



Issue Paper 4

Temperature Interaction

**Prepared as Part of EPA Region 10
Temperature Water Quality Criteria
Guidance Development Project**

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Abstract

Pacific Northwest salmon rely on many interwoven factors to maintain their health, well-being, population, and distribution. Abnormal conditions in a fish's environment may elicit a stress response. If a fish is already responding to one stressor, it is less likely to withstand another. Temperature can be a biological, physical, or chemical stressor. Biologically, temperature affects the metabolism of fish and their ability to resist disease. Physically, temperature affects properties of water and fishes' tolerance to suspended sediment. Chemically, temperature can change the concentration of substances in water and reduce a fish's ability to withstand chemical exposure. Not all these relationships are well understood, but they need to be considered when developing a temperature standard. This issue paper reviews biological, physical, and chemical properties related to temperature within salmonid ecosystems.

Introduction

An ecological system is a complex relation of numerous interconnected physical, chemical, and biological processes occurring simultaneously. These processes, which profoundly influence the structure and function of an ecosystem, are strongly affected by temperature. Animals within an ecosystem are exposed to these processes, some of which may be stressful. A stressor can generally be described as any physical, chemical, or biological entity that can produce an adverse response. This paper focuses on multiple interactions that can create stress for fish.

Temperature has a vital role in various processes that help determine whether a watershed is suitable for fish. These include aquatic plant photosynthesis and respiration, chemical reaction rates, gas solubilities, and microbially mediated processes. Temperature also is important in controlling almost all processes in fish (Jensen et al. 1993), both physiological and behavioral (see Behavior and Physiology issue papers).

Because temperature has such far-reaching influence, interrelated parameters must be considered in developing a water quality criterion for temperature. These temperature interactions are discussed below in the following questions and answers.

What is stress and how are fish affected by it?

Wedemeyer and McLeay (1981) define a stressor as an environmental change severe enough to require a physiological response on the part of a fish, population, or ecosystem. Adaptation will occur if the stress response can reestablish a satisfactory relationship between the changed environment and the ecosystem. Acclimation can be said to have occurred if the compensatory stress response enables a restoration of physiological variables to within limits that do not compromise survival (Jobling 1994).

A wide range of biological, chemical, and physical factors can challenge the physiological systems of fish. Various stressors such as handling, fright, forced swimming, anesthesia, rapid temperature changes, and scale loss all elicit a stress response characterized by physiological changes, which tend to be similar for all stressors (Wedemeyer and McLeay 1981). The stress response proceeds as follows: the central nervous system triggers the release of stress hormones (i.e., corticosteroids), changes occur in blood chemistry and hematology (i.e., reduced blood clotting time), and metabolism may be altered, which in turn can result in tissue changes (nitrogen balance and oxygen debt) followed by loss of electrolytes (Wedemeyer and McLeay 1981). These responses are expressed through changes in predator avoidance, growth, parr-smolt transformation, spawning success, migratory behavior, and incidence of disease. There also is a reduction in tolerance to subsequent stressors (Wedemeyer and McLeay 1981). At the population level, stress response may reduce recruitment and species abundance and diversity.

Chronic exposure to these sublethal stressors can result in reduced reproductive success or decreased survival of individuals, which may endanger the survival of entire populations (Jobling 1994). Thus, the success of fish and fish populations in acclimating to environmental changes depends on the compensatory abilities of individual organisms.

A fish's tolerance to environmental alterations depends on its ability to regulate stabilizing processes either physiologically or behaviorally (see Physiology and Behavior issue papers). Single or multiple stressors requiring adjustments that are beyond the fish's ability will eventually be lethal, either directly or indirectly, through secondary processes such as disease.

The stress response is usually considered in terms of primary, secondary, and tertiary changes, starting at the endocrine system and concluding at the organismal level. Jobling (1994) describes a characteristic series of responses divided into three stages: (1) alarm, which is the onset of compensation through initiation of physiological changes and homeostatic control systems; (2) resistance, the successful restoration of physiological balance, albeit with reduced performance capacity; and (3) exhaustion, or biological tolerance limits being exceeded as a result of exposure to the stressors.

To persist and ultimately reproduce, fish need to perform such necessary activities as obtaining oxygen, swimming, metabolizing, and resisting pathogens. The fish's potential capacity to carry out these activities is determined by genes and developmental changes (Schreck 1981). Stress places a physiological load on the fish, thereby reducing its capacity to cope with subsequent stresses (Schreck 1981). The appropriate recovery time depends on the severity and duration of the initial stress and on habitat conditions.

Why are these interactions important to consider in developing a temperature criterion?

To produce a temperature criterion that ensures protection and management of aquatic ecosystems, we must understand the way in which different variables affect interactions among organisms and their aquatic environment. Interactions are influenced by variables that can alter the organism's physiological condition or the physicochemical characteristics of the system. Temperature may have the most far-reaching influence of the many water-quality variables.

How do these interactions affect salmonids?

For each natural characteristic of water, such as temperature, there is an optimal zone for a given species where the species functions most efficiently. If the characteristic becomes less favorable, the organism usually has physiological processes that allow it to partially compensate, but at some metabolic cost. The change may be semipermanent, in which case the animal has acclimated to the new condition. For example, fish held at higher than normal temperature show an increase in upper lethal temperature and may, for a while, prefer a temperature above the one they normally select. Near the outer limits of the fish's range, the metabolic load on the organism becomes greater, reducing its capacity to carry out normal activities such as feeding and reproduction. The implications of these environmental conditions are quite predictable. If an organism is already under stress or metabolic loading from increased water temperature, when faced with an added stressor (e.g., toxic pollutant, disease), it would probably be less capable of dealing with it and show greatly increased susceptibility. Under these conditions, temperature may be a factor modifying the response of the fish (Rand and Petrocelli 1984).

Synergistic interactions occur among the many factors that make up the environment of a fish. The physiological and behavioral condition of a fish are very important to its ability to withstand temperature extremes (Paladino et al. 1980). A fish that is stressed by sublethal levels of a toxicant or by a disease may have a much lower temperature tolerance than a healthy fish of the same age, sex, and species.

Can you describe a situation in which a fish is facing multiple stressors?

A fish population that has recently been exposed to a prolonged series of high-temperature days is an example of a situation involving multiple stressors. After a long period of inadequate food intake, high metabolic costs, and lack of growth, which could result from exposure to high temperatures, the population may be weakened from energy depletion. Subsequently, fish may be more susceptible to disease, because many diseases attack fish that are already weakened from some other stress.

What physical aspects of aquatic ecosystems are influenced by temperature or influence temperature?

Physical aspects of aquatic ecosystems that are influenced by temperature or influence temperature include density and viscosity, current, depth, stream flow, and suspended sediment. These are discussed below.

Density and viscosity. The density, or mass per unit volume, of liquid water is affected by temperature (Gordon et al. 1992). Water reaches maximum density at 39.2°F (4°C) under normal atmospheric pressure. As water temperature increases above 39.2°F (4°C), water density decreases. Water density also decreases as temperature decreases below 39.2°F (4°C), until the freezing point is reached. Upon freezing it becomes considerably less dense as a solid than as a liquid.

Viscosity is the property of a fluid that describes how rapidly it can be deformed, or the relative rate with which a fluid can pour out of a jar. Water viscosity is strongly temperature dependent, and as temperature increases viscosity decreases (Gordon et al. 1992).

How would a change in water density and viscosity translate as an expression in the aquatic ecosystem and salmonid populations? A temperature increase would lead to decrease in water density and viscosity, and possibly a more rapid settling of the suspended sediment particles within a stream. Decreases in density and viscosity cause an increase in the settling speed of suspended particles (Hodges 1977).

The distribution of sediment sizes along a stream is one of the physical habitat factors influencing the distribution of organisms (Gordon et al. 1992). Species differ in their substrate preferences and requirements. When gravel-bed streams fill with silt, for example, they may show a shift in insect species composition that, in turn, can affect fish species composition. Infilling of gravels with finer sediments can reduce intergravel flow rates, suffocate eggs, limit burrowing activity, and trap emerging young.

Even so, density and viscosity changes associated with altered temperature regimes are generally not considered to have a major impact on salmonids or their habitats.

Depth. Water depth has an influence on water temperature, because shallow water tends to heat up and cool down more rapidly (Gordon et al. 1992).

Stream flow. Because streamflows normally decrease during the summer months in most portions of the Pacific Northwest, temperature and salinity levels may rise and plant growth within the channel can increase (Gordon et al. 1992). Some species may rely on low-flow periods for a part of their life history, but others experience stress during this time. During times of low flow when the stream may be confined to limited areas, increasing predation and competition for nutrients and space occur within the remaining waters. Generally, the concentration of dissolved ions in water (salinity) is inversely related to discharge levels, with the highest salinities during low flows and higher flows having a diluting effect. As with other factors, tolerance of saline conditions can influence the distribution and abundance of stream inhabitants.

Suspended sediment. In coho salmon, temperature is an influencing factor for level of tolerance to suspended sediments. To study this, scientists look at median lethal concentration (or LC₅₀), which is the concentration in water to which test organisms are exposed that is estimated to be lethal to 50% of the test organisms. The 96-hr LC₅₀ of yearling coho exposed to suspended fine sediment from the Fraser River (75 µm median diameter) decreased when

temperature was increased (Servizi and Martens 1991). Fish at 44.6°F (7°C) had the greatest resistance (i.e., tolerance decreased) to suspended sediment concentrations, with a 96-hr LC₅₀ of 23 mg/L. The temperature increase resulted in tolerance of suspended sediment being 33% less at 64.4°F (18°C) than at 44.6°F (7°C). Very low temperature was also found to decrease the fish's tolerance. Reduced tolerance to suspended sediments at low temperatures may be primarily related to the capacity of the fish to maintain the cough reflex and ventilation rates that are adequate to clear the gills of particles.

How are chemical constituents affected by temperature?

Temperature affects the following constituents of water: dissolved oxygen, pH, hardness and alkalinity, chemical toxicity, ammonia, organics, metals, cyanide, chlorine, and nitrogen. Each constituent is discussed below.

Dissolved oxygen. The solubility of oxygen in water is directly proportional to the temperature (Hutchinson 1957). Solubility of oxygen decreases in a nonlinear manner with increasing temperature (Figure 1).

There is an important relationship between temperature and the dissolved oxygen (DO) needs of fish. As temperature increases, metabolic rates increase, increasing the demand for oxygen by an organism. At the same time, the DO available to the organism decreases. Therefore, at times of the year when fish may experience temperature stress they also may experience stress from low DO levels.

Aquatic organisms are more likely to experience respiratory distress in warm water than in cool water. Active fish who live in cold water, such as trout, experience a sharp rise in respiratory rate at temperatures above 59°F (15°C). This is the principal reason their growth rate

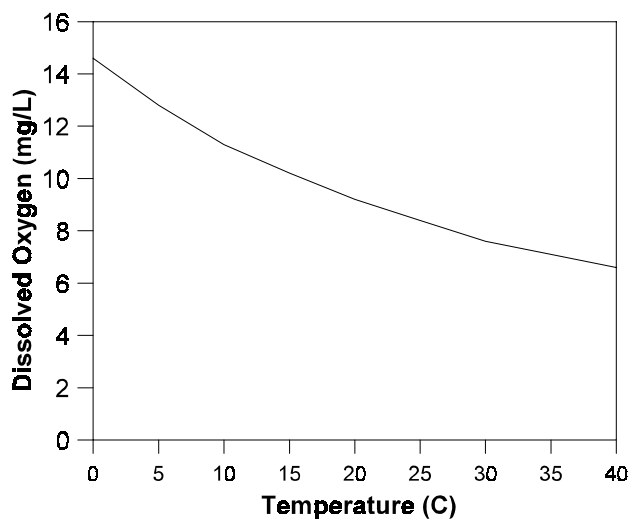


Figure 1. Oxygen saturation concentrations (mg/L) at various temperatures (°C). Derived from Hodges (1977).

declines at higher temperatures even when they are fed to excess (Elliott 1978, as cited in Allen 1995). The need to live in cold water of such species can be attributed as much to the effects of oxygen availability at higher temperatures as to temperature itself (Hynes 1970, as cited in Allen 1995).

Chinook salmon require certain conditions of temperature and DO for migration. When these conditions are not met, adult migration is prevented (McCullough 1999). In the Willamette River, a combination of average daily minimum DO of 3.3 mg/L and an average daily maximum water temperature of 72.3°F (22.4°C) resulted in cessation of upstream migration of spring chinook past Willamette Falls (Alabaster 1988). Data from Hallock et al. (1970) collected in the San Joaquin Delta showed that the average minimum DO at which chinook migrate while avoiding temperatures >66°F (18.9°C) was about 4.2 mg/L. Although combinations of temperature and DO that result in no adult migration do not indicate that adults died, these conditions at least cause stress and probable deterioration in condition, and also reduce spawning success.

If higher DO levels or colder temperatures are available to fish, negative effects of other variables could be tempered. A study conducted in Montana (Feldmeth and Eriksen 1978) found that where grayling distribution in Odell Lake extended to deep portions of the lake in which water temperatures were very cold (39.2-41°F [4-5°C]), the fish did not experience equilibrium loss until DO concentrations dropped to very low levels (1.6 mg/L). More evidence that DO mitigates negative thermal effects is suggested by Erman and Leidy (1975), who documented that trout fry can exist in isolated pools with temperatures as high as 72.3°F (22.4°C). They attribute this to the presence of DO in groundwater seeping into the pool, producing oxygen concentrations of 3.5 mg/L. These fish also swam near the surface to maximize oxygen uptake.

EPA (1986), in its Ambient Water Quality Criteria for DO guidance, described a study by Warren et al. (1973) that indicates growth of salmonids is most susceptible to the effects of low DO concentrations when the metabolic demands or opportunities are greatest. This is demonstrated by greater sensitivity of growth to low DO concentrations when temperatures are high, even with plentiful food. The greatest effects and highest thresholds of effect within the growth data occurred at high temperatures (64-71.1°F [17.8-21.7°C]). Growth data from chinook salmon tests indicate that growth tests conducted at 50-59°F (10-15°C) would underestimate the effects of low DO concentrations at higher temperatures by a significant margin. For example, at 5 mg/L DO, growth was not affected at 55.4°F (13°C), but at temperatures of 68°F (20°C) growth was reduced by 34%. Results from coho tests also support the idea that effects of low DO become severe at higher temperatures.

Fish may be able to adjust their behavior to compensate for low DO levels. Fish confronted with hypoxia (deficiency of oxygen reaching body tissue) are able to change activity level or location in a thermal gradient as a means of adjusting metabolic rate. The final temperature preference for rainbow trout under lowered oxygen conditions was significantly lower than under normal oxygen (Schurmann et al. 1991). It is to the fish's physiological advantage to select a lower temperature in a hypoxic environment, as the decrease in temperature leads to a reduced metabolic rate and a higher blood oxygen affinity (see Behavior issue paper).

pH. The modulating effect of temperature on pH primarily occurs under both acidic and alkaline conditions. Under acidic conditions, Robinson et al. (1976) found that the higher the temperature (39.2, 57.2, and 69.8°F [4, 14, and 21°C]), the shorter the survival time for brook trout in a lethal pH environment. The authors ascribe this to the elevation of metabolic functions in cold-blooded organisms held in a warm environment. Such an increase in metabolic demands would multiply the burden of physiological stress from low pH conditions. Temperature also is significant in the tolerance of rainbow trout to acidic conditions. Kwain (1975) tested rainbow trout embryos and found that at a given pH level the lowest mortality occurred at 50°F (10°C), higher mortality at 41°F (5°C) and the greatest mortality at 59°F (15°C). However, rainbow trout fingerlings showed greater tolerance to acidic conditions when acclimated at 68°F (20°C) than when acclimated at 50°F (10°C).

Other instances of synergistic pH-temperature relations are noted in the literature. Dockray et al. (1998) demonstrated that in juvenile rainbow trout that have either an unlimited or a limited food ration, the combination of warmer temperature (+3.6°F [+2°C]) and sublethal low pH appeared to have a slightly higher metabolic cost than either stressor alone.

For alkaline conditions, the combination of high pH (≥ 9) and elevated temperature (71.6°F [22°C]) has been shown to have an independent, additive effect on mortality in rainbow trout (Wagner et al. 1997). Of four treatments, two consisted of the following pH and temperature combinations: control pH 7.7-7.8 with temperature 61.3-61.9°F (16.3-16.6°C), treatment pH 9 (± 0.05) with temperature 71.1°F (± 0.5 °F) (21.7°C [± 0.3 °C]). Mortality was significantly higher with the high temperature and high pH treatment (100%) than for fish exposed to high pH only (72%). High temperature alone caused no mortality after 96 hours and all fish in the control treatment survived.

Hardness and alkalinity. Water hardness (sum of calcium and magnesium concentrations) has been shown to interact with thermal stress. In experiments with rainbow trout reared in hard water and soft water, median resistance times to thermal stress were lower in juveniles reared in hard water than in soft water (Craigie 1963).

As alkalinity (acid-neutralizing capacity) of water increases, a lowered resistance to temperature may occur in some salmonids. Vigg and Koch (1980) tested two strains of Lahontan cutthroat trout in waters with alkalinity of 1, 487, 357, and 69 mg/L and reported upper lethal temperature ranges of 65.5-68.4°F (18.6-20.2°C), 68.4-70°F (20.2-21.1°C), and 71.2-73.4°F (21.8-23°C), respectively. Different tolerances to high temperatures also were detected between the two strains.

Chemical toxicity. Despite voluminous data on temperature interactions with toxicity of various chemicals, generalizations are not possible. No single pattern explains the effects of temperature on the toxicity of pollutants to aquatic organisms. Increased water temperature can increase the solubility of many substances in water or alter their chemical form (Sprague 1985). Temperature change in a given direction may increase, decrease, or cause no change in toxicity, depending on the toxicant, the species, and the experimental design (Sprague 1985). Although limited evidence suggests that temperature may not have much effect on the chronic “no-effect”

thresholds of pollutants (Sprague 1985), temperature may alter the rate of toxification in chronic exposures (Mayer et al. 1994).

Because the physiology of fish is strongly related to temperature, their response to chemical exposures is also influenced by temperature. Salmonids are cold-blooded, thus their body temperature tracks their environmental temperature rather precisely, with little lag, even when the environmental temperature changes rapidly (Stauffer et al. 1975). Consequently, metabolic processes will exhibit increases and decreases with temperature. As metabolic demands and oxygen consumption increase, gill ventilation must also rise proportionately (Heath and Hughes 1973). A rise in water flow over the gills results in more rapid uptake of toxic chemicals through the gills (Black et al. 1991).

Tolerable temperature ranges vary among species, and to a lesser degree with age, physiological condition, and temperature to which the fish has been acclimated (Cairns et al. 1975). Sublethal exposure to toxic chemicals may reduce the upper lethal temperatures of fish, thereby constricting the tolerance zone (Paladino et al. 1980). Fish that are already weakened by other causes are expected to be much more sensitive to toxicants than are healthy individuals (Jobling 1994).

When fish are exposed to potential toxicants, the chemicals may enter the body over the gill membranes and cause damage. The toxicants may then affect the physiological functions of the fish in a variety of ways. Toxicants may also be consumed along with food and subsequently absorbed from the gastrointestinal tract. Certain chemicals may then be deposited and stored in various tissues of the body, causing tissue concentrations to rise with prolonged exposure to the chemical. A major determinant of the bioaccumulation of a toxicant under a given set of exposure conditions is the rate at which the chemical is metabolized, detoxified, and excreted from the body, processes that are controlled by temperature.

Ammonia. The concentration of ammonia (NH_3) in water depends on a number of factors in addition to total ammonia concentration; most important among these are pH and temperature. The concentration of NH_3 increases with increasing temperature (EPA 1985).

Based on this relationship of pH and temperature with ammonia, EPA's national ambient water quality criteria for ammonia are determined using ambient temperature and pH. Many States have adopted the national criteria for ammonia, which incorporate ambient temperature and pH.

EPA (1985) reported an effect of temperature on the toxicity of the un-ionized ammonia species, independent of the effect of temperature on the aqueous ammonia equilibrium. Rainbow trout were more sensitive to un-ionized ammonia at low temperatures when tested at 37.4 and 57.2°F (3 and 14°C), at 41 and 64.4°F (5 and 18°C), and at 53.6 and 66.2°F (12 and 19°C). This trend also is reported for several warm-water species.

To determine metabolic costs and physiological consequences associated with growth in a warmer environment polluted with an environmentally realistic level of ammonia, Linton et al.

(1997) took quantitative bioenergetic and physiological measurements on juvenile rainbow trout exposed over summer to a simulated warming scenario of +3.6°F (+2°C) in the presence and absence of 70 µmol total ammonia/L. They concluded that juvenile rainbow trout fed to satiation and exposed over summer (approaching 78.8°F [26°C] at hottest time) can make the metabolic adjustments necessary to maintain growth. However, in the presence of a sublethal ammonia concentration, the cost of growth will increase and growth may be compromised. The authors found that in the +3.6°F (+2°C) ammonia treatment, the stimulating effect observed in the low-level ammonia treatment alone was lost in the greater energy demands when fish had to cope with the additional stress of a small further increase in temperature.

Organics. Acute toxicity tests were conducted on rainbow trout with four chemicals (terbufos, trichlorfon, 4-nitrophenol, and 2,4 dinitrophenol) to determine interactive effects with water temperature (Howe et al. 1994). Temperature was found to significantly increase the toxicity of all chemicals except nitrophenol. Chemical bioconcentration was also significantly affected by temperature and was directly related to toxicity in most tests.

A linear relationship between uptake of a toxicant and consumption of oxygen was confirmed by a temperature-induced change in the gill membrane of rainbow trout and oxygen demand (Black et al. 1991). When temperatures were reduced from 62.6 to 46.4°F (17 to 8°C), both oxygen and toxicant uptake were reduced by 50%. Changes in oxygen consumption were correlated with changes in toxicant uptake for three compounds tested (benzo[a]pyrene, 2,2',5,5'-tetrachlorobiphenyl, and naphthalene). Acute temperature change had a proportional effect on the uptake of oxygen and the three toxicants by fish gills.

Other organic chemicals show varied reactions to temperature. Polychlorinated biphenyl (PCB) accumulation rates are enhanced by increased temperature in brown trout (Spigarelli et al. 1983, as cited in Rattner and Heath 1995). Higher temperatures provide protection to rainbow trout exposed to permithrin, an insecticide (Kumaraguru and Beamish 1981): the 96 hr LC₅₀ for 1 g trout increased an order of magnitude from 0.62 to 6.43 µg/L between 41 and 68°F (5 and 20°C). Rattner and Heath (1995) also report that some organochlorine compounds exhibit greater toxicity at cold temperatures in fish, whereas certain organophosphorus compounds elicit the opposite response.

Metals. Water temperature is important in the accumulation of metals in fish. In an oligotrophic lake, Kock et al. (1996) found that Arctic char experienced enhanced uptake of cadmium and lead as a consequence of increasing metabolic rates during the summer. Although peak metals concentrations in the water occurred in the spring with low pH and snowmelt runoff, the highest concentrations in the fish occurred during the summer (46.4-50°F [8-10°C]).

Temperature greatly influences mercury accumulation, elimination, and toxicity in fish. Mercury toxicity in rainbow trout fingerlings is shown to be related to temperature (MacLeod and Pessah 1973). Mercury toxicity was found to increase with temperature (41, 50, and 68°F [5, 10, and 20°C]), probably because of factors such as rate of chemical reaction, diffusion, active transport of toxic materials across membranes, and metabolic rate. Increased accumulation rate of mercury also was observed at higher temperatures, which can be attributed to effects on metabolic rate and uptake. Elimination of mercury from the fish flesh also

depended on temperature, with warmer temperature (68°F [20°C]) increasing the elimination rate. Time to death was found to be linearly related to temperature.

Sublethal exposure to a potentially lethal chemical agent can reduce the resistance of aquatic organisms to elevated temperatures. Exposure to as little as 1.5 mg/L nickel (Ni) for 7 to 21 days significantly suppressed resistance of rainbow trout to elevated temperatures, but 28-day exposure to 0.9 mg/L Ni did not. In a critical thermal maximum (CTM) test (10.8°F/h [6°C/h] rate of increase), equilibrium loss in fingerling rainbow trout occurred at 68°F (20°C) with prior exposure to 3 mg/L Ni after 7 of days exposure, and at 79.9°F (26.6°C) with prior exposure to 0.8 mg/L Ni in a 30-day exposure (Becker and Wolford 1980). Although a great reduction in CTM occurred after prior exposure to 3 mg/L Ni, even 5 mg/L Ni did not cause a reduction in migratory behavior or capacity for seawater survival in coho (Lorz et al. 1978, as cited in Wedemeyer 1980).

Copper can inhibit or inactivate gill ATPase function during coho smoltification at chronic exposure concentrations of 20 to 30 µg/L (Lorz and McPherson 1976). If sublethal cadmium or zinc concentrations are simultaneously present, concentrations as low as 10 µg/L copper can suppress gill ATPase, thereby reducing downstream migration (Lorz et al. 1978, as cited in Wedemeyer et al. 1980). If coho are subjected to thermal stress and chronic copper exposure during emigration, it is likely that ATPase activity and readiness to tolerate saltwater would be impaired. Lydy and Wissing (1988) found that sublethal copper exposure for 3 days significantly lowered the thermal tolerance (measured by critical thermal maximum) for two species of darter.

Temperature influences on zinc toxicity vary depending on acclimation temperature and exposure duration. Hodson and Sprague (1975), using Atlantic salmon in bioassay tests, found that salmon acclimated and exposed at a water temperature of 66.2°F (19°C) were more tolerant of zinc than were those acclimated and exposed at 37.4°F (3°C) and 51.8°F (11°C). The difference was approximately 50% from 66.2°F (19°C) compared with 37.4°F (3°C). As acclimation temperatures became lower, however, salmon were less tolerant (lower LC₅₀ values). Zinc toxicity appears to be enhanced by temperature as a stressor. Moderate (14.4°F [8°C] change in temperature) and severe (28.8°F [16°C] change in temperature) heat stresses shortened time to mortality and increased tolerance only under moderate heat stress. Severe heat stress also changed the pattern of response by altering the slope of the toxicity curves. Moderate and severe cold stresses also lengthened time to mortality, but decreased tolerance. Cold stresses decreased the slopes of the toxicity curves. Lower acclimation temperatures were associated with longer survival but less tolerance, i.e., lower LC₅₀ values. Salmon acclimated to 37.4°F (3°C) had shorter survival when exposed to heat stress (test temperatures higher than acclimation temperatures), and heat stress caused a slight decrease in the slope of the toxicity curve. Temperature may have produced this effect through transient changes in metabolic rate and the amount of water passing over the gills. Slowing of metabolism due to cold may have induced longer survival by reducing ventilation rate and consequently zinc uptake. In contrast, heat may have accelerated metabolic rate and zinc accumulation and shortened survival time. Reduction in thresholds with cold stress and increase with heat stress may result from biochemical processes that vary with temperatures, such as isoenzymes. Isoenzymes that are active during heat exposure might have different sensitivity to zinc than do those active during cold exposure.

Temperature has a marked effect on toxicity of cadmium to rainbow trout, making them more vulnerable at higher temperatures. Fish acclimated and exposed at 42.8°F (6°C) survived significantly longer than those acclimated and exposed at 53.6°F (12°C), which in turn survived significantly longer than fish exposed at 64.4°F (18°C) (Roch and Maly 1979). In addition, lethal thresholds increased with decreasing temperature. Cold-acclimated (42.8°F [6°C]) fish showed a greater 10-day lethal threshold concentration and survived approximately twice as long as warm-acclimated (64.4°F [18°C]) fish exposed to the same concentrations. Roch and Maly (1979) suggest that the rate at which cadmium affects fish is governed by temperature in a way that resembles the effect of temperature on metabolic rate following rapid temperature change.

Cyanide. An increase in temperature has been found to reduce resistance and survival time in salmon smolts and trout exposed to acutely lethal concentrations of cyanide (Cairns et al. 1975). Conversely, a higher 96-hr LC₅₀ (less toxic) was found for juvenile rainbow trout at elevated temperatures (Kovacs and Leduc 1982). Rattner and Heath (1995) hypothesize that fish are better able to tolerate cyanide at higher temperatures because of detoxifying enzyme activity, which is highly temperature dependent. At acutely toxic concentrations, death occurs more rapidly at high than low temperatures because uptake is rapid and metabolic demand accelerates as aerobic metabolism is blocked by cyanide.

Chlorine. In general, chlorine is more toxic at higher temperatures during continuous exposure. However, in an experiment where fish were exposed to a pulsed dose of chlorine, temperature had little effect on toxicity for a variety of cold-water fish species (Rattner and Heath 1995).

Nitrogen supersaturation. The Oregon Department of Environmental Quality (ODEQ) Issue Paper (1995) states that lethal and sublethal effects of nitrogen gas supersaturation on adult salmonids may be exacerbated by high temperatures and prolonged exposures (Beiningen and Ebel 1970). High temperatures can also aggravate the adverse effects of nitrogen supersaturation (Beiningen and Ebel 1970, Ebel 1969).

What aspects of salmonid biology are influenced by temperature?

Competition and predation. Temperature is a key determinant of the outcome of competitive interactions in a fish community. Predation is also keenly influenced by temperature. The Behavioral issue paper contains discussion on multiple interactions related to these topics.

Disease. The influence of water temperature on salmonid susceptibility to diseases and its control on resistance of fish exposed to disease pathogens are well documented in fish pathology literature. Fish diseases involve presence of the disease organism, infection of the host, and resistance to or progression of the disease, resulting in recovery or death. Infection rate and disease outcome depend on a variety of factors, foremost among them being water temperature, disease virulence, and genetics of the stock. During periods of warm water temperature, disease outbreaks are frequently severe.

What factors are involved in infection by disease?

Most fish diseases are favored by increased water temperatures (Ordal and Pacha 1963). The temperature regime, condition of the fish, genetic susceptibility to the disease, virulence of the disease organism, and other stressors determine the infection rate, the percentage survival, and the mean time to death. Condition of the fish can be described in terms of overall health (presence of other diseases), condition factor (a function of fat reserves, food availability, and growth rates), and presence of various points of entry of the disease. Cuts or abrasion of the skin or gills often provide routes for infection. Some diseases are either present or absent in a watershed (e.g., *Ceratomyxa*), but others appear to be ubiquitous and merely await temperature stimuli to become active.

Are warm-water vectors of disease present in rivers of the Pacific Northwest?

The bacterial infection columnaris has been observed throughout the mainstem Columbia River and in numerous tributaries: the Okanogan, Wenatchee, John Day, Umatilla, Yakima, Snake, and Similkameen Rivers. It is carried by all species of Pacific salmon and also by carp, sucker, chub, bass, northern pikeminnow, chiselmouth, and catfish (Colgrove and Wood 1966). Ordal and Pacha (1963) considered temperature-induced columnaris a major factor responsible for declines of Columbia River chinook. The system of reservoirs has been credited with the biggest increase in columnaris disease in the Columbia River (Snieszko 1964).

Although dramatic fish kills from columnaris infection have been documented, other diseases associated with warm water can also produce significant mortalities. *Aeromonas salmonicida*, *A. punctata*, and *A. hydrophila* (also known as *liquefaciens*) are common bacterial pathogens linked to organic pollution (Snieszko 1974) and high water temperatures (Groberg et al. 1978). These organisms are the infective agent for furunculosis, a pathogen affecting all Pacific salmon. *Aeromonas salmonicida* and *A. hydrophila* have been shown in laboratory studies to cause mortality in less than 3 days at 71.6-73.4°F (22-23°C) after infection and to produce survivals of 2% to 30% with constant temperature exposure in the range 69.1-73.9°F (20.6-23.3°C) (Fryer et al. 1976). Resistance to this disease varies greatly with fish strain, as revealed in studies on steelhead stocks (Wade 1986), but expression of the disease is also related to water temperature.

Likewise, there are stock variations in resistance to *Ceratomyxa shasta* but regardless of stock the effects are enhanced by warm water. The occurrence of infective units of *C. shasta* and rate of infection have also been linked to presence of slack flows (Ratliff 1981, Margolis et al. 1992). High infection frequency is associated with reduced time to death (Ratliff 1983).

Presence of the infective stage of *C. shasta* was demonstrated in many locations in the Columbia and Snake Rivers by holding disease-susceptible rainbow trout in liveboxes for 7 or 14 days at various locations (Hoffmaster et al. 1988). *C. shasta* is also a serious disease problem on the Sandy and Willamette Rivers, where 92% and 62%, respectively, of fish that died before spawning were infected with the disease. The disease appears to be controlled by water temperatures <50°F (10°C) (Sanders et al. 1970). Fryer and Pilcher (1974) found that *C. shasta* mortality in rainbow trout was 96% to 100% at ≥69.1°F (20.6°C); 92% and 96% at 59 and 54°F

(15 and 12.2°C); 84% to 75% at 48.9-44.1°F (9.4-6.7°C); and 0% at 39°F (3.9°C). In juvenile coho salmon, mortality was 92% to 100% at ≥69.1°F (20.6°C); 57% to 59% at 64°F (17.8°C); 13% to 31% at 59-54°F (15-12.2°C); and 0% to 4% at ≥48.9°F (9.4°C).

Is just a single life stage susceptible to disease?

Infection of salmon with warm-water diseases can occur at any life stage. That is, infections and mortality from disease can occur at the egg, alevin, fry, parr, smolt, or adult life stages. The alevin and fry stages are probably less likely to be significantly affected in the field because temperatures are generally below critical thresholds during these phases.

Juvenile salmon do show greater sensitivity to some diseases at higher temperatures. Uninjured fingerlings exposed for 30 minutes to *Bacillus columnaris* and then held in water at 70°F (21.1°C) all died within 3 days, whereas only 24% of fish similarly exposed for 38 days died at 64.9°F (18.3°C) for 38 days and 60% of fish at 60.1°F (15.6°C) (Fish and Rucker 1943). No mortality occurred at 50°F (10°C) over the 38 days. Susceptibility to disease is a function of concentration of columnaris organisms, length of exposure, and temperature (Fujihara et al., as cited in Parker and Krenkel 1969) as well as age of individual (increased age, increased resistance) (Olson and Fujihara 1963, as cited by Parker and Krenkel 1969). Table 1 also illustrates that young chinook salmon exposed to *C. columnaris* exhibited increased mortality on exposure to increasing temperatures (Rucker and Ordal 1944, cited by Ordal and Pacha 1963).

Wouldn't disease be obvious in salmon if it were present? Wouldn't dead fish be noticeable if this were a problem?

Rather large kills of juvenile fall chinook were reported at McNary Dam on the Columbia River in July 1994 and July 1998 during periods of high water temperature (Tiffan et al. 1996). These observations represented a small subsample of the entire migrating population that passed the dam. In the mainstem, however, dead juvenile fish may not rise to the surface and they decompose rapidly. Infected fish become easy prey to predators and may be included in predation rate rather than disease mortality rate. In addition to increased predation, diseased fish probably are less able to perform essential functions, such as feeding, swimming, and defending territories, and may not migrate or grow effectively or achieve critical size prior to ocean entry.

Mortality from disease generally requires several days. Juveniles that become infected prior to emigrating from natal habitat to the ocean may die unnoticed in mainstem reservoirs or in the ocean (Ratliff 1981, 1983). When individual adult fish are radiotracked, it is rare to find a carcass even when it is washed ashore (Schreck et al. 1994). Mammals or other scavengers are generally efficient at removing carcasses. In some rivers, such as the Fraser in Canada, that have very large adult salmon migrations, warm water has frequently produced dramatic fish kills that are very noticeable. However, run sizes in rivers of Oregon, Washington, and Idaho today are typically small enough that mortality of juveniles or adults from any source could go unnoticed.

Table 1. Increased mortality observed in chinook salmon exposed to *C. columnaris* on exposure to increased temperatures

Temperature (°C)	Time of exposure (days)	Mortality (%)
22	3	100
20	7	90
17.8	7	45
16	7	30

What are some modes of infection?

For columnaris infection, the infection rate and time to death are related to the virulence of the disease organism, the condition of the fish, and the method of infection. With highly virulent log-phase cells, contact is the most effective method of infection, but is least effective in low-virulence strains (Pacha 1961). However, Fish and Rucker (1943) showed that uninjured sockeye fingerlings exposed to columnaris became infected and died within only 48 h. Injured fish required 72 h to die. Temperatures <60.1°F (<15.6°C) prevented infection, but higher temperatures led to rapid death. Temperatures of 69.8°F (21°C) allowed the bacteria to easily penetrate the mucus coating of skin and gills, and between 60.1°F (15.6°C) and 69.8°F (21°C) bacteria invaded the body through cuts and abrasions (Fish 1948). The perils of adult migration through fish ladders and over sharp rocks, and descaling and abrasion of juveniles on fish screens, are but a few means for opening routes of infection. Prespawning mortality can be minimized by holding adults in water temperatures ≤50°F (≤10°C) to allow abrasions to heal (Fish 1944).

Contagion of columnaris has been suspected during passage of salmon through fish ladders (Pacha 1961), and increased incidence may be a result of the creation of slow-moving reservoirs (Snieszko 1964). Warm sloughs may also harbor a disease organism in coarsefish that can then infect salmonids migrating in warmed reservoirs (Fujihara et al. 1971). Likewise, ceratomyxa infection has been most severe in locations and under conditions having low or slack flows, such as reservoirs or side arms of channels (Ratliff 1981, Margolis et al. 1992).

Has infection with warm-water diseases been documented historically in streams of the region?

Problems with columnaris disease in the Columbia River were heavily documented in numerous studies conducted from the 1940s to the early 1970s. In the Fraser River, columnaris is of continued concern because of the noticeable impacts to adult sockeye on spawning grounds. As determined by surveys of antibody titers in 1964-65 and 1969-70, peak yearly columnaris infection rates in the Columbia River can be at least 70% to 80% in adults (Fujihara and Hungate 1970).

Surveys of infection frequency of sockeye and chinook in the Snake River in July and early August of 1955-1957 revealed 28% to 75% of fish infected when water temperature was $>70^{\circ}\text{F}$ ($>21.1^{\circ}\text{C}$) (Ordal and Pacha 1963). During this same period the disease was widespread in the Yakima and Okanogan Rivers. In 1958, high percentages of salmon were infected judging by samples taken at several mainstem Columbia River dams from Rock Island to Bonneville, as well as on the Yakima, Wenatchee, and Okanogan Rivers. In 1958, water temperatures in the Okanogan were so warm that the run was vastly damaged by columnaris. Thousands of adults left the Okanogan to seek the cooler temperatures of a tributary (the Similkameen River), only to die there from columnaris infection (Pacha and Ordal 1970). Over the years 1955 to 1959, the sockeye run to Redfish Lake, Idaho, declined by an order of magnitude, coincident with a large increase in Columbia River water temperatures. In 1955 and 1956, the frequency of infected sockeye was 34% and 50%, respectively, in samples taken at Clarkston, Washington (Pacha 1961). Even though these infection frequencies were high, it is likely that they became higher as the fish migrated toward their spawning grounds. Pacha (1961) reported that Anacker (1956) sequentially sampled the sockeye run into the Okanogan River, finding that columnaris frequency rose from 6.3% in August at Rock Island to 23.8% and then 38% in 9 and 15 days, respectively, further along in the migration. At the termination of the run the disease incidence was 55% (Pacha and Ordal 1970).

Chondrococcus columnaris infection was implicated in high sockeye mortalities in the Columbia River (Fish 1948). Columnaris becomes increasingly active above 60.1°F (15.6°C) (Colgrove and Wood 1966). The near obliteration of the run of 1941 at Bonneville Dam occurred when temperatures reached a high of 74.5°F (23.6°C) and an average of 68.5°F (20.3°C) (Fish 1948). For adult sockeye on Fraser River spawning grounds, mortality of females ranged from 5% to 86% from bacterial gill infections at temperatures of 71.6°F (22°C) (International Pacific Salmon Fisheries Commission 1962, as cited by Parker and Krenkel 1969). In 1970, there was a columnaris epidemic in the Hanford Reach and in the Wenatchee portion of the mainstem Columbia River, in which it appeared that coarsefish passed the disease to adult sockeye during their migration (Fujihara et al. 1971).

In 1967 and 1968, coho placed in liveboxes in the Columbia River at Bonneville Dam had ceratomyxa infection rates of 53% and 60%, respectively. At the Dalles Dam in 1967, rainbow trout in liveboxes exhibited a 13% infection rate (Sanders et al. 1970).

Are warm-water disease outbreaks in salmonids a thing of the past? Has infection with warm-water diseases been documented recently in streams of the region?

Although warm-water fish diseases were studied intensively in the past, insufficient attention has been given to them recently. This is probably because far fewer numbers of fish are present today and many other problems demand redress. Columnaris infection frequencies have been documented in fall chinook in small-scale surveys conducted in recent years on the Columbia River. After the large fish kill at McNary Dam on July 16, 1994, 125 juvenile fall chinook were collected at John Day Dam and held at the Lower Columbia River Fish Health Laboratory at the USFWS facility in Cook, Washington. In a 4-day period, 94% of these fish died from a columnaris infection considered to be a low-virulence strain (Tiffan et al. 1996). In mid-July 1998, three consecutive days of high fall chinook mortality were detected at McNary

Dam because of high water temperatures. Of 25 fish sampled from the Juvenile Fish Facility at McNary Dam that were distressed (swimming on their sides), 88% had columnaris infection. Although the fish were near death, there were no visible external signs of disease (Tiffan, USFWS, Cook, WA).

In the Rogue River, prespawning mortality from furunculosis and columnaris averaged 12% for wild and 36% for hatchery spring chinook from 1977 through 1981 (Cramer et al. 1985, as cited by Lindsay et al. 1989).

Infection frequency of rainbow trout by ceratomyxa was generally <20% in reservoirs near many of the mainstem Columbia and Snake River dams. However, in June 1984 infection frequency was 52% at the Dalles Dam, and in July 1986 at Hells Canyon and Oxbow Dams was 96% and 95%, respectively (Hoffmaster et al. 1988).

What is the relationship between increasing temperature and columnaris disease? Are there any apparent critical temperature thresholds?

Rate of infection increases with temperature. Laboratory studies are very good at identifying the influence of temperature on infection rate. Juvenile spring chinook infection with *C. columnaris* at temperatures of $\leq 54^{\circ}\text{F}$ ($\leq 12.2^{\circ}\text{C}$) was negligible, but between 59°F (15°C) and 73.9°F (23.3°C) the percentage of infected fish rose steadily with temperature from 27% to 80% (Fryer and Pilcher 1974).

Coho salmon and rainbow trout exposed to *C. columnaris* had a rapidly increasing rate of infection with increase in temperatures above 54°F (12.2°C) (Fryer and Pilcher 1974). For coho and rainbow, infection frequency of the experimental group was low at 54°F (12.2°C) (3% to 8%) but was 49% and 40%, respectively, at 59°F (15°C), and rapidly jumped to 100% at temperatures $\geq 69.1^{\circ}\text{F}$ ($\geq 20.6^{\circ}\text{C}$).

Migration effects. Prespawning survival rates among adult sockeye in the Fraser River spawning areas were as low as 10% (as found in the Chilko stock) in the early 1960s. Given that river temperatures enroute to spawning grounds for the Chilko and Stuart Lake runs averaged 62.1°F (16.7°C) during 1956 (Idler and Clemens 1959), it appears that if fish can survive migration through these adverse temperatures, it would be essential to find suitable temperatures on spawning grounds. Unfortunately, during other years, temperatures at Hell's Gate during the Horsefly sockeye migration can be even higher (up to 66.7°F [19.3°C]) (Williams et al. 1977). Bouck et al. (1970) (as cited by EPA and NMFS 1971) did not observe sockeye mortalities from columnaris at 62.1°F (16.7°C) but did note frequent lesions and death at 68°F (20°C). Temperatures of 68°F (20°C) were reported to result in 100% mortality of chinook during columnaris outbreaks (Ordal and Pacha 1963).

The evidence from the laboratory and field indicates that temperatures between 62.1°F (16.7°C) and 68°F (20°C) and greater lead to infection of adult salmon with columnaris under exposure to low-virulence strains, but infection can occur at even lower temperatures when high-virulence strains are present. Relatively short river reaches with heightened temperatures may

lead to infection that then runs its course during the prespawning period. Temperatures between 68 and 75.2°F (20-24°C) have been linked to extreme sockeye mortality in the Columbia River.

Holding effects. Columnaris becomes increasingly active above 60.1°F (15.6°C), as revealed in studies on Horsefly Creek, British Columbia (Colgrove and Wood 1966). It was found that adult sockeye held at a mean temperature of 60.1°F (15.6°C) on spawning grounds had survival rates of only 19% to 37%. However, when held at mean temperatures of 55°F (12.8°C) and a maximum <57°F (<13.9°C), the sockeye did not die and gill lesions from columnaris infection began to heal (Colgrove and Wood 1966). A mean of 55°F (12.8°C) and a maximum of <57°F (<13.9°C) is a recommended holding temperature on spawning grounds.

This evidence indicates that temperatures between 57 and 60.1°F (13.9-15.6°C) constitute a transitional temperature region below which recovery from columnaris after infection could occur, or above which infection and mortality continue to increase.

Juvenile survival rate decreases with increasing temperature. Rates of survival from columnaris infection in spring chinook were found to decrease between temperatures of 39 and 73.9°F (3.9-23.3°C) (Fryer and Pilcher 1974) (see McCullough 1999, Table 7). Survival was ≤30% for temperatures of 69.1°F (20.6°C) or greater. Time to death from the point of infection was reduced from 7 to 2.5 days as temperatures increased from 59 to 73.9°F (15-23.3°C) (see McCullough 1999, Table 8).

A temperature of 54°F (12.2°C) appears to be the threshold for initiating significant mortalities in coho. Below this temperature survival was near 100% (see McCullough 1999, Table 9). In rainbow trout survival was ≤8% for temperatures above 64°F (17.8°C). As with coho, the threshold for initiating significant mortality in rainbow occurred at ≥54°F (≥12.2°C). Experiments with steelhead infected with *Flexibacter columnaris*, a columnaris strain of intermediate virulence, showed that percentage survival was very high in the temperature range 39-54°F (3.9-12.2°C) but decreased from 44% to 0% as temperatures rose from 59 to 73.9°F (15-23.3°C) (Holt et al. 1975).

In summary, on the basis of laboratory and field studies on chinook, coho, sockeye, rainbow trout, and steelhead by numerous investigators, infection and mortality by columnaris disease were negligible at temperatures ≤55°F (≤12.8°C), but temperatures ≥59°F (≥15°C) produced significantly increased mortalities.

Time to death decreases with increasing temperature. Mean time to death after infection with columnaris varied from 4.2 to 7 days with exposure to 59°F (15°C) in juvenile chinook, coho, and steelhead. Steelhead were the most sensitive (Fryer and Pilcher 1974).

Are other salmon diseases of warm water similar to columnaris in their temperature-survival relationships?

Rates of infection, percentage survival, and time to death after infection that were reported for columnaris infection in a cross-section of salmonids are very similar to those reported for *A. salmonicida*, *A. hydrophila*, and *Vibrio* (see McCullough 1999).

What is the relationship between disease and stress?

In most cases, an equilibrium exists in the interactions among fish, their pathogens, and the aquatic environment, and this equilibrium must be altered for disease to occur (Wedemeyer et al, 1976, Esch and Hazen 1980, Walters and Plumb 1980, cited in Wedemeyer and Goodyear 1984). When the relation among host-pathogen-environment is favorable for the host, fish populations normally exhibit good health, growth, and survival. When this relation is impaired, the incidence of diseases will begin to increase. When the relation is poor, health problems accompanied by reduced growth and survival may become chronic (Roberts 1978 and Schaperclaus 1979, as cited in Wedemeyer and Goodyear 1984). A number of environmental alterations have been associated with poor fish health, including unfavorable or fluctuating temperatures (Wedemeyer et al. 1976, Knittel 1981, Malins et al. 1980 and 1982, cited in Wedemeyer and Goodyear 1984).

Fish disease incidence is potentially a very sensitive index of stress (Wedemeyer and McLeay 1981). Fish diseases do not necessarily have one cause but are the end result of the relationship among the pathogen, fish, and environment. Good examples of stress-mediated diseases are those resulting from bacterial pathogens that are continuously present in most natural waters. The presence of many of these pathogens will result in widespread disease only if unfavorable environmental conditions also exist and the host defense system has been compromised. Table 2 describes diseases that can infect salmonids and the conditions that predispose salmonids to such disease.

As warming temperatures increase the abundance or virulence of disease organisms to salmonids, they also decrease a fish's ability to withstand the stress of disease. When environmental conditions are optimal for a disease, it grows more rapidly and may be more virulent. If environmental conditions are more optimal for the disease than they are for the fish, there is a greater likelihood that the disease will overcome the host's defense systems and create serious illness (Wedemeyer and Goodyear 1984). This concept is illustrated with *Ceratomyxa shasta*, the myxosporean responsible for causing the disease of ceratomyxosis in salmonid fishes. Fish can become infected in low water temperatures, although the progress of the disease is temperature dependent and most infections are detected in warmer waters (Bartholomew et al. 1989). Juvenile rainbow trout exposed to the infective stage of *C. shasta* and held at water temperatures of 44.1-73.9°F (6.7-23.3°C) had little or no ability to overcome the infection, with mean time from exposure to death directly correlated to temperature (Udey et al. 1975). As with rainbow trout, juvenile coho salmon also showed a temperature dependence with mean time from exposure to death. However, results of coho salmon experiments that showed progressive and significant increase in mortality with increased water temperature from 48.9 to 68.9°F (9.4-20.5°C) differed from results of rainbow trout experiments in which observed mortality appeared to be independent of temperature.

