atlantic salmon often develops lesions and decreased immune response [5, 10, 11]. Atlantic salmon has shown to have hyperplastic and inflammatory reactions [12. 13], changes in their physiology such as increased plasma cortisol and glucose levels [10] and in skin morphology such as, increased apoptosis, necrosis, and leukocyte infiltration [14] in response to sea lice infection. Atlantic salmon has

also shown to have twice as many sea lice eggs than Chinook salmon, may be due to differences in nutritional status of the host or to non-specific defense mechanisms [15, 16]. Ross et al. [17] found increased mucus protease levels on sea lice-infected salmon but Firth et al. [18] observed that the increased protease activities in Atlantic salmon following sea lice infection appear to have originated from *L. salmonis*, not from the host fish. Genetically selected strains of Atlantic salmon also have shown differences in susceptibility with respect to sea lice infection. Mustafa and MacKinnon [19] have shown that differences in infection intensity with the other species of sea louse, *Caligus elongatus*, on different full sibling families of Atlantic salmon have a heritability index of 0.2 indicating that susceptibility to infection has also a genetic basis.

It is well known that stress is one of the key factors that affects the health of fish host and increases the susceptibility to disease [20–22]. Stress results in endocrinological changes in fish that activate the adrenergic system and the hypothalamus-pituitary-interrenal (HPI) axis and increase the production of catecholamines and corticosteroids [23, 24]. This neuroendocrine response causes a number of secondary effects in physiological processes that facilitates the fish's response to the stressors [25]. These in turn, suppress the immune response, which ultimately increases the susceptibility fish to infection [23, 26]. The suggestion that the stress hormone cortisol may greatly potentiate the disease process has been supported by several studies. Salmon are particularly stress sensitive whose stress levels can be compounded by aquaculture practices [22, 28]. With respect to sea lice, coho salmon implanted with cortisol, have a decreased inflammatory response and less epithelial hyperplasia when infected with sea lice, *L. salmonis* [8]. Mustafa and MacKinnon [27] showed that Atlantic salmon implanted with intraperitoneal cortisol acquired heavier infections with *C. elongatus*. While cortisol implantation experimentally stimulates the effects of stress, it has been observed on fish that affected by environmental factors acquire heavier infections with sea lice [22]. Smoltification, migration, and sexual maturation raise the stress levels in salmon. Husbandry practices in the salmon farms such as handling, crowding, and even feeding also affect the fish and raise their stress levels.

The effects of stress on salmon physiology are complex and mechanisms involved are not fully understood. It has been observed that cortisol affects thyroid hormone levels in salmon. Redding et al. [29], and Vijayan and Leatherland [30] have shown the effects of elevated plasma cortisol levels on the reduction of plasma triiodothyronine in coho salmon. Scott-Thomas et al. [31] have also shown the effects of stress due to high stocking density on the thyroid-stimulated metabolic changes in Arctic char. Mustafa and MacKinnon [27] suggested that Atlantic salmon are deficient in iodine and therefore produce less thyroid hormones and because of low thyroid hormones salmon secrets more cortisol to cope with the extra metabolic needs during smoltification which would otherwise be controlled by sufficient thyroid hormones.

It is known that a nutritional deficiency that impairs cellular response may affect the functions of both humoral and cellular, specific and non-specific, components of the fish immune system and likewise, excess nutrients may cause toxicity. Lall and Olivier [32] also indicated that nutrition of fish is important in regulating cellular functions and maintaining disease resistance. Mustafa and MacKinnon [27] showed that iodine supplementation to smoltifying salmon reduced their plasma cortisol levels and reduced infection with sea lice, *C. elongatus*.

During smoltification salmon go through physiological changes and become more vulnerable when they migrate from freshwater to seawater. At this period, the change in external salinity subjects them to physiological (osmotic) stress [33]. The consequence of this stress is increased plasma chloride concentration, muscle dehydration, and decreased plasma hematocrit levels [33]. Migrating salmon are therefore susceptible to parasitic infections including sea lice.

Infections with other parasites also cause stress. In nature, most fish are infected by parasites, but cases of parasite-inflicted mortalities are rare. Under farming conditions, however, when the fish are stressed, the number of these parasites increases causing greater levels of stress due to their cumulative effects. Because salmon reared under aquaculture conditions are almost always under stress, any additional stress due to infection with parasites is more harmful to these farm-reared salmon than their wild conspecifics. Farmed salmon can easily become infected with parasites transmitted to them, directly or indirectly, from wild salmon. In direct transmission, larval stages of parasites such as sea lice gain easy access to the sea cages and can infect their hosts. In indirect transmission, small fish infected with sea lice or other parasites may enter

sea cages and transfer the parasites on to the cultured fish. When these acquired parasites reproduce, the number of parasites increases over time and results in increased levels of stress and thereby increases the salmon susceptibility to sea lice infection.

Toxicants have also major effects on salmon in increasing stress response. Toxicants result in decreased immune function in fish, including reduced phagocytic activities of macrophages, chemotaxis, and antibody producing cells [34]. Toxicants and pollutants not only reduce the immunocompetency in fish hosts but also affect the survival of the parasites [35].

Intensity of infection with any given parasite can vary greatly between fish species and their immunocompetency. Johnson and Albright [8] found that coho salmon are less susceptible to sea lice than Chinook salmon, sockeye salmon, Atlantic salmon, or rainbow trout. The mechanisms for these differences are not yet understood, but it is the fact that the immunocompetency of fish is influenced by several factors including stress, pollutants, hormones, season, and nutrition. Fast et al. [36] indicated that the first line of defense in salmon is the skin, which secretes the layers of mucus important to host defense mechanisms as well as providing mechanical protection. Enzymes and other protective proteins are located within the mucus such as, alkaline phosphatase, a lysosomal enzyme. Levels of alkaline phosphatase in mucus have been found to increase under parasitic conditions in Atlantic salmon [17] and under stressful living conditions in carp [37]. Proteases are also potential mucus factors contributing to innate immunity. Grayson et al. [38] demonstrated that naturally infected Atlantic salmon have specific antibodies to sea lice, *L. salmonis*. However, this antibody response appears not to be protective since Atlantic salmon continue to acquire new infections. The extent to which antigens are effective in stimulating a protective response is yet to be fully established.

Other factors include shallow and enclosed water bodies where rate of water exchange is relatively low and water temperature is relatively high. Heuch et al. [39] found higher sea lice infection levels on salmon in Scotland due to shallower and more enclosed water bodies used for farming, small and shallower pens, and differences in seawater temperature. Mustafa et al. [40] from their laboratory experiments also showed that salmon kept in higher temperature have more sea lice. They also showed that salmon have lower sea lice infection kept in waters with higher flow rates and more turbulence.

The factors affecting the susceptibility of sea lice infections in Atlantic salmon issue is fascinating and yet to be discovered. But we are getting closer to reduce the sea lice infection on salmon by combining good husbandry methods with alternative treatment methods.

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