

Winter Stress Syndrome: An Important Consideration for Hazard Assessment of Aquatic Pollutants

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Winter Stress Syndrome (WSS) is a condition of severe lipid depletion in fish brought on by external stressors in combination with normal reductions in feeding and activity during cold weather. Fish can develop this syndrome in response to chemical stressors, such as water pollutants, or biological stressors such as parasites. Substantial mortality can result, potentially changing year-class strength and population structure of the affected species and altering community-level ecological interactions. Aquatic contaminants should be evaluated in the context of seasonal metabolic changes that normally occur in test organisms. WSS could be an important, but as yet unquantified, cause of mortality in many circumstances. Wastewater discharges may pose a greater toxic threat to fish during winter than at other times of the year. A comprehensive protocol for aquatic hazard assessment should include testing for WSS. © 1996 Academic Press, Inc.

INTRODUCTION

Winter Stress Syndrome (WSS) is a term coined by Lemly (1993) to describe a condition of metabolic distress in warm-water fish. The syndrome develops when external stressors that cause increased metabolic demands are present concurrently with the arrival of cold water temperature in late autumn. Cold weather and the associated short photoperiod of winter environmentally "programs" these fish for reduced activity and food intake, and they do not respond to the stressors with increased feeding. If the elevated metabolic demands persist, stored body lipid necessary for overwintering is depleted, body condition drops, and the fish may die. The key element in WSS is the influence of environment on behavior, i.e., reduced feeding as a result of low water temperature and short photoperiod.

Many species of fish in temperate regions of North America undergo a normal seasonal cycle of reduced feeding, decreased body condition, and lowered nutritional status during winter (Oliver *et al.*, 1979; Toney *et al.*, 1980; Thompson *et al.*, 1991). This, in turn, results in a seasonal cycle of ability to resist metabolic stressors. WSS is a serious threat to these fish because it occurs when their capability to

compensate is at its lowest point. Stressors that are normally tolerated during warm weather and active feeding can become lethal as water temperatures and food intake drop in late autumn (Lemly and Esch, 1984; Lemly, 1993).

Assessing the hazard and impact of aquatic pollutants is an important component of modern fisheries management (Nielsen and Johnson, 1983). WSS can be a significant but easily overlooked cause of fish mortality, particularly in circumstances in which pollution is not an obvious problem. Moreover, the occurrence of WSS during a time of year when concerns about water quality may be at a minimum makes it especially important to recognize WSS as a potential regulatory factor for fish populations. Aquatic toxicologists and fisheries professionals should understand the mechanism of WSS, know what stressors are likely to cause the condition, and be aware of the implications for aquatic hazard assessment. This paper is a concise review written to provide that information.

COMPONENTS OF WINTER STRESS SYNDROME

Three conditions must be met concurrently in order for WSS to develop: (i) a significant metabolic stressor must be present, (ii) cold water temperatures must be present (<10°C), and (iii) fish must respond to cold water temperature by reducing activity and feeding. The stressor alone is usually not sufficient to cause problems, but when combined with reduced feeding as water temperatures drop, it can cause the syndrome to develop within 60 days (Lemly, 1993). Fish in the later stages of WSS are somewhat thin and appear to be undernourished. The stressor increases the nutritional requirements of the fish by increasing activity and oxygen consumption. If food intake is adequate to offset these demands, i.e., if water temperature remains warm enough to promote active feeding, no change in condition or apparent health occurs. However, cold water temperature causes reduced feeding and a metabolic deficit can soon develop (Lemly, 1993; Fig. 1).

Several physiological factors contribute to the metabolic

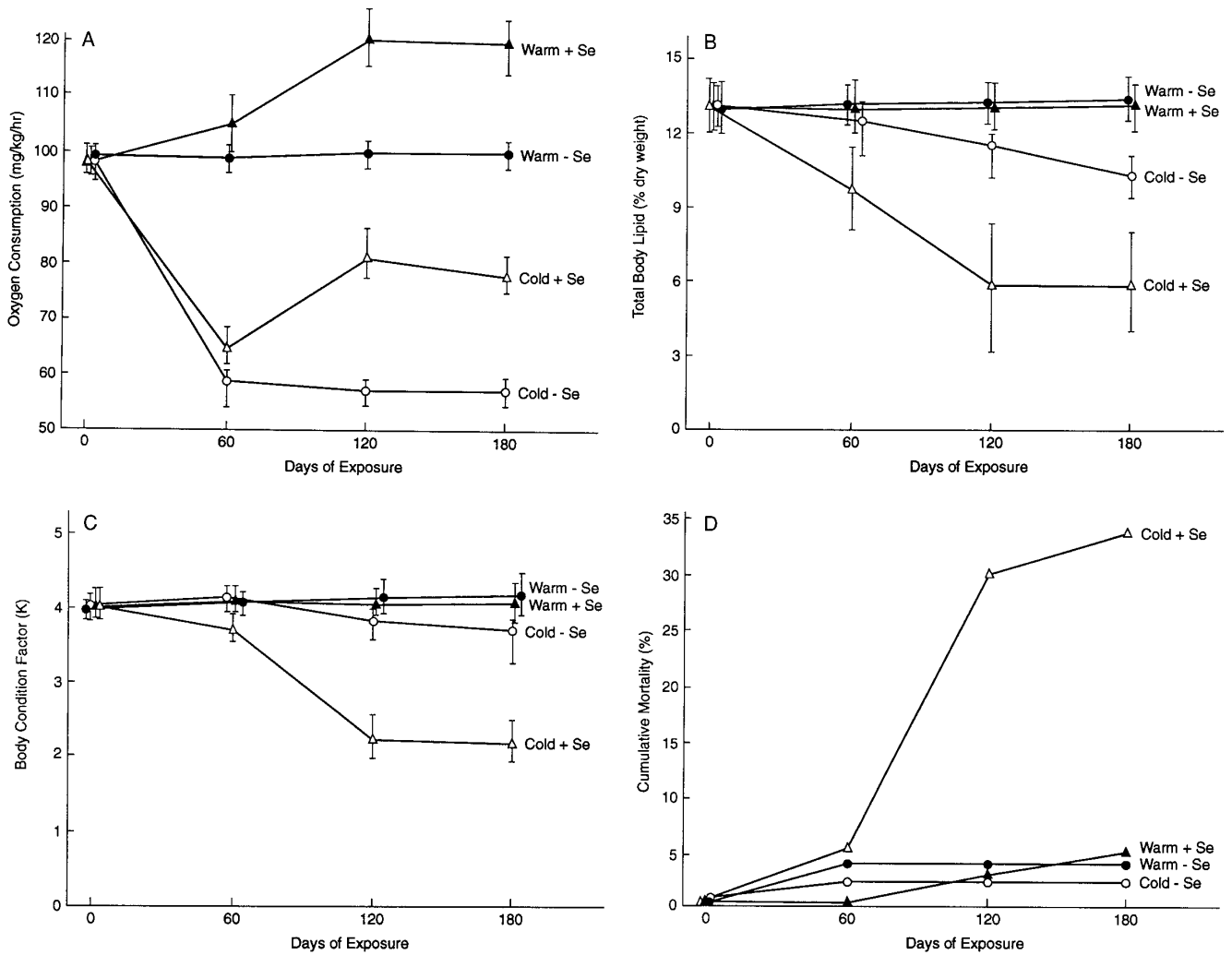


FIG. 1. An example of the key biological responses that characterize Winter Stress Syndrome in fish. These data are for bluegill (*Lepomis macrochirus*) exposed to a trace element contaminant (selenium) in water and diet. Treatments included subjecting bluegill to selenium while concurrently decreasing water temperature and photoperiod, mimicking winter conditions. Water temperature was held constant at 20°C throughout the study for exposures designated as “warm”; temperature was decreased 2°C per week for 8 weeks and then held constant at 4°C in exposures denoted “cold”. The exposures for cold treatments began with a 16:10 hr light:dark photoperiod that was gradually reversed to a 10:16 hr light:dark cycle by Day 60 of the tests. Treatments identified as + Se received 5.1 $\mu\text{g/g}$ dry wt selenium in the diet and 4.8 $\mu\text{g/liter}$ in water; - Se indicates selenium controls, which received 0.8 $\mu\text{g/g}$ in diet and 0.16 $\mu\text{g/liter}$ in water. (A) Oxygen consumption. Values are the geometric mean and range for 5 fish in the controls (warm - Se, cold - Se) and 15 fish in the treatments (warm + Se, cold + Se). The rate of oxygen consumption was significantly elevated in selenium treatments relative to the corresponding control on Days 120 and 180. Metabolic stress due to selenium exposure is indicated. (B) Whole-body lipid content. Each data point is the geometric mean and range for composite samples of 2–5 fish; $n = 2$ or 3 for controls (warm - Se, cold - Se) and $n = 3$ –5 for treatments (warm + Se, cold + Se). Lipid was an average of 7–9% lower in the cold-water selenium treatment than controls and the warm-water selenium treatment on Days 120 and 180. Lipid depletion due to selenium exposure in combination with cold water temperature is indicated. (C) Body condition (K). Values are the geometric mean and range for 10 fish in the controls (warm - Se, cold - Se) and 30 fish in the treatments (warm + Se, cold + Se). Condition was significantly lower in the cold-water treatment on Days 120 and 180. Reduced body condition due to selenium exposure in combination with cold water temperature is indicated. (D) Cumulative mortality. The two selenium treatments, warm + Se and cold + Se, had three replicates, each with 70 fish. The two controls, warm - Se and cold - Se, also had 70 fish each at the beginning of the study. Mortality due to selenium exposure in combination with cold water temperature is indicated. Most of the mortality in the cold + Se treatment took place within about 60 days after water temperature reached its low point of 4°C, which occurred between Days 50 and 60 (Lemly, 1993).

deficit and nutritional distress. Pathological conditions may develop in direct response to the stressor, leading to increased respiration and oxygen consumption and depletion

of stored body lipids (Lemly and Esch, 1984). If the stressor is a chemical contaminant, a variety of sublethal tissue and organ responses can occur and increase energy requirements

(Ribelin and Migaki, 1975; Hocutt and Stauffer, 1980; Rand and Petrocelli, 1985; Lemly, 1993). These factors impinge on the fish at a time when they are forced to be inactive by cold temperature. They do not respond behaviorally or physiologically to offset the effects of the stressor.

The end result of WSS is often mortality. As water temperatures drop, the continued presence of a metabolic stressor greatly accelerates the normal seasonal cycle of lipid depletion. Without this source of energy to draw upon, feeding is insufficient to supply adequate nutrition and fish may die. The lipid content of young bluegill (*Lepomis macrochirus*), for example, does not normally fall below about 10% (dry weight) during winter. Bluegill experiencing WSS cannot maintain as much stored lipid, and levels may fall below 5%, which is the threshold for significant mortality in this species (Lemly, 1993; Fig. 1).

IMPORTANT STRESSORS

Chemical and biological stressors can induce WSS. Two important categories that have been confirmed in experimental studies are chemical pollutants and tissue-dwelling fish parasites (Lemly and Esch, 1984; Lemly, 1993). Both these types of stressors are common, if not ubiquitous, in the aquatic environment and thus have the potential to cause WSS in fish populations throughout temperate regions of the world.

Many chemical contaminants can induce gill damage and respiratory stress, leading to increased respiratory rate and metabolic activity (Eller, 1975; Meyers and Hendricks, 1985; Lemly, 1993). Parasites are not usually lethal to their fish hosts; however, many species are known to cause tissue pathology and substantial metabolic stress (Davis, 1953; Amlacher, 1970; Ribelin and Migaki, 1975; Lemly and Esch, 1984). In addition to the more obvious candidates for consideration, such as heavy metals, synthetic organic compounds, and pesticides, there are other potentially important stressors to include in the WSS component of hazard assessment. These include strongly acid or alkaline pH, naturally occurring trace elements that are mobilized by human activities such as agricultural irrigation (e.g., selenium, boron, molybdenum, and arsenic), and water-quality conditions that cause physical irritation of fish gills, for example, siltation and high suspended sediments. Overlaying these stressors with falling water temperature may be all that is necessary to initiate the metabolic and physiological changes that characterize WSS.

The two primary components of the syndrome, i.e., increased respiratory demands and lipid depletion, appear to be a general metabolic response to stress. Therefore, the probability that a given stressor will lead to WSS depends on its potential to increase the fish's metabolic activity. The

presence of multiple stressors, for example, several chemical contaminants in a wastewater discharge or a contaminant plus a parasite, would greatly increase the probability of the syndrome developing. Time of year is also important in evaluating potential stressors. Those that have little effect and are seemingly harmless during periods of warm water and active feeding could become lethal during winter.

ECOLOGICAL SIGNIFICANCE

Experimental studies have demonstrated that as many as one-third of young centrarchids may die within 1 month following the onset of WSS (Lemly and Esch, 1984; Lemly, 1993; Fig. 1). The critical point for survival in these fish is reached when body lipid falls to about 5%, which occurs within 30–60 days once water temperatures drop below 10°C. Fish that have sufficient lipid to continue for another 60 days with limited feeding will likely survive and those with inadequate lipid will not. In nature, the continued survival of young centrarchids that are alive at about 3 or 4 months following the arrival of cold water temperatures (<10°C) in autumn is probably good. This is true because most of the mortality due to WSS occurs within 90 days. Beyond 90 days, water temperatures would soon be on the increase as spring arrived, particularly in the southeastern United States, accompanied by increased feeding by the fish.

In North America, young centrarchids appear to be at high risk of developing WSS. Field and laboratory observations indicate that the greatest threat is to young-of-the-year and yearling fish because they reduce activity and feeding during winter to a greater extent than older age classes. The population and community-level implications are serious. Lemly (1993) found 33% mortality in young bluegill due to a single contaminant stressor (selenium). It is easy to envision a situation in which two or more stressors would occur simultaneously, thereby multiplying the risk that WSS would develop. The impact of single or multiple stressors could reduce survival of young fish to the point of offsetting reproduction. If the stressors persisted for several years, fish community composition could gradually shift, with centrarchids being replaced by cool-water species in the northern parts of their range or by competing warm-water species elsewhere.

Fishes other than centrarchids are likely to experience WSS if they reduce feeding and activity during cold weather. In North America, field and laboratory observations suggest that young cyprinids and percids are also susceptible to WSS (Toneys and Coble, 1980; Thompson *et al.*, 1991). Basic knowledge of life history characteristics and feeding ecology, particularly for young-of-the-year fish, would allow identification of potentially vulnerable fish species in other temperate regions of the world.

IMPLICATIONS FOR HAZARD ASSESSMENT

In order to adequately assess the hazard of pollutants to fish, laboratory tests must include the range of temperatures that would be encountered in the environment. Warm-water sport fish such as centrarchids experience cold water temperatures for extended periods during winter over most, if not all, of their geographic range in North America. However, standard laboratory protocols for studies of chemical contaminants routinely specify that constant, warm temperatures ($\geq 20^{\circ}\text{C}$) should be maintained when testing warm-water fish (McKim, 1985; Petrocelli, 1985). Evaluating the role of mortality from WSS, which is a consequence of cold water temperature ($< 10^{\circ}\text{C}$) and short photoperiod, cannot be done if these protocols are followed. However, it is critical to determine winter stress mortality in order to establish true no-effect concentrations and develop environmentally sound water-quality criteria.

Water temperature and water chemistry are generally recognized as being important determinants of contaminant toxicity to fish (Sprague, 1985). However, previous studies have focused on how these factors affect the speciation, uptake, and direct toxicity of chemicals rather than examining how seasonal changes in temperature modify the activity and nutritional status of the organism and, thereby, its sensitivity and response. Physiological studies have also concentrated on describing direct effects. Emphasis has been placed on determining mechanisms of toxicity and developing bioindicators of effects rather than evaluating to what extent the physiological changes that result from normal seasonal fluctuations in temperature modify responses (Mehrle and Mayer, 1985). Future studies should use an experimental design that includes measures for determining the metabolic status of the test organism, i.e., oxygen consumption and lipid content. As a result, they would yield information on how changes in environmental conditions affect the physiology of the organism and, in turn, the toxicity of contaminants. The findings for selenium (Lemly, 1993) suggest that this approach should be used to reevaluate the toxicity of all contaminants to fish, particularly the early life stages. Furthermore, they suggest that the aquatic hazard associated with wastewater discharges is greater during winter than at other times of the year. WSS could be an important, but as yet unquantified, cause of mortality in many circumstances.

It is important to know the extent to which the hazard of a pollutant depends on environmental conditions that are present for only part of the year. The occurrence of WSS for a few weeks can eliminate 30% or more of a year class of fish, yet traditional approaches to toxicity testing overlook this aspect of hazard. Well-designed laboratory studies that measure responses over a range of water temperatures and photoperiods, some of which mimic winter conditions, are the first of two steps to use in identifying WSS as a cause

for concern. General procedures for these types of studies are given by Lemly (1993). If the laboratory studies determine that WSS occurs, the second step is to conduct a field assessment. Field validation can be used to verify the results of laboratory tests by placing them into an ecological context (Sanders, 1985). However, such lab-field comparisons for WSS will likely yield conflicting results unless the field studies span the entire fall and winter seasons and also include efforts to monitor concentrations of contaminants in water and food as well as body condition and lipid content of fish. Simply documenting reduced survival is not sufficient evidence to indicate WSS. The response variables for lab and field studies must match in order to produce conclusive evidence of cause and effect.

Investigations of WSS need not be contaminant specific. Whole-effluent tests can be used for both the lab and the field studies to give an indication of the relative seasonal hazard of wastewater discharges. The concept of seasonal hazard adds an important dimension to toxicity evaluations.

Of particular concern is hazard assessment of pollution under a scenario in which chemical stressors are introduced but they overlay preexisting biological stressors. For example, a fish population that is experiencing some level of stress from parasites is challenged with the additional stress of contaminants in a wastewater discharge. The parasite and contaminant stresses will be at least additive, and perhaps even synergistic, thus amplifying the potential impact of the wastewater compared to a situation in which no parasites are present. The potential hazard from aquatic pollutants must therefore be conducted on a site-specific basis in the context of the natural ecology of the fish, including its parasite fauna and associated stresses. In this situation, field validation may yield results that are quite different from those of lab tests. The field assessment will likely show significantly more impact due to the interplay of chemical and biological stresses. However, it may be the added chemical stress that tips the scale and causes WSS to develop. Because the fish-parasite community is probably at an equilibrium level below the effect threshold for WSS (Lemly and Esch, 1984), the full hazard or potential for impacts under this scenario should be attributed to the pollutants, not the parasite. Reductions in hazard will necessarily depend on modifications of the pollutant discharge.

CONCLUSIONS

The tolerance and response of fish to stress depends on many factors, some within the organism and some environmental. A host of physiological and behavioral responses may occur, sometimes leading to disease, reproductive failure, and reduced survival (Esch *et al.*, 1975; Wedemeyer *et al.*, 1976; Mazeaud *et al.*, 1977; Gerking, 1980). Among

these stress-effect possibilities, Winter Stress Syndrome should be recognized as an important seasonal cause of mortality for young fish in temperate waters. Studies that determine toxicity in the context of Winter Stress Syndrome are critical to the development of environmentally sound hazard assessments for aquatic pollutants. Wastewater discharges may pose a far greater hazard to fish during winter than at other times of the year.

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