

Stress, Growth and Survival of Juvenile Chinook Salmon

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## Introduction

Columbia River chinook salmon (*Oncorhynchus tshawytscha*) stocks have been declining for several decades. These declines have been linked to ocean conditions (Beamish and Boullion 1993, Francis and Hare 1994, Hare and Francis 1995, Adkinson et al. 1996, Beamish et al. 1997a, Beamish et al. 1997b, Mantua et al. 1997; Percy 1997, Johnson 1998, Beamish et al. 1999, Beamish et al. 2000, Anderson 2000b, Batchelder and Powell 2002), to river conditions (Zabel and Williams 2001, Budy et al. 2002) and to fish condition (Beckman et al. 1998, Beckman et al. 1999, Zabel and Williams 2001). The motivation for this research was the hypothesis that there is a link between the three. In-river experiences must affect most juvenile salmon's (*Oncorhynchus* spp.) condition and its condition when it hits the ocean must affect its further survival. At the most basic level, I tested whether the initial conditions of a fish affect its ability to survive its next experience.

Although Columbia River salmon inspired this work, the results are broadly applicable. Intuitively, most can accept that stressful events impact an individual's fitness, but quantifying how this occurs is difficult. Understanding the effects of stress on survival of an individual is critical to exploring the effects of life history events on a population. This information is crucial for conservation biology, stewardship of wild populations and aquaculture.

In the first chapter I review the literature, outlining the hypotheses that inspired my research. I investigated in-river survival, ocean conditions and their correlation with adult return rate, correlations of size and growth with survival, salmonid metabolism and

starvation, stress effects on survival and evidence that passage of hydroelectric dams is stressful. In addition I introduce the concept of vitality, which attempts to quantify the effects of stressors on an animal's survivability.

In the second chapter, I describe the methods used to investigate my hypothesis. I used several approaches. I performed two fasting and temperature challenge experiments on juvenile chinook salmon at the University of Washington (UW) to test the hypothesis that feeding history affects a fish's ability to survive under a secondary stress. I analyzed otolith samples of migrating juvenile chinook salmon from a National Marine Fisheries Service (NMFS) experiment at Bonneville Dam to investigate whether relative growth history (determined from otoliths) was related to survivability in sea water tanks. Lastly, I analyzed the survival results of all of those experiments using the vitality model.

The third chapter describes in detail the 20° C challenge experiments performed at the University of Washington. It demonstrates the effects of feeding history on survival and examines the implications of such effects.

In the fourth and fifth chapters I show the results of the otolith analysis, and the second UW challenge experiment. Although the results of these investigations were not statistically significant, they were useful for establishing methods to pursue this research in the future.

Finally I provide brief conclusions and discuss management implications. Decisions about instream flow, hatchery release timing, barging, and spill, could all be influenced by results of studies on cumulative stress and delayed mortality.

## 1. Background

**Extra Mortality:** any effect of the Snake and Columbia River hydrosystem on salmonid survival that is not measured during juvenile migration or adult upstream migration, that does not include differential delayed transportation mortality, and that does not include in-common environmental trends that are reflected in concert in stocks above and below the Snake River dams (NMFS 1999).

### 1.1 Introduction

Columbia and Snake River salmon stocks have declined for several decades. Smolt-to-adult return (SAR) rates for Snake River spring-summer chinook (*Oncorhynchus tshawytscha*) fell from greater than 4% in the mid to late 1960s to less than 2% during the 1970s. The most obvious environmental change during that time is the jump from four operating dams in the 1960s to eight by the end of the 1970s (Williams et al. 2001).

Low survival rates were once considered mainly a consequence of direct mortality during passage through the hydropower system. In the 1970s, migration survival was estimated at 10% to 30%. From 1993 to 1999, structural and operational changes in the hydropower system increased survival during migration to between 31% and 59%. However, SAR remains low. Therefore, continuing low adult returns in the 1990s do not

appear to be related to mortality of downstream migrants that occurs directly within the hydropower system (Williams et al. 2001).

This agrees with the conclusions drawn by Karieva, et al. (2000) from applying a matrix model to long-term population data. They found that dam passage improvements dramatically mitigated direct mortality, but that even if main stem survival were elevated to 100% Snake River spring/summer chinook salmon would probably continue to decline toward extinction. However, modest reductions in estuarine and first ocean year mortality would reverse current population declines. They concluded that the direct effects of dams manifested outside the migration corridor must be examined.

The potential for delayed impacts of the hydropower system and smolt transportation on the survival of smolts to adults have become a central issue in the recovery of Snake and Columbia river salmon stocks. Delayed impact is termed by the National Marine Fisheries Service as “extra mortality”. Several studies suggest a significant component of “extra mortality” is associated with climate regime shifts (Beamish and Boullion 1993, Francis and Hare 1994, Hare and Francis 1995, Adkinson et al. 1996, Beamish et al. 1997a, Beamish et al. 1997b, Mantua et al. 1997, Pearcy 1997, Beamish et al. 1999, Beamish et al. 2000, Anderson 2000b). Climate regime shifts affect all stocks, though differently depending on route of migration and distribution in the ocean. Snake River fish declined more dramatically than other stocks and therefore, appear more vulnerable to climate than the others. Studies suggest that fresh water growth patterns may affect survival to adult return (Zabel and Williams 2002, Beckman et al. 1999). Freshwater growth could mitigate effects of ocean climate. Since other

studies suggest that stress may retard growth (McCormick et al. 1998), a hypothesis emerges that freshwater stressors affect ocean survival.

I investigated the effects of freshwater growth and stress on survival. Before considering the hypothesis that freshwater stressors affect ocean survival, I examined evidence for the ocean climate and growth hypotheses.

## **1.2 Ocean Conditions**

Recent large returns of adult Columbia River salmon support the hypothesis that ocean conditions have a strong affect on salmonid survival (Anderson 2001). The synchrony of changes in abundance between stocks indicates that a large-scale process is affecting all of them together. Many cycles in oceanic conditions alter patterns of circulation, distribution of predators and prey, and productivity. El Niño (ENSO) fluctuations occur on the timescale of years; Pacific interdecadal oscillations (PDO) occur on the timescale of decades; other cycles (such as ice ages) operate on timescales of thousands of years (NMFS 1999). It is important that we understand the interactions between climatic and anthropogenic effects on Columbia River salmon populations if society intends to prevent further declines. In part we find ourselves in the current situation because we did not consider the interactions in the past. Between 1932 and 1977 favorable ocean conditions largely masked the negative effects of fishing pressure and dam construction on populations. A shift back to poor ocean conditions in 1977 largely masked mitigation efforts of the last 20 years (Anderson 2000b).

A series of studies have identified both large scale and regional linkages (Beamish and Boullion 1993, Francis and Hare 1994, Hare and Francis 1995, Adkinson et al. 1996,

Beamish et al. 1997a, Beamish et al. 1997b, Mantua et al. 1997; Pearcy 1997, Beamish et al. 1999, Beamish et al. 2000, Anderson 2000b, Batchelder and Powell 2002).

Phytoplankton abundance, which is linked to physical ocean changes, has also been correlated with Pacific salmon abundance (Brodeur and Ware 1992, Sugimoto and Tadokora 1997 *as cited in* Beamish et al. 1999). NMFS (1999) acknowledges strong indications that many salmonid stocks' survival and growth are significantly correlated to the PDO.

Shorter-term variations have been associated with ENSO. Abundance and average size of coho (*O. kisutch*) and chinook were below normal in the 1957-59 and 1940-41 ENSO events (Johnson 1988). Johnson found that the 1983 ENSO event was simultaneous with a decline in summer growth rate. Oregon Coho were half the size they were in years of good upwelling. Average weight of chinook in the Oregon troll fishery was the lowest on record, and Johnson found a 27% decline in fecundity on the Columbia River. Chinook in 1983 that reared from Vancouver Island south had poor survival. Oregon Department of Fish and Wildlife samplers reported that many of these chinook's stomachs were empty or near empty. These data indicate that the ENSO event caused or was simultaneous with extremely poor conditions for growth in the ocean (Johnson 1988).

Schaller et al. (1999) postulate that estuary and early ocean conditions do not appear to exert systematically different effects on survival of stream-type chinook stocks from upriver regions of the interior Columbia River basin. Climatic influences appear to affect populations on large geographic scales. The stocks contrasted in their analysis are

similar in life history and overlap in time and space during their early ocean residence (and beyond).

Similar trends are apparent in the Atlantic Ocean. Coherence patterns among regional and continental stock groups suggest broad scale functions are more important in defining recruitment than mortality effects associated with individual rivers (Friedland 1998). Friedland cites that recruitment in Atlantic salmon (*Salmo* sp.) populations is controlled by events that take place during their first year at sea (Salminen et al. 1995, Friedland et al. 1998, Scarnecchia et al. 1989). Survival of Atlantic salmon stocks in the North Sea appears to be affected by the distribution of spring water temperatures in the northeast Atlantic.

There are several mechanisms by which shifting ocean climate could affect salmonid recruitment. Prey availability is often associated with oceanographic processes and structural features in the water column (Levings 1994) and quality and abundance of prey are potentially related to survival (Healey 1991; Brodeur et al. 1992; Perry et al. 1996). Therefore, alterations in physical conditions affect prey density, which, in turn, affects salmonid survival. Physical features, such as temperature, of the ocean could also directly affect post-smolt survival and condition through influences on salmon metabolism. Growth increases linearly with water temperature up to a maximum rate and then decreases at high temperatures (Brett 1979). In pelagic fishes, body size is inversely related to survival (Peterson and Wroblewski 1984). The faster a juvenile salmon grows, the less likely it is to be preyed upon. This is called growth-mediated survival. Food

consumption and temperature strongly affect growth rate. Variability in growth among individuals can affect individual chances of survival.

### **1.3 Feeding and starvation metabolism**

Food consumption is a major determinant of growth. Natural diets of salmon change as they develop and move between habitats. Higgs et al. (1995) reviewed feeding habits of all Pacific salmon in relation to life stage. Chinook fry in fresh water use a wide range of food items, but primarily consume aquatic insects (particularly chironomids and ephemeropterans). Fry also consume crustaceans such as calanoid copepods, cladocerans and gammarid amphipods. Yearling chinook salmon in freshwater have similar feeding habits to fry. The primary food source of smolts is aquatic insects such as chironomids, plecopterans, and ephemeropterans. Crustaceans such as cladocerans are also common.

Estuarine fry diets include a mixture of freshwater and brackish marine organisms. Gammarid amphipods, insects, and calanoid copepods are dominant food organisms. As smolts approach the estuary and ocean, food items reflect increased encounters with marine organisms. Dominant prey are fish (such as chum salmon), epibenthic amphipods, pelagic amphipods, decapod larvae, and calanoid and cyclopoid copepods. In general, yearling chinook diets contained fewer prey taxa than noted in fry diets. In the ocean, chinook consume fish, euphasids, crab zoea, and megalopa, squid, and pelagic amphipods (Higgs et al. 1995).

Congleton et al. (2001) found that juvenile chinook salmon migrating down the Columbia River are in a negative energy balance. Blood indices indicated that this was due to restricted food intake. Fish can survive a long time without food and it is a natural

part of many lifecycles (Navarro and Gutierrez 1995). Because lack of food is a frequent condition for animals, starvation is not just an experience to be passively endured, but involves a tightly regulated reorganization of metabolism and behavior (Mendez and Weiser 1993). Many fish can survive and fully recover from starvation, so they must be adapted to mobilize metabolic reserves and body constituents to survive periods of deprivation. The pre-fasting diet may exert influence on metabolic events initiated by fasting. Generally, usage of energy reserves occurs in a pattern (Navarro and Gutierrez 1995).

Mendez and Weiser (1993) outline 4 phases of response to food deprivation in fish. In phase one, the stress phase, activity and oxygen consumption increase. During phase two, the transition phase, which is completed after two days in cyprinids, oxygen consumption decreases and energy consumption shifts from carbohydrates to lipids. Phase three, adaptation, occurs around 4 weeks of starvation. It involves settling into a stable, reduced metabolism with a high body water content, low glycogen levels and increased levels of glutamate-pyruvate transaminase (which is a major link in the glucose/alanine cycle). If the fish are then exposed to food again, phase four begins, in which there is a rapid increase in oxygen consumption and growth.

In most species, including salmonids, liver glycogen is mobilized early in experimental fasting and is often the first substrate used. However, it is a small contribution to the total energy of the fish. Blood glucose levels generally remain at a steady level during starvation. This is thought to occur at the expense of loss of liver glycogen. Muscle glycogen is thought to be a minor contribution to energy expenditure.

Due to its immediate involvement in muscular activity, it is a volatile entity more related to activity than fasting. Levels of muscle glycogen increase in cooler temperatures and decrease in higher temperatures (Navarro and Gutierrez 1995).

Lipids, which are high in energy, are used simultaneous with or following carbohydrate use, but always before proteins. There is an inverse relationship between water and lipid concentrations. As lipid is used, it is replaced by water, maintaining the weight of the fish. Lipid is stored in the liver, intestine and muscle. In salmonids, most lipid is stored in visceral fat. Visceral fat is generally mobilized before muscle or liver lipid in salmonids. However, they will use liver and muscle lipid if they are fasted long enough. Rainbow trout (*O. mykiss*) starved 27 days showed no difference in muscle fat, but after 50 days, a difference was measurable (Navarro and Gutierrez 1995).

Protein is used last. Like lipids, as protein is consumed, it is replaced by water. Fish are specially adapted for protein mobilization; They have a high level of proteolytic enzymes in their muscles coupled with the ability to excrete excess nitrogen as ammonia or ammonium ions. When fasting begins, proteolysis is slowest in the muscles, then in the spleen, liver, kidney, and intestine. As fasting continues, the order of importance of the protein sources reverses. Muscle protein is used last. Many different tissues contribute to the pool of amino acids, but skeletal muscle represents the largest store of amino acids and is therefore the main source of energy during prolonged starvation (Navarro and Gutierrez 1995).

Hormone levels are also affected by fasting. Glucagon family peptides, involved in mobilizing lipid stores, are key hormones regulating energy reserves under fasting

conditions in fish. The previous nutritional state of fish can modulate hormonal mediated lipolysis. Four weeks of fasting enhanced glucagon-stimulated lipolysis in livers of rainbow trout (Navarro and Gutierrez 1995). In rainbow trout, plasma cortisol levels seem to be representative indicators of the fish responses to stress, but are not greatly affected by fasting. Although plasma cortisol elevations in response to handling were not appreciably modified in 20 day fasted juvenile chinook salmon as compared to controls, the hyperglycemia response to stress was lower in fasted fish than fed fish (Navarro and Gutierrez 1995). There is a general decrease in thyroid activity associated with fasting which may reflect the need to limit an exaggerated metabolite mobilization of energy reserves (Navarro and Gutierrez 1995).

Fish tissue metabolism is finely regulated under fasting conditions by the actions of many hormones. Previous nutritional condition and physiological state must be considered when interpreting experimental results. Especially in the case of starvation, laboratory experiments, generally done on heavily inbred fish previously fed a diet maximizing meat production, give quite different results to those done on naturally starving fish (Navarro and Gutierrez 1995). Age, size, temperature, and ecological factors are of profound importance in setting the stage on which the reorganization of reserves can take place (Mendez and Weiser 1993).

#### **1.4 The importance of size and growth**

Size has been suggested as a factor that can influence migratory behavior and subsequent survival to adulthood in juvenile salmonids. Studies of steelhead trout (*O. mykiss*) have shown that larger smolts within a year class tend to survive better than

smaller smolts (Ward and Slaney 1988; Ward et al. 1989). Hatchery chinook and coho released at large size also have a higher survival rate than smaller ones (Hagar and Noble 1976, Bilton et al. 1982; Bilton 1984; Martin and Wertheimer 1989). The same has been shown for chum (*O. keta*) (Parker 1971, Hargreaves and Le Brasseur 1986), pink (Kobayashi 1980), and sockeye (*O. nerka*) salmon (Macdonald et al. 1987; Henderson and Cass 1991). Zabel and Williams (2002) found that spring chinook salmon survival, expressed as the ratio of returning adults to the out-migrating smolts (SAR) increased with size at tagging.

Large smolts may also be faster growing, so differences in survival of large and small fish could be due to growth rate, body size, or both (Beckman et al. 1998). A few studies have noted influence of growth rate on smoltification independent from body size. Wagner et al. (1969) noted that faster-growing fall chinook salmon exhibited higher seawater tolerance than larger, but slower-growing fish. In Atlantic salmon, measured increases in summer growth rate increased the percentage of age one fish that smolted (Thorpe 1989; Thorpe et al. 1989). Okland et al. (1993) rejected the hypothesis of a threshold size for smolting in Atlantic salmon and suggested that age at smolting was inversely related to growth rate. Dickhoff (1995) showed a relationship between growth rate of chinook salmon prior to release and return of adults.

Beckman (1999) examined several characters representing smolt quality and their relationship to SAR. The characters examined were: fish size, spring growth rate, condition factor, plasma hormone concentrations (thyroxine, cortisol, and insulin-like growth factor-I (IGF-I)), stress challenge, gill  $\text{Na}^+$ ,  $\text{K}^+$ -ATPase activity and liver

glycogen concentration. Of those nine characters, only spring growth rate, IGF-I, and gill ATPase activity were significantly correlated to SAR. Release size and SAR were not highly related in this study. Thus, some factor in addition to size at release must be contributing to survival.

### **1.5 Growth mitigating ocean conditions**

The ocean conditions may explain the general decline in salmon populations, but not the differences between stocks (Beamish and Boullion 1993, Francis and Hare 1994, Hare and Francis 1995, Adkinson et al. 1996, Beamish et al 1997a, Beamish et al. 1997b, Mantua et al 1997; Pearcy 1997, Beamish et al. 1999, Schaller et al. 1999, Beamish et al. 2000, Anderson 2000b, Batchelder and Powell 2002). Variation in growth could explain the differences between stocks, but not the general decline (Beckman 1999). From the fusing of these two ideas emerges the hypothesis that freshwater growth might mitigate the effects of poor ocean conditions.

### **1.6 Vitality and stress**

Letcher et al. (1996) investigated the effects of prey patches, which would cause variable feeding histories among individuals, on susceptibility to future starvation of yellow perch larvae (*Perca flavescens*). They found that the feeding history affected the larvae's ability to survive starvation. Regardless of initial mass, perch under starvation conditions lost mass proportionally at the same rate. However, perch fed intermittently before starvation died after losing proportionately less mass than fish that had been fed continuously. Analysis of their data showed a significant ( $p < 0.05$ ) relationship between growth rate prior to starvation and days to mortality under starvation (Days to mortality =

18.01 \*Growth rate + 6.44,  $r^2=0.57$ ). They hypothesized that this related to compositional changes that occur during short periods of food deprivation. During starvation, larvae lose carbohydrates and proteins (Erlich 1974; Molony 1993), reorganize enzymes (Mendez and Wieser 1993) and mobilize glycogen stores (O'Connell and Paloma 1981; Blasco et al. 1992). These changes might cause fish that have experienced short-term starvation to lose mass differently (mobilize different energy stores) than fish that have fed continuously (Letcher et al. 1996).

Taking this concept a step further, the vitality model (Anderson 2000a) predicted that everything about an animal's history affects its subsequent survival. The vitality model is based on the premise that each species has its own initial state or capacity for survival, and that over its life span, an animal uses up this capacity. Thus, events that happen today affect an organism's vitality and its ability to survive in the future. Vitality is an abstract property that changes with moment-to-moment experiences. The model provides a way to extrapolate the impact on vitality of stressors measured in one environment to another. By characterizing how stressors alter the vitality probability distribution, the model can quantify the degree to which a stressor differentially influences members of a population.

### **1.7 Dams are stressful**

Hydropower dams have transformed the Columbia River into a series of reservoirs. These dams and reservoirs have caused significant changes in migration timing and travel rates of juvenile salmonids (Raymond 1968, 1969, 1979). Slower migration rates may result in decreased survival due to increased predation (Gray and

Rondorf 1986; Poe et al. 1991), susceptibility to disease at higher water temperatures (Tiffan et al. 2000) and increased physiological stress due to prolonged food deprivation (Congleton et al. 2001).

There is evidence that dam passage is stressful. When a downstream migrant encounters a dam it must take one of four passage routes. It can go over the spillway, through the bypass system and back into the river, through the bypass system and onto a barge or truck, or through the turbines. Fish that pass through the turbines are exposed to extreme pressure changes and mechanical injury. Fish in the bypass system go from 1 to 3 atmospheres then back to 1 in 10 seconds. They also experience high turbulence and 90° turns in the bypass conduits. At Lower Granite Dam, fish are shuttled through a device that sorts them by size, then dewatered and passed through a tube that leads to the PIT tag detector. Fish for tagging may then be held for 24 hours, anesthetized, marked and returned to the river or to a barge (Budy et al. 2001).

Past research has demonstrated that there is an increase in stress level as spring chinook salmon move through the by-pass system from the gate wells to the transport separator (Park et al. 1986; Matthews et al. 1986). Park et al. (1986) subjected fish collected at different points in the bypass system to seawater challenge. C-slot gate well fish were collected as they entered the bypass gates. Pre-separator fish traveled through the gatewell and bypass pipe into the pre-separator. Transport fish traveled through the gatewell, pipe, and separator into a mock transportation. Mark-transport fish were marked before they were transported. When held in seawater tanks, the pre-separator fish reached mortality significantly faster than C-slot group. Transport and marked transport

fish mortality rates were higher than the pre-separator groups. This supports both the idea that fish are increasingly stressed as they move through the bypass and the concept that stress is cumulative and affects future survival.

Dams have been shown to directly delay migration. Vendetti et al. (2000) studied the behavior of radio-tagged juvenile fall chinook as they traveled from Lower Granite Dam to Little Goose dam. Fish migration rates decreased significantly as fish approached the lower dam, corresponding to reservoir water velocity. 10% to 20% of the fish spent a week or more in the forebay and lower reservoir. Radio-tagged fish displayed two behaviors in the forebay. They either crossed the forebay or swam upstream. Fish crossed the forebay between 0 and 21 times during each encounter with a dam. Upstream excursions were as long as 14.4 km in length. This delay exposes the fish to prolonged periods of high temperatures and predation exposure. Surface temperature in the Little Goose Dam forebay were observed as high as 24.6° C (Vendetti et al. 2000), which is above the lethal limit for juvenile spring chinook (Brett 1952). The mean weekly forebay temperature in 1996 was 23° C at the surface and 20° C at 20m depth (Vendetti et al. 2000). The effects of long-term exposure to these temperatures on disease resistance, predator avoidance, and bioenergetic costs are largely unexplored (Vendetti et al. 2000). The elevated temperatures add stress to an already energetically costly experience of negotiating the dam.

Delays at dams not only cause direct stress, but coupled with artificially low in-stream flow rates, may also expose the migrating salmon to marginal foraging possibilities as they arrive in the estuary and ocean later than historically (Quinn and

Adams 1996). The pre-hydrosystem hydrograph of the Columbia River was characterized by high spring run-off (April to mid-June), decreasing summer and fall flows (mid-June to December) and low winter flows (January to March). Anadromous salmon adapted to this flow pattern by migrating downstream during the spring freshet. Now, instead of migrating passively with the spring freshet, the juvenile salmon must swim through 522 km of slack water and eight dams (Budy et al. 2002). Quinn and Adams (1996) speculate that migration timing evolved to place them at the mouth of the river at an optimum time for feeding; now they arrive late. The lower river is warming earlier than in previous years, so the mismatch may be amplified (Quinn and Adams 1996). Changes in flow may also alter the ecology of the estuary and near-shore environment, which may negatively affect salmonid survival (Budy et al. 2002). Consistent with this, sub-yearling chinook migrating early in the year produced more adults than those migrating later in the year (Giorgi et al. 1990)

Information on juvenile salmon foraging in the reservoirs remains scant, but studies indicate that foraging opportunities are limited. Congleton et al. (2001) showed that juvenile chinook in the Columbia River are in negative energy balance while migrating to sea. Total lipid and protein reserves (as percentage of total body weight) decreased from upstream to downstream dams and decreased with increasing travel time. Their model indicates that a 20-day increase in travel time to John Day Dam resulted in an additional 41 % decrease in lipid reserves. Blood indices indicated that the energetic deficit was, in a large part, due to restricted food intake. Muir and Cooley (1996) found that 29% of chinook sampled at Lower Granite dam had empty stomachs.

Beckman et al. (1999) found that growth rate is an important correlational factor with SAR. Arrival in the ocean at a period of low food availability could slow growth rates. Levin et al. (2001) asserts that during years of poor ocean productivity, hatchery salmon production has a negative impact on wild salmon survival. This is most likely due to competition for limited food resources causing slowed growth rates and increased predation. In addition, the direct stress of dam passage could inhibit growth rate of migrating juveniles. It is widely accepted that stress can reduce growth rate in fish (McCormick, et al., 1998). McCormick, et al. (1998) found that short periods of intense stress resulted in reduced growth rate of juvenile Atlantic salmon. Furthermore, they found that growth depression increased with increased frequency of stress. Carey and McCormick (1998) found that smolts show a more intense response to acute stress than non-smolting juvenile salmon. Peak levels of plasma cortisol 3 hours after initiation of stress were twice as high in smolts than in non-smolts. This could be significant because many of the fish that are traveling through the dams are smolts.

### **1.8 Approach**

I investigated the possible connections between cumulative stresses, growth rate, and survivability. The project consisted of two parts. One compared growth rates, feeding history, and survival in starvation and temperature challenge experiments carried out at the fish hatchery located at the School of Aquatic and Fishery Sciences, University of Washington. Those experiments were to test the hypothesis that fish that had been fasted and had lower growth rates would not survive as long under temperature stress as those that had not been fasted. The second part of the project entailed analysis of otoliths

removed from fish in an ongoing NMFS experiment. By measuring otolith increment widths, I attempted to compare relative growth rates of the NMFS fish with their survival time in seawater tanks. The hypothesis tested was that fish with higher growth rates prior to being collected would survive longer in the seawater tanks than those with slower growth rates.

## **2. Methods**

### **2.1 University of Washington 20° C Experiment**

#### **2.1.1 Selecting fish**

All juvenile chinook salmon were selected from the same cohort of brood year 2001 at the University of Washington hatchery, which were raised under the same conditions. This maximized uniformity in the history of the young fish, and minimized the effects of confounding factors such as age differences, temperature experiences, feeding regimes, and growth rates. Fish were fed Moore-Clark, Nutra 2000, #0 to #3 crumble depending on their size. Fish weighed an average of 3.6 g when selected for the experiment.

Two groups of fish were collected with large aquarium nets. On 9 March 2002 one group of 150 fish were selected for PIT tagging (see below). On 13 March 2002, a second group of approximately 1000 fish were selected by weighing 3600 g of fish. PIT and non-PIT tagged fish were kept separate throughout the experiment in case different mortality rates resulted from handling and tagging. The two groups were placed in separate sections of a small raceway inside the hatchery.

This number of fish was required for three treatments with five replicates each of the untagged fish and three treatments with no replicates of the PIT-tagged fish. Fifty fish were in each replicate. Sample size was determined using power analysis and consultations with a statistician. However, the results of power analysis were large ranges of sample sizes. Therefore, the main determinants of sample size were availability of tanks, fish, PIT tags, and man-hours.

## 2.1.2 Handling and Tagging

### 2.1.2.1 PIT tagged fish

A passive integrated transponder (PIT) tag is a miniature single relay station that is 12 mm long and 2.1 in diameter. PIT tags allow identification of animals without disturbing them. It consists of an antenna of copper wire wound around a cylindrical, ferromagnetic core. The antenna is connected to an electronic chip, which produces a unique programmed signal when supplied with energy. The tag components are enclosed in a glass tube. The energizing system delivers energy to the tag by producing a magnetic field. When the tag enters the magnetic field, its antenna induces an electrical current in the core, which activates the chip and causes it to emit a 40 to 50 MHz signal. A radio receiver hears the signal and transforms it into a unique 10-digit alphanumeric code (Nielson 1992).

I fasted the fish for two days in preparation for tagging. On 11 March 2002, I PIT tagged 150 fish using the method described by Prentice et al. (1987, 1989, 1990). I sedated the fish by immersion in 50 to 100 mg/L of Tricaine Metansulfonate (MS-222). Sedation level was determined visually using total loss of equilibrium, loss of reflex response, and lack of response to physical stimulus as indicators. While the fish were sedated, I PIT-tagged them.

I used the hand held PIT tagging system. This is a modified 5 cm<sup>3</sup> plastic syringe. A hole drilled in the end of the barrel prevents the operator from injecting air into the fish during tagging. To implant the tag, I depressed a rod attached to the end to the syringe, forcing the tag out of the 12-gauge needle. I manually inserted each tag into the needle, sterilizing them before each insertion. I inserted the needle posterior of the pectoral fins

and just offset from the mid-ventral line. The bevel of the needle was face up with the syringe at an angle between 20 and 45 degrees with a smaller angle for smaller fish. The angle reduces sliding on the scales. Just enough needle pressure was exerted to penetrate the body wall. I then decreased the needle angle until it paralleled the fish body and inserted the needle farther to place the tag posterior to the pyloric caeca in the proximity of the pelvic girdle. Depressing the plunger implanted the tag (Figure 2.1). During tagging, equipment was disinfected with 90% ethanol. I checked each fish after implantation to ensure the tag was injected.

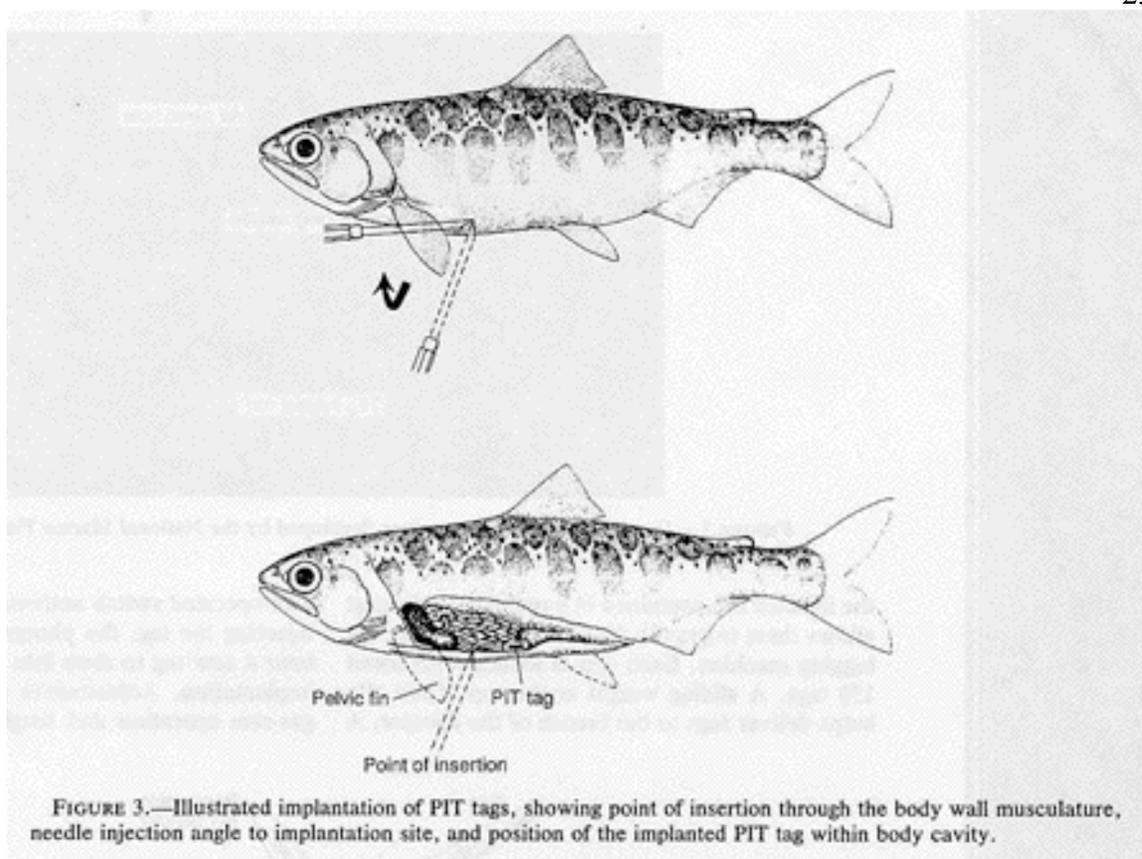


FIGURE 3.—Illustrated implantation of PIT tags, showing point of insertion through the body wall musculature, needle injection angle to implantation site, and position of the implanted PIT tag within body cavity.

Figure 2.1 Illustrated implantation of PIT tags, showing point of insertion through the body wall musculature, needle injection angle to implantation site, and position of the implanted PIT tag within the body cavity (Prentice et al.1990).

No appreciable host tissue response from tagging procedures has been observed in salmonids cavity (Prentice et al.1990). In experiments on chinook and sockeye salmon up to 19 cm in length, the needle puncture wound appeared to close almost immediately. As early as two weeks after tagging, little visible evidence of external trauma remained at the injection site (Prentice et al., 1987). This was also true of my fish.

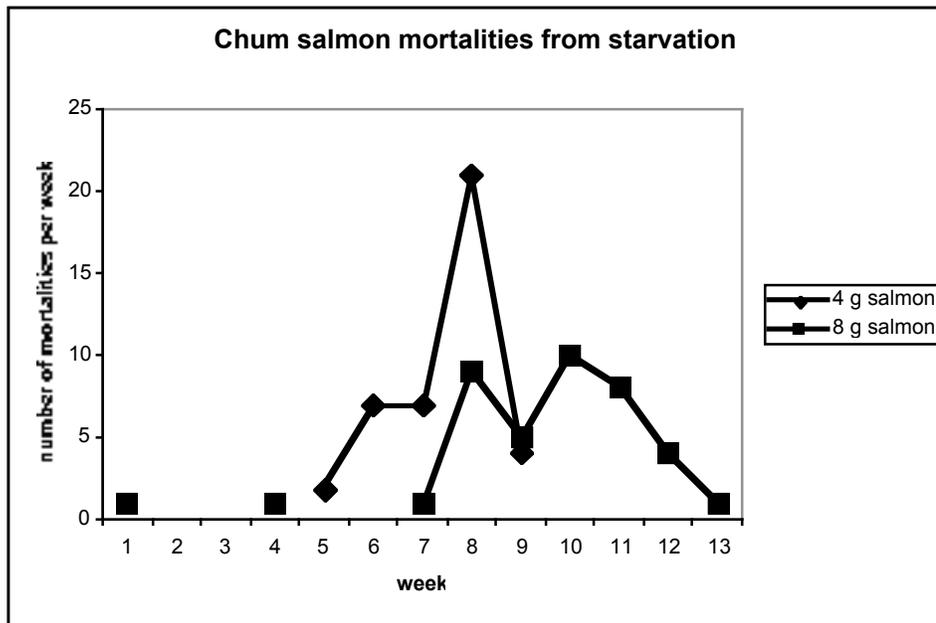
For three days after implantation, the fish were fed maintenance ration to prevent swelling stomachs from forcing the tag out of the open wound (Prentice, et al. 1987, 1989, 1990).

#### 2.1.2.2 Non-PIT tagged fish

On 13 March 2002, I collected approximately 1000 of the same brood year 2001 chinook and transferred them to a small raceway inside the hatchery. I used average fish weight to determine the number collected. Fish were maintained in this raceway and fed regularly for one month. Group weights were taken on 10 April. I weighed 3 groups of 50 to determine an average weight per fish. I then divided the fish into 3 equal sections by weight.

#### 2.1.3 Feeding Fish

Prior to the beginning of the experiment, the hatchery staff had fed the fish approximately 3% of their body weight daily. I fasted the fish for different lengths of time and treated them all to the temperature stress simultaneously. Feeding treatments were determined using past starvation studies, which show that juvenile salmon show a strong resistance to starvation. In a study by Akiyama and Nose (1980), 4 to 8 gram chum salmon fed continuously, then starved to mortality, survived up to 13 weeks. I calculated the mean time to mortality of those fish to be 7.4 and 9.45 weeks for 4- and 8- g fish, respectively (Figure 2.2). I hypothesized that four weeks of fasting would not cause mortality in 4 to 5 g fish.



**Figure 2.2** Incremental mortalities of starved chum salmon at two initial weights (Akiyama and Nose, 1980).

However, hormonal responses to ration change have been observed within two weeks. Pierce, et al. (2001) fed juvenile coho salmon high, medium and low ration diets. Significant differences in specific growth rate (weight) manifested themselves by week 2 of the treatment. Thus, I hypothesized that two weeks of starvation should be enough to alter the growth rates of juvenile chinook in this experiment.

Table 2.1 Feeding schedule for all treatments.

Treatment	10 April 2002	24 April 2002	10 May 2002
0f	Feed	Feed	Temperature Stress and fast
2f	Feed	Fast	Temperature Stress and fast
4f	Fast	Fast	Temperature Stress and fast

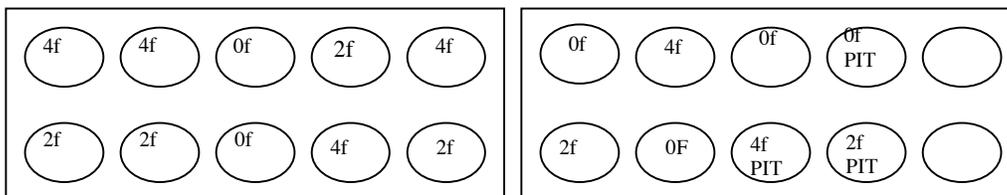
#### 2.1.4 Measuring Fish

Table 2.2 Schedule and description of measurements

	12 March	10 April	24 April	8 May	Mortality
PIT	Sedated and measured fish weight (to nearest 0.1 g) and FL (to nearest mm)	Individual weights to nearest 0.1 g and FL to nearest mm			
Non-PIT		Group weighed to nearest 0.1 g	Group weighed to nearest 0.1 g	Group weighed to nearest 0.1 g	Individual weights to nearest 0.1 g and FL to nearest mm

### 2.1.5 Fish Placement

The non-PIT-tagged fish were distributed within their treatment groups to five replicate tanks per treatment. Tank arrangement distributed randomly. All tanks received the same water supply for the duration of the experiment.



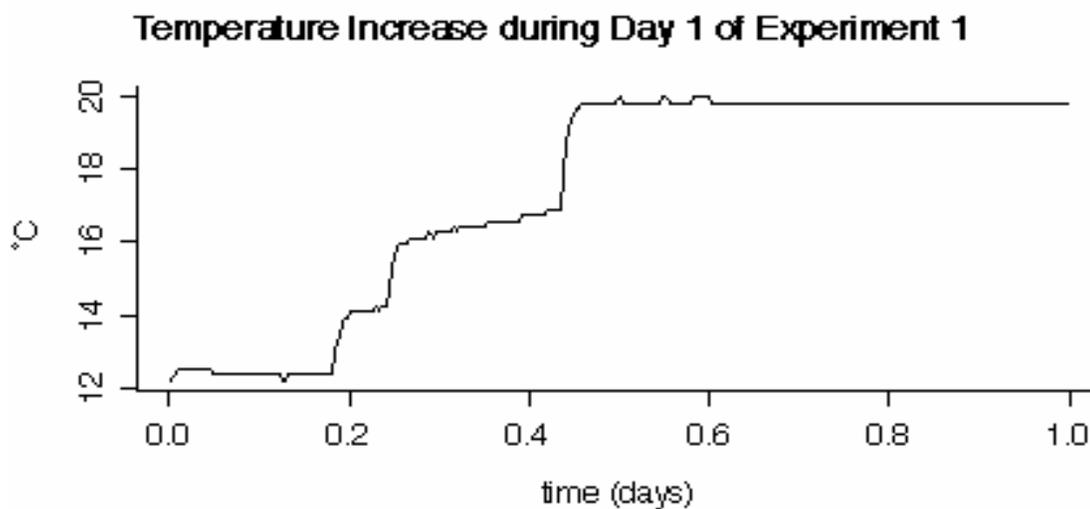
**Figure 2.3** The layout of treatment group replicate tanks on the table

### 2.1.6 Temperature Trials

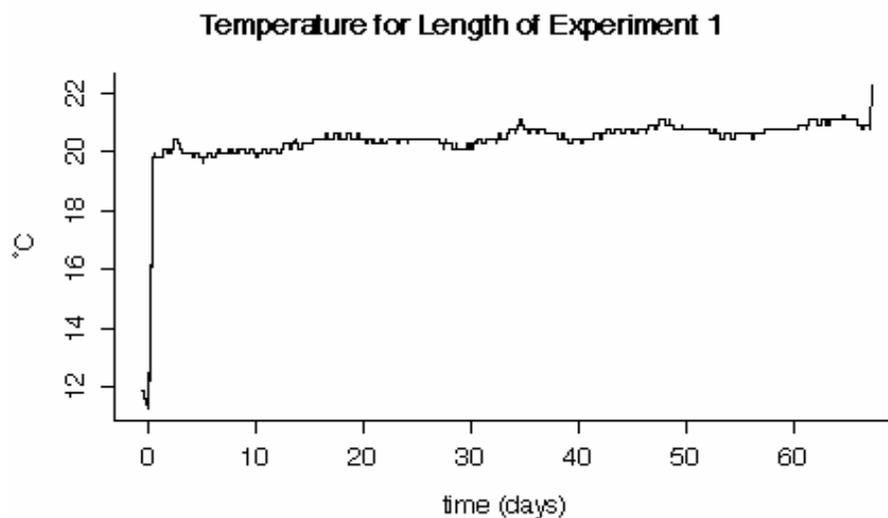
In an effort to evaluate how long it would take the fish to die at elevated temperatures, I ran a few small trials in aquarium tanks. I did not want the fish to die so quickly that I could not keep track of when they died with reasonable check intervals but I wanted them to die fast enough to ensure they were dieing from the stress of temperature. I placed ten fish in a tank with a gradual water flow. I monitored the temperature regularly. The fish went from 7 to 21° C in four hours. Two fish died the first day. By the fourth day, seven fish had died. The temperature rose to 23.2° C and 2 more fish died on the fifth day. The last fish died on the sixth day. Because the fish died within a week, I ran the trial again at 19° C. The fish went from 11 to 19° C in four hours. The max temp of 22° C was reached on the fourth day and went back down to 21.6° within a few hours. In this trial, nine of ten fish died within six days. Based on this information, I decided to run the experiment at 20° rather than the proposed 26° C.

### 2.1.7 Mortality Experiment

On 10 May 2002, I increased the water temperature by gradually changing the mix of two sources of water to the hatchery (water from Lake Washington (11°C) and water from a campus well (20° C)). The temperature increased from 11.29 in both tanks to 20.12 °C over eight hours, beginning at 07:50 (Figure 2.4). The temperature remained relatively constant from then until the point of 100% mortality (Figure 2.5). Dead fish were collected once to four times daily, their length measured, and weighed.



**Figure 2.4** Temperature Increase on May 10, 2002



**Figure 2.5** Temperature over entire experiment

### 2.1.8 Analysis and Vitality Model

An unexpected side effect of the water delivery system was gas super-saturation which may have altered early mortality rates in those tanks.. Although all tanks received the same water supply, there was some variation in dissolved oxygen levels due to the angle of the water input stream into the tanks. When the super-saturation (mean=104.4%, sd=1.3) was discovered on the fourth day of the temperature treatment, it was corrected (mean= 98.2, sd=2.1). Because the treatment placement was random, and the super-saturation was randomly distributed through the tanks, I determined to continue the experiment because it would not affect the ordering of treatment lifespan results.

However, an assumption of the vitality model is that the stress is constant and in our experiment the stress level changes during and after super-saturation. The first six days had much higher mortality rates than the rest of the experiment and I assumed that it



analysis showed that MTMs of replicates differed significantly within treatment groups, they were all combined for analysis. The non-PIT-tagged group weights and growths were not significantly different than the PIT tagged fish. The t50s of the PIT and non-PIT tagged groups were nearly identical. I therefore combined all the tanks, PIT and non-PIT, for analysis. I performed ANOVAs of MTM by treatment group. Creating box plots of lifespan by treatment, I determined the median time to death and the 95% confidence intervals around the median. I performed ANOVAs of the regression of MTM against instantaneous growth for the PIT tagged fish, and a Student's t-test comparing instantaneous growth rates of early and late mortalities in the PIT tagged groups. All tests were performed at the 95% level ( $\alpha=0.05$ ).

### ***Vitality measures***

Vitality is an abstract property that changes with moment-to-moment experience. The model is based on the premise that every species has its own initial capacity for survival and that over time an organism uses up that capacity. When vitality declines to 0, the organism is dead. Mortality can also occur independent of an organism's vitality. For example, harvest and catastrophic events induce mortality equally within a population independent of the different histories of individuals. Thus, the survival distribution,  $S$ , can be expressed in terms of the product of the probability of being alive according to the organism's vitality,  $P_v$ , and the probability of survival in avoiding random (accidental) mortality events,  $P_a$ . In this vitality framework, survival is characterized by three parameters,  $r$ ,  $s$  and  $k$ , which I calculated using the vitality fitting program. (Anderson 2000a, Salinger et al., 2003)

$$\frac{dv}{dt} = \mu + w(t) \quad \text{Change in vitality over time}$$

$$S(t) = P_v(t)P_w(t) \quad \text{The probability of being alive at age } t$$

$$r = \frac{\mu}{v_0} \quad \text{Rate of loss of vitality of a given population}$$

$$s = \frac{\mu}{v_0} \quad \text{Random component of vitality of a given population}$$

$$k \quad \text{Random mortality unassociated with vitality}$$

I used Salinger et al.'s (2003) vitality fitting program to determine the vitality parameters and the standard errors of the r-value. I had precise growth rates for PIT-tagged fish, and they did not differ significantly ( $p > 0.05$ ) from non-PIT tagged fish growth estimates, so I used the mean PIT-tagged growth rates as growth rates for each treatment. I regressed the vitality parameters from all the fish against the mean growth rates of the PIT-tagged fish and performed an ANOVA on the regression. (Zar 1999)

## 2.2 University of Washington 26° C Challenge Experiment

### 2.2.1 Selecting and transporting fish

On 5 November 2002, I collected approximately 300 fish, weighing 23.9 g/fish from the Washington State Icy Creek Hatchery ponds using a dip net. I transported the fish in an un-insulated oxygenated fish transport container from the Hatchery, in the Green River watershed, to the University of Washington, School of Aquatic and Fishery Science's hatchery. At the Icy Creek Hatchery, water temperature was 9° C. The only

available water at University of Washington at that time was 15° C. I placed the fish in circular tanks in a windowless lab. Artificial lighting matched current day length.

However, there was no dawn or dusk, only lights on or lights off.

### **2.2.2 Handling, Measuring and Tagging**

After nine days of acclimation, on November 12 and 13, I PIT tagged the fish using the methods described previously (Prentice et. al. 1987, 89, 90). Fish were also measured to the nearest mm of fork length and weighed to the nearest 0.1 g. Fish were weighed without sedation on 22 November, 10 December, 8 and January 2003. At mortality, the fish were again measured to the nearest mm FL length and weighed to the nearest 0.1 gram.

### **2.2.3 Feeding Fish**

I fed the fish a total of 3% of their weight twice daily from the time of their arrival at the hatchery until 10 December 2002. On 10 December, I began fasting the fish in tank 2. They were fasted for five weeks until 13 January, when the temperature treatment began. I continued feeding the fish in tank 1 until 1 January, when it became obvious that they would not eat; they quit feeding around 25 December 2002. Joan Thomas of Washington Department of Fish and Wildlife diagnosed the fish with *Ceratomyxa shasta*, a water-borne intestinal parasite (WDFW-fish program, 600 Capitol Way N, Olympia, WA 98501, pers. comm.).

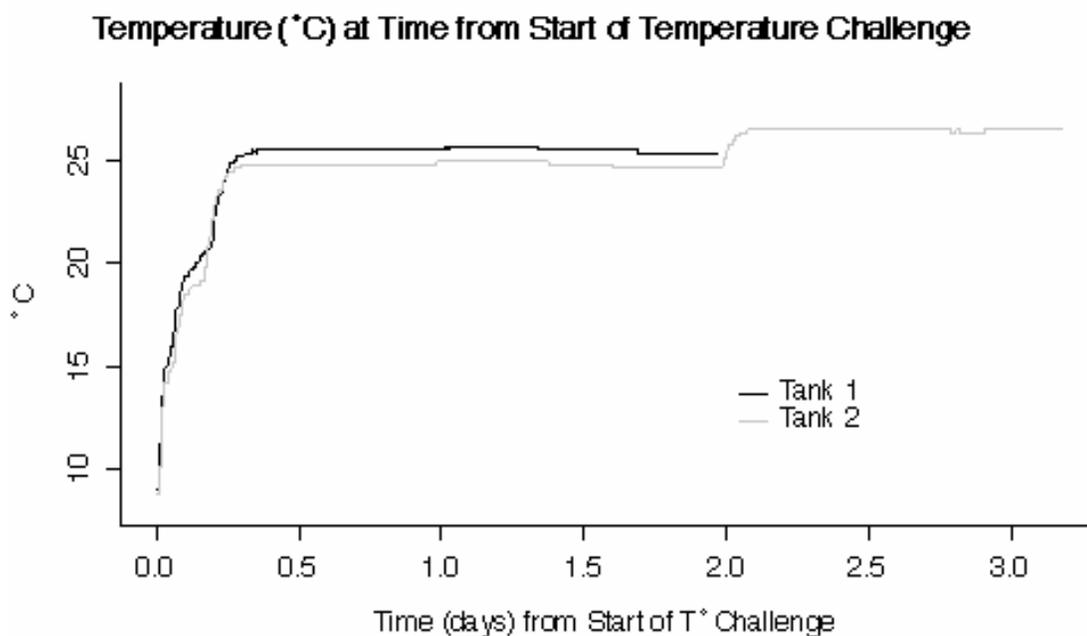
Despite feeding problems and early mortalities due to the disease, I ran the temperature challenge as planned on the remaining fish.

### 2.2.4 Fish Placement

I placed half the fish in each of two tanks. They remained in that tank throughout the feeding treatment. On 13 January, the fish were re-distributed between the tanks with half of the 0f fish and half of the 4f fish mixed in each of the two tanks.

### 2.2.5 Mortality Experiment

On 13 January, I increased the water temperature by gradually changing the mix of 9°C and 26°C water sources. Beginning at 11:50, the temperature increased from 8.9 °C in both tanks to 25.5°C and 24.8 °C in tanks 1 and 2 respectively over 8.6 hours (Figure 2.6). The temperature remained relatively constant from then until the point of 100% mortality. Dead fish were collected as they died, measured for length FL, and weighed to nearest 0.1 g.



**Figure 2.6** Temperature for all fish on 13 January

### **2.2.6 Analysis**

I performed ANOVAs testing whether weight, growth and MTM differed between treatment groups. I also performed ANOVAs of the regression of MTM against instantaneous growth and MTM against weight to test whether the slopes were significantly different from zero. I ran the data through the vitality model and produced vitality parameters.

## **2.3 Otolith Analysis of NMFS Bonneville Experiments**

Inspired by SAR data suggesting decreased survival with increased bypass system passage, NMFS conducted an experiment on the impacts of dam passage history on delayed mortality. I measured otolith increments from the resultant mortalities of their study. Their study tested whether survival in seawater tanks was related to dam passage history. Using the otolith measurements, I tested the hypothesis that survival was related to relative growth during the fishs' early life, 30 days post hatch. I hypothesized that the wider the increments were, the longer survival time would be.

### **2.3.1 NMFS Bonneville Methods**

In 2001, a trial year, they ran a limited version of the study in which 800 fish with one of two histories were held. Fish were either run-of-the-river fish collected at Bonneville Dam, or fish barged from the Snake River. All fish were reared in a closed, state-of-the-art artificial seawater holding facility that was constructed for this experiment. They were reared using standard husbandry techniques at 10°C and were fed to satiation three times daily. Mortalities were collected daily and frozen for necropsy.

During the necropsy, the sagittal otoliths were removed and donated to our study (Gilbreath et al. 2001).

### **2.3.2 Otolith Preparation**

I used a combination of standard mounting and grinding techniques outlined in Secor et al. (1992) and described to me by the Washington Department of Fisheries otolith lab (Grimm, J., WDFW-fish program, 600 Capitol Way N, Olympia, WA 98501, pers. comm.) Otoliths mounted in resin were ground on a grinding wheel half way through, until primordia were visible. The resin block was then glued otolith side down to a glass microscope slide and the excess resin was removed using an Isomet® saw. The otolith was then ground from the other side until the primordia were clearly visible and the otolith was translucent.

### **2.3.3 Otolith Increment Measurement**

Using Optimas® image analysis software, macros written for the software by Kim Larsen (United States Geological Survey, 6505 N.E. 65<sup>th</sup> Street, Seattle, WA 98115) and a compound microscope, I measured daily growth increments along a measurement axis. The axis originated and ran approximately 45° to a standard vertical axis drawn from the otolith rostrum through the otolith core. The software allows for accurate, reproducible results. I measured individual increments, and averaged all the widths, for approximately 30 days post hatch check depending on ring formation and grind quality. Volk et al. (1984) measured otolith increments in weekly periods due to large fluctuations in daily widths. Shorter durations of growth increase the uncertainty of measurements in relation to actual growth. Ca<sup>++</sup> deposition and increment width can vary due to a wide variety of

factors including quality and quantity of diet, temperature, and photoperiod (Irie, 1960, Nielson and Geen, 1982).

### **2.3.3 Analysis**

#### ***Otolith Analysis:***

I measured each otolith once on three days. I produced box plots and performed ANOVAs in an effort to find differences in average width among otoliths. I performed ANOVAs on the increment widths between samples. For each day of sampling, I regressed an individual otoliths average increment width against the  $T_{mort}$  of that fish.

### 3. Stress, Growth and Survival of Juvenile Chinook Salmon

#### Abstract

Understanding the effects of stress on survival of an individual is critical to exploring the effects of life history events on a population. Quantifying those effects would be extremely valuable for fisheries conservation and management. Using fasting and temperature challenge in a laboratory experiment, I tested the hypothesis that initial condition affects a juvenile chinook salmon's (*Oncorhynchus tshawytscha*) ability to survive under stress. The results were analyzed with traditional measures (time to 50% mortality, t50; mean time to mortality, MTM) as well as with Anderson's (2000) vitality model. I found significant differences in both MTM and t50 between fasted and non-fasted fish. In addition, there was a significant relationship between growth rate and time to mortality of individual fish. The regression of the vitality parameters,  $r$  = rate of loss of vitality and  $s$  = variability in rate of vitality loss, against growth rate was also significant. This paper demonstrates the effects of stress on survival can be quantified and explores possible effects of dams and reservoirs on stress.

#### 3.1 Introduction

Although it seems obvious that stressful events impact an individual's fitness, quantifying how stress decreases survival is difficult. More importantly, loss of individual fitness may have population level consequences. Thus, understanding the pathway of fitness from individuals to populations is critical for conservation biology, stewardship of wild populations, and aquaculture.

Fish, like other vertebrates, respond to stressors by changing physiological function to reallocate energy for the purposes of coping (Schreck et al. 2001). Responses to stresses are similar to fasting: mobilization of energy-rich substrates by depletion of hepatic glycogen stores, elevation of plasma levels of glucose, alteration of circulation levels of free fatty acids and general inhibition of protein synthesis (Mazeaud et al. 1977, Pickering 1981).

Chronic stress, or repeated acute stress with insufficient time for recovery could decrease survival through many pathways. Stress might increase an individual's susceptibility to disease, or decrease its growth rate or swimming ability, making it more vulnerable to predation. These effects may be cumulative. In Atlantic salmon, increasing the frequency of acute stress caused increasingly depressed growth (McCormick et al. 1998). In a salinity challenge, juvenile chinook traveling through the dam bypass at Lower Granite Dam in the Columbia River died more quickly the farther through the bypass system they were collected. This indicates increasing stress levels as they moved through the bypass (Park et al. 1986, Matthews et al. 1986).

Size has also been shown to be a factor influencing migratory behavior and subsequent survival to adulthood in juvenile salmonids (Zabel and Williams 2002). Large fish may grow faster, so that differences in behavior and survival of large and small fish might relate to growth rate (independent of current size), body size, or both factors (Beckman et al. 1998). Beckman et al. (1999) found that spring growth rate, but not fish size, correlated well with smolt-to-adult-return rate of juvenile chinook. Many

species exhibit pronounced individual differences in measured growth rates, often in response to variation in food resources (Hentschel 1999).

Food resource patchiness can produce discontinuous or intermittent feeding on the time scale of days may be common for juvenile fish (Letcher et al. 1996). Congleton et al. (2001) showed that Columbia River juvenile chinook migrating to sea are in a negative energy balance. Lipid and protein weight decreased from upstream to downstream dams and with increasing travel time. Blood indices indicated that the energetic deficit was, in a large part, due to restricted food intake. This was supported by a study of Muir and Cooley (1996) who found 29% of chinook sampled at Lower Granite dam had empty stomachs.

Letcher et al. (1996) found that variable feeding histories of perch larvae affected their ability to survive starvation in a laboratory experiment. Regardless of initial mass, perch lost mass proportionally at the same rate. However, perch fed intermittently before starvation died after losing proportionately less mass than fish that had been fed continuously. They hypothesized that this is related to compositional changes that occur during short periods of food deprivation.

Fasting involves adapting physiologically to limited consumption to maintain a level of metabolic homeostasis that can preserve vital organ function (Simpkins et al. 2003). During starvation, larvae lose lipids, carbohydrates and proteins (Erlich 1974; Molony 1993, Simpkins et al. 2003), reorganize enzymes (Mendez and Wieser 1993) and mobilize glycogen stores (O'Connell and Paloma 1981; Blasco et al. 1992). These changes might cause fish that have experienced short-term starvation to lose mass

differently (mobilize different energy stores) than fish that have fed continuously (Letcher et al. 1996).

These studies indicate that a fish's growth and size affect its survival within the next life stage and throughout life. Anderson (2000a) developed a model to quantify how these and other changes throughout an organism's life affect its survival. He used the term "vitality" to express where on the continuum from birth to death an animal resides.

The purpose of this study was to explore whether I could quantitatively relate growth history to a fish's ability to survive a future stress event. In this paper, I present the results of my research to estimate parameters for Anderson's (2000) vitality model and quantify the impacts of stress and growth on the survival of juvenile chinook salmon. I present the results in the context of the Columbia/Snake river hydropower system and its impacts on migrating juvenile chinook salmon.

### **3.2 Methods**

I investigated interactions between growth history, stress and survival by subjecting fish with three different growth histories to a uniform temperature stressor and measuring the mortality over time. I tested the hypothesis that faster growing, less stressed fish would survive longer in an elevated temperature challenge. I collected data to develop traditional descriptors of mortality from challenge experiments: time to 50% mortality ( $t_{50}$ ) and mean time to mortality or mean time to mortality (MTM). As these measures only provide one point on the survivorship curve, providing no information about the curve's shape, I also used the survival data to parameterize the vitality model.

All fish were selected from the same cohort of brood year 2001 chinook salmon at the fish hatchery of the School of Aquatic and Fishery Sciences, University of Washington. This ensured uniformity in every feature possible of the history of the young fish, minimizing effects of confounding factors like age differences, temperature experiences, feeding regimes, and growth rates. Fish weighed 3.6 g on average when selected.

On 11 March 2002, I implanted 150 fish with passive integrated transponder tags (PIT) using the method described by Prentice et al. (1987, 1989, 1990a, 1990b). On 13 March 2002, a second group of approximately 1,000 fish were selected by weighing out 3,600 g of fish. The two groups were placed in separate sections of a small raceway inside the hatchery. Tagged and untagged fish were kept separate throughout the experiment in case differential mortality rates were caused by handling and tagging. Fish were maintained in this raceway and fed 3% of their weight daily for one month under ambient light.

Juvenile salmon show a strong resistance to starvation. In a study by Akiyama and Nose (1980), 4 to 8 gram chum salmon (*Oncorhynchus keta*) fed continuously, then starved, survived up to 13 weeks. I calculated the mean time to mortality of those chum as 7.4 weeks and 9.5 weeks for 4- and 8-g fish, respectively. Thus, I hypothesized that a four-week period of fasting should not cause mortality in 4- to 5-g fish. On the other hand, two weeks of ration change caused hormonal responses and changes in growth for coho salmon (*O. kisutch*) (Pierce et al. 2001). Thus, I decided that two weeks of

starvation would be sufficient to alter the growth rates and initiate a stress response of juvenile chinook in this experiment.

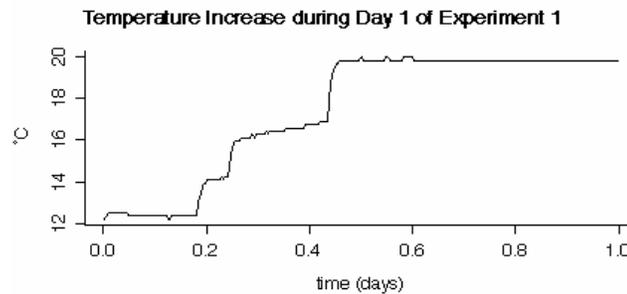
The fish were fasted for 0, 2, and 4 weeks and all were treated to the temperature stress simultaneously. On 10 April 2002, a month after tagging, feeding was stopped on the first group of fish, designated the 4f group (4-week fasted). Two weeks later, feeding was stopped for the 2f (2-week fasted) group. Feeding was not stopped for the 0f (0-week fasted) group until all fish were placed in the small circular tanks and under temperature stress on 10 May. The experiment consisted of three treatments with five replicates of untagged fish and three treatments with no replicates of PIT-tagged fish. Fifty fish were in each replicate.

At the beginning of the fasting treatment, and four weeks later, immediately before the onset of increased temperature, I weighed the fish. For PIT-tagged fish, I measured fork length to the nearest millimeter and weight to the nearest 0.1 gram. I group weighed (to the nearest 0.1 g) non-tagged fish. Individual weights of all fish were taken at the time of mortality.

The non-PIT-tagged fish were randomly distributed within their treatment groups to five replicate tanks per treatment. Tank arrangement was also random. All tanks received the same water supply for the duration of the experiment. Each PIT tagged group was placed in a separate tank on the same table with the same water supply. An unexpected side effect of the water delivery system was gas super-saturation. Although all tanks received the same water supply, there was some variation in dissolved gas saturation levels due to the angle of the stream into the tanks. This may have altered

early mortality rates in those tanks. When the super-saturation was discovered on the fourth day of the temperature treatment (mean=104.4%, sd=1.3), it was corrected to a level below super-saturation (mean= 98.2, sd=2.1).

On 10 May 2002, beginning at 07:50, I increased the water temperature 9°C (from 11.29 to 20.12°C) over 8 hours by gradually changing the mix of lake water (11°C) and well water (20°C) (Figure 3.1). The temperature remained relatively constant (<1 °C change) from then until the point of 100% mortality. Dead fish were collected and weighed once to four times daily.



**Figure 3.1** Temperature increase on 10 May 2002

From the non-PIT-tagged fish, I collected data on individual time to mortality, group weights at the beginning of the 4f feeding treatment, and group weights at the beginning of the temperature treatment. Data collected from the PIT-tagged fish were individual weights at the beginning of the 4f feeding treatment and at the beginning of the temperature treatment. Percent growth was defined by the equation

$$G = 100 \frac{\ln(W_i) - \ln(W_{i-1})}{t_i - t_{i-1}} \quad (1)$$

where:

$W_i$  = fish weight in grams at time  $t_i$ .

To characterize mortality I determined the time for 50% of a test group to die,  $t_{50}$ , and the mean time to mortality (MTM) of a test group. I performed ANOVAs of the regression of MTM against instantaneous growth for the PIT-tagged fish, and a Student's t-test comparing instantaneous growth rates of early and late mortalities in the PIT-tagged groups.

The  $t_{50}$  of the PIT and non-PIT tagged groups was nearly statistically indistinguishable. I therefore combined all the tanks, PIT and non-PIT, for analysis. I performed ANOVAs of MTM by treatment group. Creating box plots of lifespan by treatment, I determined the  $t_{50}$  and the 95% confidence intervals around the median.

Using the times of individual fish mortality ( $T_{mort}$ ) I constructed survival curves. These were fit to the vitality model survival curve in which survival at time  $t$ ,  $S(t)$  is defined (Anderson 2000a):

$$S(t) = \frac{1}{s\sqrt{t}} (1 - rt) \exp\left[-\frac{2r}{s^2} \frac{1}{s\sqrt{t}} (1 + rt)\right] e^{-kt} \quad (2)$$

where:

$t$  = age of a cohort

$r$  = average rate of loss of vitality

$s$  = variability in the rate of vitality loss

$k$  = rate of accidental vitality, which is independent of vitality

$\Phi$  = cumulative normal distribution function

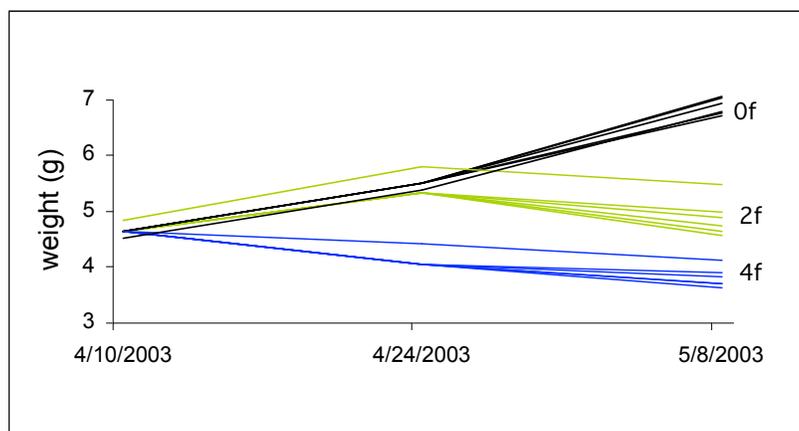
The parameters  $r$ ,  $s$  and  $k$  were obtained by fitting the equation to survival data using the method of Salinger et al. (in press).

### 3.3 Results

Growth and weight differed between treatment groups, as verified by ANOVA and Tukey's honestly significant difference post-hoc test. ANOVA analysis showed that the mean time to mortality of replicates differed significantly within treatment groups. I believe that this is related to differences in gas saturation levels between replicates within treatments (which induced gas bubble mortality). To compensate for the differences in saturation, all replicates were all combined for analysis. The non-PIT tagged group weights and growths were not significantly different than the PIT tagged fish.

The fasting treatments produced significantly ( $p < 0.05$ ) different weights between treatments at the end of the starvation treatment and beginning of the temperature treatment (Figure 3.2). Mean weights of non-PIT-tagged fish on 8 May for treatments 0f, 2f and 4f were 5.27, 4.28, and 3.27g, respectively. These average weights were within

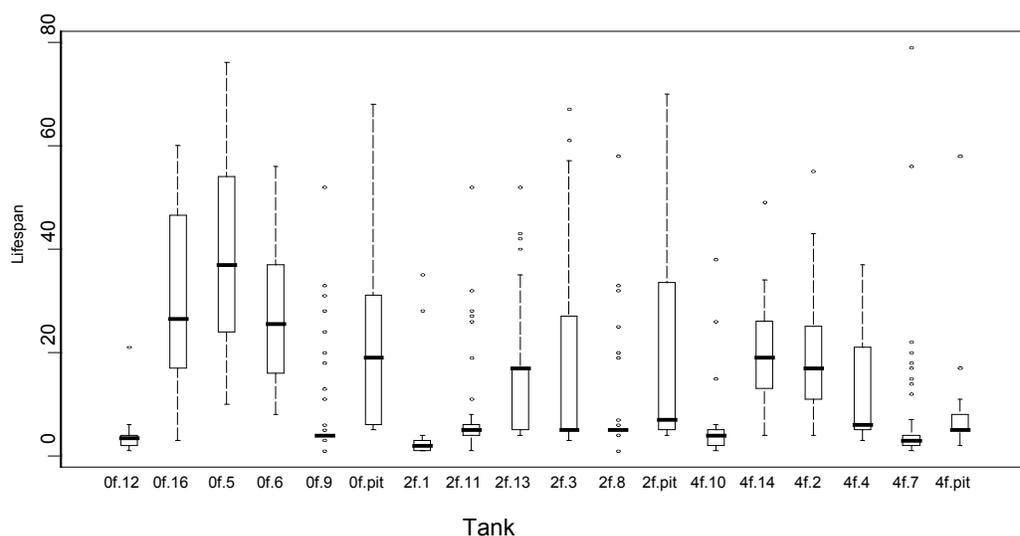
the one-quartile range of PIT-tagged fish. Mean weights of PIT-tagged fish on 8 May were 6.78, 5.46, and 4.12g for the 0f, 2f and 4f groups, respectively. Mean instantaneous growth rates during the feeding treatment for the PIT-tagged 0f, 2f, and 4f groups were 1.42, 0.42, and -0.30% per day, respectively.



**Figure 3.2** Mean fish weights determined by tank weights before, during and at the end of the fasting treatment. The 0f fish (□) were not fasted. The 2f fish (□) were fasted for 2 weeks beginning on 24 April. The 4f fish (□) were fasted for 4 weeks beginning on 10 April.

### ***Median time to mortality***

Fish t<sub>50</sub> varied within treatments (Figure 3.3). However, when treatments were combined, t<sub>50</sub> of 0f was significantly different than both 2f and 4f ( $p < 0.05$ ), but was not significantly different between 2f and 4f. Median time to mortality (t<sub>50</sub>) in days for each treatment ranged from 5.0 for 2f fish to 17.5 for 0f fish (Table 3.1). The difference in t<sub>50</sub> between 2f and 0f varied between 12.4 and 6.5 days. The difference t<sub>50</sub> between 0f and 4f varied between 11.9 and 5.9 days.



**Figure 3.3** Box plot of lifespan for all tanks sorted by treatment. With median (solid dark line), upper and lower quartiles (edges of box), and 95% confidence intervals around median (whiskers).

**Table 3.1** Statistical analysis showing t50 and MTM for treatments with all replicates combined, including PIT tagged fish. MTM std is the standard deviation around the mean time to mortality.

Treatment	Lower 95%CI	t50	Upper 95%CI	MTM	MTM Std	N
0f	14.60	17.5	20.39	20.98	18.33	250
2f	3.79	5.0	6.21	11.55	13.69	288
4f	4.59	6	7.41	12.07	11.40	282

### *Mean time to mortality*

With all replicates and PIT-tagged groups combined (Table 3.1), the MTM of the 0f group was significantly different than 2f and 4f groups ( $p < 0.05$ ). Tukey's Honestly significant difference test demonstrated that the 0f MTM was 9.4 days longer than the 2f

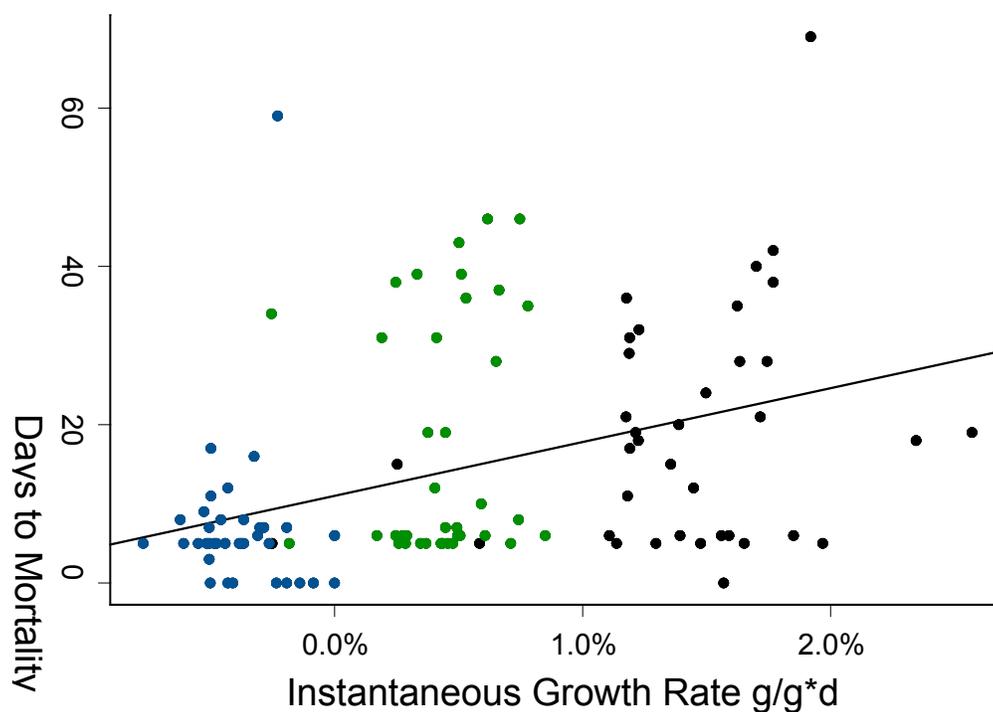
MTM and 8.9 days longer than the 4f MTM. The 95% confidence intervals around both those differences were 3 days. There was no significant difference between 2f and 4f treatments.

***Time to mortality vs. growth rate***

Individual growth rate measurements and individual times to mortality from the PIT-tagged fish allowed me to regress survival against growth history (Figure 3.4). The regression,

$$\text{Days to Mortality} = 11 + 6.8 * \text{Instantaneous Growth Rate}$$

was significant ( $p < 0.05$ ), but weak ( $r = 0.13$ ), and suggests that an increase of 1% in growth rate increased the time to mortality by an average of seven days. Comparison of fish that died in the first 10 days to those that died after 30 days showed that growth rates of the earlier mortalities were significantly slower than growth rates of the later mortalities (t-test;  $p < 0.05$ ).



**Figure 3.4** Growth rate during feeding treatment regressed against lifespan for individual PIT-tagged fish. 0f fish ( $\bullet$ ), 2f fish ( $\bullet$ ) and 4f fish ( $\bullet$ ) are marked.

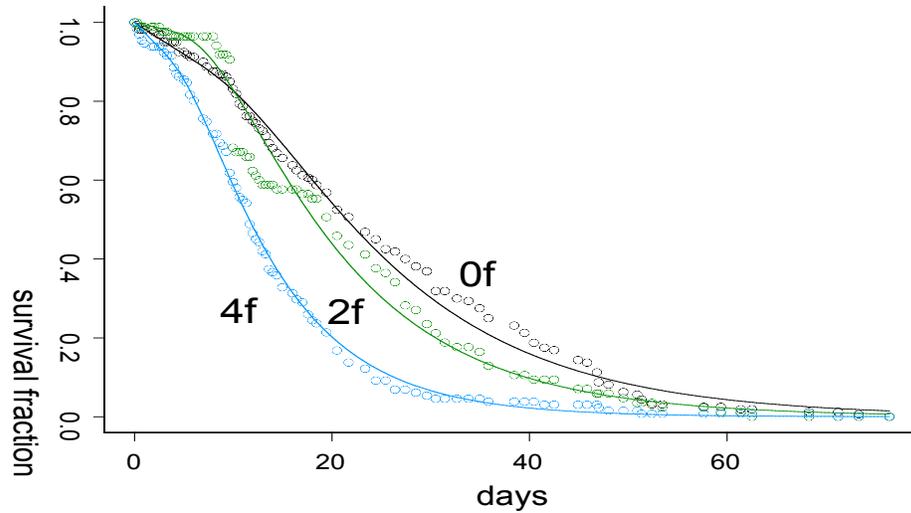
### *Vitality*

I fit the survival data to the Anderson (2000a) vitality curve to estimate the vitality parameters for each treatment group. In these analyses, however, I excluded the data from the period of super-saturation and characterized survival curves for the fish alive after the period of gas super-saturation. The exclusion was necessary to meet the model assumption that the stressor level must be essentially constant over the duration of the survival curve. The start of the challenge period was set at 16 May, six days after the beginning of temperature challenge and two days after the super-saturation was eliminated. With this adjustment, the pre-challenge stressors of these fish included 0, 2 and 4 weeks of starvation plus four days of exposure to elevated temperature and total

dissolved gas. Survival curves (with PIT-tagged and non-tagged fish combined for each treatment group) were steeper for 4f than 0f fish (Figure 3.5). I used the Salinger et al. (in press) fitting program to determine the vitality parameters and the standard errors of the parameter  $r$  for each treatment. The rate of loss of vitality,  $r$ , was highest for the 4f group and lowest for the 0f group (Table 3.2).

**Table 3.2** Vitality Parameters, for each treatment with all tanks combined and start day after super-saturation.  $G$  is instantaneous growth rate during the feeding treatment. The  $r$ -parameter is the rate of loss of vitality. The  $se(r)$  is the standard error of the  $r$ -parameter. The  $s$ -parameter is the variation in rate of loss of vitality. The  $k$ -parameter is the accidental mortality.

Treatment	$G$ (%/day)	$r$ (day <sup>-1</sup> )	$se(r)$	$s$ (day <sup>-1/2</sup> )	$k$ (day <sup>-1</sup> )
0f	1.42%	0.0282	0.0022	0.1034	0.0167
2f	0.42%	0.0422	0.0031	0.1311	0.0057
4f	-0.3%	0.0533	0.0044	0.1525	0.0262



**Figure 3.5** Vitality model fit of the cumulative survival for all tanks combined for each treatment group.

Anderson (2000a) demonstrated that the rate of vitality loss is proportional in stressor level in many stressor-organism systems. To determine if growth related to vitality, parameters derived for each treatment group were regressed against the instantaneous growth rate  $G$ . Because precise growth rates were available for PIT-tagged fish, and they did not differ significantly from non-PIT tagged fish growth estimates, I used the mean PIT-tagged growth rates for each treatment. Regressions were significant at the ( $p < 0.05$ ) for  $r$  vs.  $G$  and  $s$  vs.  $G$ . The regression of  $k$  vs.  $G$  was not significant (Table 3.3).

**Table 3.3** Regression equations and r-squared values for vitality parameters against growth.

Parameters	Equation	r-squared
$r$ vs. growth	$r = 0.048 - 1.36 * G$	0.999
$s$ vs. growth	$s = 0.142 - 2.66 * G$	0.998

### 3.4 Discussion

The results show a clear relationship between growth and survival under stress that is consistent between analyses explored. As hypothesized, fish that fed continuously survived significantly ( $p < .05$ ) longer under temperature stress than those that did not feed prior to the onset of temperature stress. Complications with gas super-saturation may have altered the results, but this effect was, by chance, higher in the 0f treatments and, if anything, would have diminished the differential mortality between groups. It is likely that the large amount of variation in time to death within treatments (Figure 3.3) was influenced by the variation in gas saturation between the tanks. When replicates were combined, the MTM and t50 results were significant despite the early deaths due to super-saturation.

The vitality model analysis supports Anderson's (2000) findings relating the rate of vitality loss to environmental stressors. That is, the survival of an organism over its life history can be characterized by how stressors affect the vitality rate parameters. My results (Table 3.3) demonstrate that the rate of vitality loss was linearly related to growth rate across the treatment group. The negative slope of  $r$  with  $G$  indicates that as the

growth rate increases the vitality loss rate decreases. Correspondingly, the lower the vitality loss rate, the longer the organisms live. Thus, as found in the MTM and t50 analyses, high growth rates improve survival. The vitality rate variance,  $s$ , was also linearly related to  $G$  and again higher growth rates correspond with lower  $s$  values. A higher value of  $s$  indicates more variability in the rate of loss of vitality in members of a treatment group. Thus, members of the treatment group that fasted four weeks exhibited greater variation in vitality than members of the treatment group that were fed continuously prior to the temperature challenge. Anderson (2000) found a similar result in feeding experiments of *Asplancha brightwelli*. In that experiment, feeding stress also increased both  $r$  and  $s$  resulting in greater variation in the vitality of starved organisms than fed organisms.

Although I found no significant difference between 2f and 4f groups with MTM and t50 analyses but did note differences in terms of  $r$ , the results were not unexpected. In the former analyses, the mortality data included the period of mortality under super-saturation, while the vitality analysis excluded this data. For all analyses, the basic conclusion is clear: lack of food decreases the ability of fish to survive a stress challenge.

These studies consistently showed that starvation decreases the ability of fish to survive a temperature challenge under laboratory conditions. The question remains, What does this finding mean in terms of the effects of feeding schedule on survival under natural conditions? Due to the temporal and spatial patchiness of food availability in the aquatic environment, periods of food deprivation may be common in the life of young fish (Mendez and Weiser 1993). Variable feeding history has been shown to affect yellow

perch larvae's ability to survive starvation (Letcher et al. 1996). In the Columbia and Snake rivers system, food availability may be limited in the reservoirs. Curet (1993) estimated that food consumption of juvenile chinook only slightly exceeded maintenance ration in the upper two reservoirs. Congleton et al. (2001) showed that chinook are in a negative energy balance as they migrate to sea. This could affect their ability to survive when they reach the ocean through size selective predation, disease, or starvation. For salmon, the majority of marine mortality occurs early in the oceanic phase (Pearcy 1992). At that time, hundreds of millions of wild and hatchery fish co-occur in a near-shore region of the ocean. Hatchery production of chinook may thus exacerbate food limitation during less productive periods (Hilborn and Eggers 2000, Levin et al. 2001). If the resources were limited on the way to the ocean, the salmon are less likely to survive competition and predation when they reach it.

My results support other studies correlating fish size with survival (e.g., Ward and Slaney 1988, Ward et al. 1989, Bilton et al. 1982, Bilton 1984, Marin and Wertheimer 1989, Hagar and Noble 1976, Zabel and Williams 2002, and others). It also supports Beckman's (1999) assertion that growth rate affects survival to returning adult.

These results are particularly applicable to the Columbia/Snake rivers system. By linking current survivability to past experiences, such as feeding history, this experiment provides a small step towards linking freshwater life history and ocean survival of juvenile salmon and steelhead. Evidence is accumulating that cumulative stress may affect a fish's ability to survive. This is important because multiple studies have shown links between dam passage and survivability (Park et al. 1986, Matthews et al. 1986,

NMFS 2000, Karieva et al. 2000, Budy et al. 2002) but the mechanism of the delayed mortality is unclear. Thus, challenge experiments interpreted in the vitality framework provide one possible approach to quantify the significance of stress on delayed mortality of fish. Such studies will be necessary for quantifying effects and making inferences on the effect of structural and operational changes in the Columbia/Snake hydropower system and elsewhere.

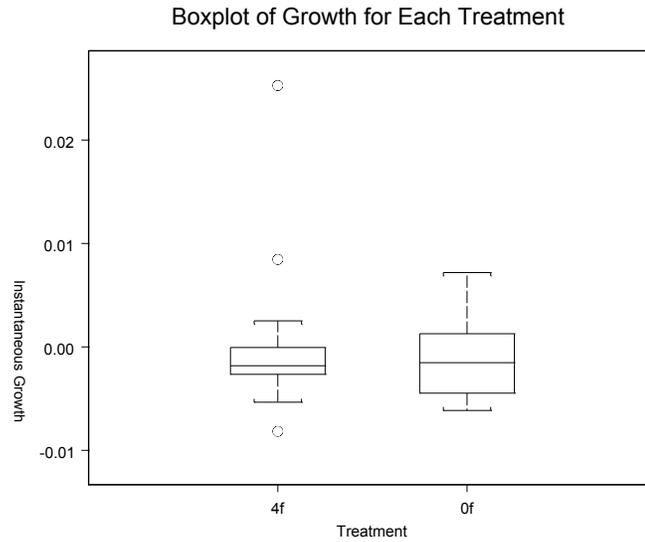
## **4. 26° C Temperature Challenge**

### **4.1 Introduction**

Due to the super-saturation complications with the 20 °C challenge experiment, and the longer than mean time to mortality of those fish, I ran the experiment a second time. The hypothesis remained the same, that faster growing, less stressed fish would survive longer in an elevated temperature challenge. However, I used 26 instead of 20 °C water. The methods are described in Chapter 2.

### **4.2 Results**

Differences in weight between the 0f and 4f treatment groups after the feeding treatment were insignificant (ANOVA and Student's t-test,  $p=0.52$  and  $0.42$ , respectively). There was no significant difference between the groups in growth (Figure 4.1).



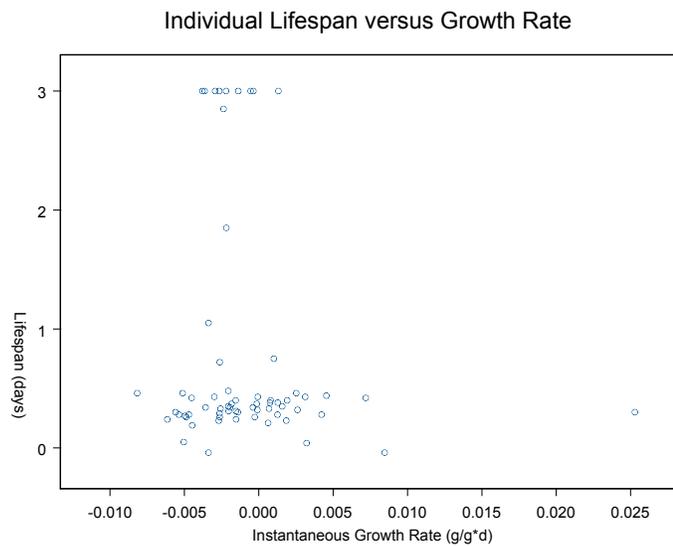
**Figure 4.1** Box plot of instantaneous growth split by treatment. The line in the box is the median, upper and lower edges of box are one quartile, and whiskers are 95% confidence intervals around median.

There was no significant difference in estimated MTM between the two treatment groups either within each tank or when both were combined. Mean MTMs were 0.666 and 0.819 days, respectively. However, there was a significant difference in MTM between the two tanks ( $p < 0.05$ ), where mean MTMs were 0.403 and 1.058 days for tank 1 and tank 2, respectively (Figure 4.2).



**Figure 4.2** Box plot of lifespan split by treatment. The line in the box is the median, upper and lower edges of box are one quartile, and whiskers are 95% confidence intervals around median.

Regression analysis of instantaneous growth rate against lifespan of individual fish showed no relationship ( $p=0.3$ ,  $r^2=0.001$ ) (Figure 4.3).



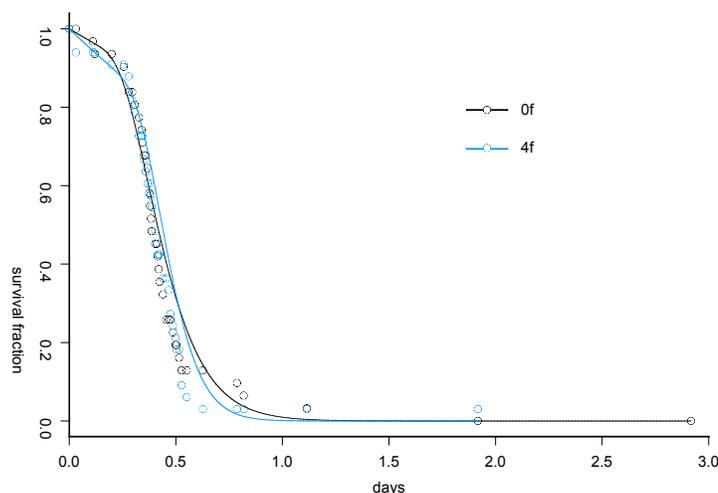
**Figure 4.3** Scatter plot of individual lifespan versus instantaneous growth rate for all fish.

Vitality analysis also shows no difference between the groups (Table 4.1, Figure 4.4).

**Table 4.1** Vitality parameters of 0f and 4f treatment groups.

	r	s	k
0f	2.112	0.555	0.321
4f	2.040	0.379	0.518

Cumulative Survival Data and Vitality Model Fitting for 26 C Challenge



**Figure 4.4** Vitality model fit of survival curve for each treatment.

### 4.3 Discussion

The lack of difference in growth rate is due to a different stressor than imposed in the experiment, i.e., an intestinal parasite that caused the fish to cease eating. Joan Thomas of Washington Department of Fisheries identified the parasite as *Ceratomyxta shasta*. I suspect that Green River salmon stocks have not evolved a resistance to *C. shasta*, because it is not present in that watershed (Joan Thomas, pers. comm. Washington Department of Fish and Wildlife, 600 Capitol Way N, Olympia, WA 98501).

Despite the lack of results from the data, this experiment clarified some experimental design issues. The higher water temperature of this experiment produced smoother survival curves and better fits with the vitality model. In addition, combining PIT tagged fish of multiple treatment groups in one tank for the temperature challenge ensured that all the groups were undergoing to same secondary stress. If I were to continue these experiments, I would pursue this approach to the experimental design.

## 5. Results and Discussion of NMFS Experiment

### 5.1 Otolith Increment Widths

I hypothesized that early life growth might affect survival. Specifically, I tested the hypothesis that the average increment width of fish from the NMFS Bonneville seawater experiment that died early would be smaller than increments of those that died later. There were no significant differences in increment widths between samples ( $p>0.05$ ) (Figure 5.1). Therefore, the regression of mean increment width against lifespan was not significant either ( $p>0.05$ ).

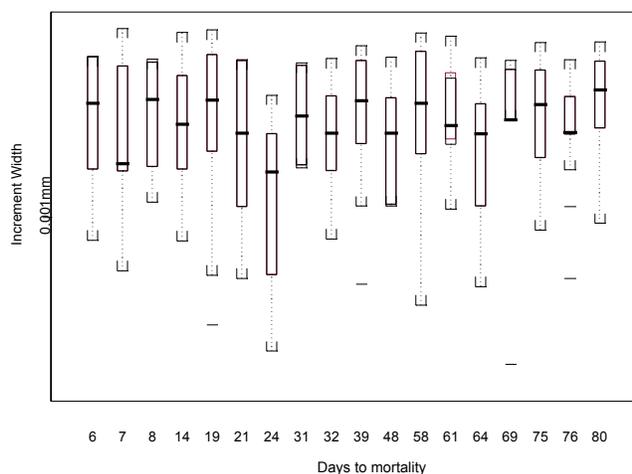


Figure 5.1 Boxplot of otolith increment widths by samples in order of day of mortality. Each box is all the increments measured on that otolith.

This might have been the case because I was only measuring the first 30 days after hatch. During this time, juvenile chinook are still receiving most of their nutrients from their yolk sac (Weatherly and Gill 1995). At this point, differences in growth rate are related to efficiency of yolk utilization and the amount of yolk present. Yolk conversion efficiency depends heavily on the regime of water temperature. Other factors

include darkness and water exchange (Weatherly and Gill 1995). All of my samples were from the same brood year from Rapid River Hatchery. Therefore, they likely experienced the same temperature and light regime, which would decrease the differences in growth rate between individuals during that time.

In addition, I only had 30 quality slides. It is possible that with more samples, a pattern might have emerged. If I were to pursue this research, I would ensure that there were more samples available and measure farther out from the hatch check. I would also use samples from multiple origins, including wild fish.

## 6. Conclusions

Salmon stock declines in the Columbia River have been independently linked to ocean condition (Beamish and Boullion 1993, Francis and Hare 1994, Hare and Francis 1995, Adkinson et al. 1996, Beamish et al. 1997a, Beamish et al. 1997b, Mantua et al. 1997; Percy 1997, Johnson 1998, Beamish et al. 1999, Beamish et al. 2000, Anderson 2000b, Batchelder and Powell 2002), river conditions (Zabel and Williams 2001, Budy et al. 2002) and fish condition (Beckman et al. 1998, Beckman et al. 1999, Zabel and Williams 2001). I investigated these links using laboratory work to establish whether the initial condition of a fish affects its ability to survive under successive stressors. The comprehensive hypothesis is that the more stresses a migrating juvenile salmon experiences, the less likely it is to survive future stressors whether they occur in the river, the estuary or the ocean.

There is evidence from both laboratory and field work that cumulative stress affects survival. Matthews et al. (1986) showed that as fish travel through the bypass system at dams, their stress level increases and that they die more quickly in a salinity challenge than fish collected before or closer to the entrance to the bypass. Letcher et al. (1996) used feeding history to create fish with different initial conditions and established that fish that had experienced fasting in their past did not survive as long under starvation stress as those that had been fed continuously. McCormick, et al. (1998) found that short periods of intense stress resulted in reduced growth rate of juvenile Atlantic salmon and that growth depression increased with increased frequency of stress. Growth has also

been shown to relate to survival. Beckman et al. (1999) found that spring growth rate, but not size, of juvenile chinook correlated to SAR.

I used several approaches to investigate the effects of stress on survival in juvenile chinook salmon. I performed two fasting and temperature experiments at the hatchery at the University of Washington. In these experiments, groups of fish with different feeding histories were subjected to temperature stress and the time to mortality was determined for the individual fish. Second, I analyzed otoliths from an experiment performed by NMFS where I used increment width as a measure of growth test the hypothesis that fish grown faster in their youth would survive longer in a seawater challenge experiment.

Statistical analyses demonstrated that there was a link between growth/feeding history and survival in challenge experiments. To further quantify the link, the resulting survival curves were fit with a vitality based survival equation that characterizes the effects of stressors on survival (Anderson 2000a). Whereas the simple statistical analyses demonstrated a link between growth and survival to a challenge stress, the fit of the data to the vitality survival model provided a way to quantify the effect of the growth stress in terms of a simple but mechanistic description of the survival processes. Thus, my work demonstrates that the link between growth and survival can be quantified in terms of vitality. This demonstration may be important because Anderson (2000a) suggests that a vitality approach can be used to relate stressors to organism survival in a natural life-history setting. Specifically, Anderson (2002) suggests laboratory based challenge

experiments interpreted in a vitality framework may be useful in understanding how freshwater stress factors affect SARs.

However, my approaches to quantify the link between growth and survival had variable success. The results of the 20° C challenge supported the hypothesis that the initial condition of a chinook, in this case its feeding history, affects its ability to survive under a secondary stress. Additionally, the experiments demonstrated that survival curves could be quantitatively related to the growth rate through the vitality model. In the 26° experiment, an intestinal parasite caused the fish to stop eating and made it impossible to create treatment groups; i.e. all fish starved. All fish lost weight in a similar manner and the two groups had similar survival curves. Although it was not possible to relate growth differences, the experiment did demonstrate again that the survival of fish subjected to a challenge stressor could be quantified with the vitality model.

Furthermore, the experience of the two experiments did provide useful information for future design of challenge experiments. Data from the 26 ° C experiment survival curves fit the vitality model more precisely than the 20 ° C experiment. 20° C, though stressful, has not been shown to be a lethal temperature. 26° is, however, above the upper lethal limit (Brett 1952). The extended length of the 20° experiment may have complicated the vitality analysis. In future challenge experiments, I recommend the higher temperature. A second recommendation from the 26 ° C experiment is that the fish be individually marked by their initial stress treatment and then mixed together before the secondary stress treatment. This would ensure uniform stress on the two treatment groups. All the vitality analyses illustrate that the stressor must be constant to

ensure reliable vitality results. In this sense, the 26° experiment was successful because one of the goals of this work was to establish whether temperature is an effective treatment for achieving mortality.

The otolith analysis showed no correlation between early growth and survival later. There was no measurable difference in early growth rate between samples. Therefore, there was no correlation with survival time. I think that it would be interesting to pursue this by measuring growth closer to the time of mortality and with fish with known histories.

Both the traditional and vitality analysis of the 20° C experiment clearly establish that certain aspects of the initial condition of a fish, such as feeding history and growth, affects its ability to survive a secondary stress. This is applicable to Columbia River juvenile salmonids that migrate hundreds of kilometers from their hatcheries and nursery streams, negotiating up to eight dams, to reach the ocean. Main influences on energy balance and growth during the migration are water temperature, flow velocity and food availability (Congelton et al. 2001). Water temperature affects metabolic rate, river velocity is inversely related to migration duration, and food availability directly affects foraging success. These three environmental factors are all affected by the hydrosystem. River velocities have been dramatically altered by impoundments and therefore the duration of migration for juvenile chinook has been extended. The ability of fish to prolong the migration period and sustain the smolt-associated metabolism for three to six weeks longer than the pre-impoundment conditions in which they evolved, may depend on the availability of food in the hydrosystem reservoirs (Congleton et al. 2001).

Information on food availability in Columbia River reservoirs is scant, but it appears that food supply for juvenile chinook is limited in the upper two Snake River reservoirs (Lower Granite and Little Goose). Muir and Cooley (1996) found 29% of fish sampled from turbine intakes at Lower Granite Dam had empty stomachs. Curet (1993) estimated that food consumption by sub-yearling chinook in the Snake River reservoirs just slightly exceeded maintenance ration in April, May and June. Comparatively, at McNary and Bonneville dams, only 3 and 5% of fish had empty stomachs, respectively (Muir and Cooley 1996).

Juvenile chinook from the Snake River Basin were in negative energy balance throughout their migration and lipid usage appeared to increase with increasing travel time (Congleton et al. 2001). Congleton et al. (2001) estimated that a twenty day increase in travel time to John Day Dam resulted in an additional 41% decrease in lipid reserves. Decreases in blood hormone indices indicate that the energetic deficit was in a large part due to restricted food intake (Congleton et al. 2001).

My results indicate that two weeks without food decreases survival under a secondary stress (in this case, temperature). This could indicate that Snake River fish are less likely to survive than lower Columbia River fish due to prolonged periods with limited food affecting their resistance to secondary stressors.

The results also provide a foundation for future research. Two main areas may be especially productive. One is the physiology of growth and starvation and its effects on survival. My work did not address the physiology of the interaction, but it did show a link between starvation and survival. Establishing the physiological mechanisms of the

link would be useful. Second, this thesis supports the Anderson (2000a) vitality model's usefulness as a tool to relate the effects of stresses on survival of fish in the wild. Stress challenge experiments conducted on fish during their hydrosystem passage may provide valuable information on their ability to survive the transition to their salt-water life history stage.

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