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FACTORS AFFECTING THE HEALTH OF FARMED AND WILD FISH POPULATIONS: A PERSPECTIVE FROM BRITISH COLUMBIA

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ABSTRACT

With the increase of mariculture, particularly netpen rearing of salmonids, has come the need to address whether these operations have a significant impact on coastal marine environments. Models have been made to assess this impact, but these models have not considered the potential risk of increased disease in wild fishes that mariculture may impose. At this time, much of the information needed to create models to assess the impact of farmed fish diseases on wild fish populations is not available. The transmission and development of disease is a complex process that involves numerous factors that affect the host, the pathogen, and their environment. The following paper describes some of these factors and compares their occurrence in wild and farmed salmonids.

RÉSUMÉ

Avec le développement de la mariculture, en particulier l'élevage de salmonidés en enclos, est né le besoin d'établir si ces installations ont un impact important sur l'environnement côtier. On a élaboré des modèles pour évaluer cet impact, mais ils ne tiennent pas compte du risque potentiel d'une incidence accrue de maladies chez le poisson sauvage imposée par la mariculture. On ne possède pas encore toute l'information nécessaire pour créer des modèles permettant d'évaluer l'impact des maladies du poisson d'élevage sur les populations de poisson sauvage. La transmission et l'évolution d'une maladie sont un processus complexe qui engage de nombreux facteurs agissant sur le pathogène, l'hôte et l'environnement. On décrit certains de ces facteurs et on compare leur fréquence chez les salmonidés sauvages et d'élevage. On décrit aussi certaines des stratégies de gestion présentement mises en oeuvre par l'industrie salmonicole de la Colombie-Britannique et des organismes de réglementation en vue d'atténuer les effets potentiels de ces facteurs.

INTRODUCTION

In recent years, the rearing of salmonid fishes in seawater netpens has become an important mariculture industry in several countries (e.g., Canada, Chile, Scotland, and Norway). With the growth of this industry, there is a need to assess the possible impact of fish farming on the coastal environment, including transmission of diseases. As with most forms of intensive agriculture, infectious diseases can be a problem in these netpen farms. Therefore, in addition to concerns that these diseases pose to the industry, there is a concern about the potential impact that these diseases may have on wild fishes. Models have been developed to assess the impact of aquaculture on coastal marine environments (Ibrekk et al. 1991; Silvert 1992), but these models have not considered the potential transmission of diseases. Before farmed fish can be incriminated as the cause of disease in wild fish, the previous geographic distribution of the pathogen and prevalence of the disease in the wild population must be known. Once the baseline prevalence is determined, then one can begin to look at aquaculture and other factors as direct or indirect causes of disease.

Although it is difficult to determine if diseases of farmed fishes have been transferred to wild fishes, it is clear that pathogens from wild fish (both salmonids and non-salmonids) have had impacts on pen-reared fish (MÖller and Anders 1986; Kent and Fournie 1992). For example, wild salmonids are reservoirs of infection for the salmon louse, *Lepeophtheirus salmonis*, which can be a problem in pen-reared Atlantic salmon (*Salmo salar*) (Pike 1989). Examples of non-salmonid reservoirs for netpen diseases in the marine environment include sea lice (*Caligus* spp.) from a variety of fishes (Margolis et al. 1975), and spiny dogfish *Squalus acanthus* for the eye tapeworm *Gilquinia squali* (Kent et al. 1991). Wild salmon also act as reservoirs for bacterial diseases. Bacterial kidney disease (BKD), caused by *Renibacterium salmoninarum*, is a serious disease in pen-reared Pacific salmon (*Oncorhynchus* spp.) in British Columbia. In the mid-1980's wild brood stock were used as the source of eggs for the industry. *Renibacterium salmoninarum* is vertically transmitted (Evelyn 1993) and this may have been a route of transfer from wild fish to cultured fish. *Loma salmonae* (Microspoidia) may be another example of a pathogen transferred from wild to farmed fish. Reports of *Loma salmonae* causing disease in farmed Pacific salmon a year after their salt water entry suggests that this parasite was acquired at sea, most likely from wild salmonids because the parasite is prevalent in ocean-caught Pacific salmon species (Kent et al. 1998), and studies indicate that non-salmonids are not a reservoir for the infection.

There are numerous factors which uniquely affect fish in captivity which may make them more prone to diseases. The fact that farmed fish become diseased when exposed to a pathogen may or may not hold true for wild fish populations. For a pathogen to affect a fish population, enough of the population has to be susceptible to the pathogen and be exposed to it for a sufficient amount of time for infection to occur (Scott and Smith 1994). For secondary infections to occur susceptible hosts must come into close enough and long enough contact with infected fish for transmission to occur (Scott and Smith 1994). The numerous disease agents, their modes of transmission, and the number of different species of fish affected by them make it difficult to assess the impact of fish farms on the spread of disease. In order to begin to address some of these questions it is important to understand what factors lead to disease in populations of animals. Moreover, it is important to note that although the health of a population is determined by the health of the individuals in that population, the presence of a few diseased animals does not necessarily lead to a diseased population or an outbreak of disease. The objective of this paper is to discuss some of the factors which can increase or decrease the chance of disease in salmon,

contrasting the occurrence of these factors in wild and farmed salmon.

FACTORS AFFECTING THE OCCURRENCE OF DISEASE

To assess the potential increased disease risk that a farm disease may have on wild populations of fish, one must consider the host, the pathogen and their environment. In other words, the occurrence and severity of disease is dependent on the status of the host and the pathogen which are affected by a number of factors including their environment. This is a very complex set of interactions, even in the apparently simple context of comparing wild and cultured salmonids. If one considers, as an example, the differences between cultured and wild fish with regard to tolerance to stress, it becomes obvious that the question of whether cultured and wild fish differ in disease resistance is a tremendously complex question. Not only does one have to describe the difference in the two groups to a standardised disease challenge, but if an attempt is going to be made to extrapolate those results to nature, then one really needs to know how cultured and wild fish differ with respect to all the factors that can modulate their immune systems. In addition, most biological systems change over time so this adds one more dimension to the interaction between the host and the pathogen. We explore those components of host and pathogen separately in this paper, in an attempt to present clearly the information relevant to the comparison of cultured and wild fish; with a particular focus on salmonids in British Columbia.

The Host

The host's innate resistance, and physiological and immunological status play an important role when exposed to a pathogen in determining whether an infection will be established and disease will ensue. The host's susceptibility is influenced by a number of factors including: species susceptibility, strain susceptibility, age of the host, nutritional status, stress, trauma (e.g., open lesions or abrasions that may facilitate entry of pathogens), sexual maturation, smoltification, pre-existing infections and other co-factors, previous exposure and acquired immunity (e.g., through vaccination) to the specific pathogen, and numerous environmental factors or stressors.

Species and Strains. It is well recognised that there is a great variability in susceptibility to diseases between species within the family Salmonidae. For example, sockeye salmon (*Oncorhynchus nerka*) are generally more susceptible to infectious hematopoietic necrosis virus (IHNV) type I than chinook salmon (*Oncorhynchus tshawytscha*) (Traxler et al. 1993). This difference in susceptibility also occurs at the strain level. One of the best examples of differences in strain susceptibility is with *Ceratomyxa shasta*. Chinook salmon from enzootic waters are much more resistant than strains from watersheds where the parasite is absent (Zinn et al. 1977). Ching and Parker (1989) have shown stock differences in resistance to this parasite in both chinook and coho salmon. As well, different stocks of chinook salmon show different susceptibility to *L. salmonae* (R. Shaw, per. comm.). McGeer et al. (1991) found differences among five stocks of coho salmon *(O. kisutch)* when challenged with *Renibacterium salmoninarum*, the causative agent for BKD. Gjedrem and Aulstad (1974), and Refstie (1982) have shown that resistance to *Vibrio anguillarum* can vary in stocks of rainbow trout in sea water, and in Atlantic salmon in fresh water, respectively. Such differences evidently have some genetic basis, and it is reasonable to think that selection can influence disease resistance. Suzumoto et al. (1977) linked differences in BKD resistance among three groups of coho salmon to different transferrin genotypes.

Naïve Hosts. Innate and acquired resistance to a pathogen would not be present when a new pathogen emerges. "New pathogens" can emerge on their own either through genetic mutations or they can be introduced to a new region. Migrating species like salmon can encounter many different pathogens through their life cycle; changing weather and temperature patterns can change the home range of different fishes, and the movement of fish into new regions allows for introduction of their pathogens. The introduction of a species of fish to a new area for aquaculture purposes could also have the potential for introducing exotic pathogens. It should be noted that the transplant of exotic pathogens via the introduction of Atlantic salmon has never been reported on the west coast of Canada despite the introduction of several stocks (Stephen and Iwama 1997). Conversely, this introduced species is likely to be exposed to "new" pathogens that are indigenous in the area and it may be more susceptible to these pathogens than native species.

Age and Endocrine Changes. It is not clear how or why changes in the immune system of fish occurs towards the end of its life cycle. The increase in immunological self-recognition (i.e., autoimmune diseases) and a decline in immune response against antigens that occurs in older mammals may also happen in fishes. However, as indicated above, there is very little knowledge about the effects of aging on fish health. There are reports of tumors in older fish (Mulcahy 1970), and there is evidence that fish can generate autoimmune responses (Laird et al. 1978). On the other hand, many fish viral diseases, such as IHNV and infectious pancreatic necrosis virus (IPNV), cause high mortality in young fish. Older fish are generally more refractory, even if they have not been previously exposed. It is beyond the scope of this paper to discuss this topic of the effects of age on the immune system in fish. The reader is referred to Iwama and Balfry (1998) for a more complete discussion.

There have been several studies on the effects of elevated sex hormones, associated with the maturation and spawning processes, on various components of the immune system in salmon species. Physiological levels of testosterone have been shown to kill white blood cells and reduce the plaque-forming ability of lymphocytes (Slater and Schreck 1993;1997). Given the immunosuppressive effects of cortisol and testosterone in salmonids, it is perhaps not surprising that migrating salmon on their way to their spawning grounds are often seen with fungus and parasites covering their body. It is noteworthy that this is not true for all steroids, as their 1993 study showed this effect at high concentrations of testosterone and cortisol, but not with 17beta-estradiol or aldosterone. It is also noteworthy that such immunosuppresion was attenuated in the winter, underscoring the importance of recognizing the environmental influences on potential interactions between the endocrine and immune systems.

The General Stress Response. Besides smoltification and sexual maturation, immunosuppression from elevated cortisol levels occurs when fish are stressed. One definition of stress is "a state produced by an environmental or other factor which extends the adaptive response of an animal beyond the normal range or which disturbs the normal functioning to such an extent that in either case, the chance of survival are reduced" (Brett 1958 in: Stephen and Iwama, 1997). Stress has been shown to affect the immune response in fish. In response to a stressor a fish will undergo a series of biochemical and physiological changes in an attempt to compensate for the challenge imposed upon it and, thereby cope with the stress. The stress response in fish has been broadly categorized into the primary, secondary and tertiary responses (Mazeaud et al. 1977; Wedemeyer

et al. 1990) and are summarized in Diagram 1 and Figure 1. In addition to these whole animal changes that have been widely studied, recent reports indicate cellular adjustments in response to different stressors which is characterized by the expression of stress proteins (Iwama et al. 1998).

Primary response. This initial response represents the preception of an altered state and initiates a neuroendocrine/endocrine response that includes the rapid release of stress hormones, catecholamines, and cortisol into the circulation. Catecholamines (CATS) are released from the chromaffin tissue situated in the head kidney of teleosts, and also from the endings of adrenergic nerves. Cortisol is released from the interrenal tissue, located in the head kidney, in response to several pituitary hormones, but most potently to adrenocorticotrophic hormone (ACTH). The physiological action of both hormones are dependent on appropriate receptors on target tissues. CATS are associated with a very rapid acute response, while the cortisol response is delayed but more prolonged (Fig. 1).

Seconday response. This response comprises the various biochemical and physiological effects associated with stress, and mediated to a large extent by the above stress hormones. The stress hormones activate a number of metabolic pathways that result in alterations in blood chemistry and haematology (see Barton and Iwama, 1991). For example, the increase in blood glucose is a common component of the generalized stres response (Fig. 2), and is used as an indicator of stressed states in fish. The increase in blood glucose concentration is caused by gluconeogenic and glycogenolytic effects of both CATS and cortisol.

Tertiary response. This response represents whole animal and population level changes associated with stress. If the fish is unable to acclimate or adapt to the stress, whole animal changes may occur as a result of energy-repartitioning by diverting energy substrates to cope with the enhanced energy demand associated with stress and away from anabolic activity such as growth and reproduction. Thus, chronic exposure to a stressor, again depending on the intensity and duration of that stressor, can lead to decreases in growth, disease resistance, reproductive success, smolting, swimming performance, etc. At a population level, decreased recruitment and productivity may alter community species abundance and diversity.

In the short term, the generalized stress response (see Barton and Iwama 1991) stimulates the immune system, but chronic stress usually compromises the immune system. Diseases such as vibriosis, furunculosis, and BKD are particularly known to be associated with stress in fish. While the immunosuppressive effects of stress is emphasized in much of the data, it is clear that the stress response in fish is adaptive and aids the animal in coping with stressors that are presented to it. Comparing wild and cultured fish, therefore, should include how the rearing history affects the stress response in fish (see below for such a discussion). There are natural changes in the temporal patterns of cortisol and the immune response of fish, but the sensitivity of the immune system to stress is not constant throughout the life span of the fish. For example, in the latter stages of life in a salmon, the immune system is particularly sensitive to the effects of the increase in cortisol caused by stress. Clearly, these temporal changes must be considered in assessing the immune competence in fishes. Such considerations should be part of management decisions that may affect or concern the health of fish in the wild as well as under intensive aquaculture conditions. It is clearly beyond the scope of this review to discuss in any detail the extensive list of known stressors to fish. We summarize a few here that are pertinent to the topic of this paper.

Environmental Stressors mainly include adverse chemical conditions of the water. Although pollutants are common environmental stressors, extreme conditions or changes in

water quality parameters such as dissolved oxygen, ammonia, hardness, pH, gas content and partial pressures, temperature etc. can unduly stress fish (Wood 1979; Meyer et al. 1983). Water of high quality is a necessary prerequisite for the intensive culture of salmonids. Such water quality criteria can be found in many of the common publications on salmonid husbandry. Any significant deviation from such water quality can be considered as a potential stressor. The forced confinement of farmed fish may in some circumstances make them more prone to be affected by environmental stressors. For example, seldom are toxic algae blooms a concern for migrating wild salmon stocks that can easily escape to better water quality. In contrast, captive fish have a limited area to escape when exposed to toxic algae or other poor water quality conditions. Furthermore, high fish densities found in aquaculture facilities may increase the likelihood of poor water quality. These two characteristic of farmed fish make them at higher risk of exposure to water quality-related stressors and may increase their susceptibility to disease when exposed to pathogens compared to wild fish. It should be noted that with increasing loss of habitat and industrialisation around rivers it may be possible that wild fish are now, too being exposed to some environmental stressors.

The physiological state of fish is closely tied to the temperature of the water. Extreme fluctuations in water temperatures can increase their susceptibility to pathogens. Farmed fish may be influenced by this parameter more than wild fish, which are not restricted to surface water and therefore may not experience extreme water temperature fluctuations. At higher temperatures within a physiologically tolerated range, the onset of the host's immune response is usually faster and its magnitude is usually greater (Ellis 1982). This is particularly the case with the primary immune response (Avtalion at al. 1976). However, these higher temperatures may also promote the proliferation of the pathogen.

Physical stressors include those that involve handling, crowding, confinement, transport, or other forms of physical disturbance (Strange et al. 1978). Many such manipulations are common to the intensive husbandry of salmon. Wild fish, may also be under stress from physical disturbances when they encounter man made obstacles such as fences, weirs, and ladders. Angling also stresses fish in this way. It is possible that farmed fish become somewhat acclimatized to frequent handling. On the other hand wild fish would not be habituated to such stimulus and therefore, any handling could potentially be very stressful on the fish.

In addition to stressing fish, handling and or crowding fish may also increase the likelihood of skin abrasions and other surface lesions by fin nipping or scraping on tank/nets. Such abrasions could provide sites of invasion for pathogens. *Cytophaga* and *Flexibacter* spp. bacteria infect the surface of marine and freshwater fishes, and infections are often initiated at the site where the skin or fins have been damaged (Kent et al. 1988). In fresh water, the opportunistic fungi, such as *Saprolegnia* and related genera, commonly infect open lesions and necrotic tissue following trauma in freshwater fishes (Pickering and Willoughby 1982).

Biological stressors can be manifest in dominance hierarchies which develop between individuals within confines such as experimental tanks or possibly in natural environments. Disease pathogens can also be considered as biological stressors.

There are few studies that have compared culture and wild fish with respect to stress and disease resistance. In one such study, Salonius and Iwama (1993) found that plasma cortisol concentrations in both coho and chinook salmon from the wild were significantly higher after a 30-60 s dip netting stress, whereas fish reared in a hatchery from the same stocks as the wild fish did not show a significant change in plasma cortisol after such handling stress. That study also

examined a group of fish, called "colonized fish" that were hatched in the hatchery but released into the wild as small fry. The colonized fish responded to this stressor in a way similar to the wild fish. When colonized fish and the wild fish were then brought into a hatchery and reared there for 7 months, and stressed again in the same manner, they retained their high stress response compared to the cultured fish. The authors speculated that the attenuated cortisol response of hatchery fish to the stressor was due to acclimation to repeated episodes of such procedures at the hatchery; thus there was likely a down-regulation of the interrenal cells to produce cortisol in the face of ACTH stimulation from the pituitary. Salonius and Iwama (1993) found no difference in resistance to *Vibrio anguillarum* challenge among the three groups of fish. However, the wild coho salmon became more susceptible to the disease after the 7 months of hatchery rearing. Another interesting observation was that wild fish had higher numbers of antibody-producing cells at rest, compared to colonized or cultured fish. Speculations about disease from such differences of one such variable of the immune system, however, should not be made; e.g., disease resistance to vibriosis were similar among the three groups.

Regardless of the type of stressor, if a parameter elicits the stress response in a fish it has the potential to increase the fish's susceptibility to disease. Besides host susceptibility, the pathogen itself plays a very important role in determining whether disease will ensue when a susceptible fish is confronted with an infectious agent.

The Pathogen

Several characteristics of pathogens are of importance for assessing the risk of disease to fish. Virulence of the pathogen, exposure dose required to initiate an infection, contact time required to initiate infection, ability of the pathogen to survive and/or multiply without a host present, and the route of transmission all affect the ability of the pathogen to cause disease. Examination of the specific properties of the pathogens is important in assessing the risk of transfer of disease to wild fish populations.

Virulence. The virulence of pathogens depends on the agent and its environment. Some pathogens cause disease at a low concentration and with a short exposure time. For example, IHN virus has been associated with mortality in Atlantic salmon exposed to low concentrations of the virus for a short period of time (Traxler et al, 1993), whereas other pathogens are much less virulent (regardless of the fish species) and require much higher concentrations and longer exposure periods before disease occurs. For example, in laboratory experiments *Renibacterium salmoninarum* is usually transmitted via intraperitoneal injection or long term cohabitation with infected fish as it is not readily transmitted by experimental water borne exposure. An experiment with *Aeromonas salmonicida* demonstrated that at low concentrations it required an extended period of time to establish disease in a tank of Atlantic salmon (Rose et al. 1989 in: Stephen and Iwama 1997). For farmed fish, exposure to pathogens may be greater and more extended than for wild fish since farmed fish are held in closer confinement and sick fish may remain in the population for a long period of time. Therefore, for pathogens that require an extended period of exposure at a relatively high concentration it is possible that the risk of a disease outbreak is much greater for a farmed fish population than a wild fish population. In order for transmission of a pathogen to occur from a farmed fish population to a wild fish population the wild fish have to remain in the area where there

is viable pathogen for a period of time sufficient for infection to occur.

Survival. The ability of a pathogen to survive and multiply in the environment is a significant factor in assessing the risk of disease. In fact, most reported disease transmissions from wild to farmed fish have been with pathogens that are able to survive for some time outside of a host. In a population of migrating fish where diseased fish quickly leave the population, pathogens that cause acute disease with a high rate of mortality may not persist long enough to cause a severe disease outbreak while the fish are migrating. Conversely, if the fish are confined to an area such as a farm, or trapped in a section of a river (e.g., below a fence or ladder) then propagation of this type of pathogen is much more likely.

Pathogens that can survive and replicate in the environment without a host (e.g., many bacteria) may be more important risks of disease transfer between wild and farmed fish than pathogens that do not survive long without a host (e.g., many viruses). However, determination of the survival of pathogens in water is not a simple task. Pathogen survival is influenced by a number of environmental parameters such as temperature, dissolved oxygen, suspended organic material, the presence of intermediate or reservoir hosts, pollutants, and other water quality parameters.

Many fish pathogens have a relatively narrow temperature regime in which they can survive, multiply, and cause disease. *Vibrio salmonicida,* the causative agent of Hitra is one example of a pathogen that prefers colder temperatures. Other bacterial agents, like *Aeromonas salmonicida* and other *Vibrio* spp., are more prolific at warmer water temperatures.

The amount of organic material may also be important for pathogen survival. For example, in controlled laboratory conditions, *Renibacterium salmoninarum* survived much longer in sea water when a small amount of peptone was added (Paclibare et al. 1993). Biotic factors also influence the survival and transmission of pathogens. Invertebrates that act as vectors or intermediate hosts may be essential for transmission of some pathogens. Conversely, invertebrates may consume pathogens, and thus reduce the possibility of transmission. Paclibare et al. (1993) demonstrated that mussels (*Mytilus edulis*) effectively removed *R. salmoninarum* from sea water, thus their presence on netpens may reduce transmission of the bacterium. Bacterial and viral antagonism influences the survival of pathogens: Austin and Rayment (1985) found that the *R. salmoninarum* could survive much longer in sterile water than unsterile water.

Wild fishes, both salmonids and non salmonids, should also be considered as biotic factors for disease transmission because they can also act as vectors, intermediate hosts or reservoir hosts. As already mentioned, wild salmonids are important reservoir hosts for a number of pathogens that are important to both wild and farmed fish.

In general, then, the environment within a farm setting may be more conducive to pathogen survival and propagation than the more open setting of most wild fish habitat. Wild salmon may be infected with a greater variety of pathogens compared to farmed fish (Kent et al. 1998); but there appears to be fewer disease outbreaks with significant mortality. To reduce the number of disease outbreaks in aquaculture facilities producers and government regulatory agencies have numerous strategies, most of which target factors that increase host resistance to pathogens and reduce the survival of pathogens in the environment.

CONCLUSIONS

Assessing the risk of increased disease in wild fish due to aquaculture activities with any significant precision is a very complicated task and requires much more information than is currently available. There is no structured disease surveillance program in place that allows us to assess trends in disease prevalence in wild fish populations[1]; therefore, it is not possible to determine whether an association exists between disease in wild and farmed fish populations much less a cause and effect relationship. In order to assess such a relationship one must evaluate a number of factors which affect the host and the pathogen, and determine how these factors affect the interaction of these two elements. Each pathogen and each fish species respond differently to different circumstances and therefore may require different management strategies. Nevertheless, reducing diseases on farms will certainly benefit the industry, and reduce the potential "real" or "perceived" risk of transmission of such diseases to wild fishes. However, it is important to remember that removing disease on fish farms will not prevent disease from occurring in wild fish populations. There are numerous accounts of diseased wild fish that occurred prior to the development of aquaculture on both coasts of Canada. In fact, data from the environmental review office suggests that in the last 10 years the percentage of IHNV, BKD, and furunculosis cases in wild fish submissions to the diagnositic lab at the Pacific Biological Station in Nanaimo, BC has not shown any increasing trends (Stephen and Iwama, 1997).

Reducing the risk of disease in wild fish should include minimizing the number of factors that that may increase the fish's susceptibility to pathogens (e.g., stressors). Finally, it is crucial to remember that the fish reared in government hatcheries for release into the wild are cultured using similar husbandry technology and practice as those used for commercial aquaculture. Research in this area will therefore serve management and policy decisions which will be relevant to natural stock enhancement as well as to the interactions between wild and salmon produced in netpens for food.

RECOMMENDATIONS

1) Establish (or improve) a surveillance system to monitor trends in the prevalence of disease agents (and disease) in wild and farmed fish populations.
2) Determine the actual level of interaction between wild and farmed fish populations (when and where it occurs) to help develop a management plan to reduce or minimize this interaction.
3) Improve fish health. Set guidelines, determine an action plan for endemic and exotic diseases based on the risk of transmission of these individual pathogens, and increase fish health research.
4) Reassess the management practices used on wild stocks. Ensure that they are not increasing susceptibility to disease.

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[1] British Columbia has an extensive database in place which includes data collected from the provincial and federal laboratories to monitor the health of enhanced stocks. Systematic sampling of wild stocks (with the exceptions of some wild broodstock returns) rely on case submissions and it is difficult to determine trends in disease prevalence from these.

Diagram 1.

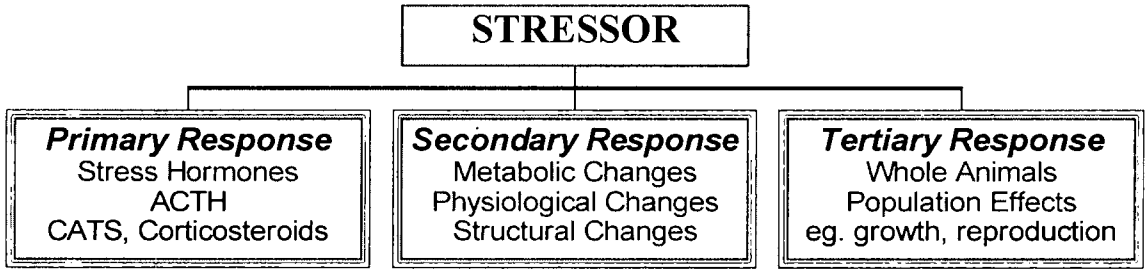
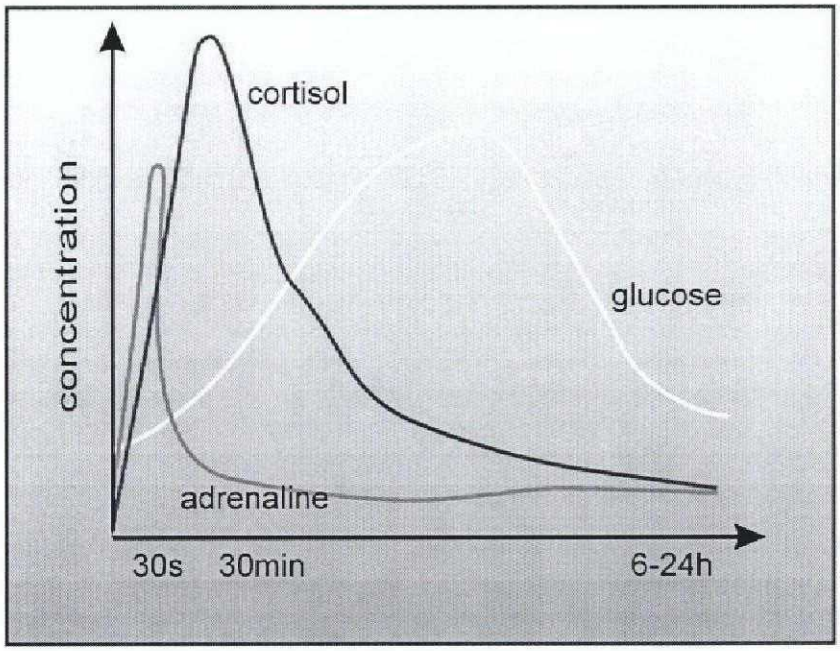

STRESSOR
Primary Response
Stress Hormones
ACTH
CATS, Corticosteroids
Secondary Response
Metabolic Changes
Physiological Changes
Structural Changes
Tertiary Response
Whole Animals
Population Effects
eg. growth, reproduction

Table 1. Comparisons of factors affecting the host's susceptibility to disease for wild and farmed salmonid fishes. ↑ = increases chance of disease, ↓ = decreases chance of disease.

Wild Fish	**Farmed Fish**
↑ - inconsistent feeding	↓ -consistent, balanced diet
↓ - low density in ocean unless crowded for some reason (e.g., counting fence)	↑ -high density increased immunosuppresion increased transfer of pathogens
↑ - predation a constant pressure	↑↓ - predation controlled, but when it occurs can not be avoided
↑ -broodstock not screened for vertically-transmitted pathogens	↓ -broodstock screened for vertically transmitted pathogens
↑ -not vaccinated or treated with drugs	↓ -vaccinated and treated as needed
↓ -resistance to indigenous pathogens	↑ -new hosts in new geographic regions may be more susceptible to indigenous pathogens
↑ -exposed to freshwater pathogens	↓ -free of many freshwater pathogens if reared on ground water
↓ -smolts can select appropriate salinity in estuaries	↑ -smolts have limited control of salinity
↓ -able to avoid poor water quality (e.g., high temperatures, toxic algae blooms)	↑ -trapped when suboptimal water conditions occur
↓ - no handling (if there is handling of wild fish at a counting fence or from anglers this may be quite stressful on fish)	↑ -frequent handling (fish may get habituated to this)

Figure 1. Schematic diagram showing relative time course of adrenaline, cortisol, and glucose concentrations that may be seen in the peripheral circulation of fish following a single episode of stress



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