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Evidence Linking Delayed Mortality of Snake River Salmon to Their Earlier Hydrosystem Experience

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Abstract.—The numbers of Snake River salmon and steelhead *Oncorhynchus* spp. have substantially declined since the completion of the Columbia River hydrosystem. We used analytical approaches to identify management options for halting the decline of these stocks, such as removal of Snake River dams and improvements to the existing hydrosystem. The benefits these actions are predicted to have in terms of salmon recovery hinge on whether the mortality that takes place in the estuary and early in their ocean residence is related to earlier hydrosystem experience during downstream migration. Evidence from the literature demonstrates numerous mechanisms that would explain this delayed mortality in relation to a fish's experience passing through the hydrosystem. Spatial and temporal comparisons of stock performance provide indirect evidence of delayed mortality and evidence that delayed mortality is linked to hydrosystem experience. Recent mark–recapture data also provide evidence of differences in delayed mortality by route of passage through the hydrosystem. The different types of evidence discussed here suggest that the delayed mortality of Snake River fish is related to the hydrosystem.

Over the last several decades, Pacific salmon *Oncorhynchus* spp. and steelhead *O. mykiss* populations have declined to extremely low levels. These declines have been especially pronounced in the Snake River and upper Columbia River regions, where the National Marine Fisheries Service (NMFS) has estimated a 55–100% probability of extinction of particular stock groups over the next 100 years (NMFS 2000). These low population levels, and the high risk of extinction, led to the recent Endangered Species Act listing of 13 evolutionarily significant units (ESUs) in the Columbia Basin (Myers et al. 1998). The decline in Columbia River Basin salmon and steelhead is probably the result of a combination of factors, including harvest, habitat degradation, hydroelectric development, an emphasis on hatchery supplementation, and climatic and oceanic conditions.

The Snake River stocks of spring and summer chinook salmon *O. tshawytscha* and steelhead once accounted for as much as 50% of the salmon produced in the interior Columbia River Basin (Hassemer et al. 1997). Because of their earlier decline relative to other Columbia River stocks, the extinction or near extinction of some stocks, and their long migration past as many as eight large hydroelectric dams (Figure 1), these Snake River stocks have been a topic of extensive research, modeling, and management efforts since the development of the hydroelectric system in the late 1960s and early 1970s. Most recently, the fate of the Snake River stocks has received national attention, and the question of whether or not to breach the lower four Snake River hydroelectric dams is being discussed among scientists, managers, and politicians (American Fisheries Society 1999a; ISG 1999; Karieva et al. 2000).

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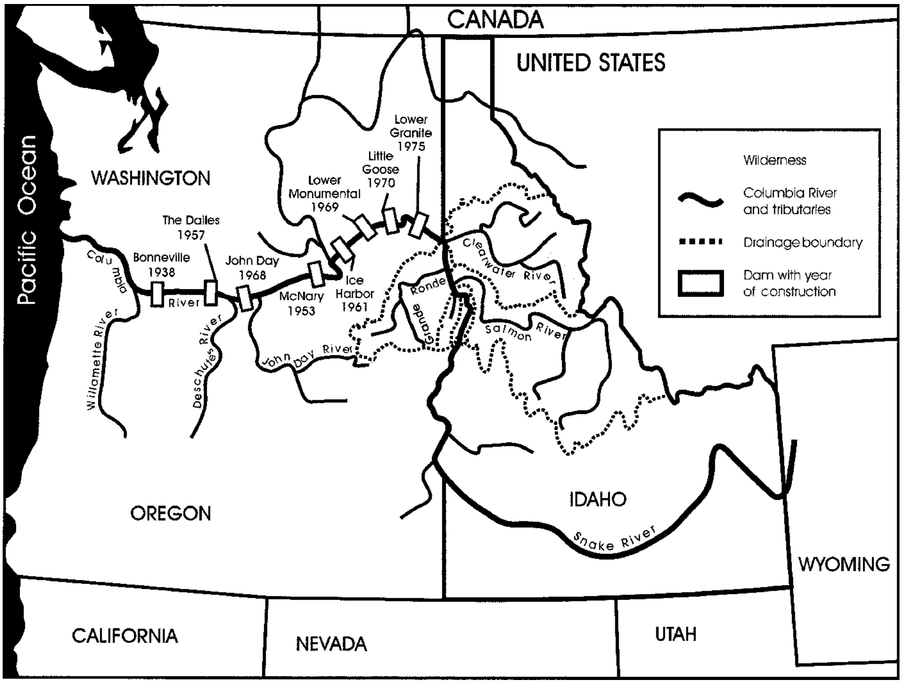

FIGURE 1.—Map of the Columbia River Basin, showing the eight hydroelectric dams (and dates of their completion) that spring and summer chinook salmon migrate past on the Snake and lower Columbia rivers.

Until recently, management efforts for Snake River salmon and steelhead stocks have focused largely on technological fixes aimed at improving the survival of juvenile and adult fish through or around the hydroelectric dams during their migrations to and from the Pacific Ocean (ISG 1999; Nemeth and Kiefer 1999). These technological fixes have included attempts to transport juvenile fish around the dams by truck or barge, allowing bypass fish to migrate in-river around each dam, spilling the fish over the tops of dams, and developing turbines having lower mortality rates for the fish that pass through them. Although these fixes have gradually improved the direct survival of fish around or through the dams (Raymond 1988; Williams and Mathews 1995), the Snake River stocks have continued to decline. Because of this continued decline, along with the results from research and modeling, many fishery scientists now believe there is no feasible technological fix, short of dam breaching, that will recover Snake River salmon and steelhead (Marmorek and Peters 1998; American Fisheries Society 1999b; ISG 1999; Nemeth and Kiefer 1999; Karieva et al. 2000).

As part of the efforts to mitigate the impacts of the hydrosystem on endangered stocks, the breaching of the four lower dams on the Snake River has been proposed (USACE 1999). This management option was evaluated through a multiagency modeling process (the Plan for Analyzing and Testing Hypotheses; PATH). The PATH forum completed a detailed biological decision analysis to evaluate the different management options recommended for the recovery of Snake River ESUs (Marmorek and Peters 1998; American Fisheries Society 1999a). In addition, NMFS, the agency responsible for protecting anadromous fishes under the Endangered Species Act, has addressed the question of dam breaching, in the context of improvements in other areas and life stages, using a series of extinction and life history models through their Cumulative Risk Initiative (NMFS 2000). The PATH decision analysis demonstrated that dam breaching would likely lead to recovery over a broad range of hypotheses and was thus the most risk-averse management option. The NMFS Cumulative Risk Initiative similarly concluded that to minimize the risk of extinction of Snake River stocks, dam breaching may be required in combination with improvements in habitat, reductions in harvest, and changes in hatchery practices (NMFS 2000).

The key point of uncertainty relates to the source of mortality that affects Snake River fish while they are in the estuary and ocean. Sources of estuary and early ocean mortality include not only elements of the natural ocean environment but also delayed effects of earlier life stage experiences. Here we focus on the hypothesis that although this mortality takes place in the estuary and ocean, it may be related to a fish's earlier experiences within the hydrosystem. Snake River salmon, for example, may die in the estuary or ocean after exiting the hydrosystem as a result of the cumulative stresses of negotiating as many as eight hydroelectric dams. Direct mortality, in contrast, is mortality that can be measured directly and takes place within the hydrosystem. Delayed hydrosystem mortality could also affect the upstream survival of returning spawners (e.g., increased straying of transported fish as a result of poor imprinting), but that component is not addressed here.

Identifying the incidence and magnitude of delayed hydrosystem mortality of Snake River stocks is crucial to the predicted outcome of a dam-breaching scenario. If delayed mortality takes place and is related to the hydrosystem, then dam breaching is likely to greatly increase the survival of Snake River stocks and ultimately may provide a high probability of recovery. In contrast, if delayed mortality does not take place or is unrelated to hydrosystem experience, dam breaching will have less of a benefit and may not be sufficient to recover these stocks. Whether research can reduce this uncertainty is unclear (Brodeur et al. 2000); under current conditions, however, extinction is highly probable and decision makers will be faced with making a decision about dam breaching soon. In this paper, we discuss evidence that Snake River stocks are affected by delayed mortality and that this outcome is related to their experience in the hydrosystem. This paper provides a comprehensive synthesis of the evidentiary points for hydrosystem-based delayed mortality. We attempt to document the different types of evidence currently available that may help fishery scientists and decision makers fairly evaluate the likely effects of a dam breaching.

Evidence for delayed hydrosystem mortality comes in several forms. In this paper, we first summarize the literature that considers mechanisms for delayed mortality and links to a fish's hydrosystem experience. In addition to our literature review, we also evaluate spatial and temporal patterns of stock performance to provide indirect evidence for the existence and the extent of delayed hydrosystem mortality. Finally, we evaluate direct evidence of delayed hydrosystem mortality from recent passive-integrated-transponder (PIT) tag data, which documents the hydrosystem passage and survival histories of individual fish. The combination of information from different life stages, however, provides evidence and estimates of delayed hydrosystem mortality and links this mortality to factors of the earlier hydrosystem experience. In this paper, we synthesize these pieces of evidence for delayed hydrosystem mortality.

Study Area

A Natural River

As the Independent Scientific Group noted (ISG 1999), the predevelopment Columbia River salmonid ecosystem was a network of complex interconnected habitats that had been created, altered, and maintained by natural physical processes. Flow regimes produced by local and regional climates (unencumbered by hydrosystem regulation) provided natural spawning, rearing, and migration conditions. Before hydrosystem development, passage to and from the spawning and rearing sites was unimpeded. The historical hydrograph of the Columbia River Basin was characterized by high spring runoff (April through mid-June), decreasing summer and fall flows (mid-June through December), and low winter flows (January through March). Anadromous salmon adapted to this flow pattern by migrating downriver during the spring freshet. These riverine habitats, now altered by human activity, were and are critical for salmonid spawning, rearing, migration, feeding, and predator avoidance (ISG 1999).

Snake River salmon and steelhead have complex life histories, encompassing adaptations to a variety of freshwater and saltwater habitats over a broad geographic range. These salmon spawn, emerge, and rear in tributary streams 800–1,540 km inland from the Pacific Ocean at elevations ranging from 300 to 2,000 m. Because yearling smolts passively migrate to the estuary during the spring freshet, water velocity determines their arrival time at the estuary. Numerous morphological, physiological, and behavioral changes (e.g., change in coloration, increased saltwater tolerance, and becoming semipelagic) are associated with smoltification such that, as the smolt migration progresses, the smolts are no longer adapted to remain in freshwater (Ebel 1977; Thorpe et al. 1981). The salmon spend several years in the ocean, covering thousands of kilometers in their ocean migration, before returning to freshwater

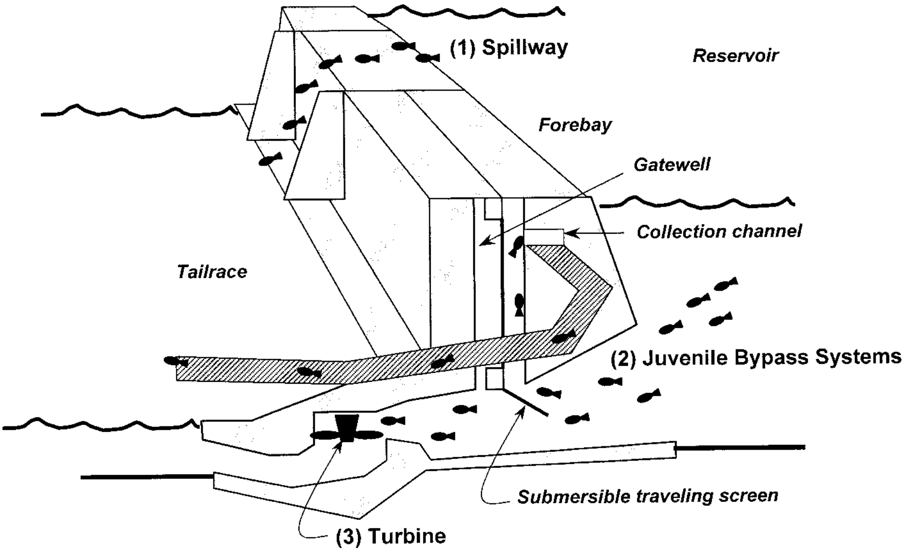

FIGURE 2.—Cross-sectional view of possible routes for juvenile fish past the dams: (1) over the spillway; (2) through the juvenile fish bypass system; and (3) through the turbine. Some fish go into the juvenile bypass system and collection channel and are either returned to the river below the dam or transported past the dams by barge and truck.

and migrating to their natal tributaries to spawn. Each of these life history stages poses natural challenges for survival. Anthropogenic conditions may increase stress and perhaps contribute to decreased survival at each life history stage. Our focus is on the smolt migration and the effects of altered conditions and stresses imposed by the hydrosystem.

The Impounded River

In the Columbia River Basin, main-stem dam construction began in 1938 with the completion of Bonneville Dam near the mouth of the Columbia River. All seven dams on the lower Columbia and Snake rivers had been completed by the time Lower Granite Dam, the eighth and uppermost dam on the lower Snake River, was completed in 1975 (Figure 1). Juvenile fish attempting to migrate to the ocean and adult fish migrating back to their natal Snake River freshwater tributaries to spawn must now pass through eight large hydroelectric dams and 522 km of slack water (Figure 1). The juveniles have four different possible routes past the dams: (1) over the spillway; (2) through the juvenile bypass system, with routing back to the river; (3) into the juvenile bypass system and being collected for transport in barges or trucks; or (4) through the turbines (Figure 2). Snake River fish may be collected for transportation at three of the four dams on the Snake River (Lower Granite, Little Goose, and Lower Monumental) and at McNary Dam on the Columbia River. Transported fish are then released below Bonneville Dam.

Radio-tagging studies indicate that migration rates decrease significantly as smolts approach a dam and a considerable proportion of fish are delayed in the forebay for a week or more (Venditti et al. 2000). Venditti et al. (2000) also believed that increased forebay crossings and upstream excursions of fish (delay) were associated with reduced water velocity. Fish that pass through turbines are exposed to extreme pressure changes and mechanical injuries while going through the turbines (Long et al. 1968; Mathur et al. 1996; Navarro et al. 1996; Bickford and Skalski 2000). Some smolts are guided into a collection and bypass system by intake screens. From the intake screens, the guided smolts are returned in 3 s back to the surface (about 20 m) into a turbulent gatewell (Figure 2). Smolts go from 1 to 3 and back to 1 atmospheric pressures in about 10 s. At Lower Granite Dam (S. Pettit, Idaho Department of Fish and Game, personal communication), they are then piped from the fish collection area nearly 0.4 km to below the dam at high velocity (9 m/s) and pressured through two 90° turns, experiencing high turbulence and rapid deceleration at the end. Smolts are passed through a device to separate fish by size, dewatered, and then passed through a tube that leads to a PIT-tag detector. Remember, this account describes the passage through only one of eight dams. Typically, for transportation effectiveness evaluations, smolts are then held in raceways for 24 h, transferred to a sample room, anesthetized, marked, returned to the raceways for recovery, and then barged, trucked, or flushed through a 20-cm-diameter pipe for release to the river. In recent bypass and collection operations at the dams (since 1994), bypassed smolts may not be held in the raceway but still experience the same stressors up to the point of diversion. In summary, several problems are associated with passage through the dams by way of collection, bypass, and turbines: (1) delay of smolts in the forebay; (2) concentration of smolts in the forebay and in the tailrace at the bypass outflow; (3) high predation rates of smolts near the dams, where predators tend to congregate; 4) large pressure changes; and (5) mechanical injury.

The fish that pass dams through the spillways face different conditions: (1) They appear not to be delayed as long as fish that pass by the other routes, (2) they avoid the mechanical injury and dewatering of bypass and the severe pressure changes of the other routes, and (3) predators are more dispersed in the tailwater of the spillway (Faler et al. 1988). Therefore, passing a dam by the spillway has been hypothesized to be the least stressful route and cause less mortality than passing through the turbines (although gas supersaturation may present problems during uncontrolled spills in high-flow years). In low-flow years, most or all of the water is diverted for power production, forcing the majority of fish into the collection system (for transport or bypass) or through the turbines. In high-flow years, a larger proportion of water will go over the spillway, such that a greater proportion of fish will pass the dam by a possibly less stressful route. Passage past a dam claims the lives of as much as 10% of the smolt population (NMFS 2000), and the remaining population has probably been stressed by the experience.

Reservoirs behind the dams may also create stressful conditions for smolts. Water velocity is greatly reduced by accumulating behind the dams, and thus the time and energy the fish expend to get through the reservoirs are greater than that experienced in the free-flowing conditions for which these fish evolved (Williams and Mathews 1995). The increased freshwater residence time of in-river migrants may result in untimely physiological changes for survival in saltwater that are less suitable for the freshwater environment (Conte and Wagner 1965; Wagner 1974; Woo et al. 1978). The decrease in water velocity also results in an increase in the residence time and temperature of the water, further stressing fish (Raymond 1979). In addition, reservoir environments are better suited for certain predators, which can have a direct and indirect effect on smolts (Ruggerone 1986; Beamsderfer and Rieman 1991; Poe et al. 1991; Rieman et al. 1991; Vigg et al. 1991; Schreck and Stahl 1998; Collis et al. 1999). As much as 20% of the smolt population can be lost during the migration through a reservoir (NMFS 2000). Similarly, hydrosystem operation and its effects on the estuary and near-shore environment may affect salmonid survival by altering the annual flow cycle and associated ecological (e.g., increased smolt residence time) and environmental (e.g., reduced productivity) changes (Sherwood et al. 1990; Pearcy 1992). This migration experience through an impounded river presents the potential for an accumulation of stress events, which may lead to delayed mortality.

Evidence from the Literature: A Focus on Impacts of Stress

Impacts of Stress

Stress and its impacts on fish and fish populations have been widely studied and documented. For further discussions of the general impacts of stress on fish, see major reviews by Wedemeyer et al. (1990) and Fagerlund et al. (1995). Much of the focus on stress in Pacific salmon in the available literature describes acute factors or events such as toxicology (Meyers and Hendricks 1982; Cairns et al. 1984; Larsson et al. 1985) or obstacles to migration (Bentley and Raymond 1976; Fagerlund et al. 1995). However, many of these studies provide a narrow perspective on stress and tolerance in the context of delayed or latent mortality; they do not consider sublethal and chronic stress. In this section, we focus on evidence and potential pathways for the impacts of the hydrosystem on stress, the accumulation of stress, and delayed mortality associated with stress.

Fish migrating through a reservoir and past a dam have three possible experiences: (1) They migrate successfully past the dam and experience no negative impacts from passage, (2) they die immediately from mechanisms discussed above, or (3) they are physiologically or behaviorally stressed (Figure 3). This set of outcomes is possible for each of the eight hydroelectric dams that a Snake River fish must negotiate. Acute stress is generally a high-intensity discrete event (e.g., bypass system at the dam), whereas chronic stress is

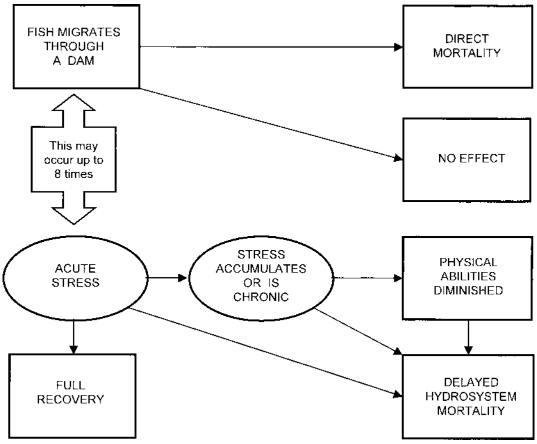

FIGURE 3.—Flow diagram of mortality and survival options as a juvenile fish migrates past a dam for as many as eight times. As a fish passes a dam, it either dies, experiences no effect, or encounters stress. If stressed, the fish may fully recover or the stress may accumulate or have chronic health effects on the fish, leading to decreased physical abilities. Acute stress can lead directly to delayed mortality, or diminished abilities may lead to death through mechanisms of delayed mortality.

either an accumulation of acute events or a longer term, low-intensity experience (e.g., repeated exposure to high temperatures). A fish may recover from acute or chronic stressors, eventually die from the stressor, or experience diminished physical abilities, which may increase vulnerability to sources of mortality. Here we describe the combination of the effects of stress and its associated mortality as delayed mortality. Although delayed mortality can take place within the hydrosystem, for our discussion this portion of mortality is included in direct hydrosystem mortality estimates and not included in our definition of delayed hydrosystem mortality. Studies linking stress and mortality that happens within the hydrosystem, however, provide evidence that delayed mortality can happen outside the hydrosystem.

Measuring Stress

Stressful events related to the hydrosystem can be categorized as follows: (1) dam passage, (2) migration conditions, and (3) collection and transportation around the hydrosystem. Visible external signs of injury, trauma, and disease in fish are observed and recorded regularly at each dam collection facility (e.g., descaling, hemorrhaging on fins, curved spine, fungal infections, and visible gas bubble trauma; Williams and Mathews 1995; Hetherman et al. 1998; FPC 2000; Mesa et al. 2000). Other studies, evaluating nonvisual signs such as swimming performance, growth, and blood chemistry, also suggest that smolts are stressed by these events (Dawley and Ebel 1975; Dawley et al. 1975; Wright and McLean 1985; Krise et al. 1990). However, these latter signs of stress are not routinely measured and are probably underestimated in terms of the total effect of these passage experiences.

Although several experiences are theoretically stressful, determining whether these events are actually stressful can be difficult. Sometimes stress effects are visually observable, but physiological and biochemical tests generally are needed to detect stress in fish. Despite the great strides in measuring stress from these tests, however, interpretation of their significance remains difficult (Kruzynski and Birtwell 1994). This is due, in part, to the problem of obtaining background indicator values: Either reference fish have already experienced a stressor or the capture event itself was stressful (Congelton et al. 2000). For example, Harman et al. (1980) demonstrated that stress brought on by the capture process was, if not immediately fatal, a major contribution to delayed mortality in freshwater drum *Aplodinotus grunniens*. Documenting stress may also be difficult because single indicators (e.g., cortisol levels) or challenge tests used to measure stress may not demonstrate the full effect of stress (Wood et al. 1983; Wedemeyer et al. 1990). The use of multiple indicators may ameliorate this problem but may also result in equivocal results, given that multiple indicators are often conflicting (McLeay and Brown 1979). Finally, some responses may be initially beneficial and so interpreted (Maule et al. 1989); if a stress becomes chronic, however, the responses may be maladaptive and their impact may be expressed outside the time frame of the evaluation (Wedemeyer et al. 1990). Because many of these problems produce only partial evidence of stress, the prevalence of stress in fish populations may be underestimated.

Measuring the magnitude of stress experienced by a fish is difficult enough; relating the level of mortality brought on by stress may be even more problematic. Even if stress is observed in a fish, this stress may not be ecologically significant (Sprague 1971). Indeed, stressed individuals can often recover fully (Mesa et al. 1994). Conversely, individuals that no longer show signs of stress through specific indicators may still die from the cumulative effects of stress (Wood et al. 1983;

Wedemeyer et al. 1990). Moreover, death from these cumulative effects may not take place for some time after the stress events. Jensen et al. (1980) suggested that for half of the population of juvenile salmonids to die from the stress of exposure to increased total gas pressures may take as long as 50 d. Such time frames for stress-induced mortality would often put smolts past the hydrosystem, thus making it difficult to observe and link hydrosystem-related stress to mortality.

Physiological stress experiments are often performed in some type of holding facility (e.g., challenge tests) and evaluate an indicator of stress (e.g., cortisol levels) from a single stressor (e.g., high temperatures) or the interaction between the stress event and another source of mortality (e.g., vulnerability to predation under high temperature; Wedemeyer et al. 1990). However, these experiments generally do not capture additional stresses that an organism may encounter in the wild, nor do they evaluate the vulnerability of stressed individuals to multiple sources of mortality (Barton et al. 1986). Although these experiments do provide insight into the effects of stress, they probably do not capture the total effects of stress on delayed mortality of individuals (Vaughan et al. 1984; Adams et al. 1985; Barton et al. 1986). In addition, the laboratory conditions and experiments themselves may cause stress. These potential limitations of stress experiments done in holding facilities may need to be considered, given that NMFS has proposed to estimate the degree of delayed mortality associated with different routes of dam passage by holding fish collected at Bonneville Dam in tanks (USACE 2000). The salmon in these experiments would not be subject to the additive or multiplicative effects of all mortality agents experienced in the wild (e.g., piscine and avian predators, diseases, parasites, foraging, and salt-water transitions). Although stress has been demonstrated to lead to mortality eventually, the total effect of stress has probably been underestimated because of the complex interaction between one or more stressors and multiple interactions with other sources of mortality.

Stress and Energetic Condition

Stress can cause metabolic disturbances and decreased energetic condition (Leach and Taylor 1980). For example, if an extended migration through reservoirs increases metabolic requirements by more than the energy intake, a smolt's energy reserves would be depleted, which might eventually lead to lower survival rates (Rondorf et al. 1985). Also, physical disturbances (acute stressors) have been demonstrated to increase the metabolic rate of fish (Barton and Schreck 1987). Cumulative increases in energy demand in response to repeated disturbances may result in depleted energy reserves (Mazeaud et al. 1977; Barton et al. 1986). Decreased energy reserves may lead to decreased swimming performance, disease resistance, foraging ability, growth, reproductive success, predator avoidance, and ultimately survival (Wedemeyer et al. 1990).

Stress and Predation

The concept of increased vulnerability to predators as a result of acute or chronic stress is ubiquitous in ecology (Errington 1946; Peterson 1977; Rice et al. 1987). Many examples can be found for fish (Coutant 1973; Sigismondi and Weber 1988; Kruzynski and Birtwell 1994; Mesa et al. 1994; Mesa and Warren 1997), specifically increased vulnerability to predation because of hydrosystem-induced stress (Rieman et al. 1991; Mesa 1994; Schreck and Stahl 1998). Mesa (1994) found that the more stress events experienced by a smolt, the greater its vulnerability to predation by northern pikeminnow *Ptychocheilus oregonensis*, the most abundant piscivore in the lower Columbia River (Rieman et al. 1991). In addition, Schreck and Stahl (1998) followed radio-tagged smolts released from barges below Bonneville Dam and found that stressed smolts avoided saltwater entry by remaining on the floating freshwater lens at the saltwater–freshwater interface. That forced these smolts to the surface, where they were subject to predation by avian predators. Given the greater densities of piscine and avian predators below Bonneville Dam (Ward et al. 1995; Roby et al. 1998), the interaction between predation and stress is probably a mechanism involved in hydrosystem-related delayed mortality.

Stress and Disease

Various types of environmental stress can also affect fish health and condition by lowering resistance to disease (Wedemeyer 1970). Stress may decrease mucus production (Mazeaud et al. 1977) and impair immune function (Morgan et al. 1996), which may increase vulnerability to disease. Raymond (1988) found that during downriver migration, transportation and seawater transition can cause stress and activate bacterial kidney disease. For example, fish exposed to dissolved gas levels of 100% or more have shown increased susceptibility to disease and fungal infections (Weitkamp and Katz 1980; White et al. 1991).

Stress and Physiology

Many investigators have concluded that salmonids undergo physiological changes, such as changes in osmoregulatory capacity, to successfully enter saltwater (Zaugg and Wagner 1973; Wagner 1974; Zaugg 1981; Zaugg et al. 1985; Rodgers et al. 1987). The proper timing of this physiological change has been referred to as the ‘‘biological window’’ of successful saltwater entry (Walters et al. 1978; Bilton et al. 1982; Boeuf and Harache 1982; Holtby et al. 1989). The hydrosystem has prolonged the migration time for Snake River salmon populations by decreasing water velocity and delaying fish at dams (Bentley and Raymond 1976). Extended freshwater residency is associated with reversion to parrlike characteristics due to a lower salinity tolerance in some species of salmon (Conte and Wagner 1965; Wagner 1974; Woo et al. 1978). In contrast, transport of fish in trucks and barges greatly shortens the time it takes fish to travel through the hydrosystem relative to historical free-flowing conditions and may result in premature saltwater entry. The effects of premature saltwater entry (e.g., incomplete smoltification) can result in high mortality and, in many of the survivors, a reduction in or cessation of growth (Fagerlund et al. 1995).

Accumulation of Stress

Although stresses are generally measured as single-point–specific events, stress may accumulate in fish and these additive effects may cause mortality at a later stage. Kruzynski and Birtwell (1994) have referred to mortality resulting from exposure to a legacy of physiologically sublethal stressors as an ‘‘ecological death.’’ Barton et al. (1986) observed an accumulation of stress when juvenile chinook salmon subjected to repeated handling exhibited more stress indicators (e.g., increases in plasma lactate, sodium, potassium, anaerobic metabolism, or ionoregulatory performance) than after a single disturbance. In fact, the accumulation of stress resulting in ‘‘sudden death syndrome,’’ induced by multiple handling, has been documented for rainbow trout (Lanno and Dixon 1996). The cumulative effects of multiple stress events on fish populations has been suggested to cause the decline or collapse of fish populations, even though the effects of a single stress may be insignificant (Vaughan et al. 1984; Adams et al. 1985; Wedemeyer et al. 1990; Korman et al. 1994).

The effects of cumulative stress have been observed in smolts as they migrate through the Snake and Columbia River hydrosystem. Mathews et al. (1986) documented an increase in stress levels as chinook salmon smolts moved through the collection and transport system at Lower Granite Dam. Similarly, evidence exists for cumulative effects of increased temperature and dissolved gas concentrations in the hydrosystem on smolt condition and ultimately on survival. For example, Coutant (1999) suggested that the cause of a massive fish kill at McNary Dam was the result of cumulative exposure to high-temperature water over several days. Ebel et al. (1975) reviewed several laboratory and in situ studies and concluded that a substantial number of smolts die as a result of chronic versus acute exposure to high concentrations of dissolved gas. Given that the accumulation of stress and often the resulting mortality have been observed in the hydrosystem where these studies are feasible, one can reasonably expect that this stress may not result in mortality until after smolts leave the hydrosystem and thus would be considered delayed hydrosystem mortality.

Indirect Evidence for Delayed Hydrosystem Mortality: Retrospective Analyses and Downstream Stock Comparisons

Retrospective comparisons of stock performance among regions and time periods provide indirect evidence of delayed mortality linked to hydrosystem experience (Deriso et al. 1996; Schaller et al. 1999). The comparisons are based on spawner and recruit (offspring of spawners) data describing the overall survival (or mortality) over the life cycle of spring and summer chinook salmon for brood years 1957–1990 (Figure 4). For the comparisons, life cycle mortality was separated into two components. The first component, direct mortality (25–73%) takes place while juvenile and adult salmon pass by dams and travel through reservoirs that constitute the hydrosystem and are transported around the hydrosystem by barge or truck (discussed in more detail below; Bouwes et al. 1999). The second component of mortality is what occurs in the Columbia River downstream from Bonneville Dam, in the estuary, and in the ocean. After accounting for direct hydrosystem mortality, natural spawner and recruit processes (e.g., density-dependent mortality), and environmental or ocean effects, the additional mortality that Snake River fish experience may be 37–68%

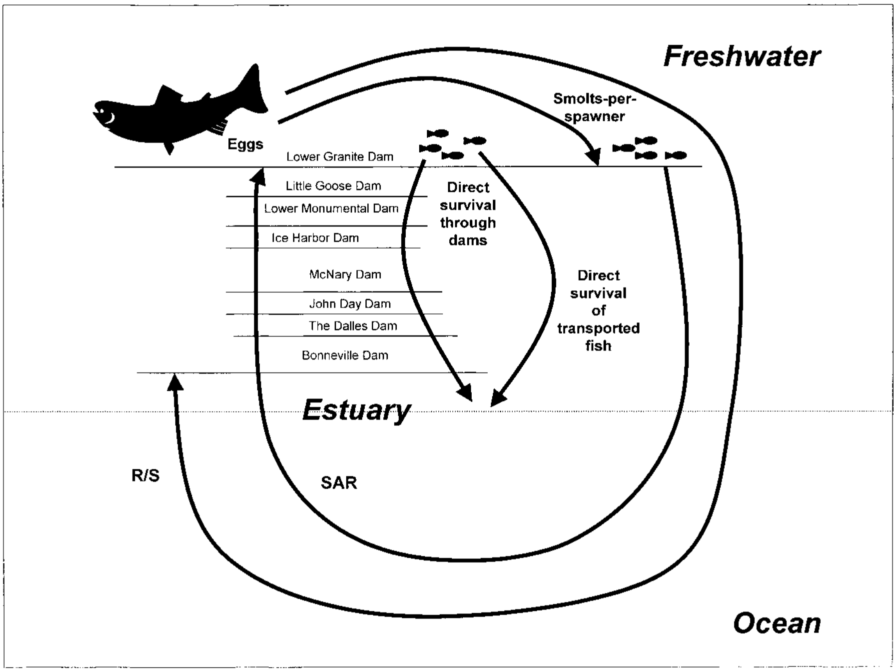

FIGURE 4.—Schematic life cycle diagram for Snake River salmon and steelhead showing passage past the dams and the different indices of survival across different life stages. Smolts/spawner provide an estimate of freshwater and rearing spawning survival. The survival rate of smolts to returning adults (smolt-to-adult), or SAR, is measured at Lower Granite Dam; R/S is recruits per spawner, measured by back-calculating the number of recruits (progeny) from the number of spawners (parents) based on redd counts, age structure, and harvest. Direct and overall survival of fish that migrated through the hydrosystem and in barges is based on PIT tag mark–recapture experiments.

(Deriso et al. 1996). Two types of comparisons yield information on the degree or source of this additional mortality for Snake River stocks of spring and summer chinook salmon. We discuss comparisons of overall survival rates for Snake River stocks before and after the development of the hydrosystem and of stocks originating from different regions with different migration experiences but similar life histories.

Comparison of historical and current empirical life stage survival estimates provide evidence that changes in delayed mortality have taken place in Snake River spring and summer chinook salmon coincident with the completion of the hydrosystem (Schaller et al. 1999). Retrospective comparisons of stocks originating from different regions demonstrate that completion of the hydrosystem in the late 1960s through the mid-1970s was immediately followed by considerably sharper declines in overall survival rates of Snake River stocks than of downstream stocks over the same period (Schaller et al. 1999). The sharper decline in overall survival observed for the upstream stocks probably reflected the increasing amounts of direct and hydrosystem-related delayed mortality (Schaller et al. 1999). These declines continued despite reduction or elimination of freshwater harvest and decreases in direct mortality from transportation. In addition, spring and summer chinook salmon stocks experience little ocean harvest (Berkson 1991). Most degradation of Snake River spawning and rearing habitats occurred before the start of stock declines, and populations in degraded and good habitat responded similarly (Schaller et al. 1999; Petrosky et al. 2001). Furthermore, no climatic index was found that could explain why Snake River stock declines were greater than their downstream counterparts (Marmorek et al. 1996). Evidence suggests, however, that each stock group experienced common-year effects, presumably from climatic conditions experienced by both (Deriso et al. 1996). Moreover, although there was some correspondence between overall hatchery production levels and declines in Snake River spring chinook salmon, overall hatchery production showed little to no correspondence to declines in the downstream counterparts. Similarly, little to no correspondence was seen between hatchery production and population decreases in individual subbasins (Wilson et al. 1996; Budy et al. 1998; Williams et al. 1998). Therefore, the lack of support for other hypotheses indicates that the hydrosystem is probably partially responsible for the greater mortality that was observed in the Snake River stocks, relative to their downstream counterparts, after the completion of the hydrosystem.

The potential for stressful migration conditions is high under low-flow conditions because a majority of fish will experience increased delays at dams (given that a majority of fish pass dams through turbines and bypass systems) and longer travel times through reservoirs. In years with high flows, fish experience fewer delays at dams (more fish pass over spillways) and shorter travel times through reservoirs, which would reduce stressful conditions. Columbia River salmon stocks generally respond favorably to good water years with high runoff during smolt migration, but the response is relatively greater for upriver stocks than for downriver stocks (Deriso et al. 1996). This supports the notion that poor migration conditions caused by the additional mainstem dams are the primary cause of delayed mortality of upriver stocks relative to their downriver counterparts. In fact, in recent years, the returns of wild Snake River spring and summer chinook salmon have increased coincident with the relatively high flow and spill (the route of passage thought to be the least stressful) during their downstream migration.

The overall survival estimated from spawner and recruits described above can be further divided into an egg-to-smolt survival rate (smolts per spawner) and a smolt-to-adult survival rate (SAR; Figure 4). Empirical estimates for these two indices of survival from the period before three of the four Snake River dams were in place (1975) indicate that the number of smolts per spawner has not changed in a pattern consistent with the steep decline in SAR for the Snake River stocks (Petrosky et al. 2001). The decline in survival overall cannot be attributed to changes in freshwater spawning and rearing survival (e.g., habitat degradation) and instead may be explained in part by the combination of direct and delayed mortality during downstream migration. This result is not surprising, given that several of these populations spawn and rear in wilderness areas that have remained relatively pristine since before the completion of the Snake River hydrosystem (Figure 1). Alternative explanations for increased mortality have emphasized overfishing, degradation of freshwater and marine habitats, interaction between hatchery fish and wild fish, and increases in exotic predators (Schaller et al. 2000; Zabel and Williams 2000). The evidence discussed above suggests that delayed effects of the hydrosystem are likely to be partially responsible for overall declines in survival.

Direct Evidence: Relationship to Hydrosystem Experience

Comparisons of overall survival of different groups of fish that experience different combinations of passage routes (Figure 2) and transportation around the dams provide direct evidence of delayed hydrosystem mortality in relation to hydrosystem experience. In 1994–1996, NMFS expanded their studies on transportation and in-river survival of Snake River spring and summer chinook salmon by using PIT-tagged fish, thus estimating rates of survival by route of passage around each dam (e.g., bypass or transport; Figure 4; NMFS 1999; Sandford and Smith, in press). The tagged smolts are not detected if they pass through turbines or over spillways. When the adults return, the detectors record their successful passage past the uppermost dam on the Snake River, Lower Granite Dam (Figures 1 and 4). This detection record allows estimation of the overall SAR of various groups of fish that experience different passage routes throughout the Snake and Columbia rivers (Bouwes et al. 1999; NMFS 1999; Sandford and Smith, in press).

For fish that are not transported and migrate in-river, evidence for delayed hydrosystem mortality in relation to hydrosystem experience can be evaluated by comparing the overall SARs of fish that were not collected and bypassed around the dams with those fish that were collected and bypassed one or more times. Spill over the top of a dam is the route of passage most similar to a natural river, whereas collection and bypass have a much greater potential of stressing the fish (discussed above). However, direct mortality is generally estimated to be similar between the spill and bypass routes, both of which have lower rates than the turbine route (Marmorek and Peters 1998). We would expect direct survival rates for collected and bypassed fish to be generally higher because a portion of the fish that are not collected go through the turbines. Therefore, the more times fish go through the bypass system, the lower their direct mortality attributable to the hydrosystem and thus a higher expected smolt-to-adult return rate (in the absence of delayed mortality).

The apparent direct survival benefits of the by-

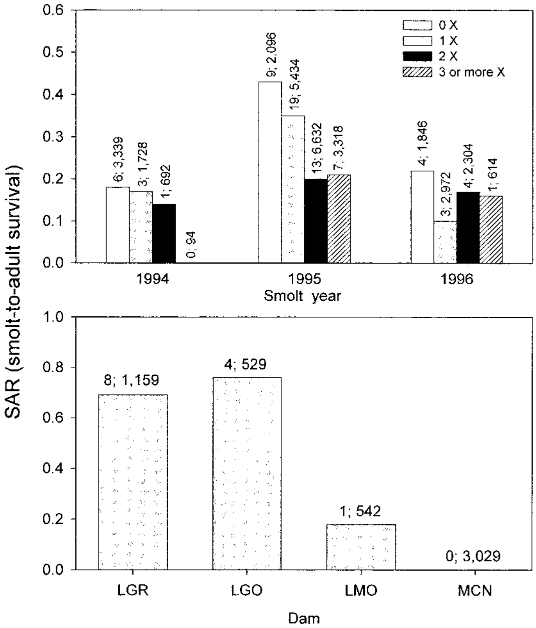

FIGURE 5.—The top panel shows estimated SAR of wild spring and summer chinook salmon by in-river passage history: nondetected (0×), and bypassed one, two, and three or more times (1×, 2×, 3 or more ×, respectively), 1994–1996. The bottom panel shows estimated SAR of wild spring and summer chinook salmon for fish that were transported from Lower Granite (LGR), Little Goose (LGO), Lower Monumental (LMO), and McNary (MCN) dams, respectively. Data are shown for 1994, the only year with sufficient sample sizes for analysis.

pass route of passage, however, do not translate well into SARs of wild spring and summer chinook salmon. The SARs of uncollected fish were significantly higher than those of the bypassed group in 1995–1996 and may have been higher, or no different, in 1994 (R. Kiefer, Idaho Department of Fish and Game, personal communication). Fish in the uncollected group used a combination of spill and turbine routes and have a lower expected direct survival than those in the bypassed group (bypassed from one to four times). In addition, the uncollected group returned at rates as good as or better than those for the transported group in 1995 and 1996, but in 1994 the transported smolts may have had the higher return rates. Similarly, estimates of SARs for in-river migrating smolts were examined each year for groups that were collected and bypassed 0–3 times or more through the four collector dams (Bouwes et al. 1999). These PIT-tag data indicate that SARs decreased when the number of times fish were bypassed increased (Figure 5; NMFS 1999; Sandford and Smith, in press). This same general pattern is also observed for steelhead (Sandford and Smith, in press). These data further indicate that although direct mortality may be lowest for fish that are bypassed, some delayed mortality related to the hydrosystem may explain the patterns of overall survival. These results explain only the difference in delayed mortality between different routes of passage rather than the total effect of dam passage on delayed mortality.

Furthermore, when fish are transported from lower dams (and avoid the bypass systems in the Snake River), the overall SAR appears to decrease as the number of navigated hydrosystem dams increases (to the point of transport). The SARs were greater for smolts transported from the upper two dams (Lower Granite and Little Goose) than for those transported from the lower dams (Lower Monumental and McNary) in 1994, the only year with an adequate sample size (Figure 5; Bouwes et al. 1999). This pattern, in part, provides evidence of delayed hydrosystem mortality for nonbypassed smolts in that all groups were collected only once (for transportation). If we assume that survival in the barge is always high, this pattern is probably the result of the increasing stress of multiple project passage before transportation.

In addition to comparing the overall survival of fish that migrate by different routes in-river, the SAR of transported fish, relative to in-river fish, provides evidence of hydrosystem-related delayed mortality. The direct mortality of fish that are transported through the hydrosystem appears to be very low (about 2%), whereas the direct mortality of the fish that migrate in-river is generally greater than 50%. Therefore, if no additional delayed mortality resulted from being collected and transported, we would expect the transported fish to return as adults at a rate nearly twice that of the in-river fish (given almost all transported fish survived through the hydrosystem and half of the in-river migrants died while migrating). A posthydrosystem SAR can be estimated after accounting for this estimated direct mortality. The ratio of transport to in-river posthydrosystem SARs is termed *D*. If the posthydrosystem SARs of transported and in-river fish were the same, *D* would be equal to one. A ratio of less than one means that the transported fish die at a higher rate in the estuary and ocean than do the in-river migrants.

The degree of the delayed mortality of transported fish relative to fish that migrate in-river (*D*) is controversial. For Snake River spring and summer chinook salmon, estimates of *D* from two

PATH-developed models were 0.48 and 0.66 (Marmorek et al. 1998). When estimates were made directly from the PIT-tag data, estimates of *D* for spring and summer chinook salmon ranged from 0.52 (1994–1998; Budy 2001) to 0.63–0.73 (1994–1997; NMFS 2000). NMFS used similar methods and assumptions to estimate *D* for Snake River steelhead and obtained values ranging from 0.52 to 0.58 (NMFS 2000). PIT-tag estimates for fall chinook salmon are not as reliable, but the recent estimate of *D* is approximately 0.24, with very wide confidence intervals (Marmorek and Peters 1998; NMFS 2000). Despite some disagreement about the true *D* values, all methods indicate that *D* is less than 1.0, which is evidence of delayed mortality of transported fish. Although *D* describes the difference in delayed mortality between transported fish and fish that migrate in-river, the absolute amount of delayed mortality for both groups may plausibly be substantially greater.

The differences in hydrosystem experience may help explain why delayed mortality is greater for transported fish than for nontransported fish. The stressful experience of being delayed in the forebay, collected, and bypassed is most relevant to transported fish at collection dams because most of the fish entering the collection and bypass system at these dams are subsequently transported. In contrast, in-river smolts may be bypassed, go through turbines, or over the spillway. In addition, transported smolts are subjected to the stress of crowding and injury during collection, holding, and transport (e.g., each liter of water would hold up to approximately the equivalent of one wild chinook salmon and one wild steelhead; Congelton et al. 2000). These high loading densities, in combination with the stressed state of the fish, provide an effective environment for disease transmission or manifestation (IDFG 1998). Also, transported smolts may be delivered to the estuary without regard for their physiological readiness to enter saltwater (Fagerlund et al. 1995). Because fall chinook salmon are still rearing while migrating, the difference in the time required to be transported (approximately 1–2 d) versus migrating in-river (up to 6 weeks) is far greater than for spring and summer chinook salmon. Thus premature saltwater entry for fall chinook may explain why *D* is much lower for fall chinook salmon than for spring and summer chinook salmon. All these factors may be responsible for the greater delayed mortality experienced by transported fish.

As discussed above, the hypothesis of delayed mortality resulting from hydrosystem passage has an empirical basis as well as biological rationale. Recent PIT-tag data are direct evidence that delayed mortality of both in-river and transported smolts is related to the hydropower system. More specifically, the evidence suggests that, at least for collected and bypassed smolts, there is a difference between the patterns of direct passage survival rates and SARs. These findings provide direct evidence that the delayed mortality of Snake River spring and summer chinook salmon is related to the juvenile migration hydrosystem experience.

Conclusion

Substantial evidence supports the existence of delayed mortality for Snake River chinook salmon and steelhead and links delayed mortality to hydrosystem experience. This evidence comes in the form of published literature on causes of stress and any resulting delayed mortality, indirect evidence from life cycle modeling based on historical data and known mechanisms, and direct evidence from fish-tagging experiments. The literature supplies numerous mechanisms that explain how the observed stressors of the hydrosystem could be cumulative and eventually lead to mortality at a later life stage. The literature also demonstrates that the effects of stress can be hard to detect and difficult to link empirically to delayed mortality in field experiments. Nevertheless, there is little debate that fish condition and stress affect overall survival to adulthood or that the hydrosystem causes direct mortality and imposes stress on those fish that survive. Similarly, the retrospective life cycle analysis provides indirect evidence of increases in delayed mortality in Snake River spring and summer chinook salmon coincident with completion of the Snake River hydrosystem. The decreases in survival rates of Snake River fish were considerably sharper than those of downriver stocks over the same period. Further, most decreases in survival rate were in the smolt-to-adult life stage, rather than the spawner-to-smolt life stage. Data from PIT tags provide direct evidence that delayed mortality of both in-river–migrating and transported smolts was related to the hydropower system. The patterns of SAR could not be explained on the basis of estimates of direct survival within the hydrosystem but instead must result from delayed mortality related to specific hydrosystem experience.

This evaluation has implications for the analyses of management options for the recovery of Snake River salmon and steelhead. In those analyses, dam breaching appears to increase the survival of

Snake River chinook salmon and may perhaps lead to eventual recovery of these stocks if delayed mortality is related to hydrosystem experience. Dam breaching is predicted to have less of an effect and be insufficient for recovery if delayed mortality is unrelated to hydrosystem experience. Regardless of the action chosen to recover these stocks, the amount of hydrosystem mortality to compensate for must include both direct and delayed components. Given the evidence discussed here, we find significant support for the hypothesis that Snake River fish are subject to delayed mortality and that this effect is related to the hydrosystem.

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References

Adams, S. M., J. E. Breck, and R. B. McLean. 1985. Cumulative stress-induced mortality of gizzard shad in southeastern U.S. reservoir. Environmental Biology of Fishes 13:103–112.

American Fisheries Society. 1999a. Breaching dams is best alternative to recover salmon, says report. Fisheries 24(2):37.

American Fisheries Society. 1999b. Western division: dam removal resolution passed. Fisheries 24(10):28.

Barton. B. A., and C. A. Schreck. 1987. Metabolic cost of acute physical stress in juvenile steelhead. Transactions of the American Fisheries Society 116:257–263.

Barton. B. A., C. A. Schreck, and L. A. Sigismondi. 1986. Multiple acute disturbances evoke cumulative physiological stress responses in juvenile chinook salmon. Transactions of the American Fisheries Society 115:245–251.

Beamsderfer, R. C., and B. E. Rieman. 1991. Abundance and distribution of northern squawfish, walleyes, and smallmouth bass in John Day Reservoir, Columbia River. Transactions of the American Fisheries Society 120:439–447.

Bentley, W. W., and H. L. Raymond. 1976. Delayed migrations of yearling chinook salmon since the completion of Lower Monumental and Little Goose dams on the Snake River. Transactions of the American Fisheries Society 105:422–424.

Berkson, J. 1991. Coded wire tag analysis on Snake River spring, summer, and fall chinook. Letter dated February 19, 1991 to the U.S. National Marine Fisheries Service for Endangered Species Act record, Columbia River Inter-Tribal Fisheries Commission, Portland, Oregon.

Bickford, S. A., and J. R. Skalski. 2000. Reanalysis, and interpretation of 25 years of Snake-Columbia River juvenile salmonid survival studies. North American Journal of Fisheries Management 20:53–68.

Bilton, H. T., D. F. Alderdice, and J. T. Schnute. 1982. Influence of time and size at release of juvenile coho salmon (*Oncorhynchus kitsutch*) on returns at maturity. Canadian Journal of Fisheries and Aquatic Sciences 39:426–447.

Boeuf, G., and Y. Harache. 1982. Criteria for adaptations of salmonids to high salinity seawater in France. Aquaculture 28:163–176.

Bouwes, N., H. Schaller, P. Budy, C. Petrosky, R. Kiefer, P. Wilson, O. Langness, E. Weber, and E. Tinus. 1999. An analysis of differential delayed mortality experienced by stream-type chinook salmon of the Snake River. Oregon Department of Fisheries and Wildlife, Technical Report, Portland.

Brodeur, R. D., G. W. Boehlert, E. Casillas, M. B. Eldridge, J. H. Helle, W. T. Peterson, W. R. Heard, S. T. Lindley, and M. H. Schiewe. 2000. A coordinated research plan for estuaries, and ocean research on Pacific salmon. Fisheries 25(6):7–16.

Budy, P. 2001. Analytical approaches to assessing recovery options for Snake River chinook salmon. Utah Cooperative Fish and Wildlife Research Unit (UCFWRU) 2001(1):1–86.

Budy, P., C. Petrosky, P. Wilson, O. P. Langness, and N. Bouwes. 1998. Hatchery ‘‘extra mortality’’ hypothesis: response to submissions 1 and 2 of the WOE. Pages 19.1–19.4 *in* D. R. Marmorek and C. N. Peters, editors. PATH submissions to weight of evidence report. ESSA Technologies, Ltd., Vancouver.

Cairns, V. W., P. V. Hodson, and J. D. Nriangu, editors. 1984. Contaminant effects on fisheries. Wiley, New York.

Collis, K., S. Adamany, D. D. Roby, D. P. Craig, and D. E. Lyons. 1999. Avian predation on juvenile salmonids in the lower Columbia River. 1998 Annual Report to Bonneville Power Administration, Project 9702400, Portland, Oregon.

Congelton, J. L., W. J. La Voie, C. B. Schreck, and L. Davis. 2000. Stress indices in migrating juvenile chinook salmon and steelhead of wild and hatchery origin before and after barge transportation. Transactions of the American Fisheries Society 129:946–961.

Conte, F. P., and H. H. Wagner. 1965. Development of osmotic and ionic regulation in juvenile steelhead trout (*Salmo gairdneri*). Comparative Biochemistry and Physiology 14:603–620.

Coutant, C. C. 1973. Effect of thermal shock on vulnerability of juvenile salmonids to predation. Journal of the Fisheries Research Board of Canada 30: 965–973.

Coutant, C. C. 1999. Perspectives on temperature in the Pacific Northwest’s fresh waters. Oak Ridge National Laboratory, Report ORNL/TM-1999/44, Oak Ridge, Tennessee.

Dawley, E., T. Blahm, G. Snyder, and W. Ebel. 1975. Studies on effects of supersaturation of dissolved atmospheric gas on juvenile chinook salmon and steelhead trout. U.S. National Marine Fisheries Service, Northwest Fisheries Center Final report to the Bonneville Power Administration, U.S. Bureau of Reclamation, and U.S. Army Corp of Engineers, Seattle.

Dawley, E. M., and W. J. Ebel. 1975. Effects of various concentrations of dissolved atmospheric gas on juvenile chinook salmon and steelhead trout. U.S. National Marine Fisheries Service Fishery Bulletin 73: 787–796.

Deriso, R., D. Marmorek, and I. Parnell. 1996. Retrospective analysis of passage mortality of spring chinook of the Columbia River. *In* D.R. Marmorek and 21 coauthors, compilers, and editors. Plan for analyzing and testing hypotheses (PATH): final report of retrospective analysis for fiscal year 1996. ESSA Technologies, Ltd., Vancouver.

Ebel, W. J. 1977. Panel 2: fish passage problems and solutions—major passage problems. Pages 33–39 *in* E. Schweibert, editor. Columbia River salmon and steelhead. American Fisheries Society, Special Publication 10, Bethesda, Maryland.

Ebel, W. J., H. L. Raymond, G. E. Monan, W. E. Farr, and G. K Tanoka. 1975. Effects of atmospheric gas saturation caused by dams on salmon and steelhead trout of the Snake and Columbia rivers. U.S. National Marine Fisheries Service, Seattle.

Errington, P. L. 1946. Predation and vertebrate populations. Quarterly Review of Biology 21:144–177.

Fagerlund, U. H. M., J. R. McBride, and I. V. Williams. 1995. Stress and tolerance. Pages 461–503 *in* C. Groot, L. Margolis, and W. C. Clarke, editors. Physiological ecology of Pacific salmon. UBC Press, Vancouver.

Faler, M. P., L. M. Miller, and K. I. Welke. 1988. Effects in variation in flow on distribution of northern squawfish in the Columbia River below McNary Dam. North American Journal of Fisheries Management 8:30–35.

FPC (Fish Passage Center). 2000. Fish passage center smolt monitoring and gas bubble trauma monitoring program protocols. FPC, Portland, Oregon.

Harman, B. J., D. L. Johnson, and L. Greenwald. 1980. Physiological responses of Lake Erie freshwater drum to capture by commercial shore seine. Transactions of the American Fisheries Society 109:544–551.

Hassemer, P. F., S. W. Kiefer, and C. E. Petrosky. 1997. Idaho's salmon: can we count every last one? Pages 113–125 *in* D. G. Stouder, P. A. Bisson, and R. J. Naiman, editors. Pacific salmon and their ecosystems: status and future options. Chapman and Hall, London.

Hetherman, L., and 18 coauthors. 1998. Juvenile fish transportation program. 1997 annual report to the U.S. Army Corps of Engineers, Walla Walla, Washington.

Holtby, L. B., T. E. McMahon, and J. C. Scrivener. 1989. Stream temperatures and inter-annual variability in the emigration timing of coho salmon (*Oncorhynchus kitsutch*) smolts and fry and chum salmon (*O. keta*) fry from Carnation Creek, British Columbia. Canadian Journal of Fisheries and Aquatic Sciences 46:1396–1405.

IDFG (Idaho Department of Fish and Game). 1998. Issue paper: fish health and stress related to transportation. Appendix 3.3 *in* Idaho's anadromous fish stocks: their status and recovery options. IDFG, IDFG 98-13, Boise.

ISG (Independent Scientific Group). 1999. Return to the river: scientific issues in the restoration of salmonid fishes in the Columbia River. Fisheries 24(3):10–19.

Jensen, J. O. T., J. Schnute, and D. F. Alderice. 1980. Assessing juvenile salmonid response to gas supersaturation using a general multivariate dose–response model. Canadian Journal of Fisheries and Aquatic Sciences 43:1694–1709.

Karieva, P., M. Marvier, and M. McClure. 2000. Recovery and management options for spring/summer chinook in the Columbia River basin. Science 290: 977–979.

Korman, J., D. R. Marmorek, G. L. Lacroix, P. G. Amiro, J. A. Ritter, W. D. Watt, R. E. Cutting, and D.C. E. Robinson. 1994. Development and evaluation of a biological model to assess regional-scale effects of acidification on Atlantic salmon (*Salmo salar*). Canadian Journal of Fisheries and Aquatic Sciences 51:662–680.

Krise, W. F., J. W. Meade, and R. A. Smith. 1990. Effect of feeding rate and gas supersaturation on survival and growth of lake trout. Progressive Fish-Culturist 52:45–50.

Kruzynski, G. M., and I. K. Birtwell. 1994. A predation bioassay to quantify the ecological significance of sublethal responses of juvenile chinook salmon (*Oncorhynchus tshawytscha*) to the antisapstain fungicide TCMTB. Canadian Journal of Fisheries and Aquatic Sciences 51:1780–1790.

Lanno, R. P., and D. G. Dixon. 1996. The induction of lethality in rainbow trout exposed to thiocyanite by application of a stressor: acute or chronic response? Canadian Journal of Fisheries and Aquatic Sciences 1774:241–242.

Larsson, A., C. Haux, and M. L. Sjobeck. 1985. Fish physiology and metal pollution: results and experiences from laboratory and field studies. Ecotoxicology and Environmental Safety 9:250–281.

Leach, G. J., and M. H. Taylor. 1980. The role of cortisol in stress-induced metabolic changes in *Fundulus heteroclitus*. General and Comparative Endocrinology 42:219–227.

Long, C. W., R. F. Krcma, and F. J. Ossiander. 1968. Research on fingerling mortality in Kaplan turbines. U.S. National Marine Fisheries Service, Northwest and Alaska Fisheries Center, Seattle.

Marmorek, D. R., and 21 coauthors. 1996. Plan for analyzing and testing hypotheses (PATH): final report on retrospective analyses for fiscal year 1996. ESSA Technologies, Ltd., Vancouver.

Marmorek, D. R., and C. N. Peters, editors. 1998. Plan for analyzing and testing hypotheses (PATH): final report for fiscal year 1998. ESSA Technologies, Ltd., Vancouver.

Mathews, G. M., D. L. Park, S. Achord, and T. E. Ruehle. 1986. Static seawater challenge test to measure relative stress levels in spring chinook salmon smolts. Transactions of the American Fisheries Society 115: 236–244.

Mathur, D., P. G. Heisey, E. T. Euston, J. R. Skalski, and S. Hays. 1996. Turbine passage survival estimation for chinook salmon smolts (*Oncorhynchus tshawytscha*) at a large dam on the Columbia River. Canadian Journal of Fisheries and Aquatic Sciences 53:542–549.

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*). Journal of Endocrinology 120: 135–142.

Mazeaud, M. M., F. Mazeaud, and E. D. Donaldson. 1977. Primary and secondary effects of stress in fish: some new data with a general review. Transactions of the American Fisheries Society 106:201–212.

McLeay, D. J., and D. A. Brown. 1979. Stress and chronic effects of untreated and treated bleached kraft pulpmill effluent on the biochemistry and stamina of juvenile coho salmon (*Oncorhynchus kisutch*). Journal of the Fisheries Research Board of Canada 36:1049–1059.

Mesa, M. G. 1994. Effects of multiple acute stressors on the predator avoidance ability and physiology of juvenile chinook salmon. Transactions of the American Fisheries Society 123:786–793.

Mesa, M. G., T. P. Poe, D. M. Gadomski, and J. H. Petersen. 1994. Are all prey created equal? a review and synthesis of differential predation on prey in substandard condition. Journal of Fish Biology 45: 81–96.

Mesa, M. G., and J. J. Warren. 1997. Predator avoidance ability of juvenile chinook salmon (*Oncorhynchus tshawytscha*) subjected to sublethal exposures of gas-supersaturated water. Canadian Journal of Fisheries and Aquatic Sciences 54:757–764.

Mesa, M. G., L. K. Weiland, and A. G. Maule. 2000. Progression and severity of gas bubble trauma in juvenile salmonids. Transactions of the American Fisheries Society 129:174–185.

Meyers, T. R., and J. D. Hendricks. 1982. A summary of tissue lesions in aquatic animals induced by controlled exposures to environmental contaminants, chemotherapeutic agents, and potential carcinogens. U.S. National Marine Fisheries Service Marine Fisheries Review 44(1):1–17.

Morgan, J. D., S. K. Balfry, M. M. Vijayan, and G. K. Iwama. 1996. Physiological responses to hyposaline exposure and handling and confinement stress in juvenile dolphin (mahimahi: *Coryphaena hippurus*). Canadian Journal of Fisheries and Aquatic Sciences 53:1736–1740.

Myers, J. M., R. G. Kope, G. J. Bryant, D. Teel, L. J. Lierheimer, T. C. Wainwright, W. S. Grand, F. W. Waknitz, K. Neely, S. T. Lindley, and R. S. Waples. 1998. Status review of chinook salmon from Washington, Idaho, Oregon, and California. NOAA Technical Memorandum NMFS-NWFSC-35, Seattle.

Navarro, J. E., D. J. McCauley, and A. R. Blystra. 1996. Turbine passage at four low-head hydroelectric facilities in northeast Michigan. North American Journal of Fisheries Management 16:182–191.

Nemeth, D. J., and R. B. Kiefer. 1999. Snake River spring and summer chinook salmon—the choice for recovery. Fisheries 24(10):16–23.

NMFS (U.S. National Marine Fisheries Service). 1999. An assessment of lower Snake River hydrosystem alternatives on survival and recovery of Snake River salmonids. Anadromous fish appendix *in* Draft lower Snake River juvenile salmon migration feasibility study/environmental impact statement. U.S. Army Corps of Engineers, Walla Walla, Washington.

NMFS (U.S. National Marine Fisheries Service). 2000. Biological opinion of the Federal Columbia River power system including the juvenile fish transportation program and the U.S. Bureau of Reclamation's 31 dams including the entire Columbia Basin project. NMFS, Seattle, Washington.

Pearcy, W. G. 1992. Ocean ecology of North Pacific salmonids. University of Washingon Press, Seattle.

Peterson, R. O. 1977. Wolf ecology and prey relationships on Isle Royale. U.S. National Park Service Scientific Monograph Series 11.

Petrosky, C. E., H. A. Schaller, and P. Budy. 2001. Productivity and survival rate trends in the freshwater spawning and rearing stage of Snake River chinook salmon (*Oncorhynchus tshawytscha*). Canadian Journal of Fisheries and Aquatic Sciences 58:1196–1207.

Poe, T. P., H. C. Hansel, S. Vigg, D. E. Palmer, and L. A. Prendergast. 1991. Feeding of predaceous fishes on out-migrating juvenile salmonids in John Day Reservoir, Columbia River. Transactions of the American Fisheries Society 120:405–420.

Raymond, H. L. 1979. Effects of dams and impoundments on migrations of juvenile chinook salmon and steelhead from the Snake River, 1966 to 1975. Transactions of the American Fisheries Society 108: 505–529.

Raymond, H. L. 1988. Effects of hydroelectric development and fisheries enhancement on spring and summer chinook salmon and steelhead in the Columbia River basin. North American Journal of Fisheries Management 8:1–24.

Rice, J. A., L. B. Crowder, and F. P. Binkowski. 1987. Evaluating potential sources of mortality for larval bloater (*Coregonus hoyi*): starvation and the vulnerability to predation. Canadian Journal of Aquatic Sciences 44:467–472.

Rieman, B. E., R. C. Beamsderfer, S. Vigg, and T. P. Poe. 1991. Estimated loss of juvenile salmonids to predation by northern squawfish, walleyes, and smallmouth bass in John Day Reservoir, Columbia River. Transactions of the American Fisheries Society 120:448–458.

Roby, D. D., D. P. Craig, K. Collis, S. L. Adamany.

1998. Avian predation on juvenile salmonids in the lower Columbia River. 1997 Annual Report to Bonneville Power Administration, Contract 97BI33475, and U.S. Army Corps of Engineers, Contract E96970049, Walla Walla, Washington.

Rodgers, J. D., R. D. Ewing, and J. D. Hall. 1987. Physiological changes during seaward migration of wild juvenile coho salmon (*Oncorhynchus kisutch*). Canadian Journal of Fisheries and Aquatic Sciences 44:452–457.

Rondorf, D. W., M. S. Dutchuk, A. S. Kolok, and M. L. Gross. 1985. Bioenergetics of juvenile salmon during spring out-migration. 1983 Annual Report to Bonneville Power Administration, Project 82-11 (DE-AI70-82BP35346), Walla Walla, Washington.

Ruggerone, G. T. 1986. Consumption of migrating juvenile salmonids by gulls foraging below a Columbia River dam. Transactions of the American Fisheries Society 115:736–742.

Sandford, B. P., and S. G. Smith. In press. Estimation of smolt-to-adult return percentages for Snake River Basin anadromous salmonids, 1990–1997. Journal of Agricultural, Biological, and Environmental Statistics.

Schaller, H. A., C. E. Petrosky, and O. P. Langness. 1999. Contrasting patterns of productivity and survival rates for stream-type chinook salmon (*Oncorhynchus tshawytscha*) populations of the Snake and Columbia rivers. Canadian Journal of Fisheries and Aquatic Sciences 56:1031–1045.

Schaller, H. A., C. E. Petrosky, and O. P. Langness. 2000. Reply to Zabel and Williams' comments on ''Contrasting patterns of productivity and survival rates for stream-type chinook salmon (*Oncorhynchus tshawytscha*) populations of the Snake and Columbia rivers'' by Schaller et al. (1999). Canadian Journal of Fisheries and Aquatic Sciences 57:1742–1746.

Schreck, C. B., and T. P. Stahl. 1998. Evaluation of migration and survival of juvenile salmonids following transportation. Annual Report to the U.S. Army Corps of Engineers, Project MPE-W-97-4, Walla Walla, Washington.

Sherwood, C. P., D. A. Jay, R. B. Harvey, P. Hamilton, and C. A. Simestad. 1990. Historical changes in the Columbia River estuary. Progressive Oceanography 25:299–352.

Sigismondi, L. A., and L. J. Weber. 1988. Changes in avoidance response time of juvenile chinook salmon exposed to multiple acute handling stresses. Transactions of the American Fisheries Society 117:196–201.

Sprague, J. B. 1971. Measurement of pollutant toxicity to fish III. Sub-lethal effects and ''safe'' concentrations. Water Research 5:245–266.

Thorpe, J. E., L. G. Ross, G. Struthers, and W. Watts. 1981. Tracking Atlantic salmon *Salmo salar L.* migration. Journal of Fish Biology 19:519–537.

USACE (U.S. Army Corps of Engineers). 1999. Draft lower Snake River juvenile salmon migration feasibility report/environmental impact statement. USACE, Walla Walla, Washington.

USACE (U.S. Army Corps of Engineers). 2000. Evaluate comparative delayed mortality of juvenile salmon utilizing different routes of passage at the dams on the lower Columbia and Snake rivers. USACE, Northwest Division, Walla Walla, Washington.

Vaughan, D. S., R. M. Yoshiyama, J. E. Breck, and D. L. DeAngelis. 1984. Modeling approaches for assessing the effects of stress on fish populations. Pages 259–278 *in* V. W. Cairns, P. V. Hodson, and J. E. Nriagu, editors. Containment effects on fisheries. Wiley, Toronto.

Venditti, D. A., D. W. Rondorf, and J. M. Kraut. 2000. Migratory behavior, and forebay delay of radio-tagged juvenile fall chinook salmon in a lower Snake River impoundment. North American Journal of Fisheries Management 20:41–52.

Vigg, S., T. P. Poe, L. A. Pendergast, and H. C. Hansel. 1991. Rates of consumption of juvenile salmonids and alternative prey fish by northern squawfish, walleyes, smallmouth bass, and channel catfish in John Day Reservoir, Columbia River. Transactions of the American Fisheries Society 120:421–438.

Wagner, H. H. 1974. Photoperiod and temperature regulation of smolting in steelhead trout (*Salmo gairdneri*). Canadian Journal of Zoology 52:219–234.

Walters, C. J., R. Hilborn, R. M. Peterman, and M. J. Staley. 1978. Model for examining early ocean limitation of Pacific salmon production. Journal of the Fisheries Research Board of Canada 35:1303–1315.

Ward, D. L., J. H. Peterson, and J. J. Loch. 1995. Index of predation on juvenile salmonids by northern squawfish in the lower and middle Columbia River and in the lower Snake River. Transactions of the American Fisheries Society 124:321–334.

Wedemeyer, G. 1970. Stress of anesthesia with MS-222 and benzocaine in rainbow trout (*Salmo gairdneri*). Journal of the Fisheries Research Board of Canada 27:909–914.

Wedemeyer, G. A., B. A. Barton, and D. J. McLeay. 1990. Stress and acclimation. Pages 451–489 *in* C. B. Schreck and P. B. Moyle, editors. Methods for fish biology. American Fisheries Society, Bethesda, Maryland.

Weitkamp, D. E., and M. Katz. 1980. A review of dissolved gas supersaturation literature. Transactions of the American Fisheries Society 109:659–702.

White, R. G., G. Phillips, G. Liknes, G. Brammer, W. Conner, L. Fidler, T. Williams, and W. Dwyer. 1991. Effects of supersaturation of dissolved gases on the fishery of the Bighorn River downstream of the Yellowtail Afterbay Dam. Final report to the U.S. Bureau of Reclamation, Bozeman, Montana.

Williams, J., G. Mathews, J. Myers, S. G. Smith, T. Cooney, and C. Toole. 1998. Hatchery extra mortality hypothesis. Pages 1–16 *in* D. R. Marmorek and C. N. Peters, editors. PATH submissions to weight of evidence report. ESSA Technologies, Ltd., Vancouver.

Williams, J. G., and G. M. Mathews. 1995. A review of flow and survival relationships for spring and summer chinook salmon, *Oncorhynchus tshawytscha*

cha, from the Snake River basin. U.S. National Marine Fisheries Service Fishery Bulletin 93:732–740.

Wilson, P. With support form the hatchery evaluation group. 1996. Chapter 11. PATH: hatchery impacts. *In* D.R. Marmorek and 21 coauthors, compilers, and editors. Plan for analyzing and testing hypotheses (PATH): final report of retrospective analysis for fiscal year 1996. ESSA Technologies, Vancouver.

Woo, N. Y. S., H. A. Bern, and R. S. Nishioka. 1978. Changes in body composition associated smoltification and premature transfer to seawater in coho salmon (*Oncorhynchus kitsutch*) and king salmon (*O. tshawytscha*). Journal of Fish Biology 13:421–428.

Wood, C. M., J. D. Turner, and M. S. Graham. 1983. Why do fish die after exercise? Journal of Fish Biology 22:189–201.

Wright P. B., and W. E. McLean. 1985. The effects of aeration on the rearing of summer chinook fry (*Oncorhynchus tshawytscha*) at the Puntledge hatchery. Canadian Technical Report of Fisheries and Aquatic Sciences 1390.

Zabel, R. W., and J. G. Williams. 2000. Comments on ‘‘Contrasting patterns of productivity and survival rates for stream-type chinook salmon (*Oncorhynchus tshawytscha*) populations of the Snake and Columbia rivers’’ by Schaller et al. (1999). Canadian Journal of Fisheries and Aquatic Sciences 57:1739–1741.

Zaugg, W. S. 1981. Advanced photoperiod and water temperature effects on gill Na^+-K^+ adenosine triphasphatase activity and migration of juvenile steelhead (*Salmo gairdneri*). Canadian Journal of Fisheries and Aquatic Sciences 38:758–764.

Zaugg, W. S., E. F. Prentice, and F. W. Waknitz. 1985. Importance of river migration to the development of seawater tolerance in Columbia River anadromous salmonids. Aquaculture 51:33–47.

Zaugg, W. S., and H. H. Wagner. 1973. Gill ATPase activity related to par–smolt transformation and migration in steelhead trout (*Salmo gairdneri*) influence of photoperiod and temperature. Comparative Biochemistry and Physiology 45:955–965.