CUIMR-R-93-076 C2

Stress Induced Pathologies in Fish: The Cost of Stress

GARY P. MOBERG

Department of Animal Science
and
Fisheries Program
University of California, Davis
Davis, CA 95616

ABSTRACT

Pathology is the consequence of prolonged stress. Maintaining fish under aquaculture conditions intensifies the problems of stress by adding the impact of stressors that are unique to culture conditions. Unlike terrestrial domestic animals, fish have not benefitted from the genetic pressures of generations of domestication that favor those individuals most suitable for culture. The physiological mechanisms by which stress induces the development of pathology remain unknown, although it is recognized that one of the major factors affecting health during stress is the response of the neuroendocrine system, a stress responsive system that directly regulated growth, reproduction, and the immune system. During stress, the neuroendocrine system shifts biological resources from pre-stress activities to new functions at a biological cost to the fish. As the biological cost of shifting these resources rises during stress, the fish is placed into a prepathological state, rendering it vulnerable to the development of pathology. It is by focusing on this biological cost of stress that it is possible to develop strategies to reduce stress in aquaculture and to understand the biological basis for the development of disease.

Introduction

Pathology is the consequence of prolonged stress. While we can not identify the exact mechanisms responsible for the development of pathologies during stress, there is indisputable evidence that stress can result in the development of a diverse number of pathologies that include not only disease, but the loss of reproduction, the failure to grow normally, and even the development of abnormal, deleterious behaviors (Moberg 1985).

Fish in culture are especially at risk to the adverse effects of stress. Unlike domesticated mammals, fish have not benefitted from generations of genetic selection for traits that would assist them in adapting to the restraints of confinement. In essence, fish are still wild animals, and when culture conditions do not duplicate their natural habitat, they are forced to make biological adjustments to survive. Exacerbating this problem is the failure of aquaculturists to make any serious effort to systematically define those culture conditions that the fish would find least stressful. Practical considerations have dictated most culture conditions with emphasis on such factors as the ease of cleaning, maximizing the number of fish, or using any holding facility that is readily available. Little concern is given to the fish, at least until stress becomes so severe that pathology occurs. Certainly this is an area of aquaculture that needs to be addressed, especially as increasingly intensified culture conditions are adopted.

Problem of Defining Stress

Although there is considerable evidence demonstrating the disruptive effects of stress on the well-being of fish (Schreck 1982; Pickering and Pottinger 1987; Maule et al. 1989; Vijayan and Leatherland 1990), it is surprising that there is not a better understanding of how stress actually leads to the development of pathologies. One reason for the difficulty in discovering the mechanisms involved is a lack of consistent responses to stress. Most fish do not develop a pathol-

From: Proceedings of the 19th U.S.-Japan Natural Resource Meeting. NOAA Technical Report NMFS 111: 131-134. 1993.

ogy during stress. In fact, it is not possible to predict which fish will become vulnerable to a pathology due to stress. This is because stress, per se, is not necessarily harmful to an individual. Animals have developed defenses for coping with stress. Stress is a part of life, and for a species to be successful, it must have evolved biological defenses to stress. Therefore, stress responses are not bad. In fact, they are desirable and necessary. Pathology occurs only when the animal is confronted with stress of such a magnitude that the very biological responses that evolved for defense result in a biological cost to the individual and renders it vulnerable to pathology. To understand how the biological cost of stress leads to the development of pathology, it is necessary to first examine how fish respond to stress.

Model of the Stress Response

Numerous studies have identified the various biological responses that fish use in responding to and coping with stress (for monographs summarizing this work see Pickering 1981 and Adams 1990). Because these responses are so varied and complex, I will use a model of animal stress (Fig. 1) to organize current concepts of stress biology and examine how the biological cost of stress results in the development of pathology. The development of this model has been discussed elsewhere (Moberg 1985, 1987), but, in brief, the model divides the biological response to stress into three major components: recognition of a threat to homeostasis, the stress response, and the consequences of stress. It is the central nervous system that perceives a threat to homeostasis and organizes the fish's biological defense. If a threat (also referred to as a stressor) is perceived, three general types of biological responses are available to the fish: behavioral, autonomic nervous system, and neuroendocrine system responses. In the wild, behavior can be the most biologically efficient way for a fish to cope by simply allowing the animal to remove itself from the stressor. However, under the confinement of culture conditions, this option is severely limited. If the fish is unable to avoid the stressor, then the roles of the autonomic nervous and neuroendocrine systems become critical. While activation of the autonomic nervous system represents an important way for a fish to avoid or cope with a stressor, its effects are rapid in onset, of short duration, and relatively specific. For this reason, the importance of the autonomic nervous system in inducing stress-related pathologies is questionable. In contrast to the autonomic nervous system, the hypophyseal hormones of the neuroendocrine system undoubtedly play an important role in stress-induced pathology (Moberg 1985). These hormones have a widespread action on the animal and long-lasting effects on such diverse biological functions as reproduction, growth, metabolism, resistance to diseases, and behavior. Each of these functions are vital to the fish's well-being.

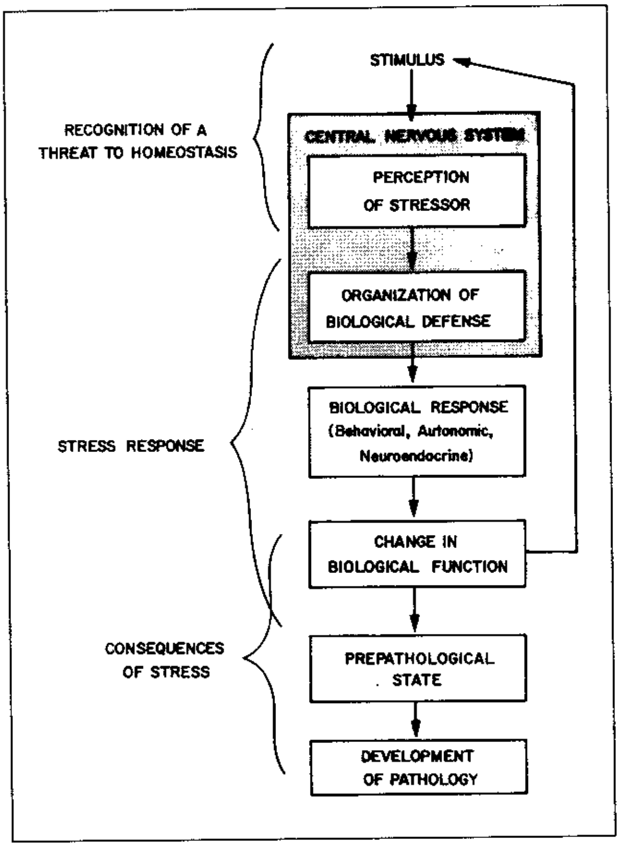

Figure 1

Model for the response of animals to a stressful event (from Moberg 1985).

When the central nervous system organizes the biological defense to a stressor, it is a combination of these three general biological systems that alters function. In mammals, we have found that a number of factors influence the pattern of this biological response, and we have found it impossible to predict how the individual systems will respond (Moberg 1985). While the data for fish is limited, there is evidence indicating that in fish there are also a number of factors that can alter the stress response. For example, water quality and temperature (Pickering and Pottinger 1987, 1989), genetics (Refstie 1982), and social interactions (Peters et al. 1988) have all been found to influence the stress response. Thus, measurement of the three general biological systems have not proved to be reliable as an indicator of stress

(Moberg 1987). However, what is important to the fish is not the pattern of the biological response but the resulting change in biological function—the ultimate consequence of stress.

Regardless of which of the general biological systems the fish chooses in response to the stressor, the result is a change in biological function. Depending upon the appropriateness of the response, this change in biological function may alleviate, or even eliminate the stressor. But the change in biological function can also lead to the development of a prepathological state which can eventually result in the development of a pathology (Fig. 1). For example, if the change in biological function during stress results in the suppression of the immune system (a prepathological state), then the animal is vulnerable to pathogens, and the opportunity for disease (a pathological state) exists. The longer the fish is stressed, the longer the immune system is suppressed and the greater the opportunity for disease to occur. However, disease is only one possible pathological state. Other examples would be the inability to reproduce, abnormal behavior, or the failure to grow normally.

Biological Cost of Stress

The change in biological function that occurs as part of the stress response results in a biological cost to the fish. The fish's resources are diverted from such pre-stress activities as growth to new activities. For example, the glucocorticosteroid hormone cortisol secreted during stress will induce gluconeogenesis, diverting metabolic resources supporting such functions as growth to the production of glucose. Barton and Schreck (1987) found in juvenile steelhead (*Oncorhynchus mykiss*) that acute stress reduced by about 25% the amount of energy available for other activities. Likewise, the biological cost of responding to a stressor may suppress the ability of the immune system to respond to pathogens, rendering the fish vulnerable to disease (Maule et al. 1989). It does not matter which pattern of responses the animal chooses; a change in biological function occurs that imposes a cost, whether or not the change in function is effective in helping the animal to cope with the stressor.

Fortunately, most stressors last for only a brief time and the biological cost of coping with them is relatively small. However, if a stressor is severe, if it persists, or if the fish experiences a series of stressors, the resulting biological cost may be sufficient to induce a prepathological state which in turn can lead to the development of pathology.

The concept that there is a biological cost associated with coping with stress also explains how the effects of subclinical stressors can accumulate, resulting in a significant stress that results in pathology (Moberg 1985, 1992). Separately, none of the subclinical stressors would result in a significant expenditure of the fish's resources, but combined, the subclinical stressors could cost the animal sufficient resources to induce a prepathological state and lead to the development of a pathology. For example, Jarvi (1989) found a greater mortality rate in Atlantic salmon smolts (*Salmo salar*) that were exposed simultaneously to both osmotic stress and the presence of predators than if the smolts experienced only one of the stressors. Because of the accumulation of biological costs, a series of apparently innocuous events can lead to disease, loss in reproduction, diminished growth, or mortality.

Conclusion

The biological cost of stress is the key to understanding why fish exposed to stress develop pathologies. For a fish to cope with a stressor, it must expend biological resources. The more prolonged the stressor or the greater the effort needed to cope with a severe stressor, the greater the diversion of biological resources, and thus the greater the biological cost of the stress. This diversion of resources occurs at the expense of other biological functions, leading to a prepathological state where the fish is at risk to the development of pathology. For some fish, the biological cost of coping with a stressor is greater than for other fish and these are the first to succumb to the stressor. As we manage fish in culture, every effort must be made to reduce stress and, as a result, lower the biological cost for fish living in culture.

Citations

Adams, S.M.
1990. Biological indicators of stress in fish. Am. Fish. Soc. Symposium 8, Bethesda, Maryland, 191 p.

Barton, B.A. and C.B. Schreck.
1987. Metabolic cost of acute physical stress in juvenile steelhead. Trans. Am. Fish. Soc. 116:257–263.

Jarvi, T.
1989. Synergistic effect on mortality in Atlantic salmon, *Salmo salar*, smolt caused by osmotic stress and presence of predators. Environ. Biol. Fishes 26:149–152.

Maule, A.G., R.A. Tripp, S.L. Kaattari, and C.B. Schreck.
1989. Stress alters immune function and disease resistance in chinook salmon (*Oncorhynchus tshawytscha*). J. Endocrinol. 120:135–142.

Moberg, G.P.
1985. Biological response to stress: key to assessment of animal well-being? *In* Animal stress (G.P. Moberg, ed.), p 24–29. American Physiological Society, Bethesda, MD.
1987. Problems in defining stress and distress in animals. J. Am. Vet. Med. Assoc. 191:1207–1211.
1992. Stress: diagnosis, cost and management. *In* The well-being of agricultural animals in biomedical and agricultural research (J.A. Mench, S.J. Mayer and L. Krulisch, eds.), p 58–61. Scientists Center for Animal Welfare, Bethesda, MD.

Peters, G., M. Faisal, T. Lang, and I. Ahmed.
1988. Stress caused by social interaction and its effect on susceptibility to *Aeromonas hydrophila* infection in rainbow trout *Salmo gairdneri*. Dis. Aquat. Org. 4:83–89.

Pickering A.D.
1981. Stress and fish. Acad. Press. New York, NY, 354 p.

Pickering, A.D., and T.G. Pottinger.
1987. Poor water quality suppresses the cortisol response of salmonid fish to handling and confinement. J. Fish Biol. 30:363–374.

Pickering, A.D. and T.G. Pottinger.
1989. Stress responses and disease resistance in salmonid fish: effects of chronic elevation of plasma cortisol. Fish Physiol. and Biochem. 7:253–258.

Refstie, T.
1982. Preliminary results: differences between rainbow trout families in resistance against vibriosis and stress. Dev. Comp. Immunol. Supplement 2: 205–209.

Schreck, C.B.
1982. Stress and rearing of salmonids. Aquaculture 28:241–249.

Vijayan, M.M., and J.F. Leatherland.
1990. High stocking density affects cortisol secretion and tissue distribution in brook charr, *Salvelinus fontinalis*. J. Endocrinol. 124:311–318.