PHYSIOLOGY, BEHAVIOUR AND MORTALITY OF ADULT FRASER RIVER SOCKEYE SALMON AS THEY MIGRATE UPSTREAM TO SPAWN

by

JEFFERY YOUNG
B.Sc. (Hons), University of Calgary, 2000

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE STUDIES
(Forestry)

THE UNIVERSITY OF BRITISH COLUMBIA
August 2006

© Jeffery Young, 2006

ABSTRACT

Sockeye salmon (*Oncorhynchus nerka*) undertake arduous upstream spawning migrations to complete their anadromous lifecycle. The success of this migration requires intensive energy use, complex physiological changes, and reproductive development. For the first time, I examine the relationships between individual physiology, migration timing, and mortality for a meta-population of sockeye salmon (late-run adults of the Adams-Shuswap stock complex) as they passed a single point in their upstream migration (i.e., in the Thompson River situated 270 km from the coast). Since 1995, large segments of the late-run sockeye salmon stock complex from the Fraser River, British Columbia, Canada, have been initiating spawning migrations several weeks earlier than normal. Most early migrants die before spawning. To evaluate the mechanisms underlying the mortality, I non-lethally assessed physiological and energetic status, and tracked individuals using gastrically-inserted radio transmitters. Early migrants had higher gross somatic energy and lower reproductive hormone titres. However, aberrantly early migrants that failed to reach the spawning grounds had lower gross somatic energy and higher plasma reproductive hormone titres than aberrant migrants that successfully reached spawning grounds. Aberrant migrants that did not reach spawning grounds also had higher average migration ground speeds and higher plasma osmolality. Plasma lactate was higher in early migrants, which experienced higher water temperatures. Other physiological measures of stress were not related to migration timing, mortality, or environmental conditions. Plasma glucose was lower in early migrants, possibly influenced by reproductive development rather than stress. Fish surgically fitted with electromyogram radio transmitters did not continue their migration and fell downstream. These fish

displayed excessive bleeding during transmitter implantation, an unusual phenomenon that likely contributed to the fish's inability to resume migration. Blood clotting time decreased steadily throughout the migration period. Collectively, these data support the strong inverse relationship between somatic energy and reproductive development during upstream migration and implicate a combination of energy depletion, premature reproductive development, and blood loss from wounds as potential contributors to mortality in aberrantly early migrating sockeye salmon of the Fraser River late-run stock complex.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

ACKNOWLEDGEMENTS

I would like to thank Dr. Scott Hinch for his considerable guidance and support. I would also like to acknowledge Dr. Tony Farrell and Dr. Steven Cooke for assistance in the design and execution of this research. Many thanks are needed to the members of the Hinch lab, who were always willing to provide advice and unconditional support.

All procedures used in this study were approved by the Canadian Council on Animal Care administered by the University of British Columbia, Simon Fraser University, and Fisheries and Oceans Canada. I thank Steve Macdonald, Al Cass, Laura Richards, Jim Cave, Jim Woodey, Mike Lapointe, Timber Whitehouse, Merv Mochizuki ,and others from Fisheries and Oceans Canada and the Pacific Salmon Commission for facilitating this project. Tagging and physiological sampling support was provided by Ray Drynock and Jim Ferguson. I also acknowledge the invaluable assistance of Ray Drynock and the Nicomen Band with fish capture on their lands. Telemetry data management and receiver maintenance was conducted by Jim Ferguson, Bill Koski, Nathan Blakley, and Cezary Sliwinski. Physiological assays were conducted by Jayme Davidson and Jeanette Garries. Funding for the telemetry component of the study was provided by a contract to LGL Limited from Fisheries and Oceans Canada. The bio-sampling component of the project was funded by a Natural Sciences and Engineering Research Council (NSERC) Strategic grant and NSERC Discovery grants, and the Fraser River Environmental Watch Program. I was supported by an NSERC graduate scholarship.

CO-AUTHORSHIP STATEMENT

This thesis forms an integral component of a multi-disciplinary research program. Although the design, fieldwork, and analyses for research supporting this thesis was led by Jeffery Young, the identification and design of the broader research program was led by Dr. Scott Hinch, Dr. Steven Cooke, and Dr. Tony Farrell. Co-authorship of manuscript versions of Chapters 2 and 3 of this thesis credit the role of these scientists along with those individuals providing expertise and methodological support for the multi-disciplinary components of each manuscript. For all research provided in this thesis Jeffery Young held primary responsibility for the identification and design of the research program, as well as undertaking the field research, performing most of the subsequent physiological analyses, and writing of manuscripts. Those individuals that assisted with the performance of the research and analyses are mentioned in the acknowledgement section of this thesis.

CHAPTER 1 - INTRODUCTION

Background

Pacific salmon (*Oncorhynchus* spp.) are an integral component of freshwater and marine ecosystems across the North Pacific. In the marine environment Pacific salmon travel across vast coastal and deep ocean regions providing an abundant food source for marine mammals, fish, invertebrates and seabirds, while playing a significant role as nektonic and piscivorous predators (Cederholm et al. 2000). Pacific salmon provide a unique input of marine-derived nutrients and energy to coastal and inland freshwater and terrestrial ecosystems (Naiman et al. 2002; Schindler et al. 2003). Sockeye salmon (*O. nerka*) are one of seven species in the Pacific salmon genus, but play a unique and disproportionately important role in British Columbia as the second most abundant anadromous species and through their unique use of lake ecosystems for early rearing (Groot and Margolis 1991). Sockeye salmon in British Columbia typically rear in lakes for two years, migrate to the ocean in spring, and rear in the ocean for two to three years before returning to their natal stream or lake to spawn.

The life cycle of all anadromous Pacific salmon involves reproduction in freshwater and adult rearing in the marine environment. Species vary in the amount of time spent in these environments, the timing of their migration, and the specific habitat types they utilize (Quinn 2005). Unique life history and physiological attributes are exhibited by Pacific salmon including anadromous migration, the ability to "home" to their natal spawning area, semelparity, rapid growth but short lifespan, large eggs, female parental care, highly productive populations and generalist feeding/habitat use.

Migration between freshwater and the ocean occurs twice in this life cycle and both migrations present significant physiological and energetic challenges. The migration of adult salmon from the open ocean, through coastal waters, estuary, and into freshwater streams requires unique physiological capabilities. Of fundamental importance is the energy necessary to meet the exercise demands of this often long distance and arduous upstream journey, while also partitioning energy to reproductive development (Brett 1995). These complex energy demands occur as feeding ceases and the salmon enter a catabolic state.

Despite the importance of migration success to the survival and reproduction of Pacific salmon there remains a lack of understanding of migration physiology and the factors responsible for migration success and failure (reviewed in Hinch et al. 2005). This information gap limits the ability to manage fisheries for sustainable benefits and implement effective actions to conserve endangered species or stocks. Although many aspects of migration physiology have been intensively studied, the vast majority of this research has not integrated multiple physiological components or related these components back to other critical migration factors, such as timing, environmental condition, or behaviour.

This study uses telemetry and the measurement of a suite of physiological variables to refine my understanding of the migration of Fraser River sockeye salmon. In particular, this study evaluates the relationships between migration timing, behaviour, and mortality with physiological measures of reproductive development, energy stores, stress, and osmoregulation. Greater understanding of these mechanisms and the identification of indicators suitable for increasing the predictability of migration success or failure will

improve the ability to plan fisheries, develop recovery plans for depleted populations, and hone in-season management measures.

Literature review

Migration energetics

The exercise demands of upstream migration can be very high, a concept well demonstrated in sockeye salmon of the Fraser River (reviewed in Hinch et al. 2005). Sockeye salmon exhibit local adaptions to river conditions (MacNutt et al. 2006) and individuals from populations with more distant freshwater spawning grounds tend to start the freshwater migration with higher energy stores. Although exercise demands far exceed the energy needed for standard metabolism (Crossin et al. 2004), energy must be maintained for reproduction, including displays, competition, nest digging, mating, and nest defence by the female (Groot and Margolis 1991). During the migration sockeye salmon must effectively use energy to alter their osmoregulatory state and reproductively develop, including gonad growth and body morphology changes associated with secondary sexual characteristics (e.g., male kype and dorsal hump). Challenging migration conditions, such as high flows or temperatures, can delay migration and contribute to the depletion of energy to levels unlikely to support successful migration (Rand and Hinch 1998; Lee et al. 2003).

The development of gonads is energetically costly, particularly for females, which allocate approximately 14% of their energy stores to egg development (reviewed in Brett 1995). Stored energy must be effectively partitioned between gonad development and swimming with potential trade-offs between the two. Individuals that encounter adverse environmental conditions that deplete energy stores will have less energy available for

gonad development and are more likely to reach a critical energy threshold associated with death (i.e., ~4 $MJ \cdot kg^{-1}$; Crossin et al. 2003). However, Patterson et al. (2004) also demonstrated that although adults not subjected to exercise partition more energy to eggs, these non-exercised fish had lower egg deposition rates, were more likely to die prior to ovulation, and produced eggs with lower survival rates. Thus, energy used for migration must fall within an upper and lower threshold to maximize success of migration and reproduction.

Migration physiology

Reproductive development occurs during the freshwater migration and is controlled by reproductive hormones (Ando et al. 1985; Ueda et al. 2000). The three reproductive hormones measured in this study: testosterone, 11-ketotestosterone, and 17β-estradiol stimulate upstream migration behaviour (Munakata et al. 2001) and generally begin to increase during the freshwater migration and peak in concentration close to the time of spawning (Hinch et al. 2005). These hormones are implicated in both the process of locating natal spawning areas, also referred to as "homing", and in the development of gonads and secondary sexual characteristics (e.g., male kype and dorsal hump). Plasma cortisol is a substrate for the production of reproductive hormones and plays a further role in both the senescence of sockeye salmon and in osmoregulation (Barry 2001; Carruth et al. 2002). However, cortisol is also an important stress hormone and may become elevated during periods of migration stress, such as encountering high water temperature (Macdonald et al. 2000). During these periods of stress reproductive hormones can decrease, possibly delaying reproductive development and altering migration behaviour.

In addition to cortisol, lactate and glucose are metabolites that can increase in the blood in response to stressors (Fagerlund 1967; Farrell et al. 2000; Barton 2002). Plasma ion concentrations provide a secondary physiological stress response as osmoregulation is compromised (Barton 2002). Anaerobic exercise in fish, such as burst swimming, increases plasma lactate. Burst swimming is energy inefficient but is common in migrating salmon as they encounter challenging migration conditions, such as high flows or elevated temperatures (Black 1958; Farrell et al. 1998). Glucose is mobilized in response to stress, although the mechanisms for glycogenolysis can vary (Axelrod and Reisine 1984; Vijayan et al. 1997; Kubokawa et al. 1999).

Evaluating relationships between these stress indicators and other physiological variables, environmental conditions, migration behaviour, and migration success can reveal potential mechanisms for migration behaviour and en route or prespawning mortality. Further refining my understanding of sockeye salmon migration through the use of integrative physiology provides a unique opportunity to rapidly assess conservation issues and evaluate management actions (Wikelski and Cooke 2006).

Migration timing

The primary influence on upstream migration timing is spawn timing, which is a heritable trait optimized for offspring survival and development (Quinn 2005). Migration initiation and travel rate must provide sufficient time to reach spawning grounds. Another important heritable influence on migration timing is the avoidance of detrimental migration conditions, such as high flows or temperatures (Quinn et al. 1997; Hodgson and Quinn 2002). Higher river temperatures, which were correlated with elevated marine temperatures, have been associated with earlier migration timing in Atlantic salmon

(*Salmo salar*) of the Baltic Sea (Dahl et al. 2004). Although the physiological mechanisms for the initiation of freshwater migration are not well understood Fraser River sockeye demonstrate relatively consistent timing of migration initiation, often with timing windows of less than one week (Woodey 1987). More distant populations generally initiate migration earlier and often migrate faster. A relatively consistent and narrow spawn timing window in migration timing would result in early migrants spending more time in freshwater and in a catabolic state prior to spawning.

Purpose of this study

This study uses nascent bio-telemetry and integrative physiological sampling methods to improve our understanding of the spawning migration of sockeye salmon and to elucidate potential mechanisms for en route mortality (Cooke et al. 2005). En route mortality can comprise a significant proportion of mortality in the life cycle of sockeye salmon prior to spawning, and has reached elevated levels in the past decade amongst Fraser River sockeye salmon (Cooke et al. 2004). En route mortality has been difficult to predict (Williams 2005) and represents a considerable challenge for both sustainable harvest management and effective endangered species recovery. Identifying potential mortality mechanisms may increase the ability to predict en route mortality. Refining my understanding of the complex physiology of migrating adult sockeye salmon may also reveal physiological indicators that provide effective surrogates for predicting migration behaviour and survival.

The migration of Fraser River sockeye salmon has been intensively studied, including some of the earliest research on salmon migration swimming and energetics by Brett (reviewed in Brett 1995) and the more recent evaluation of sockeye migration swimming

and energetics using conventional and electromyogram biotelemetry (reviewed in Hinch 2005). By utilizing this relatively well-researched system, this study was able to use established capture, sampling, and telemetry methods to evaluate a complex problem affecting an important meta-population of sockeye salmon.

Research questions/hypotheses

The primary objective of this study was to identify physiological correlates and potential mechanisms for en route mortality in late-run Fraser River sockeye salmon. A secondary objective of this study was to further refine understanding of the role of migration timing on migration physiology.

I tested the role of migration timing, and early migration particularly, on energy stores, reproductive development, migration behaviour, and stress of adult Pacific salmon. The first hypothesis is that early migrants have higher energy stores and lower reproductive development, corresponding to a relatively fixed reproductive lifespan associated with a narrow spawn timing window. However, to evaluate the influence of early migration on en route mortality I also hypothesized that early migrants that died en route to spawning grounds do not follow the first hypothesis, with lower energy stores and higher reproductive development than migrants from the more traditional migration timing period.

A distinct set of hypotheses considered the role of adverse environmental conditions, particularly high water temperatures, on migration physiology and en route mortality. I predicted physiological measures of stress would be higher in early migrants as freshwater temperatures traditionally peak before migration and decline through the migration period. As an investigative study I did not confine my data collection and

analyses to testing only these hypotheses. For example, osmoregulatory status was assessed as a further measure of stress but also to consider potential interactions between osmoregulatory status, other physiological measures, migration timing, and behaviour. In addition to the outcome of hypothesis testing this study may reveal further hypotheses that could direct more specific field or empirical studies.

References

Ando, S., Hatano, M., and Zama, K. 1985. Deterioration of chum salmon (*Oncorhynchus keta*) muscle during spawning migration – I. Changes in proximate composition of chum salmon muscle during spawning migration. Comp. Biochem. Physiol. B: Biochem. Mol. Biol. 80: 303-307.

Axelrod, J., Reisine, T.D., 1984. Stress hormones: their interaction and regulation. Science 224, 452–459.

Barry, T. P., Unwin, M. J., Malison, J. A., and Quinn, T. P. 2001. Free and total cortisol levels in semelparous and iteroparous chinook salmon. J. Fish. Biol. 59: 1673-1676.

Barton, B. A. 2002. Stress in fishes: a diversity of responses with particular reference to changes in circulating corticosteroids. Integr. Comp. Biol. 42: 516-525.

Black, E. C. 1958. Hyperactivity as a lethal factor in fish. J. Fish. Res. Board. Can. 15: 573-584.

Brett, J. R. 1995. Energetics. In Physiological Ecology of Pacific Salmon. Edited by C. Groot, L. Margolis, and W. C. Clarke. University of British Columbia Press, Vancouver. 3-68 pp.

Carruth, L. L., Jones, R. E., and Norris, D. O. 2002. Stress and Pacific salmon: A new look at the role of cortisol in olfaction and home-stream migration. Int. Comp. Biol. 42: 574-581.

Cederholm, C. J., D. H. Johnson, R. E. Bilby, L. G. Dominguez, A. M. Garrett, W. H. Graeber, E. L. Greda, M. D. Kunze, B. G. Marcot, J. F. Palmisano, R. W. Plotnikoff, W. G. Pearcy, C. A. Simenstad, & P. C. Trotter. 2000. Pacific Salmon and Wildlife - Ecological Contexts, Relationships and Implications for Management. Special Edition Technical Report, Prepared for D. H. Johnson and T. A. O'Neil, Wildlife - Habitat Relationships in Oregon and Washington. Washington Department of Fish and Wildlife, Olympia, Washington). 115 pp.

Cooke, S. J., Hinch, S. G., Farrell, A. P., Lapointe, M. F., Jones, S. R. M., Macdonald, J. S., Patterson, D. A., Healey, M. C., and Van Der Kraak, G. 2004. Abnormal migration timing and high en route mortality of sockeye salmon in the Fraser River, British Columbia. Fisheries 29(2): 22-33.

Cooke, S. J., Crossin, G. T., Patterson, D. A., English, K. K., Hinch, S. G., Young, J. L., Alexander, R. F., Healey, M. C., Van Der Kraak, G., and Farrell, A. P. 2005. Coupling non-invasive physiological assessments with telemetry to understand inter-individual variation in behaviour and survivorship of sockeye salmon: development and validation of a technique. J. Fish. Bio. 67: 1342-1358.

Crossin, G. T., Hinch, S. G., Farrell, A. P., Whelly, M. P., and Healey, M. C. 2003. Pink salmon (*Oncorhynchus gorbuscha*) migratory energetics: response to migratory difficulty and comparisons with sockeye salmon (*Oncorhynchus nerka*). Can. J. Zool. 81: 1986-1995.

Crossin, G. T., Hinch, S. G., Farrell, A. P., Higgs, D. A., Lotto, A. G., Oakes, J. D., and Healey, M. C. 2004. Energetics and morphology of sockeye salmon: effects of upriver migratory distance and elevation. J. Fish Biol. 65: 788-810.

Dahl, J., Dannewitz, J., Karlsson, L., Petersson, E., Löf, and Ragnarsson, B. 2004. The timing of spawning migration: implications of environmental variation, life history, and sex. Can. J. Zool. 82: 1864-1870.

Fagerlund, U. H. M. 1967. Plasma cortisol concentration in relation to stress in adult sockeye salmon during the freshwater stage of their life cycle. Gen. Comp. Endocrinol. 8: 197-207.

Farrell, A. P., Gamperl, A. K., and Birtwell, I. K. 1998. Prolonged swimming, recovery and repeat swimming performance of mature sockeye salmon *Oncorhynchus nerka* exposed to moderate hypoxia and pentachorophenol. J. Exp. Biol. 201: 2183-2193.

Farrell, A. P., Gallaugher, P., Clarke, C., DeLury, N., Kreiberg, H., Parkhouse, W., and Routledge, R. 2000. Physiological status of coho salmon (*Oncorhynchus kisutch*) captured in commercial nonretention fisheries. Can. J. Fish. Aquat. Sci. 57: 1668-1678.

Groot, C., and Margolis, L. 1991. Pacific Salmon Life Histories. UBC Press, Vancouver, BC, Canada. 564 pp.

Hinch, S.G., Cooke, S., Healey, M.C., and Farrell. A.P. 2005. Behavioural Physiology of Fish Migrations: salmon as a model approach. Pp. 239-295, in K. Sloman, S. Balshine and R. Wilson, [eds.]. Fish Physiology Volume 24: Behaviour and Physiology of Fish. Elsevier Press.

Hodgson, S., and Quinn, T. P. 2002. The timing of adult sockeye salmon migration into freshwater: adaptations by populations to prevailing thermal regimes. Can. J. Zoology 80: 542-555.

Kubokawa, K., Watanabe, T., Yoshioka, M., and Iwata, M. 1999. Effects of acute stress on plasma cortisol, sex steroid hormone and glucose levels in male and female sockeye salmon during the breeding season. Aquaculture 172: 335-349.

Lee, C. G., Farrell, A. P., Lotto, A., MacNutt, M. J., Hinch, S. G., and Healey, M. C. 2003. The effect of temperature on swimming performance and oxygen consumption in adult sockeye (*Oncorhynchus nerka*) and coho (*O. kisutch*) salmon stocks. J. Exp. Biol. 206: 3239-3251.

Macdonald, J. S. 2000. Mortality during the migration of Fraser River sockeye salmon (*Oncorhynchus nerka*): a study of the effect of ocean and river environmental conditions in 1997. Can. Tech. Rep. Fish. Aquat. Sci. 2315. 120 pp.

MacNutt, M. J., Hinch, S. G., Lee, C. G., Phibbs, J. R., Lotto, A. G., Healey, M. C., and Farrell, A. P. 2006. Temperature effects on swimming performance, energetics, and aerobic capacities of mature adult pink salmon (*Oncorhynchus gorbuscha*) compared with those of sockeye salmon (*Oncorhynchus nerka*). Can. J. Zool. 84: 88-97.

Munakata, A., Amano, M., Ikuta, K., Kitamura, S., and Aida, K. 2001. The involvement of sex steroid hormones in downstream and upstream migratory behavior of masu salmon. Comp. Biochem. Physiol. B: Biochem. Mol. Biol. 129: 661-669.

Naiman, R. J., Bilby, R. E., Schindler, D. E., and Helfield, J. M. 2002. Pacific salmon, nutrients, and the dynamics of freshwater and riparian ecosystems. Ecosystems 5: 399-417.

Patterson, D. A., MacDonald, J. S., Hinch, S. G., Healey, M. C., and Farrell, A. P. 2004. The effect of exercise and captivity on energy partitioning, reproductive maturation and fertilization success in adult sockeye salmon. J. Fish Bio. 64: 1039-1059.

Quinn, T. P., Hodgson, S., and Peven, C. 1997. Temperature, flow, and the migration of adult sockeye salmon (*Oncorhynchus nerka*) in the Columbia River. Can. J. of Fish. Aquat. Sci. 54: 1349-1360.

Quinn, T. P. 2005. The Behavior and Ecology of Pacific Salmon & Trout. UBC Press, Vancouver, BC, 378 pp.

Rand, P. S., and Hinch, S. G. 1998. Swim speeds and energy use of upriver-migrating sockeye salmon (*Oncorhynchus nerka*): simulating metabolic power and assessing risk of energy depletion. Can. J. Fish. Aquat. Sci. 55: 1832-1841.

Schindler, D. E., Scheuerell, M. D., Moore, J. W., Gende, S. M., Francis, T. B., and Palen, W. J. 2003. Pacific salmon and the ecology of coastal ecosystems. Frontiers in Ecology and the Environment 1: 31-37.

Ueda, H., Urano, A., Zohar, Y., and Yamauchi, K. 2000. In: Hormonal control of homing migration in salmonid fished "Proceedings of the 6th International Symoposium on the Reproductive Physiology of Fish," pp. 95-98. Department of Fisheries and Marine Biology, University of Bergen, Bergen, Norway.

Vijayan, M.M., Pereira, C., Grau, E.G., Iwama, G.K., 1997. Metabolic responses associated with confinement stress in tilapia: the role of cortisol. Comp. Biochem. Physiol. C 116, 89–95.

Wikelski, M., and Cooke, S. J. 2006. Conservation physiology. Trends Ecol. Evo. 21: 38-46.

Williams, Hon. B. 2005. 2004 Southern Salmon Fishery Post-season Review, Part One: Fraser River Sockeye Report. 91 pp.

Woodey, J. C. 1987. In-season management of Fraser River sockeye salmon (*Oncorhynchus nerka*): meeting multiple objectives. In: Sockeye salmon (*Oncorhynchus nerka*) population biology and future management. Edited by H. D. Smith, L. Margolis, and C. C. Wood. Can. Spec. Publ. Fish. Aquat. Sci. 96. pp.

CHAPTER 2

Physiological and energetic correlates of en route mortality for abnormally early migrating adult sockeye salmon (*Oncorhynchus nerka*) in the Thompson River, British Columbia[1]

Introduction

The spawning migrations of Pacific salmon (*Oncorhynchus* spp.) are physiologically challenging, using limited energy reserves to adjust osmoregulation from saltwater to freshwater, migrate upstream, develop gonads, and spawn (Brett 1995). Despite having a general understanding of migration rates, movement patterns and survival for some species and stocks during migrations (Groot and Margolis 1991, Quinn 2005), there is little information on how the physiological state of migrants affects their ability to reach spawning grounds. This knowledge gap may hinder the explanation of year-to-year variation in spawning abundance and subsequent juvenile production.

A recent alteration in river migration behaviour and mortality in Fraser River (British Columbia, Canada) sockeye salmon (*O. nerka*) permitted an examination of the role of physiological state in migration success. Though over 150 distinct spawning sites are used by sockeye salmon in the Fraser River system, fisheries managers identify four broad run timing groups based on entry timing of maturing adults into freshwater: early

---

[1] A version of this chapter has been published: Young, J. L., Hinch, S. G., Cooke, S. J., Crossin, G. T., Patterson, D. A., Farrell, A. P., Van Der Kraak, G., Lotto, A. G., Lister, A., Healey, M. C., and English, K. K. 2006. Physiological and energetic correlates of en route mortality for abnormally early migrating adult sockeye salmon (*Oncorhynchus nerka*) in the Thompson River, British Columbia. Can. J. Fish. Aquat. Sci. 63: 1067-1077.

stuart, early summer, summer, and late (Woodey 1987). Sockeye enter the Fraser River between June and October. The late-run timing group is the last to enter and unlike the other timing aggregates, typically holds in the outer estuary for several weeks prior to starting river migration. This estuarine delay occurs despite arrival time near the river mouth at approximately the same time as early summer and summer stocks, which enter the river immediately upon arrival. Since 1995, large segments of late-run sockeye have ceased their holding behaviour and have entered freshwater 3-6 weeks earlier than historically observed (Cooke et al. 2004a; Lapointe et al. 2004). However, these aberrant migrants have experienced high rates of mortality prior to spawning, which in some years exceeded 90% (Cooke et al. 2004a; Lapointe et al. 2004). In contrast, prior to 1995 and aberrant migration, mortality during river migration rarely exceeded 20% in any year for late-run Fraser River sockeye (Macdonald and Williams 1998).

To date, there has been no direct study of the underlying causes of the high mortality associated with the aberrant early migration of late-run sockeye salmon. However, most proposed hypotheses suggest that certain physiological systems are malfunctioning during freshwater migration (Cooke et al. 2004a). One hypothesis is based on the observed association between depletion of somatic energy reserves and mortality in migrating adult sockeye (Rand and Hinch 1998). Migrating adult salmon are in a catabolic state, having ceased feeding prior to river entry, in fact Fraser River sockeye have stopped feeding at least 200-400 km from the river mouth (Hinch et al. 2005). Thus, late-run sockeye salmon depend on a fixed energy reserve for migration, reproductive development, and spawning. By entering the Fraser River early, late-run sockeye are more likely to encounter higher water temperatures and flows than they would normally

experience in the fall. Increased flow and water temperatures increase transport costs and would accelerate the depletion of fixed energy reserves. Further, by encountering higher flows, I might expect to see erratic swimming patterns and considerable burst swimming (Macdonald 2000), leading to elevated levels of plasma lactate, glucose and cortisol (Fagerlund 1967; Farrell et al. 2000; Barton 2002). High levels of lactate and the associated metabolic acidosis have long been associated with post-exercise mortality (Black 1958). Related studies have shown that a freshwater myxosporan parasite (*Parvicapsula minibicornis*) contributes to mortality and reduced exercise capacity in adult late-run salmon if they accumulate more than approximately 450 degree days in freshwater (Wagner et al. 2005). Although individuals in this study were captured at a location that would traditionally be encountered by migrating adults prior to accumulating 450 degree days, elevated temperatures and flow along with early freshwater entry may allow development of the parasite to an extent that it affects individuals prior to or at the location of capture for this study.

A second hypothesis is based on the notion that reproductive hormone concentrations involved in the initiation of spawning migrations (Ueda et al. 1998; Munakata et al. 2001) can influence migration behaviour and mortality (Høgåsen and Prunet 1997; Ueda et al. 1998). Early river entry and en route mortality could be associated with advanced reproductive development. Secondary sexual characteristics and egg production typically develop during the freshwater migration (Hendry and Berg 1999). Premature maturation may reduce energy stores required for migration or compromise swimming efficiency. Reproductive maturation is also closely linked with rapid senescence and tissue degeneration in Pacific salmon (Dickhoff 1989; Finch 1990; Hendry and Berg 1999).

We intercepted adult late-run sockeye salmon of the Adams-Shuswap stock complex in the Thompson River canyon, approximately halfway along their freshwater migration, and used radio telemetry coupled with biological sampling to link the fate of individual fish with their behaviour and physiological condition. I focused on sockeye salmon that spawn in or near Shuswap Lake because of their importance to fisheries, their relative abundance during the year of sampling, and their relatively long freshwater migration (~485 km, the furthest of all late-run stocks) and the associate need of high energy. Gross somatic energy was measured and blood samples taken prior to implanting either a conventional or electromyogram radio transmitter. I examined whether either of the two hypotheses, (i) energy use, associated with inefficient swimming and elevated stress; and (ii) premature reproductive development, were associated with mortality in late-run sockeye salmon with aberrant early migration.

Methods

Study site

Fish were captured, biologically sampled and released at one site in the Thompson River canyon, British Columbia. The site was located 10 km upstream of the confluence of the Fraser and Thompson Rivers, 270 km upstream of the ocean, and about halfway along the approximately 480 km freshwater migration route of the Adams River and Shuswap Lake sockeye salmon stocks (Figure 2.1).

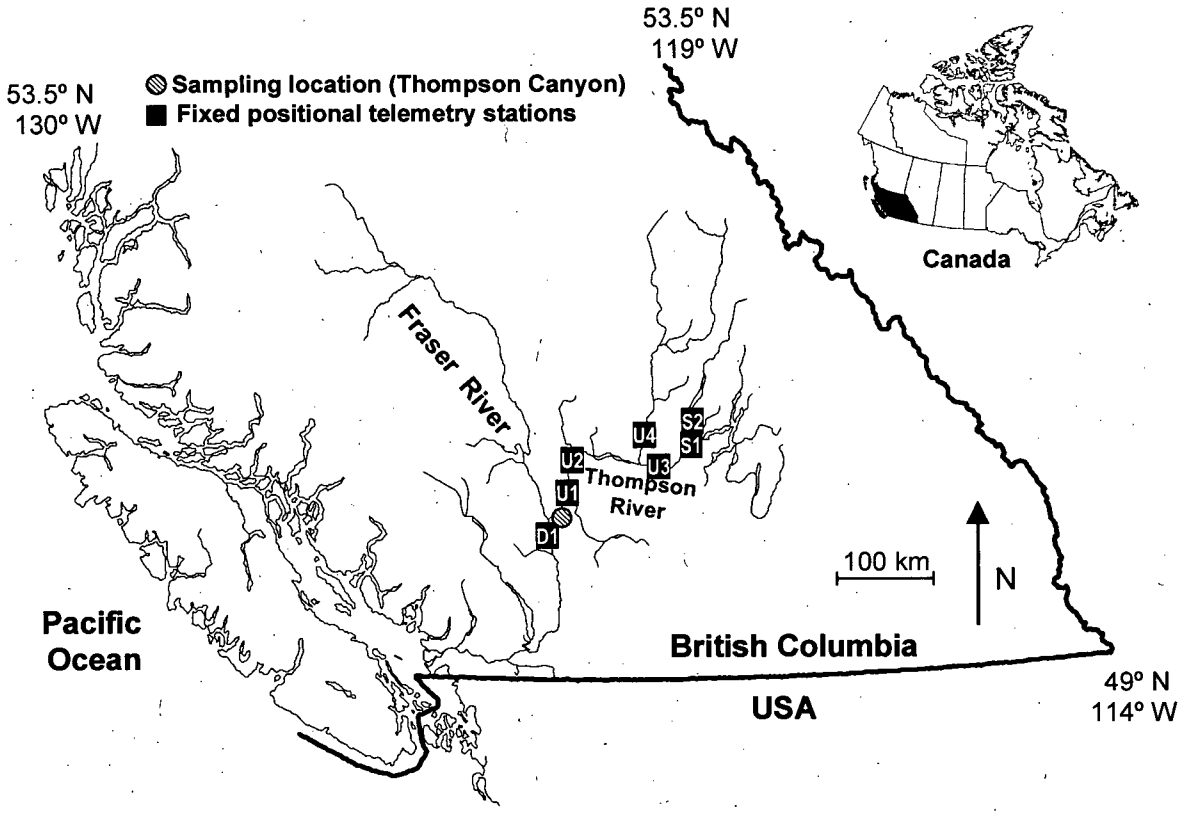

Figure 2.1. Map of study system with insert showing relative location within Canada. Fish were implanted with transmitters and biologically sampled in the Thompson River 10 km upstream of the junction of the Thompson River and Fraser River (Lat/Long: 50.3° N, 121.4° W). Fixed positional telemetry stations were positioned downstream of the release location (D1), upstream and en route to spawning grounds (U1, U2, U3), at the junction to the North Thompson River to assist with stock complex identification (U4), and at spawning grounds (S1, S2).

Downstream of this confluence, the Fraser River presents migration obstacles in the form of high loads of suspended sediment (Macdonald 2000), areas of high flow velocities (Hinch and Rand 1998) and gill net fisheries. The Thompson River has lower discharge rates and suspended sediment loads than the Fraser River, but the first 20 km of the Thompson River from the mouth contains areas of constricted bedrock channels with large steps along the bed, where currents are complex and fast. Previous research using electromyogram (EMG) telemetry in the Fraser River (eg., Hinch and Rand 1998; Hinch

et al. 2002; Standen et al. 2002) indicated that sockeye are most physically challenged during upstream migration through these types of habitats.

Fish capture and biological sampling

Sockeye salmon were collected between mid-August and early October 2003, a period that spanned the entire migration period for late-run sockeye salmon through the Thompson River. During the sampling period, average river temperature at my site displayed a seasonal decline from 19.5 to 16.0 °C. Fish were captured within 0.5 m of the shore by dip nets lowered to a maximum depth of 1 m. Successful fishing locations were associated with areas of constricted flow and where fish were observed to travel primarily along this edge area, presumably avoiding areas of higher flow near the middle of the river channels. Fish capture methods were consistent throughout the sampling period. Within 30 seconds of capture, single fish were placed ventral side up in a foam-lined 'V'-shaped trough, which supplied flowing river-water to the mouth of the fish, submerging the entire head. Individual fish were restrained by 1 or 2 people, while another person collected a blood sample via a caudal puncture (Houston 1990) using a syringe (1.5", 21 gauge) and vacutainer (3 mL), which was immediately stored in an ice-water slurry. Pressure was applied to the puncture site to facilitate blood clotting. If blood was not drawn within 1 minute, the fish was excluded from the study and immediately sacrificed by cerebral percussion. A portion of the adipose fin was collected and stored in ethanol for DNA analysis and a fork length measurement was made. A micro-wave energy meter (Distell Fish Fatmeter model 692; Distell Inc., West Lothian, Scotland, UK) was used to assess somatic energy levels following the methods in Crossin and Hinch (2005). While in the trough, the left side of the fish was partially lifted out of water to permit the energy

meter to be placed on two locations of the body wall near the dorsal fin. Gender was assessed using external secondary sexual characteristics or with the aid of reproductive hormones (see below). Blood samples were centrifuged within 10 minutes of storage on ice and two 0.5 ml plasma aliquots were immediately removed, stored on dry ice in the field and transferred to -80 °C upon return to the lab. Of 60 late-run sockeye gastrically implanted with radio transmitters, gross somatic energy was assessed on 54 and blood collected from 36. The sampling approach described has been shown to have no detrimental effects to sockeye migration rates or survival (Cooke et al. 2005).

Radio telemetry

Fish were gastrically implanted with positional radio transmitters (MCFT-3A, Lotek Wireless Inc.) via the mouth using a plastic tag applicator (Ramstad and Woody 2003; English et al. 2004). Transmitters were implanted either immediately after capture (n = 24) or immediately after blood sampling (n = 36). No anaesthesia was used on fish released with a positional radio transmitter. Transmitters weighed 16.1 g in air and 6.2 g in water and measured 16 mm in diameter and 51 mm in length. The antenna trailed out of the mouth of the fish and 30 mm of tubing from a Floy anchor tag was affixed to the end of the antenna. The tagging and sampling procedures were terminated if either the procedure took longer than 150 sec or the fish escaped from the trough. Fish were released immediately after tagging into a deep pool with a back eddy, slightly downstream of the capture location.

Six receiver stations (Lotek receiver models SRX400 or SRX400A) capable of logging information from the positional transmitters were installed at locations along the Thompson River migratory route upstream of my site (Figure 2.1). Each station

comprised up to three antennas (3-4 element Yagi). Two receivers were positioned upstream of the release site on the Thompson River (U1 and U2, 22 and 47 km from release site, respectively), and two receivers were positioned at late-run spawning streams (199 and 205 km from release site). One receiver was positioned on the North Thompson River (U4, 134 km from release site), which enabled detection of early summer-run fish that co-migrate with aberrantly early-timed late-run fish and which may have been inadvertently sampled and tagged. To detect fish that headed downstream after release, one station was positioned 10 km downstream of the release site at the confluence of the Thompson and Fraser Rivers (D1). Receivers collected data from 25 August until the last transmitter detection on 24 October 2003 (English et al. 2004). Fish were classified as 'casualty' if they were detected at an upstream detection station but not at a spawning ground detection station, 'survivor' if they were detected at a spawning ground detection station, or 'drop out' if they were not detected at a fixed station upstream of the release location.

Following biological sampling (same approaches as described above), fish destined for EMG transmitter implantation were transferred to a net-pen (~1.5 x 1.5 x 1.5 m^3) constructed from PVC pipe and plastic fencing placed in the river. Only females were used for EMG telemetry to limit inter-individual variability. Within 48 hours of capture, fish were anaesthesized (buffered MS222; 40-50 mg L^{-1}), implanted with an EMG transmitter and released after a short recovery period (15-60 min). I began EMG surgery on 29 fish. Surgeries on 11 fish were halted before transmitter insertion due to excessive bleeding from the surgical incision. Of the 18 fish implanted with transmitters, 14 also displayed bleeding from the incision, which was deemed excessive for 4 fish (loss of

greater than 20 mL). As a result, I released only 10 fish with EMG transmitters. After release, fish were tracked by hand using a mobile radio receiver (Lotek model SRX400) and single 3-element Yagi antenna. EMG transmitters measure activity of main swimming muscles that can be used to estimate swimming speed and energy expenditure (Standen et al. 2002; Cooke et al. 2004b). Full details on the EMG pulse interval transmitter and surgical procedures for sockeye are found in Hinch et al. (1996) with more generic detail in Cooke et al. (2004b).

We investigated the unusual bleeding phenomenon by measuring blood clotting time in an additional 62 sockeye captured at my site between 7 September to 7 October 2003. Clotting time was determined on blood sampled immediately after cerebral percussion by cutting the gill arch and dripping 10 drops of fresh blood on a clean, shade-stored, glass slide within one minute of fish death. With a stopwatch I measured the time for the blood sample to form into a singular, gelled mass, while keeping the slide out of direct sunlight. This method proved to be precise and was applied over consistent air temperatures. Other methods, including filling a haematocrit tube with blood and timing the formation of a connected string of clotted blood between broken sections of the tube, proved inaccurate and difficult to repeat.

Stock and timing group classification

Stock identification of individual fish was determined by microsatellite DNA variation (Beacham et al. 1995). DNA analyses revealed that of all fish captured for radio transmitter implantation 17 fish were early summer-run, and 60 fish were late-run. Late-run sockeye captured before 16 September 2003 were classified as 'aberrant', and those captured after this date as 'normal'. This delineation date was used because: i) two peaks

in abundance occurred in at the location of sampling that were clearly separated by this date, and ii) periods after 16 September represent the traditional arrival time of late-run sockeye at the sampling location in the Thompson River, as determined from long-term average passage times of late-run sockeye at a downstream location (August 29 at Mission, B.C.; English et al. 2004) and average migration travel times from this location to the sampling location (18 days, Patterson, unpublished data). Although DNA analysis was not conducted on the group of fish used for blot clotting measurements, this test was limited to fish captured after September 7. The late sampling dates occur after the majority of identified early summer-run fish passed my site suggesting that the majority were late-run fish.

Plasma analyses

Plasma ion, cortisol and osmolality measurements followed the procedures described by Farrell et al. (2000). The measurements were repeated if there was disagreement between duplicates >2.5 mequiv. L^{-1}. Concentrations of plasma Na^+ and K^+ were measured using a model 510 Turner flame photometer. Plasma aliquots (5 μL) were diluted 1:200 with a prepared 15 mEq lithium L^{-1} solution. The photometer was calibrated prior to use and checked against a standard approximately every five samples. Measurements were repeated if the disagreement between duplicates was >2%. Plasma lactate and glucose concentrations were measured using a YSI 2300 StatPlus lactate/glucose analyzer (Yellow Springs Instruments). Plasma osmolality was measured in duplicate on 10 μL samples using a model 5500 Wescor vapour pressure. Measurements were repeated if the disagreement between duplicates was >3%. Plasma cortisol concentrations were measured in duplicate using 96-well ELISA kits (Neogen Corp., Lexington, Ky.).

Testosterone (T), 17ß-estradiol (E2), and 11-ketotestosterone (11-KT) were measured by radioimmunoassay (Van Der Kraak and Chang 1990; McMaster et al. 1992). The inter-assay variabilities for the T, E2, and 11-KT radioimmunoassays were 6.6, 11.6, and 8.8%, respectively. I regressed plasma E2 values against T values to assign gender to fish, which resulted in two distinct clusters of the data corresponding to male and female fish.

Data analysis

A series of factorial two-way analysis of variance (ANOVA) tests were used to examine for differences in gross somatic energy, plasma metabolites, and plasma ions between aberrant- and normal-timed migrants and between survivors, casualties, and drop outs. An all pairwise multiple comparisons *a posteriori* procedure was used (Tukey test) to evaluate those groups that contributed to main effects. I estimated individual fish ground speeds using distances, time of travel between telemetry receiver stations, and fish body length. Migration ground speeds were compared between aberrant (both casualties and survivors) and normal-timed migrants using two-way ANOVA and Tukey post-hoc tests to evaluate significant effects. Plasma cortisol required log10 transformation to meet statistical normality and equal variance requirements. The segregation of reproductive hormone results by migration timing, migration fate, and gender created sample sizes too small for comprehensive analyses. To specifically evaluate the potential role of reproductive development on mortality in aberrant migrants select t-tests were used to compare female plasma reproductive hormone levels between aberrant survivors and casualties. I used linear regression and a t-test to compare blood clotting time with date of capture. A Chi-square test was used to compare survival between fish that were released

with a radio transmitter and blood sampled or not. All analyses were conducted using Sigmastat 2.03 (SPSS Inc.) and were assessed for significance at $\alpha = 0.05$.

Results

Positional radio telemetry

Of the 60 late-run fish implanted with a positional radio transmitter, 63% (38 fish) were captured before September 16 and classified as 'aberrant-timed' and 37% (22 fish) were captured after this date and classified as 'normal-timed' (Figure 2.2). Of aberrant-timed migrants, 50% (19 fish) were 'survivors', having reached spawning grounds, 32% (12 fish) were 'casualties', detected upstream but not at spawning grounds, and 18% (7 fish) were 'drop outs', detected downstream but never upstream of release. Of normal-timed migrants, 18% (4 fish) were 'survivors', 36% (8 fish) were 'casualties', and 46% (10 fish) were 'drop outs'. There was no difference in migration fate between fish that were sampled for blood ($n = 36$) and those that were not ($n = 24$; $\chi^2 = 2.658$, $P = 0.265$).

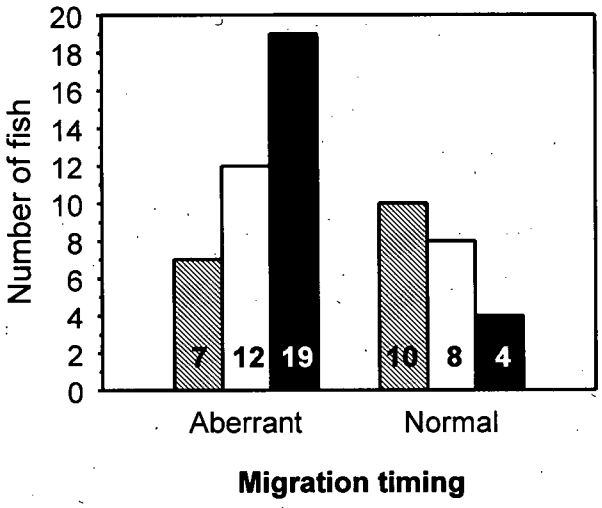

Figure 2.2. Number of aberrant and normal timed late-run sockeye grouped by migration fate as determined by positional radio-telemetry detections (▨ drop out: fell downstream after release and never detected upstream, □ casualty: detected upstream but not at spawning grounds, and ■ survivor: detected at spawning grounds). Sample sizes (n) shown within each bar.

Average ground speed from the release site to the second upstream detection station (U2) did not differ by migration timing ($F = 0.109$, $P = 0.743$) but did differ by migration fate with higher ground speeds in aberrant casualties than survivors ($F = 3.544$, $P = 0.030$; Figure 2.3a). Migration ground speed from release to spawning grounds was higher in normal- than aberrant-timed migrants ($t = 82$, $P = 0.007$; Figure 2.3b).

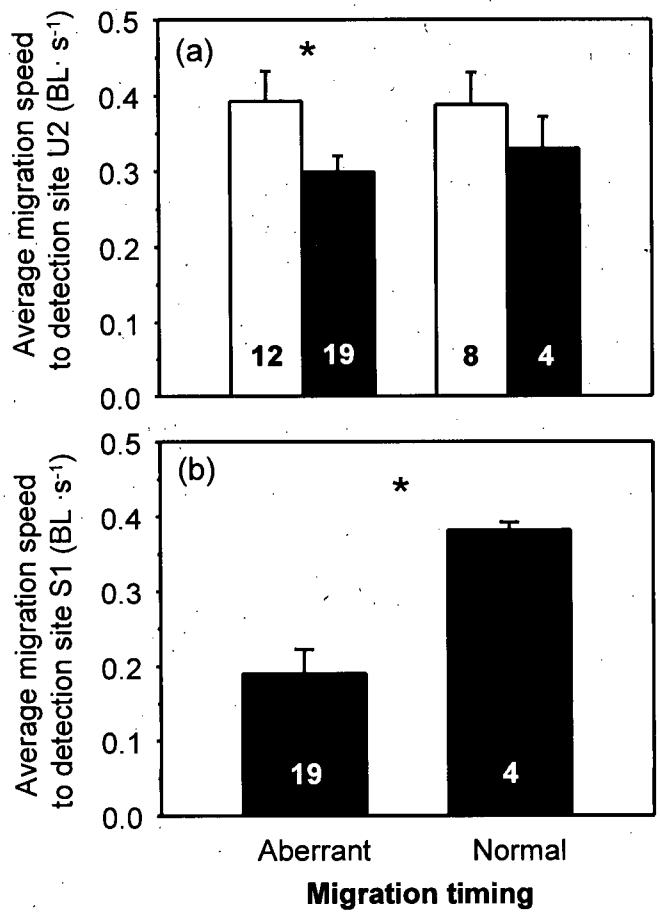

Figure 2.3. Mean ± standard error: (a) average migration ground speed (BL s^{-1}) estimated from travel between release and upstream detection site U2 (47 km upstream) for □ casualties, and ■ survivors (b) average swimming speed (BL s^{-1}) estimated from travel between upstream detection site U2 and spawning grounds (detection station S1, 163 km upstream). Sample sizes (n) within each bar. Statistically significant differences indicated by symbol (*).

Physiological and energetic analyses

Gross somatic energy (GSE) differed by migration timing and fate (Table 2.1), with lower GSE in aberrant-timed casualties than both aberrant-timed survivors ($P<0.001$) and dropouts ($P=0.004$), and a higher GSE in aberrant- compared with normal-timed survivors ($P=0.005$), casualties ($P=0.005$) and dropouts ($P=0.007$). Plasma lactate differed by migration fate (Table 2.1) with higher lactate in aberrant-timed casualties than

dropouts (P=0.020). Plasma glucose also differed by migration fate with lower glucose in casualties than dropouts for both timing groups combined (Table 2.1). Plasma cortisol did not differ by migration fate or timing (Table 2.1).

Plasma osmolality differed by migration timing (Table 2.1), with higher osmolality in aberrant-timed casualties than survivors (P=0.021) and higher osmolality in aberrant- than normal-timed drop outs (P=0.002) and casualties (P=0.003). More generally, plasma K^+ differed by migration timing with higher potassium in aberrant-timed migrants across all migration fates combined (Table 2.1). Other plasma ions (Na^+ and Cl^-) did not differ by migration timing or fate.

Table 2.1. Comparison of biological variables between late-run sockeye salmon that exhibited aberrant-or normal-timed migrations and reached spawning grounds (survivor), were detected upstream of release but not at spawning grounds (casualty), or fell downstream after release and were never detected upstream of release (drop out). Analyses were conducted using two-way ANOVA with fate as the main effect. Italicized statistical output indicate significant models ($\alpha = 0.05$). For analyses exhibiting significant main effects multiple comparisons were evaluated using a Tukey test ($\alpha = 0.05$). Dissimilar superscript indicates significant differences between migration fates within timing groups (^{a,b,c}) and between migration timing within fate groups (^{x,y}).

Physiological variable	Migration fate	Aberrant timing	N	Normal timing	N	ANOVA output		
						Timing	Fate	Interaction
Gross somatic energy	Survivor	$7.92 \pm 0.20^{a,x}$	17	6.56 ± 0.42^{y}	4	*F=25.168, P<0.001*	*F=9.831, P<0.001*	F=0.100, P=0.905
($MJ \cdot kg^{-1}$)	Casualty	$6.61 \pm 0.25^{b,x}$	11	5.45 ± 0.30^{y}	8			
	Drop out	$7.97 \pm 0.42^{a,x}$	4	6.39 ± 0.27^{y}	10			
Plasma lactate	Survivor	5.19 ± 0.85^{ab}	6	4.50 ± 1.04	4	F=1.384, P=0.249	*F=3.609, P=0.040*	F=1.972, P=0.158
($mmol \cdot L^{-1}$)	Casualty	7.78 ± 0.79^{a}	7	5.03 ± 0.85	6			
	Drop out	3.67 ± 1.12^{b}	3	4.46 ± 0.73	8			
Plasma cortisol*	Survivor	133 ± 71	6	56 ± 101	3	F=1.276, P=0.269	F=0.521, P=0.600	F=0.035, P=0.966
($ng \cdot mL^{-1}$)	Casualty	185 ± 66	7	152 ± 78	5			
	Drop out	149 ± 101	3	133 ± 58	8			
Plasma glucose	Survivor	4.43 ± 0.20	6	4.68 ± 0.24	4	F=0.085, P=0.772	*F=3.453, P=0.046***	F=0.980, P=0.388
($mmol \cdot L^{-1}$)	Casualty	4.37 ± 0.18	7	4.40 ± 0.20	6			
	Drop out	5.14 ± 0.28	3	4.74 ± 0.17	8			
Plasma osmolality	Survivor	329 ± 8^{a}	6	321 ± 10	4	*F=17.962, P<0.001*	F=2.236, P=0.126	F=2.434, P=0.106
($mosmol \cdot L^{-1}$)	Casualty	$360 \pm 7^{b,x}$	7	325 ± 8^{y}	6			
	Drop out	$356 \pm 11^{ab,x}$	3	309 ± 7^{y}	8			

Physiological variable	Migration fate	Aberrant timing	N	Normal timing	N	ANOVA output		
						Timing	Fate	Interaction
Plasma K^+	Survivor	2.54 ± 0.43	7	1.08 ± 0.57	4	*F=7.249, P=0.011***	F=0.624, P=0.543	F=0.409, P=0.668
(mequiv·L^{-1})	Casualty	2.87 ± 0.43	7	1.78 ± 0.47	6			
	Drop out	2.18 ± 0.57	4	1.60 ± 0.40	8			
Plasma Cl^-	Survivor	130.6 ± 1.1	7	128.8 ± 2.0	2	F=0.007, P=0.933	F=0.268, P=0.768	F=1.849, P=0.185
(mequiv·L^{-1})	Casualty	129.1 ± 1.1	7	132.6 ± 2.0	2			
	Drop out	131.1 ± 1.4	4	129.1 ± 1.7	3			
Plasma Na^+	Survivor	161.6 ± 2.4	7	166.6 ± 3.2	4	F=1.097, P=0.303	F=3.177, P=0.382	F=3.177, 0.056
(mequiv·L^{-1})	Casualty	163.6 ± 2.4	7	160.8 ± 2.6	6			
	Drop out	164.8 ± 3.2	4	155.6 ± 2.3	8			

* log10 transformed for analysis

**Drop out greater than casualty for both timing groups combined

***Aberrant greater than normal timing for all fate groups combined

Reproductive hormones

Plasma testosterone (T), 11-ketotestosterone (11-KT), and 17ß-estradiol (E2) were compared only between aberrant-timed female survivors and casualties due to low sample sizes. However, significant differences were observed for T and 11-KT, with higher reproductive hormone levels in aberrant-timed casualties than survivors (Table 2.2).

Table 2.2. Comparison of female reproductive hormone levels between late-run sockeye salmon that exhibited aberrant-timed migrations and either reached spawning grounds (survivor) or were detected upstream of release but not at spawning grounds (casualty). Analyses were conducted using t-tests. Italicized statistical output indicate significant models ($\alpha = 0.05$).

	Aberrant survivor		Aberrant casualty				
Female reproductive hormone	Mean	N	Mean	N	t	Power	P
Plasma testosterone ($pg \cdot ml^{-1}$)	16080 ± 3 880	4	30 550 ± 3570	5	-2.738	0.592	*0.029*
Plasma 11-Ketotestosterone ($pg \cdot ml^{-1}$)	953 ± 218	4	1745 ± 183	5	-2.807	0.617	*0.026*
Plasma 17ß-estradiol ($pg \cdot ml^{-1}$)	3398 ± 656	4	4589 ± 464	5	-1.526	0.163	0.171

Electromyogram (EMG) telemetry

Only one fish released with an EMG transmitter successfully resumed its upstream migration and DNA analysis confirmed that it was an early summer-run fish. The remaining 9 EMG tagged and released late-run fish dropped out and were never detected near or upstream of the release location during the entire sampling period. These fish typically and gradually moved downstream to the confluence of the Thompson and Fraser Rivers over a 48-72 hour period and remained downstream or not detected for the rest of

the sampling period. I collected intermittent EMG data on these fish during their fall back. EMG pulse intervals for each fish were converted to instantaneous swimming speeds using equations from Healey et al. (2003). Swimming speeds in excess of $1.5 \cdot Bl\ s^{-1}$ were common and average speeds among individuals ranged from 0.48-0.52 $Bl \cdot s^{-1}$ (detailed EMG and swimming speed data are not shown). These data indicate that fish were not passively carried downstream but were likely alive and actively swimming.

Excessive bleeding was observed in 14 of 18 fish subjected to surgery. Of the five fish that did not bleed excessively during surgery one was the early summer-run fish that I successfully tracked, two were aberrant-timed late-run fish, and two were normal-timed late-run fish. Necropsies on eight fish that bled excessively but were not released revealed that bleeding was not due to damage to internal viscera or organs, or by severing large blood vessels during surgery. The time required for blood to clot decreased over the sampling period (R^2=0.215, P<0.001; Figure 2.4), with higher blood clotting time in aberrant- (133 s ± 13, n=37) than normal-timed (77 s ± 5 s, n=61) migrants (t=3.393, P=0.001).

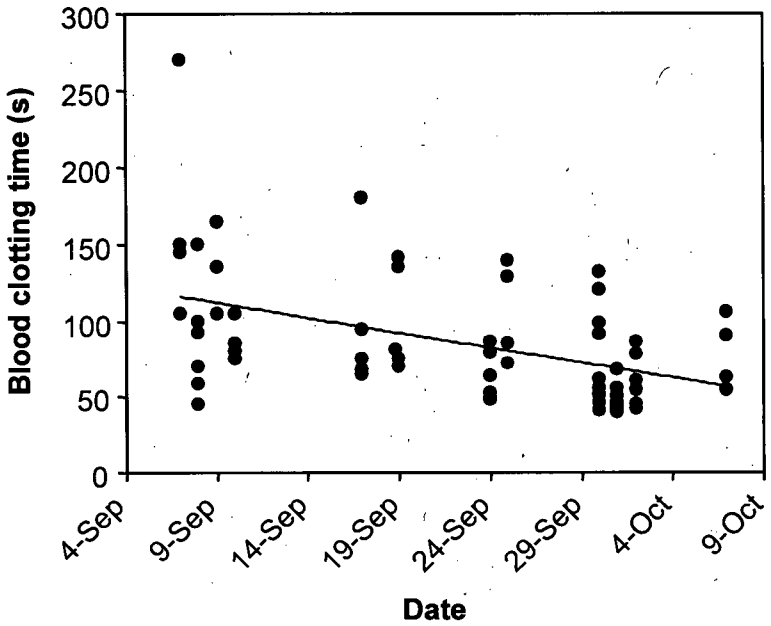

Figure 2.4. Time required for blood to clot for sockeye salmon captured between 7 September 2003 and 7 October 2003. Linear regression line shown (R^2=0.215, P<0.001).

Discussion

We evaluated potential linkages between energetic and physiological variables and mortality during the spawning migration of late-run Fraser River sockeye salmon. When intercepted half-way through their freshwater migration aberrant-timed late-run migrants had higher energy levels than normal-timed, late-run migrants. This result refutes the hypothesis that lower energy reserves are contributing to early entry behaviour, but still permits the possibility of a low energy mechanism for mortality in aberrant migrants. Other work by my group (Cooke, unpublished data) indicated a similar pattern of energy density when these fish first enter the Fraser River from the ocean, with lower energy in normal-timed migrants. These results suggest that the marine holding behaviour of normal-timed late-run salmon contributes to this energy differential through both routine metabolism and reduced feeding prior to freshwater entry. Feeding has significantly

slowed during the coastal migration and has largely ceased about 200 km from the river mouth (Hinch et al. 2005). Hendry et al. (2004) suggests a genetic link between migration and energetic-reproductive state with high somatic energy and small ovaries characterizing the earliest arriving and spawning females within a sockeye population.

An important new finding in the present study was that casualties which were aberrantly-timed had lower energy reserves prior to release and faster ground speeds through the first 47 km of upstream migration than aberrantly-timed survivors. Several mechanisms could account for these differences. First, faster ground speeds likely imply faster swimming speeds that would contribute to increased metabolic costs. Aberrant-timed survivors had lower migration ground speeds than aberrant-timed casualties and normal-timed survivors. Given that sockeye salmon have a minimum cost of transport at a swimming speed of approximately 1 $BL \cdot s^{-1}$ (Lee et al. 2003), it is possible that unless aberrant-timed fish properly pace upstream migration, they may not reach spawning grounds because of energetic exhaustion. Second, aberrant-timed female casualties had higher reproductive hormones than aberrant-timed survivors thus the former likely diverted more energy to gonads, potentially leaving them energetically deficient for the swimming challenges that remained. These ideas are discussed below.

Fraser River sockeye reach spawning grounds with barely enough energy to ripen gonads, perform courtship and spawn (Crossin et al 2004a). Normal-timed Adams sockeye (a major component of the late-run stock grouping that migrates through the Thompson River) begin the freshwater migration with about 8 MJ kg^{-1} of somatic energy and complete the migration with about 5 MJ kg^{-1}; approximately 4 $MJ.kg^{-1}$ of which is required to sustain their life throughout spawning and thus they have only a 1 MJ kg^{-1}

energy 'buffer' (Crossin et al. 2004a). For female sockeye salmon, swimming metabolism and gonad development utilize similar amounts of energy (~1.5 MJ kg^{-1} each) (Crossin 2004b). Thus only modest increases in energy demands for either of these processes could conceivably exhaust energy reserves by depleting the 1 MJ kg^{-1} buffer.

Upriver migration can be triggered by injection of testosterone (T) and 11-ketotestosterone (11-KT) in precocious castrated male masu salmon (*Oncorhynchus masou*) and by T, 11-KT, and 17ß-estradiol (E2) in immature parr (Munakata et al. 2001). T and 11-KT concentrations also increase during river migration of salmonids (Tveiten et al 1998; Munakata et al. 2001; Leonard et al. 2002). Based on physiological samples from fish from the marine environment several hundreds of kilometers from the mouth of the Fraser River, it has been suggested that the early migration phenomenon in late-run sockeye may be linked with an acceleration of reproductive development (Cooke et al 2004a). My results suggest that this accelerated reproductive development may also play a role in the abnormally high level of en route mortality. I found aberrant late-run sockeye that died en route to spawning grounds had higher levels of (T) in females and higher levels of (11-KT) in both sexes. Furthermore, elevated levels of reproductive hormones are believed to be partially responsible for tissue degradation and senescence during and after spawning by Pacific salmon (Dickhoff 1989). Thus, more advanced reproductive development during the migration could have reduced energy available for swimming or compromised migration ability in another way, such as affecting tissue degradation, migration behaviour, or swimming ability as a result of premature development of secondary sexual characteristics.

Physiological stress can accelerate energy use and impair swimming performance in migrating salmon (Farrell et al. 1998; Wagner et al. 2003). Stress can also reduce a fish's ability to maintain blood ion concentrations (Eddy 1981; Ackerman et al. 2000). I thus evaluated osmoregulatory status but found no consistent patterns among plasma ion levels linking osmoregulatory dysfunction with en route mortality. I did however find that plasma osmolality was higher in aberrant-timed casualties than survivors and higher plasma K^+ in aberrant- than normal-timed migrants. These differences could be associated with the normal decline in plasma ion concentrations that occurs after entry into freshwater or during maturation and senescence. Adult chum salmon (*O. keta*) exhibited reduced, but stable ion concentrations in freshwater following transfer from saltwater (Morisawa 1979; Hasegawa et al. 1987). Higher osmolality in aberrant-timed casualties than survivors could indicate that the former had spent less time in freshwater prior to capture. This result is consistent with the migration ground speed differences discussed above, suggesting that aberrant casualties are moving upstream more quickly, possibly resulting in energetic exhaustion. However, the lack of difference in plasma ions that most strongly contribute to osmolality (e.g., Na^+ and Cl^-) may suggest other causes for this difference, which I was unable to identify.

The occurrence of excessive bleeding during EMG transmitter implantation surgeries was unexpected. The volume of blood lost regularly exceeded 20 mL, which could represent more than a third of total blood volume. Dozens of studies have used EMG transmitter implantation techniques similar to that used in this study to examine river migrations of adult sockeye (eg., Hinch et al. 1996; Hinch and Bratty 2000), pink (*Oncorhynchus gorbuscha*) (Standen et al. 2002), chinook (*O. tshawytscha*) (Brown and Geist 2002), and

Atlantic salmon (*Salmo salar*) (Økland et al. 2000). However, none have reported excessive bleeding. Excessive bleeding may have been related to an impaired blood clotting mechanism, since the time required for blood to clot increased over the period of sampling, such that blood from normal migrants clotted in approximately half the time of aberrant migrants. The time required for blood to clot generally declines when healthy Pacific salmon become diseased or stressed and the number of circulating thrombocytes increases (Casillas and Smith 1977). A naturally occurring parasite (*Parvicapsula minibicornis*) that affects kidney function is contracted in the estuary by all homeward migrating sockeye salmon (St-Hilaire et al. 2002). This parasite can lead to disease and kidney malfunction if water temperatures are relatively high as can be experienced by early migrating late-run sockeye (Wagner et al. 2005). However, I do not know whether this or other parasite infections could influence blood clotting.

Although I cannot assign a mechanism to poor blood clotting and excessive bleeding from wounds, it is clear that any level of physical rupture of the fish's exterior surface (skin and gills) could provide an additional mechanism for mortality. All but one of the fish implanted with EMG transmitters either could not be released or fell back. The EMG-tagged fish that fell back after release did not have lower energy reserves, nor did EMG data indicate that these fish were exhibiting constant burst swimming, which would have rapidly drained their energy reserves. Sockeye salmon typically encounter rocky bed conditions in both the Fraser and Thompson canyons, a gauntlet of gill nets, and intraspecific interactions from encounters with thousands of conspecifics and congenerics (i.e. migrating pink salmon) during their upstream migration, all of which could cause abrasions or lesions leading to excessive bleeding or more rapid infections in early

migrants. Unusual bleeding from injuries received during migration could provide an additional mechanism for mortality during migration. Further investigation is required to determine the cause of reduced blood clotting times and to confirm relationships between physical injury, excessive bleeding, and mortality in aberrant migrants.

It is unlikely that physiologically sampling these fish contributed unduly to en route mortality because migration fate for telemetered fish was similar for fish that were sampled for blood or not. Although 28% of fish released with a radio transmitter failed to move 22 km upstream, my reported levels of plasma lactate were lower than the threshold (<15.0 $mmol \cdot L^{-1}$) suggested by Jain et al. (1998) for detecting impaired critical swimming ability for mature sockeye salmon. Delayed mortality as a result of handling effects is generally restricted to 24 h and rarely more than 48 h (Wertheimer 1988; Farrell et al. 2000). Fish were classified as casualty (en route mortality) only if they reached the second upstream detection station, which was 47 km from the release site. Travel times to this location averaged more than 60 hours.

In summary, by individually tracking fish after physiological sampling, I investigated a complex phenomenon affecting the migration success of Fraser River sockeye salmon. Furthermore, by linking physiology during mid-migration with fate, this study has revealed potentially important roles of energetic status and reproductive development in influencing the en route mortality associated with a aberrant migration behaviour. Aberrant migrants that died en route to spawning grounds had lower energy stores and faster migration ground speeds than aberrant migrants that reached their spawning grounds. Concurrently, aberrant migrants that died en route had higher levels of reproductive hormones, suggesting that advanced maturation may have contributed to

reduced migratory ability and death prior to spawning. Sockeye that fell back downstream after being released did not exhibit higher stress, impaired osmoregulatory function, low energy stores, or energetic exhaustion. Excessive bleeding during EMG transmitter implantation surgery suggests a possible injury mechanism that could also contribute to high en route mortality. Further studies evaluating this bleeding phenomenon are necessary to refine this hypothesis.

References

Ackerman, P. A., Forsyth, R. B., Mazur, C. F., and Iwama, G. K. 2000. Stress hormones and the cellular stress response in salmonids. Fish Physiol. Biochem. 23: 327-336.

Barry, T. P., Unwin, M. J., Malison, J. A., and Quinn, T. P. 2001. Free and total cortisol levels in semelparous and iteroparous Chinook salmon. J. Fish. Biol. 59: 1673-1676.

Barton, B. A. 2002. Stress in fishes: a diversity of responses with particular reference to changes in circulating corticosteroids. Integr. Comp. Biol. 42: 516-525.

Beacham, T. D., Withler, R. E., and Wood., C. C. 1995. Stock identification of sockeye salmon by means of minisatellite DNA variation. N. Am. J. Fish. Manage. 15: 249-265.

Black, E. C. 1958. Hyperactivity as a lethal factor in fish. J. Fish. Res. Board. Can. 15: 573-584.

Brett, J. R. 1995. Energetics. In Physiological Ecology of Pacific Salmon. Edited by C. Groot, L. Margolis, and W. C. Clarke. University of British Columbia Press, Vancouver. 3-68 pp.

Brown, R. S., and Geist, D. R. 2002. Determination of swimming speeds and energetic demands of upriver migrating fall chinook salmon (*Oncorhynchus tshawytscha*) in the Klickitat River, Washington. Pacific Northwest Laboratory, Bonneville Power Administration, Richland, Washington. Project 22063, Contract 42663A. 52 pp.

Casillas, E., and Smith, L. S. 1977. Effect of stress on blood coagulation and haematology in rainbow trout (*Salmo gairdneri*). J. Fish Biol. 10: 481-491.

Cooke, S. J., Hinch, S. G., Farrell, A. P., Lapointe, M. F., Jones, S. R. M., Macdonald, J. S., Patterson, D. A., Healey, M. C., and Van Der Kraak, G. 2004a. Abnormal migration timing and high en route mortality of sockeye salmon in the Fraser River, British Columbia. Fisheries 29: 22-33.

Cooke, S. J., Thorstad, E. B., and Hinch, S. G. 2004b. Activity and energetics of free-swimming fish: insights from electromyogram telemetry. Fish Fish. 5: 21-52.

Cooke, S. J., Crossin, G. T., Patterson, D. A., English, K. K., Hinch, S. G., Young, J. L., Alexander, R. F., Healey, M. C., Van Der Kraak, G., and Farrell, A. P. 2005. Coupling non-invasive physiological assessments with telemetry to understand inter-individual variation in behaviour and survivorship of sockeye salmon: development and validation of a technique. J. Fish Biol. 67: 1342-1358.

Crossin, G. T., Hinch, S. G., Farrell, A. P., Higgs, D. A., and Healey, M. C. 2004a. Somatic energy of sockeye salmon at the onset of upriver migration: a comparison among ocean climate regimes. Fish. Oceanogr. 13: 345-349.

Crossin, G. T., Hinch, S. G., Farrell, A. P., Higgs, D. A., Lotto, A. G., Oakes, J. D., and Healey, M. C. 2004b. Energetics and morphology of sockeye salmon: effects of upriver migratory distance and elevation. J. Fish Biol. 65: 788-810.

Crossin, G. T., and Hinch, S. G. 2005. A non-lethal method for assessing the somatic energy content of freely migrating adult Pacific salmon. Trans. Am. Fish. Soc. 134: 184-191.

Dickhoff, W. W. 1989. Salmonids and annual fishes: death after sex. In Development, Maturation, and Senescence of Neuroendocrine Systems. Edited by M. P. Schreibman and C. G. Scanes. Academic Press. 253-266 pp.

Eddy, F. B. 1981. Effects of stress on osmotic and ionic regulation in fish. In Stress and Fish. Edited by A. D. Pickering. Academic Press. 77-101 pp.

English, K. K., Sliwinski, C., Labelle, M., Koski, W. R., Alexander, R., Cass, A., and Woodey, J. C. 2004. Migration timing and in-river survival of Late-run Fraser River sockeye in 2003 using radio-telemetry techniques. Department of Fisheries and Oceans Canada, Nanaimo. EA1620. 45 pp.

Fagerlund, U. H. M. 1967. Plasma cortisol concentration in relation to stress in adult sockeye salmon during the freshwater stage of their life cycle. Gen. Comp. Endocrinol. 8: 197-207.

Farrell, A. P., Gamperl, A. K., and Birtwell, I. K. 1998. Prolonged swimming, recovery and repeat swimming performance of mature sockeye salmon *Oncorhynchus nerka* exposed to moderate hypoxia and pentachorophenol. J. Exp. Biol. 201: 2183-2193.

Farrell, A. P., Gallaugher, P., Clarke, C., DeLury, N., Kreiberg, H., Parkhouse, W., and Routledge, R. 2000. Physiological status of coho salmon (*Oncorhynchus kisutch*) captured in commercial nonretention fisheries. Can. J. Fish. Aquat. Sci. 57: 1668-1678.

Finch, C. E. 1990. Longevity, Senescence, and the Genome. University of Chicago Press. 938 pp.

Groot, C., and Margolis, L. 1991. Pacific Salmon Life Histories. UBC Press, Vancouver, BC. 510 pp.

Hasegawa, S., Hirano, T., Ogasawara, T., Iwata, M., Akiyama, T., and Arai, S. 1987. Osmoregulatory ability of chum salmon, *Oncorhynchus keta*, reared in freshwater for prolonged periods. Fish Physiol. Biochem. 4: 101-110.

Healey, M. C., Lake, R., and Hinch, S. G. 2003. Energy expenditures during reproduction by sockeye salmon (*Oncorhynchus nerka*). Behaviour 140: 161-182.

Hendry, A. P., and Berg, O. K. 1999. Secondary sexual characters, energy use, senescence, and the cost of reproduction in sockeye salmon. Can. J. Zool. 77: 1663-1675.

Hendry, A. P., Morbey, Y. E., Berk, O. K., and K., W. J. 2004. Adaptive variation in senescence: reproductive lifespan in a wild salmon population. Proc. R. Soc. Ser. B. Bio 271: 259-266.

Hinch, S. G., Diewert, R. E., Lissimore, T. J., Prince, A. M. J., Healey, M. C., and Henderson, M. A. 1996. Use of electromyogram telemetry to assess difficult passage areas for river-migrating adult sockeye salmon. Trans. Am. Fish. Soc. 125: 253-260.

Hinch, S. G., and Rand, P. S. 1998. Swim speeds and energy use of upriver-migrating sockeye salmon (*Oncorhynchus nerka*): role of local environment and fish characteristics. Can. J. Fish. Aquat. Sci. 55: 1821-1831.

Hinch, S. G., and Bratty, J. 2000. Effects of swim speed and activity pattern on success of adult sockeye salmon migration through an area of difficult passage. Trans. Am. Fish. Soc. 129: 598-606.

Hinch, S. G., Standen, E. M., Healey, M. C., and Farrell, A. P. 2002. Swimming patterns and behaviour of upriver-migrating adult pink (*Oncorhynchus gorbuscha*) and sockeye (*O. nerka*) salmon as assessed by EMG telemetry in the Fraser River, British Columbia, Canada. Hydrobiologia 483: 147-160.

Hinch, S.G., Cooke, S., Healey, M.C., and Farrell. A.P. 2005. Behavioural Physiology of Fish Migrations: salmon as a model approach. Pp. 239-295, in K. Sloman, S. Balshine and R. Wilson, [eds.]. Fish Physiology Volume 24: Behaviour and Physiology of Fish. Elsevier Press.

Houston, A. H. 1990. Blood and Circulation. In Methods for Fish Biology. Edited by C. B. Schreck and P. B. Moyle. American Fisheries Society, Bethesda, MD. 273-334 pp.

Høgåsen, H. R., and Prunet, P. 1997. Plasma levels of thyroxine, prolactin, and cortisol in migrating and resident wild arctic char, *Salvelinus alpinus*. Can. J. Fish. Aquat. Sci. 54: 2947-2954.

Jain, K. E., Birtwell, I.K., and Farrell, A.P. 1998. Repeat swimming performance of mature sockeye salmon following a brief recovery period: a proposed measure of fish health and water quality. Can. J. Zool. 76: 1488-1496.

Lapointe, M., Cooke, S. J., Hinch, S. G., Farrell, A. P., Jones, S., Macdonald, J. S., Patterson, D. A., Healey, M. C., and Van Der Kraak, G. 2004. Early upstream migration and mortality of late-run sockeye salmon in the Fraser River, British

Columbia. In Georgia Basin/Puget Sound Research Conference. Edited by T. Droscher and D. A. Fraser, Vancouver. 1-14 pp.

Lee, C. G., Farrell, A. P., Lotto, A., MacNutt, M. J., Hinch, S. G., and Healey, M. C. 2003. The effect of temperature on swimming performance and oxygen consumption in adult sockeye (*Oncorhynchus nerka*) and coho (*O. kisutch*) salmon stocks. J. Exp. Biol. 206: 3239-3251.

Leonard, J. B. K., Iwata, M., and Ueda, H. 2002. Seasonal changes of hormones and muscle enzymes in adult lacustrine masu (*Oncorhynchus masou*) and sockeye salmon (*O. nerka*). Fish Physiol. Biochem. 25: 153-163.

Macdonald, J. S., and Williams, I. V. 1998. Effects of environmental conditions on salmon stocks: the 1997 run of early Stuart sockeye salmon. In Speaking for the salmon workshop proceedings. Edited by P. Gallaugher and L. Wood. Simon Fraser University, Burnaby. 46-51 pp.

Macdonald, J. S. 2000. Mortality during the migration of Fraser River sockeye salmon (*Oncorhynchus nerka*): a study of the effect of ocean and river environmental conditions in 1997. Can. Tech. Rep. Fish. Aquat. Sci. 2315. 120 pp.

McMaster, M., Munkittrick, K., and Van Der Kraak, G. 1992. Protocol for measuring circulating levels of gonadal sex steroids in fish. Can. Tech. Rep. Fish Aquat. Sci. 1836. 29 pp.

Morisawa, M., Hirano, T., and Suzuki, K. 1979. Changes in blood and seminal plasma composition of the mature salmon (*Oncorhynchus keta*) during adaptation to freshwater. Comp. Biochem. Physiol., A: Mol. Integr. Physiol. 64: 325-329.

Munakata, A., Amano, M., Ikuta, K., Kitamura, S., and Aida, K. 2001. The involvement of sex steroid hormones in downstream and upstream migratory behavior of masu salmon. Comp. Biochem. Physiol. B: Biochem. Mol. Biol. 129: 661-669.

Økland, F., Flemming, I. A., Thorstad, E. B., Finstad, B., Einum, S., and McKinley, R. S. 2000. EMG telemetry to record the intensity of swimming- and breeding-related behaviour in Atlantic salmon. Pages pp. 51-58 in Proceedings of the Third Conference on Fish Telemetry in Europe. CEFAS, Lowestoft.

Quinn, T. P. 2005. The Behavior and Ecology of Pacific Salmon & Trout. UBC Press, Vancouver, BC, 378 pp.

Ramstad, K. M., and Woody, C. A. 2003. Radio tag retention and tag-related mortality among adult sockeye salmon. N. Am. J. Fish. Manage. 23: 978-982.

Rand, P. S., and Hinch, S. G. 1998. Swim speeds and energy use of upriver-migrating sockeye salmon (*Oncorhynchus nerka*): simulating metabolic power and assessing risk of energy depletion. Can. J. Fish. Aquat. Sci. 55: 1832-1841.

St-Hilaire, S., Boichuk, M., Barnes, D., Higgins, M., Devlin, R., Withler, R., Khattra, J., Jones, S., and Kieser, D. 2002. Epizootiology of Parvicapsula minibicornis in Fraser River sockeye salmon, *Oncorhynchus nerka* (Walbaum). J. Fish Dis. 25: 107-120.

Standen, E. M., Hinch, S.G., Healey, M.C., and Farrell, A.P. 2002. Energetic costs of migration through the Fraser River Canyon, British Columbia, in adult pink (*Oncorhynchus gorbuscha*) and sockeye (*Oncorhynchus nerka*) salmon as assessed by EMG telemetry. Can. J. Fish. Aquat. Sci. 59: 1809-1818.

Tveiten, H., Mayer, I., Johnsen, H. K., and Jobling, M. 1998. Sex steroids, growth and condition of Arctic charr broodstock during an annual cycle. J. Fish Biol. 53: 714-727.

Ueda, H., Kaeriyama, M., Mukasa, K., Urano, A., Kudo, H., Shoji, T., Tokumitsu, Y., Yamauchi, K., and Kurihara, K. 1998. Lacustrine sockeye salmon return straight to their natal area from open water using both visual and olfactory cues. Chem. Senses 23: 207-212.

Van Der Kraak, G., and Chang, J. 1990. Arachidonic acid stimulates steroidogenesis in goldfish preovulatory ovarian follicles. Gen. Comp. Endocrinol. 77: 221-228.

Wagner, G. N., McKinley, R. S., Bjorn, P. A., and Finstad, B. 2003. Physiological impact of sea lice on swimming performance of Atlantic salmon. J. Fish Biol. 62: 1000-1009.

Wagner, G. N., Lotto, A. G., Kuchel, L., Jones, S. R. M., Patterson, D. A., Cooke, S. J., Macdonald, J. S., Van Der Kraak, G., Healey, M. C., Shrimpton, M., English, K., Hinch, S. G., and Farrell, A. P. 2005. Metabolic rates and swimming performance of adult Fraser River sockeye salmon (*Oncorhynchus nerka*) after a controlled infection with *Parvicapsula minibicornis*. Can. J. Fish. Aquat. Sci. 62: 2124-2133.

Wertheimer, A. 1988. Hooking mortality of chinook salmon released by commercial trollers. N. Am. J. Fish. Manage. 8: 346-355.

Woodey, J. C. 1987. In-season management of Fraser River sockeye salmon (*Oncorhynchus nerka*): meeting multiple objectives. In Sockeye salmon (*Oncorhynchus nerka*) population biology and future management. Edited by H. D. Smith, L. Margolis, and C. C. Wood. Can. Spec. Publ. Fish. Aquat. Sci. 96. pp.

CHAPTER 3

The influence of migration timing and stream temperature on the physiology of upstream migrating adult sockeye salmon (*Oncorhynchus nerka*) in the Thompson River, British Columbia[2]

Introduction

The spawning migration of Pacific salmon (*Oncorhynchus* spp.) is a life history event requiring specialized physiological capabilities. Pacific salmon must effectively partition limited energy reserves to the osmoregulatory shift from salt to freshwater, upstream travel, reproductive maturation, and spawning (Brett 1995). Spawn timing is an important heritable trait primarily optimized for offspring survival, growth, and development that is dependent on the unique environmental conditions of the spawning and early rearing habitat (Quinn 2005). Migration timing is also an important heritable trait, ensuring mature adults are at the spawning grounds at the optimal time by incorporating sufficient time for travel, physiological adjustment to environmental conditions, and reproductive development (Hodgson and Quinn 2002). Migration timing may also be selected to avoid adverse migration conditions, such as relatively predictable periods of high flows or temperatures (Quinn et al. 1997; Lee et al. 2003).

The migration physiology of adult Pacific salmon has undergone intense study. Energy use (e.g., Brown et al. 2002; Standen et al. 2002; Crossin et al. 2004), osmoregulatory

---

2 A version of this chapter is in preparation for publication: Young, J. L., Hinch, S. G., Cooke, S. J., Crossin, G. T., Patterson, D. A., Van Der Kraak, G., and Farrell, A. P. The influence of migration timing and water temperature on the physiology of upstream migrating adult sockeye salmon (*Oncorhynchus nerka*) in the Thompson River, British Columbia.

function (e.g., Morisawa 1979; Hasegawa et al. 1987; Shrimpton et al. 2005), and reproductive physiology (e.g., Tveiten et al. 1998; Hendry and Berg 1999; Patterson et al. 2004) have been evaluated in Pacific salmon and in some cases data are available across different species, populations, and regions (Reviewed in Hinch et al. 2005). However, many questions remain regarding the association of migration timing and physiology. By measuring a suite of physiological variables in a group of sockeye salmon (*O. nerka*) over the range of time they are observed migrating through one location I evaluated the role of migration timing and associated changes in water temperature on energy stores, plasma ions, stress indicators, and reproductive development. In particular I was interested in evaluating a relatively recent migration phenomenon where some Fraser River sockeye salmon have been migrating abnormally early and dieing en route to spawning grounds at rates much higher than traditionally observed (Cooke et al. 2004).

Since 1995, late-run sockeye salmon of the Fraser River have contracted their traditional estuarine holding behaviour by up to six weeks, resulting in early freshwater entry and upstream migration (Cooke et al. 2004). Fraser late-run sockeye are one of four broad aggregations, segregated primarily by freshwater entry timing, used for fisheries management (Woodey 1987: early Stuart, early summer, summer, and late summer). Early migration by late-run sockeye has been associated with an increase in en route mortality, reaching 95% in some years (Cooke et al. 2004). Biotelemetry/biopsy studies have revealed a link between energy status, reproductive development and en route mortality in aberrantly early migrants (Young et al. 2006). Early migrants that died en route to spawning grounds had lower gross somatic energy levels and higher reproductive hormone levels compared to survivors. A biotelemetry / biopsy study that tagged and

sampled late-run sockeye in the marine environment prior to entering the Fraser River also showed that individuals that entered freshwater but died en route to spawning grounds had lower energy and higher reproductive hormones at the point of capture in the marine environment (Cooke et al. 2006).

Abnormally early migrating sockeye are not spawning outside of the traditional range of spawn timing for these stocks (Cooke et al. 2004), but because sockeye have a relatively fixed maturation trajectory (e.g., Hendry and Berg 1999; Patterson et al. 2004), early migrants would be expected to be less mature at freshwater entry. Since somatic energy is required to develop gonads as feeding does not occur during the migration (Gilhousen 1980; Patterson et al. 2004), earlier migrants would also be expected to have higher somatic energy levels at freshwater entry. My first hypothesis is that early migrants will have an inverse relationship between somatic energy and measures of reproductive development, with the earliest migrants exhibiting the highest energy and the lowest level of reproductive maturity. Hendry et al. (2004) identified higher somatic energy and smaller ovaries in the earliest migrating females within a population of sockeye. However, it is not known whether this condition applies to late-run sockeye of the Fraser River and if aberrantly early migrants will display this trend.

Higher energy levels may be beneficial to early migrants because river conditions are often more energetically challenging in mid-summer relative to end of summer (e.g. higher temperatures or flows). However, these harsher en route environments may also rapidly deplete energy stores necessary for migration and spawning completion. Stress indicators will determine how adverse conditions, particularly high water temperatures, affect migrants. For instance, plasma cortisol and lactate levels are elevated in sockeye

migrating through warm or fast flowing water (Hinch et al. 2005). Elevated cortisol levels can impair reproductive development and elevated lactate levels can contribute to mortality (Hinch et al. 2005). Plasma ions provide a further measure of stress as disrupted osmoregulatory function may result the loss of ions in freshwater (Eddy 1981; Ackerman et al. 2000). My second hypothesis is that early migrants will have higher levels of stress indicators due to greater exposure to higher temperatures and/or a greater number of degree days up to their point of capture. If my first hypothesis is rejected because early migrants do not have higher somatic energy levels, stress indicators may refine understanding of the environmental mechanisms contributing to premature decline of energy stores necessary for migration completion and maturation.

Whether early migrants have higher energy levels or not, a relatively fixed range of spawn timing requires early migrants to spend more time in freshwater, increasing their exposure to freshwater impacts. These impacts include further potential encounters with challenging flow or temperature conditions, a longer period of time in a catabolic state, and exposure to freshwater parasites. For example, in the Fraser River, a naturally occurring Myxosporan parasite (*Parvicapsula minibicornis*) contracted while adults migrate through the river mouth is known to reduce swimming performance and is thought to increase rates of mortality in relation to accumulated temperature exposure (Wagner et al. 2005).

The first and second hypotheses I will test are based on the prevalence of a fixed maturation trajectory with relatively consistent spawn timing. More generally, that regardless of the location of sampling individuals of the same meta-population will be in a similar state of reproductive development. The first hypothesis is that early migrants

will have higher energy stores and lower reproductive hormone levels and less developed gonads. The second hypothesis is that early migrants will have higher levels of stress indicators associated with their encounter with more adverse environmental conditions. In addition to evaluating stress metabolites and a stress hormone, I also measured plasma ions as an indicator of stress. A challenge for this study was the ability to separate the relative influence of migration timing and water temperature on salmon physiology as both variables are changing at the same time. However, by independently assessing the influence of migration timing and temperature on physiology, as well as attempting to isolate the influence of timing alone, I will further resolve the more strongly contributing factor. Further, results from this study will determine what physiological variables and migration conditions require more targeted field or empirical studies.

Methods

Study site

Fish were captured and sampled at one site in the Thompson River, British Columbia. The site was 10 km upstream of the confluence of the Fraser and Thompson Rivers, 270 km upstream of the ocean, and about two-thirds along the approximately 480 km freshwater migration route of the Adams River and Shuswap Lake sockeye salmon stocks (Figure 3.1).

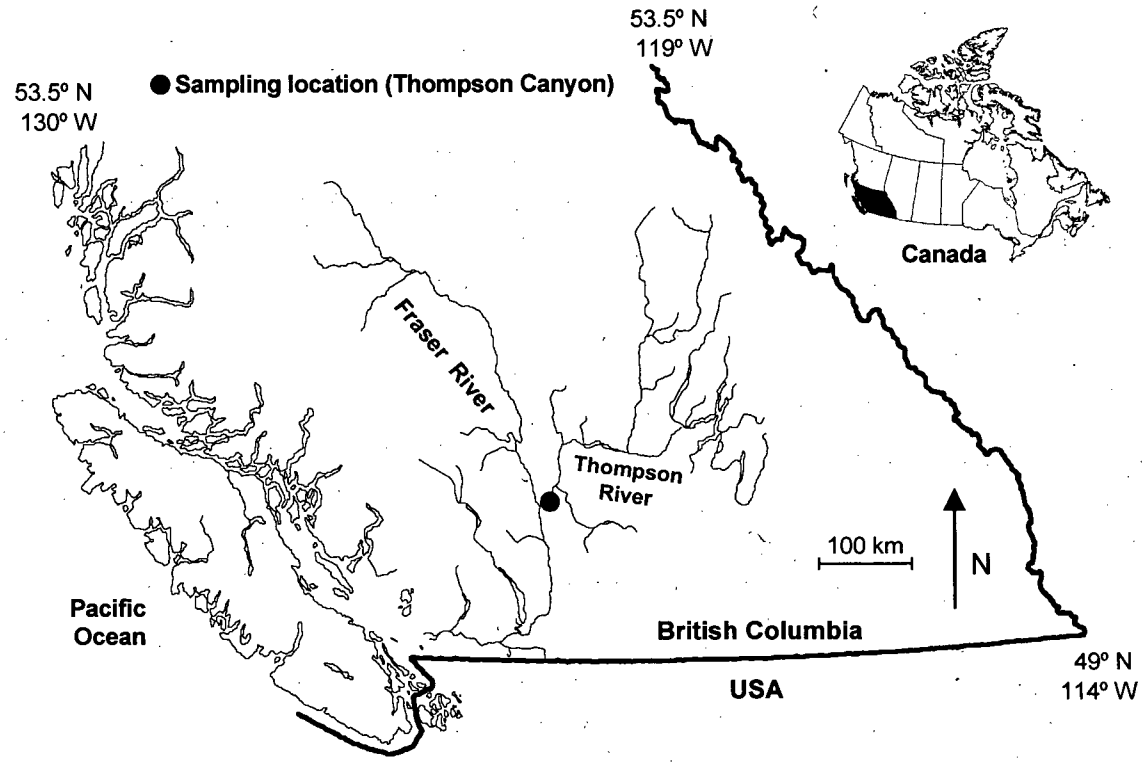

Figure 3.1. Map of study system with insert showing location within Canada. Fish were captured and sampled in the Thompson River 10 km upstream of the junction of the Thompson River and Fraser River (black circle, Latitude/Longitude: 50.3° N, 121.4° W).

Prior to reaching this confluence, migrating sockeye face migration obstacles in the form of high suspended sediment loads in the lower Fraser River (Macdonald 2000), regions of very high flow velocities through Hell's Gate, and gill net fishers along the Fraser Canyon (Hinch and Rand 1998). The first 20 km of the Thompson River presents complex and fast currents. Previous research using electromyogram (EMG) telemetry in the Fraser River (e.g., Hinch and Rand 1998; Hinch et al. 2002; Standen et al. 2002) indicated that these types of habitat are where sockeye are most physically challenged during upstream migration making this study site a logical locale to assess the relationship between physiology and timing.

Fish collection and biological sampling

A total of 218 sockeye salmon were collected between mid-August and early October 2003, a period that spanned the entire migration period for late-run sockeye salmon through the Thompson River. Of the 218 sockeye salmon sampled, 106 fish were captured and destructively sampled exclusively for this study, while 106 were physiologically sampled and released with a radio transmitter for a parallel study (Young et al. 2006). Radio-tagged fish were not euthanized or anaesthesized prior to blood collection, gill biopsy and somatic energy measurements, but were placed ventral side up in a foam-lined V-shaped trough with continuous water flow-through. For more detailed methods see Young et al. (2006) and Cooke et al. (2005). Gender was determined by body morphology and/or reproductive hormone analysis (described below in detail). Gender for the remaining 26 fish could not be assessed because reproductive hormones were not evaluated and these fish lacked sufficient body morphology characteristics (e.g., male kype and dorsal hump) needed for gender determination. Water temperature at my site was highest at the start of sampling (mean daily temperature at start ~19.5 °C) and declined throughout the sampling period (mean daily temperature at completion = ~16.0 °C, Figure 3.2). Water temperatures in the Fraser River downstream of my sampling location changed consistently throughout the migration period suggesting that mean daily water temperatures in the Thompson River used in analyses provide a general surrogate for temperatures experienced by migrating salmon prior to sampling. As demonstrated in Figure 3.2, the temperature peak occurred more than two weeks prior to the start of the late-run migration period through my sample site and declining relatively consistently beyond the this migration period. Thus, early migrants in my study would have

experienced higher degree days with successive migrants consistently experiencing fewer degree days over time. Further supporting this approach is the relatively consistent travel times from the Lower Fraser River to my sampling location (Wagner et al. 2005).

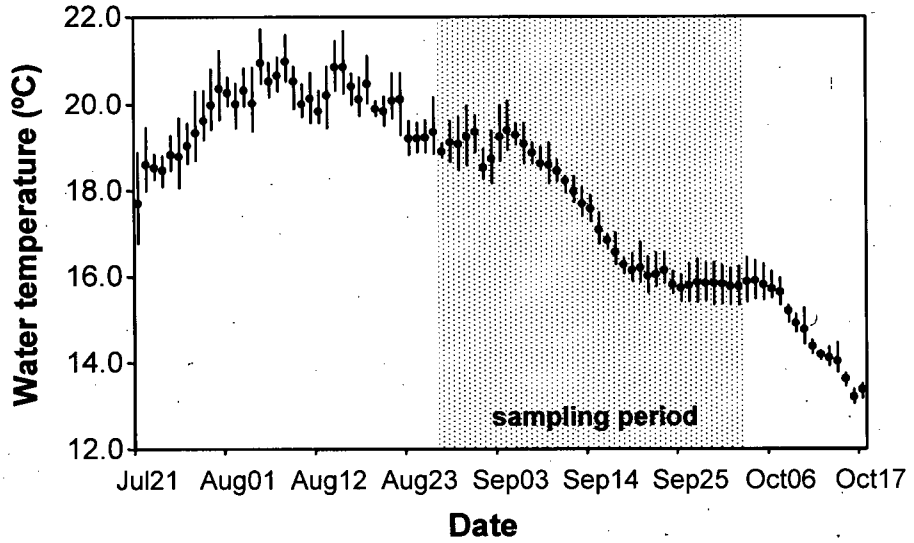

Figure 3.2. Average daily water temperature measured hourly of Thompson River at Ashcroft from 21 July to 17 October 2003. Lines indicate the range from daily minimum to maximum temperature. Shaded area indicates period of sampling.

Fish were captured within 0.5 m of the shore by First Nations dip nets lowered to a maximum depth of 1 m. Successful fishing locations were associated with areas of constricted flow and where fish were observed to travel primarily along this edge area, presumably avoiding areas of higher flow near the middle of the river channels. Fish capture methods were consistent throughout the sampling period. Of all potential fish collection methods used to sample migrating sockeye salmon (e.g., gill net, troll, recreational angling, seine, fish wheel), dip net is the most rapid way of obtaining physiological samples that are reflective of the animal's status prior to capture rather than the condition of the fish as a result of capture technique (Cooke, Unpublished data).

Immediately after capture (<10 sec), fish were euthanized by cerebral concussion and a blood sample was collected via a caudal puncture (Houston 1990) using a syringe (1.5", 21 gauge) and vacutainer (10 mL). Immediately after blood collection, < 4 mm from the tips of 6 to 8 gill filaments (0.03 g) were removed from the first gill arch (McCormick 1993) for assessing gill enzyme activity for another study. A portion of the adipose fin was collected and stored in ethanol for DNA analysis and a fork length measurement was made. A micro-wave energy meter (Distell Fish Fatmeter model 692; Distell Inc., West Lothian, Scotland, UK) was used to assess somatic energy levels following the methods in Crossin and Hinch (2005). Total body mass and gonad mass were measured using an electronic scale with accuracy to 1 g. Blood samples were centrifuged within 10 minutes of storage on ice and four plasma aliquots (5 µl) were immediately removed, stored on dry ice in the field and transferred to -86 °C freezers upon return to the laboratory.

Sample distribution and stock identification

Of 218 fish captured gross somatic energy was assessed for 194, blood was collected from 177, and a gill biopsy collected from 106. Stock identification of individual fish was determined using molecular genetic techniques (Beacham et al. 1995). DNA analyses focused on early migrants due to the potential overlap between late-run sockeye and early summer-run sockeye during this period of the migration. Those fish identified as early summer-run fish were excluded from the analysis (n = 28). DNA testing was not performed on fish captured after 11 September, representing the point at which I assumed all fish to be from the late run meta-population (M. Lapointe, Pacific Salmon Commission, Suite 600, 1155 Robson Street, Vancouver, BC, V6E 1B5, unpublished data).

Plasma analyses

Plasma variables were used as indicators of physiological stress and osmoregulatory status. Plasma ion, cortisol and osmolality measurements followed the procedures described by Farrell et al. (2000). The measurements were repeated if there was disagreement between duplicates >2.5 mequiv.$\cdot L^{-1}$. Concentrations of plasma Na+ ($[Na^+]$) were measured using a model 510 Turner flame photometer. Plasma aliquots (5 μL) were diluted 1:200 with a prepared 15 mEq lithium$\cdot L^{-1}$ solution. The photometer was calibrated prior to use and checked against a standard approximately every five samples. Measurements were repeated if the disagreement between duplicates was >2%. Plasma lactate and glucose concentrations Plasma cortisol concentrations were measured in duplicate using 96-well ELISA kits (Neogen Corp., Lexington, Ky.). Testosterone (T), 17ß-estradiol (E2), and 11-ketotestosterone (11-KT) were measured by radioimmunoassay (Van Der Kraak and Chang 1990; McMaster et al. 1992). The inter-assay variabilities for the T, E2, and 11-KT radioimmunoassays were 6.6, 11.6, and 8.8%, respectively.

Statistical analyses

Linear regression was used to independently assess the relationship between each physiological variable (dependent variables) with capture date and mean daily water temperature (independent variables). Linear regression was also used to assess the relationship between capture date and the residuals from each of the temperature regressions in an attempt to isolate the effect of migration timing. All dependent variables were assessed for heteroscedasticity and normality. Further, energy partitioning for reproductive development is much more substantive in females due to the relatively high

energy requirements of egg development. Plasma lactate and glucose concentrations were log-transformed to meet normality and equal variance assumptions. It was not possible to meaningfully transform plasma cortisol concentrations to meet regression assumptions, however, scatterplots were still presented for visual consideration. Regression analyses were conducted using Sigmastat 2.03 (SPSS Inc.). The linear regression for gross somatic energy was assessed at α=0.05 while Bonferroni corrections were applied to groups of analyses: stress indicators (lactate, glucose, cortisol; α=0.017), plasma ions (Na^+, Cl^-; α=0.025), and measures of reproductive development (female gonadosomatic index, testosterone, 11-ketotestosterone, 17β estradiol; α=0.0125). Corrected coefficients of variation of female reproductive hormone levels were calculated (Sokal and Rohlf 1981) and compared using *Z* tests (Zar 1999) between early and normal-timed migrants. Only female reproductive development variables were analyzed due to poor distribution of available data for male hormone analyses.

Results

Of the 218 sockeye salmon captured, 171 were identified as 'late-run'. Of these 171 late-run sockeye salmon, 97 were females (mean mass = 2.29 kg, SE = 0.056; mean fork length = 60.5 cm, SE = 0.28) and 74 were males (mean mass = 2.87 kg, SE = 0.086; mean fork length = 63.8 cm, SE = 0.32). Complete data sets for physiological variables are shown in Appendices 1-3.

Gross somatic energy

Higher gross somatic energy was found in early migrants (R^2=0.351, P<0.0001; Figure 3.3a) and in sockeye salmon that encountered higher water temperatures (R^2=0.350, P<0.0001; Figure 3.3b).

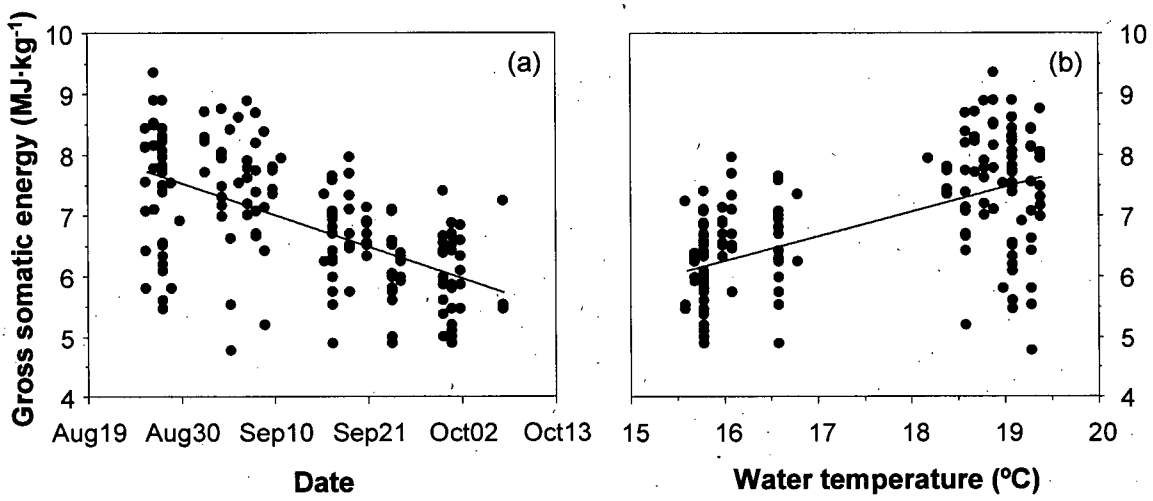

Figure 3.3. Gross somatic energy for late-run Fraser River sockeye salmon sampled at a single location by time (a) and by average stream water temperature on the date of sampling (b). Linear regression lines presented when significant ($p \leq 0.05$).

Stress indicators

Plasma lactate concentrations were higher in early migrants (R^2=0.136, P<0.001; Figure 3.4a) and in warmer water temperatures (R^2=0.125, P<0.001; Figure 3.4b). Conversely, plasma glucose concentrations were higher in later migrants (R^2=0.0544, P=0.003; Figure 3.4c) and in cooler water temperature (R^2=0.0656, P=0.001; Figure 3.4d). Figure 3.4e and Figure 3.4f show plasma cortisol concentrations with migration capture date and water temperature.

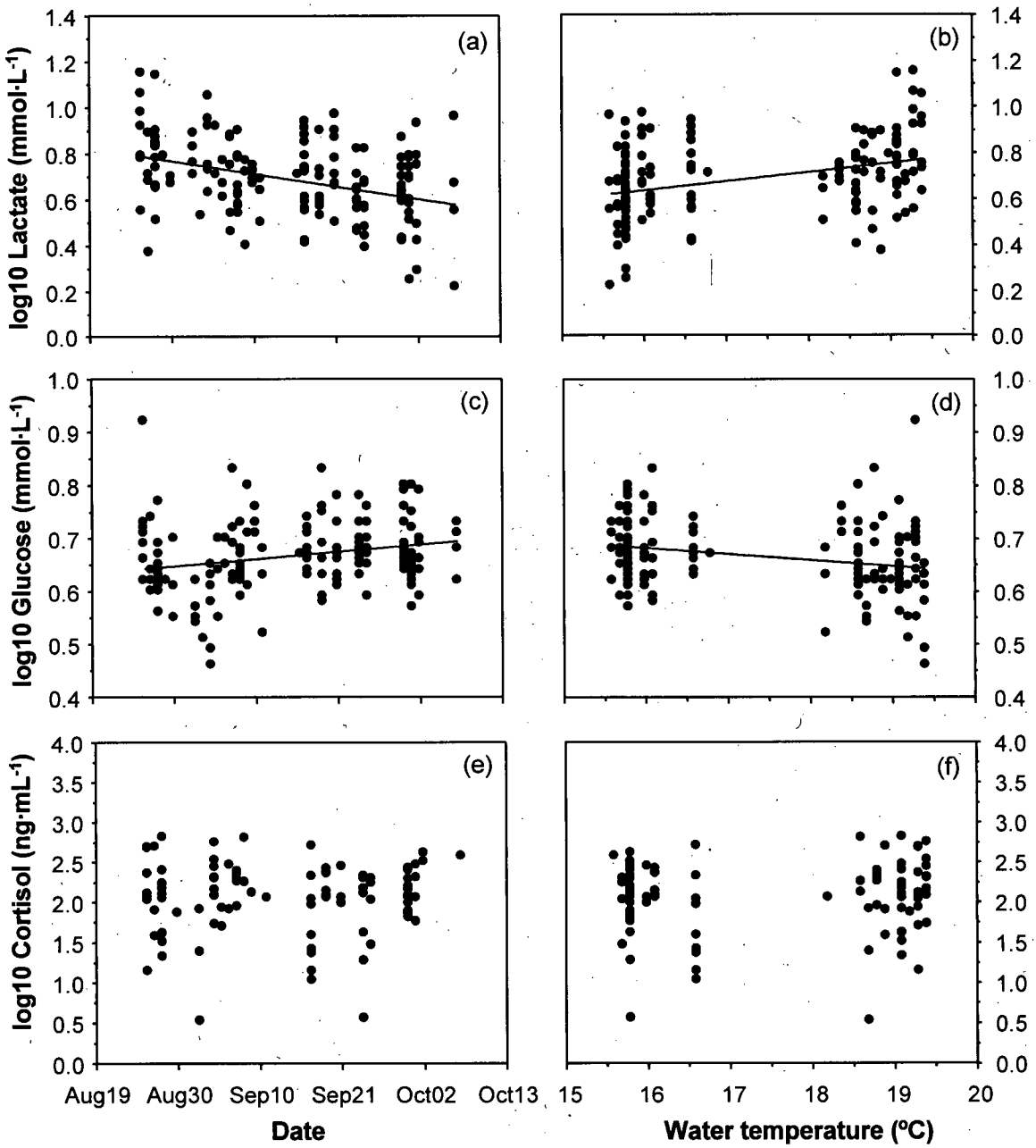

Figure 3.4. Physiological indicators of stress by time and by average stream water temperature on the date of sampling, including log10 plasma lactate (a,b), log10 plasma glucose (c,d), and log10 plasma cortisol (e,f). Linear regression lines presented when significant ($p \leq 0.017$).

Plasma ions

Plasma Na^+ and Cl^- concentrations were higher in early migrants (Na^+: $R^2=0.198$, $P<0.001$; Figure 3.5a; Cl^-: $R^2=0.285$, $P<0.001$; Figure 3.5c) and in warmer water

temperatures (Na^+: R^2=0.116, P<0.001; Figure 3.5b; Cl^-: R^2=0.319, P<0.001; Figure 3.5d).

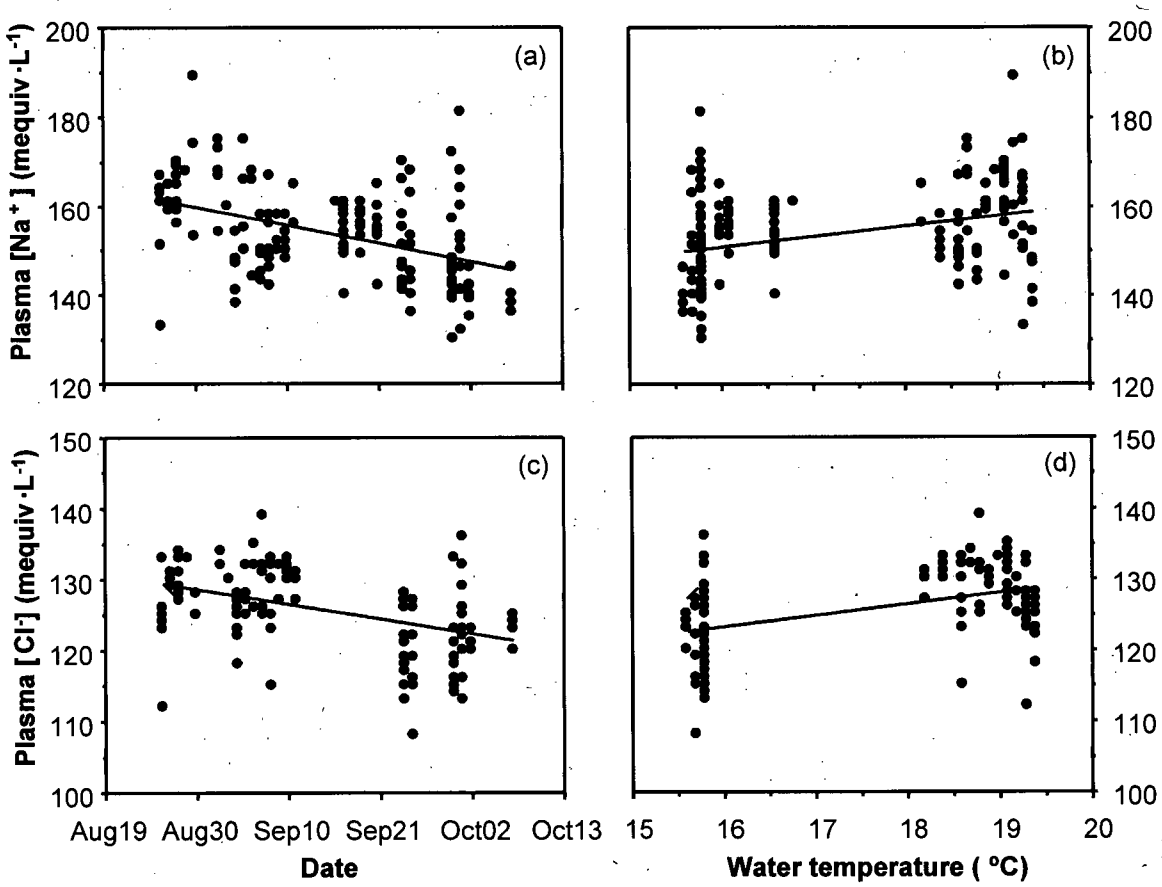

Figure 3.5. Plasma ions by time and by average stream water temperature on the date of sampling, including Na^+ (a,b) and Cl^- (c,d). Linear regression lines presented when significant ($p \leq 0.025$).

Female reproductive hormones and gonadosomatic index

Gonadosomatic index was higher in later migrants (R^2=0.230, P=0.002; Figure 3.6a) and in cooler water temperature (R^2=0.171, P=0.010; Figure 3.6b). Plasma testosterone was also higher in later migrants (R^2=0.224, P=0.003; Figure 3.6c) and in cooler water temperature (R^2=0.204, P=0.004; Figure 3.6d). Plasma 11-ketotestosterone did not differ with migration timing (R^2=0.121, P=0.065; Figure 3.6e) or water temperature (R^2=0.131,

P=0.053; Figure3.6f). Plasma 17β estradiol was higher in later migrants (R^2=0.190, P=0.006; Figure 3.6g) and in cooler temperatures (R^2=0.172, P=0.010; Figure 3.6h).

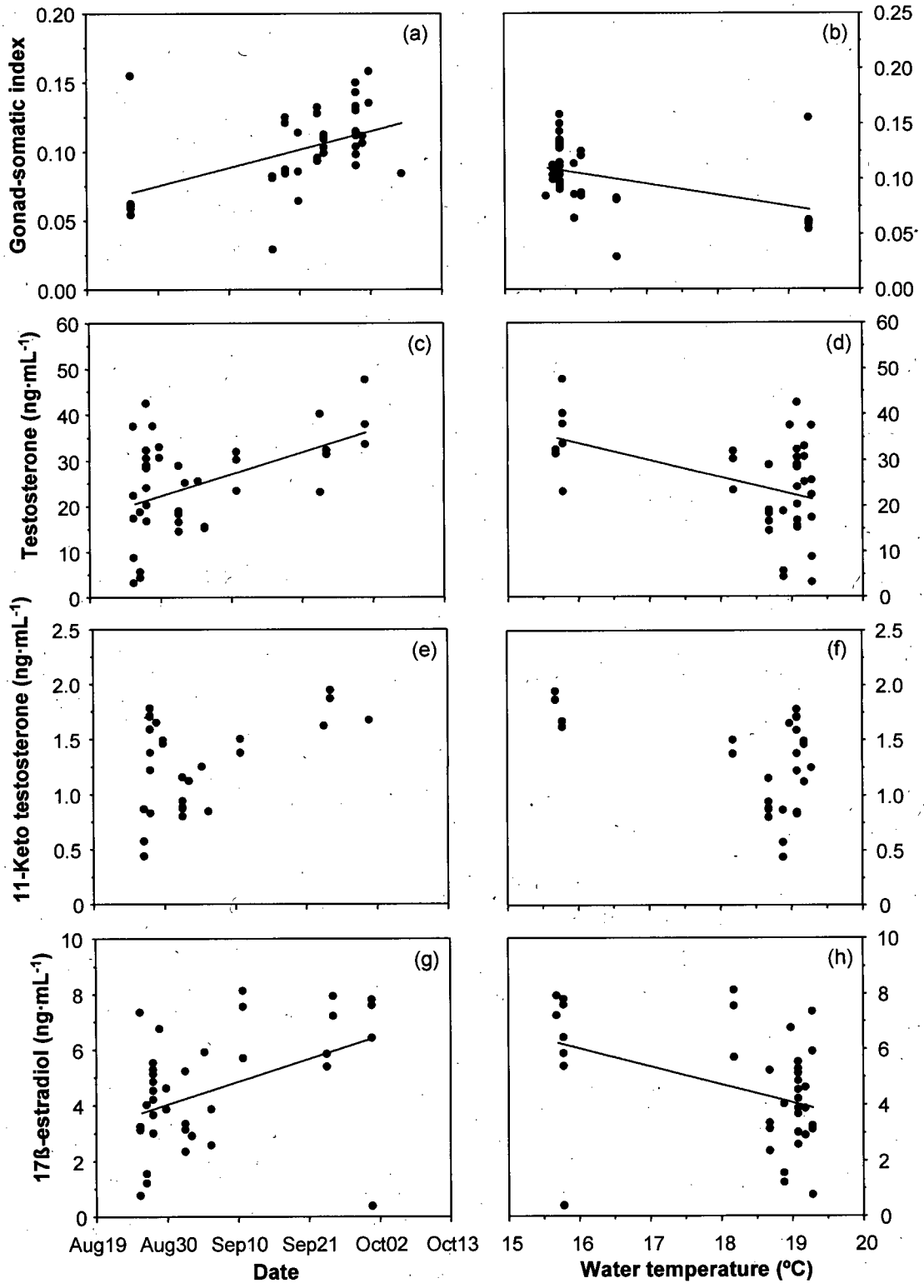

Figure 3.6. Physiological indicators of female reproductive development by time and by average stream water temperature on the date of sampling, including gonadosomatic index (a,b), plasma testosterone (c,d), plasma 11-ketotestosterone (e,f), and plasma 17β-estradiol. Linear regression lines presented when significant ($p \leq 0.0125$).

Separate regressions to isolate influence of capture date

Additional linear regression analyses of the residuals from the water temperature vs. physiological variable regression analyses did not yield any significant results. Plasma Na^+ was the only variable that was close to the α value used to assess significance (P=0.096; Table 3.1).

Table 3.1. Regression results for capture date vs. the residuals from each physiological variable that was regressed against mean daily water temperature in the Thompson River on the date of capture.

Physiological variable	R^2	P
Gross somatic energy	0.0218	0.237
log10 Plasma lactate	0.00148	0.632
log10 Plasma glucose	0.00824	0.919
Plasma Na^+	0.0175	0.096
Plasma Cl^-	0.00872	0.923
Female gonadosomatic index	0.00776	0.599
Female plasma testosterone	0.00298	0.745
Female plasma 11-ketotestosterone	0.00005	0.97
Female plasma 17ß-estradiol	0.00174	0.804

Variability in reproductive hormones

The coefficient of variation was higher in early migrants for all three female reproductive hormones analyzed (Table 3.2).

Table 3.2. Corrected coefficients of variation and Z-test results for early- (sampled on or before 16 September) and normal-timed (sampled after 16 September) late-run sockeye salmon captured in the lower Thompson River in 2003.

Female reproductive hormone	Early-timed migrant		Normal-timed migrant		Z-test	
	Coefficient of variation (V^*)	N	Coefficient of variation (V^*)	N	Z	P
Plasma testosterone	44.39%	31	22.92%	7	679.6	<0.001
Plasma 11-ketotestosterone	36.24%	26	9.28%	4	684.8	<0.001
Plasma 17ß-estradiol	44.00%	30	15.22%	7	744.6	<0.001

Discussion

By sampling migrating adult late-run sockeye at a location two thirds of the way to their spawning grounds, I investigated the influence of migration timing and water temperature on energetic status, reproductive development, osmoregulation, and stress. My findings support my first hypotheses: earlier migrants have higher energy density while being less reproductively advanced. Energy density and measures of reproductive development appeared to change consistently over the migration period, further supporting the importance of the interaction between reproductive development and energy stores in driving the availability of somatic energy at this point in the migration. Higher water temperatures were not associated with decreased energy stores, suggesting that these adverse conditions did not prematurely deplete energy for most individuals. Stress indicators provided mixed results, with higher plasma lactate but lower plasma glucose in early migrants and no association between migration timing and plasma cortisol. Plasma ions did not indicate higher stress for early migrants or migrants that encountered higher temperature, instead showing an opposite relationship in which early migrants had higher

plasma ion concentrations, possibly indicating a stronger association of ions with the level of maturation.

Higher energy stores and lower reproductive development in early migrants supports the likely role of a fixed maturation trajectory in driving the timing of reproductive development during the migration and subsequent energy availability. As revealed by Young et al. (2006) early migrants that died en route to spawning grounds had higher reproductive hormones and lower gross somatic energy part way through the migration than those early migrants that reached spawning grounds. Thus, en route mortality may be associated with those individuals with comprised migration ability due to maturation, such as reduced swimming performance (Williams and Brett 1987) or an insufficient reproductive lifespan to survive and spawn successfully. In this study early migrants had higher variance in reproductive hormones than normal-timed migrants. This higher variance in early migrants could be due to a proportion of early migrants with advanced maturation and subsequently reduced energy stores. If this is true, higher variance in early migrants further suggests that early reproductive development is more common in early migrants and thus, early reproductive development may be a unique en route or pre-spawning mortality mechanism amongst early migrants.

Plasma lactate was higher in early migrants, suggesting that higher temperatures or challenging water flows may have contributed to increased anaerobic metabolism associated with burst swimming (Black 1958; Hinch and Bratty 2000). Overall, lactate levels were higher than resting levels (<2 $mmol \cdot L^{-1}$) but usually less than 10 $mmol \cdot L^{-1}$). A previous study evaluating physiological stress in commercially caught coho salmon (*O. kisutch*) found plasma lactate levels post-capture of more than 11 $mmol \cdot L^{-1}$ and more than

20 $mmol \cdot L^{-1}$ after 60 minutes of retention (Farrell et al. 2000). Thus, sockeye salmon captured in this study were exhibiting elevated plasma lactate levels greater than resting, as would be expected given the challenging migration conditions at and immediately downstream of the capture location. However, lactate levels were usually less than half of the maximum level observed by Farrell et al. (2000), suggesting that these fish may not have been impeded by exercise-related stress and that capture methods used in this study contributed less to anaerobic metabolism than commercial ocean capture methods. Plasma lactate levels did not exceed the 15.0 $mmol \cdot L^{-1}$ threshold identified by Jain et al. (1998) for impaired critical swimming ability and only four fish exceeded 10 $mmol \cdot L^{-1}$. The lack of change in plasma cortisol with migration timing or temperature further suggests that anaerobic metabolism did not contribute to other indicators of stress. Plasma glucose levels were slightly lower in early migrants confounding the results for lactate, and possibly supporting the first hypothesis instead. Kubokawa et al. (1999) showed that plasma glucose levels increased in sockeye salmon through the breeding season. Higher glucose in later migrants may indicate that these individuals are closer to spawning.

No change in plasma cortisol associated with migration timing suggests that individuals were not acutely stressed as a result of capture or recent environmental conditions. Further, individuals with higher levels of reproductive development were not associated with hypercortisolism, which has been linked with senescence in salmonids (Barry et al. 2001). $[Na^+]$ and $[Cl^-]$ was actually higher in early migrants, rather than lower as might be expected if freshwater osmoregulation was failing as a result of physiological challenge and loss of homeostasis (Hasegawa et al. 1987; Ackerman et al. 2000).

Higher plasma [Na^+] and [Cl^-] in early migrants is difficult to explain. Other studies have shown that during the freshwater migration plasma ions initially decline, followed by a relatively stable level through migration and early spawning, and finally a sharp decline with post-spawning and senescence (Shrimpton et al. 2005). Early migrants may still be within the initial period of decline, although the mechanism is difficult to determine since both early and normal migrants that were successful in reaching spawning grounds spent similar amounts of time in freshwater and reaching the point of sampling (Wagner et al. 2005).

Average somatic energy and reproductive hormone levels in this study were more similar to early migrating survivors and generally higher than early migrants that did not reach spawning grounds in Young et al. (2006). Thus, although this en route mortality mechanism is relatively constrained to aberrantly early migrants it did not likely contribute to the majority of en route mortality amongst early migrants in 2003. Thus, research to date suggests that the elevated mortality in early migrating late-run sockeye is comprised of at least two mortality mechanisms: en route mortality due to early maturation and late en route and pre-spawning mortality due to accumulation of more than approximately 450 degree days in freshwater, with *P. minibicornis* (St-Hilaire et al. 2002, Jones et al. 2003) as a potential agent of mortality (Wagner et al. 2005). Future investigations should also consider potential interactions between these two mechanisms, including the possibility that accumulation of degree days beyond the 450 degree day range influences reproductive development, senescence, and associated mortality prior to spawning. By refining the associations between premature reproductive development and energy levels with en route mortality over a number of migration seasons it may be

possible to develop tools based on physiological measures to predict en route mortality levels in late-run Fraser River sockeye salmon that will enhance management and conservation of these important populations. These mechanisms may vary at different locations with different meta-populations, and in years with different river conditions. Further efforts are necessary to determine migration timing and temperature effects that are relatively consistent across environments and stocks, and those that are more specific to one group of salmon in a particular location.

In summary, results from this study suggest that a relatively fixed reproductive lifespan timed to meet a traditional spawn timing has a strong influence on the energy stores and level of reproductive development in adult sockeye salmon sampled through their migration period. Individuals that deviate from this pattern, in particular early migrants with advanced reproductive development and subsequently lower energy stores, are more likely to fail to complete the migration and spawn (Young et al. 2006). Aberrantly early migrants in this study had more variable levels of reproductive development. It is possible that this higher variability was influenced by a sub-set of early migrants with advanced reproductive development that more likely to fail the migration. Elevated water temperatures, which occurred earlier in the migration period in this study, were associated with higher plasma lactate levels. However, other measures of stress were not related to these potential stressors and may not provide suitable physiological indicators for in-season management purposes.

References

Ackerman, P. A., Forsyth, R. B., Mazur, C. F., and Iwama, G. K. 2000. Stress hormones and the cellular stress response in salmonids. Fish Physiol. Biochem. 23: 327-336.

Beacham, T. D., Withler, R. E., and Wood., C. C. 1995. Stock identification of sockeye salmon by means of minisatellite DNA variation. N. Am. J. Fish. Manage. 15: 249-265.

Black, E. C. 1958. Hyperactivity as a lethal factor in fish. J. Fish. Res. Board. Can. 15: 573-584.

Brett, J. R. 1995. Energetics. In Physiological Ecology of Pacific Salmon. Edited by C. Groot, L. Margolis, and W. C. Clarke. University of British Columbia Press, Vancouver. 3-68 pp.

Brown, R. S., Geist, D. R., and Mesa, M. G. 2002. The Use of Electromyogram (EMG) Telemetry to Assess Swimming Activity and Energy Use of Adult Spring Chinook Salmon Migrating through the Tailraces, Fishways, and Forebays of Bonneville Dam, 2000 and 2001. U.S. Army Corps of Engineers, Portland, Oregon. CENWP MIPR No. W66QK10432368. 94 pp.

Cooke, S. J., Hinch, S. G., Farrell, A. P., Lapointe, M. F., Jones, S. R. M., Macdonald, J. S., Patterson, D. A., Healey, M. C., and Van Der Kraak, G. 2004. Abnormal migration timing and high en route mortality of sockeye salmon in the Fraser River, British Columbia. Fisheries 29(2): 22-33.

Cooke, S.J., S.G. Hinch, G.T. Crossin, D.A. Patterson, K.K. English, J. M. Shrimpton, G. Van Der Kraak, and A.P. Farrell. 2006. Physiology of individual late-run Fraser River sockeye salmon *Oncorhynchus nerka* sampled in the ocean correlates with fate during spawning migration. Can. J. Fish. Aquat. Sci. 63: 1469-1480.

Cooke, S.J., G.T. Crossin, D. Patterson, K. English, S.G. Hinch, J.L. Young, R. Alexander, M.C. Healey, G. Van Der Kraak, and A.P. Farrell. 2005. Coupling non-

invasive physiological and energetic assessments with telemetry to understand inter-individual variation in behaviour and survivorship of sockeye salmon: development and validation of a technique. J. Fish. Biol. 67:1342-1358.

Crossin, G. T., Hinch, S. G., Farrell, A. P., Higgs, D. A., Lotto, A. G., Oakes, J. D., and Healey, M. C. 2004. Energetics and morphology of sockeye salmon: effects of upriver migratory distance and elevation. J. Fish Biol. 65: 788-810.

Crossin, G. T., and Hinch, S. G. 2005. A non-lethal method for assessing the somatic energy content of freely migrating adult Pacific salmon. Trans. Am. Fish. Soc. 134: 184-191.

Eddy, F. B. 1981. Effects of stress on osmotic and ionic regulation in fish. In Stress and Fish. Edited by A. D. Pickering. Academic Press. 77-101 pp.

Farrell, A. P., Gallaugher, P., Clarke, C., DeLury, N., Kreiberg, H., Parkhouse, W., and Routledge, R. 2000. Physiological status of coho salmon (*Oncorhynchus kisutch*) captured in commercial nonretention fisheries. Can. J. Fish. Aquat. Sci. 57: 1668-1678.

Gilhousen, P. 1980. Energy sources and expenditures in Fraser River sockeye salmon during their spawning migration. International Pacific Salmon Fisheries Communication Bulletin 22: 1-51.

Hasegawa, S., Hirano, T., Ogasawara, T., Iwata, M., Akiyama, T., and Arai, S. 1987. Osmoregulatory ability of chum salmon, *Oncorhynchus keta*, reared in freshwater for prolonged periods. Fish Physiol. Biochem. 4: 101-110.

Hendry, A. P., and Berg, O. K. 1999. Secondary sexual characters, energy use, senescence, and the cost of reproduction in sockeye salmon. Can. J. Zool. 77: 1663-1675.

Hendry, A. P., Morbey, Y. E., Berk, O. K., and K., W. J. 2004. Adaptive variation in senescence: reproductive lifespan in a wild salmon population. Proc. R. Soc. Ser. B. Bio 271: 259-266.

Hinch, S. G., and Rand, P. S. 1998. Swim speeds and energy use of upriver-migrating sockeye salmon (*Oncorhynchus nerka*): role of local environment and fish characteristics. Can. J. Fish. Aquat. Sci. 55: 1821-1831.

Hinch, S. G., and Bratty, J. 2000. Effects of swim speed and activity pattern on success of adult sockeye salmon migration through an area of difficult passage. Trans. Am. Fish. Soc. 129: 598-606.

Hinch, S. G., Standen, E. M., Healey, M. C., and Farrell, A. P. 2002. Swimming patterns and behaviour of upriver-migrating adult pink (*Oncorhynchus gorbuscha*) and sockeye (*O. nerka*) salmon as assessed by EMG telemetry in the Fraser River, British Columbia, Canada. Hydrobiologia 483: 147-160.

Hinch, S.G., Cooke, S.J., Healey, M.C. and Farrell, A.P. 2005. Behavioural physiology of fish migrations: salmon as a model approach. In: Behaviour and Physiology of Fish, Fish Physiology Series, Vol. 24 (ed. K.A. Sloman, R.W. Wilson, and S. Balshine) Academic Press, London.

Hodgson, S., and Quinn, T. P. 2002. The timing of adult sockeye salmon migration into freshwater: adaptations by populations to prevailing thermal regimes. Can. J. Zoology 80: 542-555.

Houston, A. H. 1990. Blood and Circulation. In Methods for Fish Biology. Edited by C. B. Schreck and P. B. Moyle. American Fisheries Society, Bethesda, MD. 273-334 pp.

Jain, K. E., Birtwell, I.K., and Farrell, A.P. 1998. Repeat swimming performance of mature sockeye salmon following a brief recovery period: a proposed measure of fish health and water quality. Can. J. Zool. 76: 1488-1496.

Jones, S.R.M., Prosperi-Porta, G., Dawe, S., and Barnes, D. 2003. Distribution, prevalence and severity of *Parvicapsula minibicornis* infections among anadromous salmonids in the Fraser River, British Columbia, Canada. Dis. Aquat. Org. 54: 49–54.

Kubokawa, K., Watanabe, T., Yoshioka, M., and Iwata, M. 1999. Effects of acute stress on plasma cortisol, sex steroid hormone and glucose levels in male and female sockeye salmon during the breeding season. Aquaculture 172: 335-349.

Lee, C. G., Farrell, A. P., Lotto, A., MacNutt, M. J., Hinch, S. G., and Healey, M. C. 2003. The effect of temperature on swimming performance and oxygen consumption in adult sockeye (*Oncorhynchus nerka*) and coho (*O. kisutch*) salmon stocks. J. Exp. Biol. 206: 3239-3251.

Macdonald, J. S. 2000. Mortality during the migration of Fraser River sockeye salmon (*Oncorhynchus nerka*): a study of the effect of ocean and river environmental conditions in 1997. Can. Tech. Rep. Fish. Aquat. Sci. 2315. 120 pp.

McCormick, S. D. (1993). Methods for the nonlethal gill biopsy and measurements of Na^+, K^+-ATPase activity. Can J. Fish. Aquat. Sci. 50: 656-658.

McMaster, M., Munkittrick, K., and Van Der Kraak, G. 1992. Protocol for measuring circulating levels of gonadal sex steroids in fish. Can. Tech. Rep. Fish Aquat. Sci. 1836. 29 pp.

Morisawa, M., Hirano, T., and Suzuki, K. 1979. Changes in blood and seminal plasma composition of the mature salmon (*Oncorhynchus keta*) during adaptation to freshwater. Comp. Biochem. Physiol., A: Mol. Integr. Physiol. 64: 325-329.

Munakata, A., Amano, M., Ikuta, K., Kitamura, S., and Aida, K. 2001. The involvement of sex steroid hormones in downstream and upstream migratory behavior of masu salmon. Comp. Biochem. Physiol. B: Biochem. Mol. Biol. 129: 661-669.

Patterson, D. A., MacDonald, J. S., Hinch, S. G., Healey, M. C., and Farrell, A. P. 2004. The effect of exercise and captivity on energy partitioning, reproductive maturation and fertilization success in adult sockeye salmon. J. Fish Bio. 64: 1039-1059.

Quinn, T. P., Hodgson, S., and Peven, C. 1997. Temperature, flow, and the migration of adult sockeye salmon (Oncorhynchus nerka) in the Columbia River. Can. J. of Fish. Aquat. Sci. 54: 1349-1360.

Quinn, T. P. 2005. The Behavior and Ecology of Pacific Salmon & Trout. UBC Press, Vancouver, BC, 378 pp.

Shrimpton, J.M., D.A. Patterson, J.G. Richards, S.J. Cooke, P.M. Schulte, S.G. Hinch, and A.P. Farrell. 2005. Ionoregulatory changes in different populations of maturing sockeye salmon (*Oncorhynchus nerka*) during ocean and river migration. J. Exp. Biol. 208:4069-4078.

Sokal, R. R, and Rohlf, F. J. 1981. Biometry: The Principles and Practices of Statistics in Biological Research, Second Edition. W.H. Freeman and Company, New York. p. 59.

St-Hilaire, S., Boichuk, M., Barnes, D., Higgins, M., Devlin, R., Withler, R., Khattra, J., Jones, S., and Kieser, D. 2002. Epizootiology of Parvicapsula minibicornis in Fraser River sockeye salmon, Oncorhynchus nerka (Walbaum). J. Fish Diseases 25: 107-120.

Standen, E. M., Hinch, S.G., Healey, M.C., and Farrell, A.P. 2002. Energetic costs of migration through the Fraser River Canyon, British Columbia, in adult pink (*Oncorhynchus gorbuscha*) and sockeye (*Oncorhynchus nerka*) salmon as assessed by EMG telemetry. Can. J. Fish. Aquat. Sci. 59: 1809-1818.

Tveiten, H., Mayer, I., Johnsen, H. K., and Jobling, M. 1998. Sex steroids, growth and condition of Arctic charr broodstock during an annual cycle. J. Fish Biol. 53: 714-727.

Van Der Kraak, G., and Chang, J. 1990. Arachidonic acid stimulates steroidogenesis in goldfish preovulatory ovarian follicles. Gen. Comp. Endocrinol. 77: 221-228.

Wagner, G. N., Hinch, S. G., Kuchel, L. J., Lotto, A., Jones, S. R. M., Patterson, D. A., Macdonald, J. S., Van Der Kraak, G., Shrimpton, M., English, K. K., Larsson, S., Cooke, S. J., Healey, M. C., and A. P. Farrell. 2005. Metabolic rates and swimming performance of adult Fraser River sockeye salmon (*Oncorhynchus nerka*) after a controlled infection with *Parvicapsula minibicornis*. Can. J. Fish. Aquat. Sci. 62: 2124-2133.

Williams, I. V. and Brett, J. R. 1987. Critical swimming speed of Fraser and Thompson River pink salmon (*Oncorhynchus gorbuscha*). Can. J. Fish. Aquat. Sci. 44: 348-356.

Woodey, J. C. 1987. In-season management of Fraser River sockeye salmon (*Oncorhynchus nerka*): meeting multiple objectives. In: Sockeye salmon (*Oncorhynchus nerka*) population biology and future management. Edited by H. D. Smith, L. Margolis, and C. C. Wood. Can. Spec. Publ. Fish. Aquat. Sci. 96. pp.

Young, J. L., Hinch, S. G., Cooke, S. J., Crossin, G. T., Patterson, D. A., Farrell, A. P., Van Der Kraak, G., Lotto, A. G., Lister, A., Healey, M. C., and English, K. K. 2006. Physiological and energetic correlates of en route mortality for abnormally early migrating adult sockeye salmon (*Oncorhynchus nerka*) in the Thompson River, British Columbia. Can. J. Fish. Aquat. Sci. 63: 1067-1077.

Zar, J. H. 1999 Biostatistical Analysis, Fourth Edition. Prentice-Hall Inc., New Jersey. Pp 141-145.

CHAPTER 4 - CONCLUSIONS

This study effectively integrated complex and diverse components of sockeye salmon physiology and behaviour during their upstream spawning migration. The first chapter utilized bio-telemetry, sampling physiological measures of energetic status, reproductive development, osmoregulatory status, and stress to identify links between migration behaviour, en route mortality, and physiology. The second chapter intensively sampled a broad suite of physiological variables in more than 200 adult sockeye salmon at a single site part-way along the freshwater spawning migration. Individuals were sampled continuously over the entire range of time that sockeye salmon were migrating past this location. Together, these studies identified potential mechanisms and causes of en route mortality and further resolved the role of reproductive lifespan in influencing migration physiology and mortality.

The first set of conclusions relates to the role of reproductive development on the availability of energy stores and en route mortality. Reproductive development and energy stores showed a strong inverse relationship throughout the migration period, with early migrants on average having higher energy stores and lower reproductive development. This result strongly supports the concept of a fixed reproductive lifespan timed (e.g., Hendry 2004) to ensure adults are mature at the optimal time for spawning, more generally suggesting that reproductive development and subsequent energy stores are most strongly influenced by the time left before spawning, rather than the migration location or environmental conditions (Dahl et al. 2004).

Bio-telemetry results suggest that deviations from this pattern in aberrantly early migrants, with relatively high reproductive development and reduced energy stores,

contribute to en route mortality. Potential mechanisms for this mortality could be increased migration ground speeds, possibly driven by premature reproductive development (Munakata et al. 2001) and resulting in inefficient energy use. However, early reproductive development may also reduce reproductive success by increasing exposure to degree days in freshwater (Wagner et al. 2005) and resulting in reproductive maturity prior to the optimal spawn timing window (Quinn et al. 2002).

Although the primary conclusion from this study indicates that migration timing, and associated differences in environmental conditions, did not play a significant a role in migration success, some results did support the role of challenging migratory conditions in affecting migration physiology and providing mechanisms for en route mortality. An attempt to surgically implant electromyogram telemetry transmitters in a subset of adult sockeye salmon resulted in abnormally high bleeding, with longer blood clotting times in early migrants. Although the mechanisms for this bleeding phenomenon are unknown, it provides an additional mechanism for en route mortality that could be exacerbated by challenging water flows or high temperature. Water temperatures declined through the migration period in both the Thompson River, and the Fraser River prior to the confluence with the Thompson River, in 2003. Early migrants had higher plasma lactate, possibly due to these higher temperatures (Farrell et al. 2000) or more difficult flow conditions, which were not directly assessed. However, other stress indicators were not associated with temperature or migration timing. Although these results suggest a secondary role for temperature in en route mortality results from a broader research initiative, of which this thesis is a part, has identified increased mortality prior to spawning as a result of accumulating degree days in freshwater, particularly over a

threshold of approximately 450 degree days (Wagner et al. 2005). None of the sockeye salmon captured and sampled in this study would have accumulated more than 400 degree days when sampled. Thus, this temperature-dependent mechanism for mortality may not have manifested itself in the migrants captured in this study. Overall, the combination of this thesis and other work of the research group suggests at least two major mechanisms for en route mortality: 1) elevated reproductive development and reduced energy stores in early migrants, and 2) accumulation of more than 450 degree days in freshwater prior to spawning.

The primary strength of this research is the integration of analyses of sockeye salmon physiology with the migration behaviour, mortality, and environmental conditions of sockeye salmon during their spawning migration. The concept of reproductive lifespan in semelparous sockeye salmon was refined and supported while more specific potential mechanisms and indicators of en route mortality were identified. However, this research was largely investigative in nature and used relatively untested methods of bio-telemetry. Results and conclusions identify the need for more specific field and empirical studies to further refine mechanisms of en route mortality and the causal links between physiology and behaviour. For example, my attempt to integrate physiological sampling with EMG telemetry largely failed and revealed an unexpected bleeding phenomenon. Causes for this phenomenon are unknown, but further investigations should evaluate the influence of *Parvicapsula minibicornis* infection (St-Hilaire et al. 2002, Jones et al. 2003) on osmoregulatory function, behaviour (Barber et al. 2000) and the role of migratory stressors on blood clotting ability. Further research is needed to identify the physical mechanisms responsible for en route mortality in early migrants with elevated

reproductive development. Reduced energy stores and prolonged time spent in freshwater provide likely mechanisms, but further efforts could identify whether early reproductive development is associated with premature senescence.

References

Barber, I., Hoare, D., and Krause, J. 2000. Effects of parasites on fish behaviour: a review and evolutionary perspective. Rev. Fish Biol. Fish. 10: 131-165.

Dahl, J., Dannewitz, J., Karlsson, L., Petersson, E., Löf, and Ragnarsson, B. 2004. The timing of spawning migration: implications of environmental variation, life history, and sex. Can. J. Zool. 82: 1864-1870.

Farrell, A. P., Gallaugher, P., Clarke, C., DeLury, N., Kreiberg, H., Parkhouse, W., and Routledge, R. 2000. Physiological status of coho salmon (*Oncorhynchus kisutch*) captured in commercial nonretention fisheries. Can. J. Fish. Aquat. Sci. 57: 1668-1678.

Hendry, A. P., Morbey, Y. E., Berk, O. K., and K., W. J. 2004. Adaptive variation in senescence: reproductive lifespan in a wild salmon population. Proc. R. Soc. Ser. B. Bio 271: 259-266.

Jones, S.R.M., Prosperi-Porta, G., Dawe, S., and Barnes, D. 2003. Distribution, prevalence and severity of *Parvicapsula minibicornis* infections among anadromous salmonids in the Fraser River, British Columbia, Canada. Dis. Aquat. Org. 54: 49–54.

Quinn, T. P., Peterson, J. A., Gallucci, V. F., Hershberger, W. K., and Brannon, E. L. 2002. Artificial selection and environmental change: Countervailing factors

affecting the timing of spawning by coho and chinook salmon. Trans. Am. Fish. Soc. 131: 591-598.

St-Hilaire, S., Boichuk, M., Barnes, D., Higgins, M., Devlin, R., Withler, R., Khattra, J., Jones, S., and Kieser, D. 2002. Epizootiology of Parvicapsula minibicornis in Fraser River sockeye salmon, Oncorhynchus nerka (Walbaum). J. Fish Diseases 25: 107-120.

Wagner, G. N., Hinch, S. G., Kuchel, L. J., Lotto, A., Jones, S. R. M., Patterson, D. A., Macdonald, J. S., Van Der Kraak, G., Shrimpton, M., English, K. K., Larsson, S., Cooke, S. J., Healey, M. C., and A. P. Farrell. 2005. Metabolic rates and swimming performance of adult Fraser River sockeye salmon (*Oncorhynchus nerka*) after a controlled infection with *Parvicapsula minibicornis*. Can. J. Fish. Aquat. Sci. 62: 2124-2133.

APPENDICES

Appendix 1 - Physiology and morphology data for all sockeye salmon captured in the Thompson River, summer/fall 2003

FISH #	Date	Sex	FL (cm)	Body depth (cm)	Weight (g)	Liver (g)	Gonads (g)	Gross somatic energy (MJ kg-1)	Plasma Na (mEq L-1)	Plasma K (mEq L-1)	Plasma Cl (mEq L-1)	Plasma osmolality (mOsmol L-1)	Plasma cortisol (ng mL-1)	Plasma glucose (mmol L-1	Plasma lactate (mmol L-1)	Time to 100% blod clot (sec)	Male testosterone (pg/mL)	11-ketotestosterone (pg/mL)	17-beta estradiol (pg/mL)	Female testosterone (pg/mL)
635	26-Aug	F	58		2271	59	321	6.99	178	4.5	141	312	285	3.630	13.200				3512	28750
636	26-Aug	M	62		2757	24	82.7	6.40	167	6.0	123	279	125	4.930	3.580		21490	9276		
637	26-Aug	M	63		2503	64	47.5	7.05	151	6.1	112	288	476	5.070	11.450		2820			
638	26-Aug	F	57		3020	45	270	5.78	147	2.7	113	299	412	5.265	11.250				871.9	16420
639	26-Aug	F	58		2247	55	134	8.41	167	2.6	133	298	14	4.520	6.005				3203	17100
640	26-Aug	F	58		1444	47	223	5.78	163	2.6	124	302	223	4.125	8.330				7304	37270
641	26-Aug	M	64		2926	54	88.8	5.78	160	3.7	124	284	14	5.345	3.610		29880	16420		
642	26-Aug	F	60		2950	59	170	8.10	161	5.0	126	309	114	5.225	6.225				3076	22170
643	26-Aug	F	59		2246	56	120	7.53	133	5.2	91	254	468	8.255	14.250					2957
644	26-Aug	F	61		2516	57	154	8.11	164	2.6	125	284	105	5.315	9.480				711.3	8491
645	26-Aug	F	61		2254	52	293	6.36	153	1.4	119	291	489	5.890	8.995				885.8	6844
6002	28-Aug	F	60					8.87	165	1.8	131	326	246	4.095	5.480			1364	4805	28750
6003	29-Aug	F	60					6.75	166	2.6	128	318	86	3.715	5.585			1726	6378	35580
6004	29-Aug	F	65					6.53	170	1.6	128	322		3.290	7.855			2043	6747.5	43130
6005	29-Aug	F							168	1.3	133	329		4.135	6.145			1640	6711	37290
6006	30-Aug	F	59					5.44	177	3.7	132	330	19	3.565	6.355			2336	6681	46880
6007	30-Aug	F	58					6.12	167	3.8	133	312		3.460	4.995			1491	5605	34640
6008	30-Aug	F							174	4.3	128	310		4.055	4.710			1447	4577	32750
6009	30-Aug	F	56					6.89	153	3.7	128	313	73	3.555	5.055			1479	3824	30470
6010	30-Aug	F							189	0.0	125	318		5.005	5.010					
6011	2-Sep	F							167	1.3	132	327		3.515	5.805			788.3	2305	14320
6012	3-Sep	F							160	2.2	130	310		3.205	3.410			1111	2865	24900

FISH #	Date	Sex	FL (cm)	Body depth (cm)	Weight (g)	Liver (g)	Gonads (g)	Gross somatic energy (MJ kg-1)	Plasma Na (mEq L-1)	Plasma K (mEq L-1)	Plasma Cl (mEq L-1)	Plasma osmolality (mOsmol L-1)	Plasma cortisol (ng mL-1)	Plasma glucose (mmol L-1	Plasma lactate (mmol L-1)	Time to 100% blod clot (sec)	Male testosterone (pg/mL)	11-ketotestosterone (pg/mL)	17-beta estradiol (pg/mL)	Female testosterone (pg/mL)
6013	2-Sep	F	61					8.20	168	2.6	132	323	24	3.500	6.780			927.7	3099	18050
6014	2-Sep	F	62					7.69	175	3.4	134	325	79	3.465	7.765			857.9	2300	18730
6015	2-Sep	F	60					8.68	173	5.7	134	328		4.190	5.805			869.7	3304	16320
6016	2-Sep	F	63					8.27	154	3.6	132	333	3	3.745	5.150			1144	5190	28690
6017	5-Sep	F	61					8.39	175	0.1	128	313		3.530	5.115			1239.5	5868	25300
6018	6-Sep	F							144	1.1	126	331		4.500	5.920			833.4	2527	15000
6019	11-Sep	F	61					7.92	156	1.7	130	324	112	4.255	4.905			1363	7508	31720
6020	11-Sep	F							165	3.1	127	313		3.330	3.165			1365	5658	30010
6021	11-Sep	F							165	2.8	131	317		4.810	4.415			1490	8081	23130
6022	16-Sep	F	63					7.33	161	2.1		339		4.695	5.140					
6023	17-Sep	F							161	1.9		318		4.730	7.655					
6024	21-Sep	F	60					6.68	153	1.0		309		4.120	3.170					
6025	21-Sep	F	64					6.49		1.7		313		4.205	4.660					
6026	24-Sep	F	63					7.08	158	3.1	122	310	4	4.455	3.880			1609	5350	22880
6027	25-Sep	F	65					5.90	163	-0.1	126	308		4.635	3.730			1932	7889	32010
6028	25-Sep	F	65					6.36	168	0.6	127	326		3.935	2.470			1857	7172	31150
6029	1-Oct	F	59					6.68	160	3.1	126	304	111	3.680	3.950			1662	6377	33360
6101	25-Aug		57																	
6102	25-Aug	F	55					6.49	159	-0.5	125	367	207	6.590	5.410			1185.5	6268	20000
6103	26-Aug		56																	
6104	27-Aug	F	55					7.20	165	-0.3	131	346	226	5.105	8.855			1115	4083	24570
6105	27-Aug		66																	
6106	27-Aug	F	62					6.96	166	0.5	129	325	684	3.405	13.250			1563	3816	30810
6107	27-Aug		57																	
6108	27-Aug	F	63					9.33	160	0.3	130	311	492	3.985	5.145			561.8	1503	5399
6109	27-Aug		60																	
6110	27-Aug	M	62					7.76	159	2.3	131						8128	2704	174.2	
6111	27-Aug		57																	

FISH #	Date	Sex	FL (cm)	Body depth (cm)	Weight (g)	Liver (g)	Gonads (g)	Gross somatic energy (MJ kg-1)	Plasma Na (mEq L-1)	Plasma K (mEq L-1)	Plasma Cl (mEq L-1)	Plasma osmolality (mOsmol L-1)	Plasma cortisol (ng mL-1)	Plasma glucose (mmol L-1	Plasma lactate (mmol L-1)	Time to 100% blod clot (sec)	Male testosterone (pg/mL)	11-ketotestosterone (pg/mL)	17-beta estradiol (pg/mL)	Female testosterone (pg/mL)
6112	27-Aug	F	58					8.13	161	2.4	130	344	77	4.190	7.800			854.7	3985	18520
6113	27-Aug		61					8.50												
6114	27-Aug	F	61					8.87	159	3.5	131	374	78	5.455	2.340			427.8	1163	4097
6115	27-Aug		62					8.47												
6116	27-Aug	M	74					7.08	165	2.2	129	373	37	4.335	4.755		18220	8566	74.92	
6117	28-Aug		66					5.96												
6118	28-Aug	F	62					6.07	161	1.3	129	323	124	5.895	4.495			1578	3619	23830
6119	28-Aug		66					8.20												
6120	28-Aug	F	53					6.53	170	2.0	127	349	161	4.635	6.845			2356.5	4167	42190
6121	28-Aug		63					8.20												
6122	28-Aug	F	57					7.31	157	2.7	131	359	106	3.965	3.615			1417	6493	22180
6123	28-Aug		62					6.12												
6124	28-Aug	F	57					6.17	159	4.3	128	310	145	4.940	7.450			1768	5491	28210
6125	28-Aug		61					7.46												
6126	28-Aug	M	64					7.69	161	3.5	129	327	40	3.665	3.200		11490	3611	215.9	
6127	28-Aug		60					6.86												
6128	28-Aug	M	62					7.51	168	1.8	131	382	18	3.875	6.285		19460	7050	227.7	
6129	28-Aug	F	63					6.17	164	3.8	131	328	286	4.530	3.820				3181	22340
6130	28-Aug	F	57					7.48	169	3.2	133	371	645	4.275	13.750			1701	5080	32010
6131	28-Aug		62					7.94												
6132	28-Aug	M	60					7.36	165	4.0	134	327	21	4.355	5.980		17310	5873	82.2	
6133	28-Aug		61					8.41												
6134	28-Aug	F	66					7.80	167	1.0	129	403	168	3.970	7.930			1208	2962	20000
6135	28-Aug		60					8.29												
6136	28-Aug	F	64					6.31	156	3.5	131	384	110	4.270	6.715			1693	5246	30340
6137	28-Aug		61					7.72												
6138	28-Aug	F	60					8.31	160	3.6	131	328	68	4.470	3.715			758.55	2205	12990
6139	28-Aug		61					6.49												

FISH #	Date	Sex	FL (cm)	Body depth (cm)	Weight (g)	Liver (g)	Gonads (g)	Gross somatic energy (MJ kg-1)	Plasma Na (mEq L-1)	Plasma K (mEq L-1)	Plasma Cl (mEq L-1)	Plasma osmolality (mOsmol L-1)	Plasma cortisol (ng mL-1)	Plasma glucose (mmol L-1	Plasma lactate (mmol L-1)	Time to 100% blod clot (sec)	Male testosterone (pg/mL)	11-ketotestosterone (pg/mL)	17-beta estradiol (pg/mL)	Female testosterone (pg/mL)
6140	28-Aug	M	65					5.58	160	3.9	127	334	32	4.150	7.045		17990	7916	230.3	
6141	28-Aug		63					8.04												
6142	28-Aug	M	66					8.62	166	2.1	132	368	31	4.680	7.900		16340	5297	86.3	
6143	28-Aug		62					5.44												
6144	28-Aug	F	59					8.26	165	4.0	131	345	41	4.505	4.545			818.6	4485	16550
6145	28-Aug		64					7.80												
6146	29-Aug		64					7.51												
6147	29-Aug		59					9.14												
6148	29-Aug		61					5.78												
6151	5-Sep	M	63						166	0.4	127						20460	6406		
6152	6-Sep	M	66					7.51	166	1.9	135	350	79	4.980	4.035		10940	4915		
6153	6-Sep	F	63					8.59	168	2.9	132	344	290	4.975	4.635				3820	15310
6154	9-Sep	M	62					5.18												
6155	11-Sep	M	62																	
6156	11-Sep	M	63																	
6157	11-Sep	M	64																	
6158	11-Sep	F	60																	
6159	16-Sep	M	63					6.22												
6160	17-Sep	M	64					6.27	153	1.0	153	311	0	4.620	3.905					
6161	17-Sep	M	64					6.40	150	1.2		306	25	5.185	2.570					
6162	17-Sep	M	65					6.92	160	1.5		315	14	5.520	8.080					
6163	17-Sep	M	66					6.99	151	1.4		310	11	4.720	3.875					
6164	17-Sep	F	62					7.05	151	0.6	154	309	209	5.290	5.340					
6165	17-Sep	M	66					7.63	156	1.4		310	106	4.570	5.450					
6166	17-Sep	F	63					7.56	160	0.7		318	23	5.080	7.115					
6167	17-Sep	F	64					4.87	158	2.2		320	500	4.350	8.655					
6168	17-Sep	F	63					4.87	159	2.2		307	38	4.290	4.070					
6169	19-Sep	F	57					6.68	155	1.1	155	318	136	3.895	4.090					

FISH #	Date	Sex	FL (cm)	Body depth (cm)	Weight (g)	Liver (g)	Gonads (g)	Gross somatic energy (MJ kg-1)	Plasma Na (mEq L-1)	Plasma K (mEq L-1)	Plasma Cl (mEq L-1)	Plasma osmolality (mOsmol L-1)	Plasma cortisol (ng mL-1)	Plasma glucose (mmol L-1	Plasma lactate (mmol L-1)	Time to 100% blod clot (sec)	Male testosterone (pg/mL)	11-ketotestosterone (pg/mL)	17-beta estradiol (pg/mL)	Female testosterone (pg/mL)
6170	19-Sep	F	60					7.08	153	1.7		306	113	4.600	3.695					
6171	24-Sep	F	57					5.72	166	0.2	127	307	19	4.285	2.875				5802	39900
6172	24-Sep	M	65					5.96	170	0.6	128	340	40	4.600	3.655		20840	9023		
6173	30-Sep	M	64					5.90	172	0.6	133	370	63	4.625	4.020		10470	3074		
6174	1-Oct	M	65					5.08	181	1.6	129	318		4.340	1.760		21260	11730		
6175	1-Oct	F	63					4.98												
6176	1-Oct	F	55					4.98	164	2.7	136	320	202	4.185	6.015				7548	47390
6177	1-Oct	M	65					5.84												
6178	1-Oct	F	64					6.40	150	3.5	125	304		4.095	3.250				7760	37660
6179	1-Oct	F	60					6.49												
6180	1-Oct	F	60					4.87	168	3.2	132	324		4.625	5.455		27710		329.5	
6181	1-Oct	M	65					5.18												
6201	4-Sep	M	67					7.46	154	0.7	126	296	53	4.290	4.245					
6202	4-Sep	F	63					8.00	141	2.7	122	290	272	4.090	5.340					
6203	4-Sep	M	62					8.02	154	0.3	127		198							
6204	4-Sep	M	62					7.14	147	5.1	128	300	195	3.120	8.820					
6205	4-Sep	F	64					7.92	154	0.7	123	305	333	4.060	11.150					
6206	4-Sep	M	63					7.28	141	2.0	127	304	140	3.810	5.680					
6207	4-Sep	F	57					6.96	138	6.0	118	294	118	2.905	8.245					
6208	4-Sep	F	56					4.98	145	5.6	120	295	204	4.655	10.100					
6209	4-Sep	M	63					5.72	149	4.5	123	280	119	3.885	3.830					
6210	4-Sep	F	63					8.73	148	4.4	125	303	547	4.435	8.230					
6211	5-Sep	M	64					5.51												
6212	5-Sep	M	61					4.75	150	0.2	125	300	49	4.360	5.120					
6213	5-Sep	M	66					6.60	155	1.3	132	305	83	5.025	8.315					
6214	7-Sep	M	64					7.17	158	0.1	139	316	241	6.835	7.615	270				
6215	7-Sep	F	59					8.86	143	6.0	126	292	176	4.160	7.485					
6216	7-Sep	M	66					7.60	143	4.4	132	297	88	5.230	2.855	150				

FISH #	Date	Sex	FL (cm)	Body depth (cm)	Weight (g)	Liver (g)	Gonads (g)	Gross somatic energy (MJ·kg-1)	Plasma Na (mEq·L-1)	Plasma K (mEq·L-1)	Plasma Cl (mEq·L-1)	Plasma osmolality (mOsmol·L-1)	Plasma cortisol (ng mL-1)	Plasma glucose (mmol L-1	Plasma lactate (mmol L-1)	Time to 100% blod clot (sec)	Male testosterone (pg/mL)	11-ketotestosterone (pg/mL)	17-beta estradiol (pg/mL)	Female testosterone (pg/mL)
6217	7-Sep	M	64					6.99	149	0.4	125	298		4.230	3.435					
6218	7-Sep	F	63					7.88	150	1.6	125	297	196	4.855	5.645	145				
6219	7-Sep	F	63					7.76	145	4.0	131		221			105				
6220	8-Sep	M	67	16				6.64	167	1.0	133	309		4.720	3.750	150				
6221	8-Sep	F	63	15				8.17	146	1.0	115	289	627	12.800	3.475	100				
6222	8-Sep	F	59	14				8.66	158	1.2	125	305	178	4.130	4.175					
6223	8-Sep	M	61	15					148	0.8	123	303		4.800	3.760	45				
6224	8-Sep	M	61	15					149	2.6	123	298		4.410	5.975	70				
6225	8-Sep	M	59	14				7.36	156	0.9	130	315		5.375	4.285					
6226	8-Sep	F	66	15				7.72	142	2.0	125	306	173	3.915	8.035					
6227	8-Sep	M	62	15				7.05	148	2.6	125	306		4.255	6.170	93				
6228	8-Sep	M	61	13				6.68	150	2.8	132	306		4.430	4.595	58				
6229	9-Sep	F	60	15				8.32	149	2.6	126	307	104	4.505	5.895	165				
6230	9-Sep	F	59	15				8.36	150	3.0	127	305	130	6.245	2.500	135				
6231	9-Sep	M	61	15				6.40	158	1.9	132	316		4.045	5.885					
6232	9-Sep	M	65	16				7.11	152	3.5	132	308		5.160	5.210	105				
6233	10-Sep	M	64	15				7.72	158	0.3	132	317		5.160	5.255					
6234	10-Sep	M	62	14				7.33	154	0.9	133	321		5.400	5.670	105				
6235	10-Sep	M	68	16				7.41	152	1.6	132	318		5.400	5.350	85				
6236	10-Sep	M	65	16				7.76	148	2.3	130	297		5.695	4.930	75				
6237	10-Sep	M	65	15				7.78	150	2.2	131	305		5.080	4.670	80				
6238	17-Sep	M	62					5.96	149	1.9		284		4.365	6.155					
6239	17-Sep	M	64		2449	26	70.9	5.72	161	0.9		302		4.360	3.520	180				
6240	17-Sep	F	64		2734	30	77.2	5.51	140	1.2		298	92	4.355	3.620	65				
6241	17-Sep	F	62		2687	51	215	6.22	151	3.6		309		4.615	5.270	95				
6242	17-Sep	M	66		3008	29	90.4	6.79	154	1.0		293		4.310	2.615					
6243	17-Sep	M	65		3181	31	95	5.96	161	0.0		332		4.800	2.660	68				
6244	17-Sep	F	61		2433	44	198	6.68	160	0.0		306	107	5.070	3.535	75				

FISH #	Date	Sex	FL (cm)	Body depth (cm)	Weight (g)	Liver (g)	Gonads (g)	Gross somatic energy (MJ kg-1)	Plasma Na (mEq L-1)	Plasma K (mEq L-1)	Plasma Cl (mEq L-1)	Plasma osmolality (mOsmol L-1)	Plasma cortisol (ng mL-1)	Plasma glucose (mmol L-1	Plasma lactate (mmol L-1)	Time to 100% blod clot (sec)	Male testosterone (pg/mL)	11-ketotestosterone (pg/mL)	17-beta estradiol (pg/mL)	Female testosterone (pg/mL)
6245	19-Sep	F	59	12	1883	43	226	5.72	159	1.6		311	228	3.780	3.930					
6246	19-Sep	F	60	15	2492	50	215	7.31	159	0.7		319	121	4.280	7.885	75				
6247	19-Sep	M	63	15	2855	27	91.7	6.49	161	1.5		307		5.735	5.015	70				
6248	19-Sep	F	61	13	2176	39	181	7.67	149	4.1		302	226	6.720	3.355	81				
6249	19-Sep	F	61	14	2383	47	296	6.44	156	1.7		294	258	4.915	5.425	142				
6250	19-Sep	M	68	16	3334	29	101	7.94	158	0.7		318		5.585	3.915	135				
6251	21-Sep	F	56	13	1975	45	223	6.31	142	3.1		293	113	4.275	4.550					
6252	21-Sep	M	63	15	2850	30	107	6.53	165	1.9		316		4.800	5.125					
6253	21-Sep	M	63	16	3023	30	88.1	6.53	160	1.3		332		6.035	7.455					
6254	21-Sep	M	64	16	3005	33	70.3	6.86	154	2.2		315		5.355	5.995					
6255	21-Sep	F	62	15	2556	49	216	6.89	155	3.3		305	95	4.535	9.380					
6256	21-Sep	F	58	13	2195	39	139	7.11	157	2.0		309	272	4.650	7.975					
6257	24-Sep	F	58	12	1630	29	154	4.98	151	0.5	126	307	145	4.940	6.575	52				
6258	24-Sep	M	68	17	3809	40	104	6.49	155	0.1	121	304		5.985	3.935	52				
6259	24-Sep	F	63	14	2559	54	336	5.78	142	0.0	118	301	208	5.365	4.395	79				
6260	24-Sep	F	62	14	2416	50	223	6.57	141	0.7	121	302	126	4.735	5.185	49				
6261	24-Sep	M	61	15	2610	30	92.2	5.58	143	2.4	113	302		4.955	3.980					
6262	24-Sep	M	54	12	1555	20	56.9	4.87	146	2.5	115	301		5.025	4.465	48				
6263	24-Sep	M	68	16	3259	35	112	7.05	147	1.9	117	307		4.475	3.890	86				
6264	24-Sep	F	59	14	2290	52	290	6.02	142	2.8	119	298	193	4.625	2.935	64				
6265	25-Sep	F	59	14	2251	47	244	6.27	143	0.3	119	291	105	5.310	2.745	140				
6266	25-Sep	M	68	15	3182	36	92.4	5.96	145	4.3	108	350		4.725	3.030	85				
6267	25-Sep	F	58	12	2083	42	213	6.22	153	0.4	122	293	172	5.730	4.810	129				
6268	25-Sep	M	63	16	2963	28	93.3	6.22	151	0.2	119	292		4.770	4.665	72				
6269	25-Sep	F	61	14	2254	50	221	6.31	140	2.1	116	299	29	4.425	3.660					
6270	25-Sep	F	62	15	2802	56	312	6.31	136	5.1	115	288	194	5.035	6.625					
6271	30-Sep	F	61	13	2066	38	229	6.40	146	0.6	121	305	261	4.805	5.450	61				
6272	30-Sep	F	61	14	2336	45	308	5.90	139	0.6	119	318	155	5.770	7.345	132				

FISH #	Date	Sex	FL (cm)	Body depth (cm)	Weight (g)	Liver (g)	Gonads (g)	Gross somatic energy (MJ kg-1)	Plasma Na (mEq L-1)	Plasma K (mEq L-1)	Plasma Cl (mEq L-1)	Plasma osmolality (mOsmol L-1)	Plasma cortisol (ng mL-1)	Plasma glucose (mmol L-1	Plasma lactate (mmol L-1)	Time to 100% blod clot (sec)	Male testosterone (pg/mL)	11-ketotestosterone (pg/mL)	17-beta estradiol (pg/mL)	Female testosterone (pg/mL)
6273	30-Sep	M	64	15	2670	29	95.3	5.58	145	0.4	116	309		5.405	2.715	92				
6274	30-Sep	F	58	13	2368	53	305	7.38	143	2.6	115	301	69	4.825	4.375					
6275	30-Sep	F	61	15	2803	52	250	6.64	140	4.4	119	302	119	6.125	4.305					
6276	30-Sep	F	58	13	2110	42	240	5.84	142	5.7	123	307	192	4.865	4.310	99				
6277	30-Sep	F	60	12	1906	39	284	5.35	140	6.2	115	295	244	5.775	4.135					
6278	30-Sep	M	65	17	3279	38	107	4.98	157	3.9	118	305		4.645	6.035	55				
6279	30-Sep	F	58	13	1985	36	226	5.58	147	4.7	121	296	113	4.430	5.065	41				
6280	30-Sep	F	56	12	1623	32	158	6.53	148	5.8	121	305	142	4.360	4.555	121				
6281	30-Sep	F	58	13	2060	42	212	6.36	146	4.8	119	301	96	4.545	2.625	51				
6282	30-Sep	F	62	13	2585	47	367	5.96	130	7.0	114	293	76	6.370	3.870	46				
6283	1-Oct	M	67	16	3277	29	133	6.86	141	3.6	120	313		5.655	3.790	40				
6284	1-Oct	F	60	15	2585	52	286	6.68	146	4.6	123	306	56	4.240	6.105	55				
6285	1-Oct	F	59	15	2501	49	264	5.78	132	7.0	113	291	285	4.505	5.030	46				
6286	1-Oct	M	66	15	3231	35	85.2	6.64	150	4.0	125	308		4.620	3.485	43				
6287	1-Oct	M	60	13	2344	33	59.7	5.18	153	3.0	116	298		6.350	4.885	50				
6288	1-Oct	M	60	15	2659	29	74.6	5.44	152	3.9	122	314		5.215	5.505	68				
6289	2-Oct	M	64	14	2543	28	98.6	6.07	135	2.8	123	307		4.350	2.655	42				
6290	2-Oct	M	61	16	2788	34	83.1	6.31	142	4.0	120	305		4.565	6.170	86				
6291	2-Oct	F	64	15	2641	48	415	5.84	139	3.9	121	301	406	5.020	1.960	45				
6292	2-Oct	M	62	16	3048	31	81.3	5.44	146	2.8	123	296		6.230	8.475	54				
6293	2-Oct	F	58	13	1890	36	254	6.57	139	2.0	123	302	315	3.925	5.590	60				
6294	2-Oct	M	69	17	3676	31	148	6.82	140	3.9	120	291		4.895	3.100	78				
6295	7-Oct	M	63	14	2405	24	76.3	5.44	138	2.1	124	296		4.210	1.655	91				
6296	7-Oct	F	59	14	2207	35	184	7.22	140	3.4	123	302	367	5.395	9.110	106				
6297	7-Oct	M	64	14	2590	37	41.6	7.22	136	4.8	125	300		5.130	3.570	63				
6298	7-Oct	M	62	14	2225	18	50.8	5.51	146	3.2	120	308		4.775	4.710	54				

Appendix 2 - Data for fish released with a conventional radio transmitter

FISH #	Date of capture	DNA results	Scale ID results	Ashcroft daily water temperature max (°C)	Ashcroft daily water temperature min (°C)	Ashcroft daily temperature mean (°C)	Travel time (hours) to:			Last Seen	Fate	Fate Zone
							Ashcroft	Little River	Adams			
6101	25-Aug	4 Lower_Adams	lo shu	18.82	18.23	18.55	52			27-Aug	casualty	Thompson - Below Ashcroft
6102	25-Aug	2 Fennell____	n thomr	18.82	18.23	18.55	57.75			27-Aug	casualty	Thompson - Below Ashcroft
6103	26-Aug	2 Fennell____	la shu	19.89	18.58	19.2	49.25			28-Aug	casualty	Thompson - Below Ashcroft
6104	27-Aug	2 Fennell____	n thomr	19.89	18.72	18.91	61			30-Aug	casualty	Thompson - Below Ashcroft
6105	27-Aug	2 Seymour___	.	19.89	18.72	18.91	55.25	187.5		4-Sep	spawn	Little River
6106	27-Aug	2 Seymour___	la shu	19.89	18.72	18.91	64			30-Aug	casualty	Thompson - Below Ashcroft
6107	27-Aug	2 Seymour___	la shu	19.89	18.72	18.91	73.25			30-Aug	casualty	Thompson - Below Ashcroft
6108	27-Aug	4 Lower_Adams	la shu	19.89	18.72	18.91	110.75	585		22-Sep	spawn	Little River
6109	27-Aug	4 Lower_Adams	n thomr	19.89	18.72	18.91	77.5	508.25		17-Sep	spawn	Little River
6110	27-Aug	4 Lower_Adams	la shu	19.89	18.72	18.91	102.75	610	1052.3	19-Oct	spawn	Adams River
6111	27-Aug	4 Lower_Adams	la shu	19.89	18.72	18.91	118.5	532.75	861	15-Oct	spawn	Adams River
6112	27-Aug	4 Lower_Adams	la shu	19.89	18.72	18.91	91	513.25	1046	23-Oct	spawn	Mobile Lake Shuswap Near Adams' Mouth
6113	27-Aug	4 Lower_Adams	la shu	19.89	18.72	18.91	95.25	664.5	1110.3	23-Oct	spawn	Mobile Lake Shuswap Near Adams' Mouth
6114	27-Aug	4 Lower_Adams	la shu	19.89	18.72	18.91				27-Aug	dropout	Never detected
6115	27-Aug	4 Lower_Adams	la shu	19.89	18.72	18.91	195.5	618.75		23-Sep	spawn	Little River
6116	27-Aug	4 Lower_Adams	la shu	19.89	18.72	18.91	52.5			5-Sep	casualty	Thompson - North Thompson
6117	28-Aug	2 Seymour___	.	19.89	17.56	18.25	53.25	173		4-Sep	spawn	Little River
6118	28-Aug	4 Lower_Adams	.	19.89	17.56	18.25	43	131		2-Sep	spawn	Little River
6119	28-Aug	4 Lower_Adams	la shu	19.89	17.56	18.25	77.5	452.25		17-Sep	spawn	Little River
6120	28-Aug	4 Lower_Adams	n thomr	19.89	17.56	18.25	47.75			2-Sep	casualty	Thompson - North Thompson
6121	28-Aug	2 Fennell____	.	19.89	17.56	18.25	70.5			31-Aug	casualty	Thompson - Below Ashcroft
6122	28-Aug	2 Seymour___	n thomr	19.89	17.56	18.25	77.25			8-Sep	spawn	Thompson - North Thompson
6123	28-Aug	2 Seymour___	la shu	19.89	17.56	18.25	46.75	180.75		4-Sep	spawn	Little River
6124	28-Aug	4 Lower_Adams	.	19.89	17.56	18.25	115.5			9-Sep	casualty	Thompson - North Thompson
6125	28-Aug	4 Lower_Adams	la shu	19.89	17.56	18.25	83.75	540.75		23-Sep	spawn	Little River
6126	28-Aug	4 Lower_Adams	.	19.89	17.56	18.25	73	508	763.5	7-Oct	spawn	Adams River
6127	28-Aug	2 Seymour___	la shu	19.89	17.56	18.25	63.25	268.5		8-Sep	spawn	Little River
6128	28-Aug	2 Seymour___	n thomr	19.89	17.56	18.25	50.5			8-Sep	spawn	Thompson - North Thompson

FISH #	Date of capture	DNA results	Scale ID results	Ashcroft daily water temperature max (°C)	Ashcroft daily water temperature min (°C)	Ashcroft daily temperature mean (°C)	Travel time (hours) to:			Last Seen	Fate	Fate Zone
							Ashcroft	Little River	Adams			
6101	25-Aug	4 Lower_Adams	lo shu	18.82	18.23	18.55	52			27-Aug	casualty	Thompson - Below Ashcroft
6129	28-Aug	2 Seymour___	la shu	19.89	17.56	18.25	43			31-Aug	casualty	Thompson - Below Ashcroft
6130	28-Aug	4 Lower_Adams	la shu	19.89	17.56	18.25	76			31-Aug	casualty	Thompson - Below Ashcroft
6131	28-Aug	4 Lower_Adams	.	19.89	17.56	18.25	107	489.25	1025.8	18-Oct	spawn	Adams River
6132	28-Aug	4 Lower_Adams	la shu	19.89	17.56	18.25	74	384	408.75	7-Nov	spawn	Mobile Adams above receiver site #11
6133	28-Aug	4 Lower_Adams	la shu	19.89	17.56	18.25	76.25	601.75		16-Oct	spawn	Little River
6134	28-Aug	4 Lower_Adams	la shu	19.89	17.56	18.25	81.5			31-Aug	casualty	Thompson - Below Ashcroft
6135	28-Aug	4 Lower_Adams	lo shu	19.89	17.56	18.25	139.5			3-Sep	casualty	Thompson - Below Ashcroft
6136	28-Aug	4 Lower_Adams	n thom	19.89	17.56	18.25	51			10-Sep	casualty	Thompson - North Thompson
6137	28-Aug	4 Lower_Adams	la shu	19.89	17.56	18.25	86.5	584		23-Sep	spawn	Little River
6138	28-Aug	2 Seymour___	n thomr	19.89	17.56	18.25	147.5	599.5		6-Oct	spawn	Little River
6139	28-Aug	4 Lower_Adams	n thomr	19.89	17.56	18.25	52.5			8-Sep	casualty	Thompson - North Thompson
6140	28-Aug	4 Lower_Adams	.	19.89	17.56	18.25	67.75			4-Sep	casualty	Thompson - North Thompson
6141	28-Aug	4 Lower_Adams	.	19.89	17.56	18.25	71.75	480.25		19-Sep	spawn	Little River
6142	28-Aug	2 Scotch____	.	19.89	17.56	18.25	75.5	443.5	1041	19-Oct	spawn	Adams River
6143	28-Aug		.	19.89	17.56	18.25	64.75	288.5		24-Oct	spawn	Little River
6144	28-Aug	4 Lower_Adams	la shu	19.89	17.56	18.25	119.5	633.75		23-Sep	spawn	Little River
6145	28-Aug	4 Lower_Adams	.	19.89	17.56	18.25	70.75	474.75	1014.3	23-Oct	spawn	Mobile Lake Shuswap Near Adams' Mouth
6146	29-Aug	4 Lower_Adams	la shu	19.89	17.68	18.46	112.75	580.5		22-Sep	spawn	Little River
6147	29-Aug	2 Seymour___	la shu	19.89	17.68	18.46				31-Aug	dropout	Fraser - Thompson Confluence
6148	29-Aug	4 Lower_Adams	lo shu	19.89	17.68	18.46	52.5			6-Sep	casualty	Thompson - Above Kamloops Lake
6151	5-Sep	4 Lower_Adams		19.29	18.56	18.9				7-Sep	dropout	Fraser - Thompson Confluence
6152	6-Sep	4 Lower_Adams		19.69	18.05	18.91				8-Sep	dropout	Fraser - Thompson Confluence
6153	6-Sep	4 Lower_Adams		19.69	18.05	18.91				8-Sep	dropout	Fraser - Thompson Confluence
6154	9-Sep	4 Lower_Adams		18.77	17.39	17.97				10-Sep	recovery	Freshwater Recovery Native
6155	11-Sep	2 Seymour___		18.58	17.67	17.99				11-Sep	dropout	Never detected
6156	11-Sep	4 Lower_Adams		18.58	17.67	17.99				11-Sep	dropout	Never detected
6157	11-Sep	4 Lower_Adams		18.58	17.67	17.99	112.25			16-Sep	casualty	Thompson - Below Ashcroft
6158	11-Sep	4 Lower_Adams		18.58	17.67	17.99				12-Sep	dropout	Fraser - Thompson Confluence
6159	16-Sep	4 Portage_Creek		16.7	15.64	16.3				16-Sep	dropout	Never detected
6160	17-Sep			16.37	14.97	15.63	105			21-Sep	casualty	Thompson - Below Ashcroft

FISH #	Date of capture	DNA results	Scale ID results	Ashcroft daily water temperature max (°C)	Ashcroft daily water temperature min (°C)	Ashcroft daily temperature mean (°C)	Travel time (hours) to: Ashcroft	Little River	Adams	Last Seen	Fate	Fate Zone
6101	25-Aug	4 Lower_Adams	lo shu	18.82	18.23	18.55	52			27-Aug	casualty	Thompson - Below Ashcroft
6161	17-Sep			16.37	14.97	15.63				17-Sep	dropout	Never detected
6162	17-Sep			16.37	14.97	15.63				17-Sep	dropout	Never detected
6163	17-Sep			16.37	14.97	15.63				22-Sep	dropout	Fraser - Thompson Confluence
6164	17-Sep			16.37	14.97	15.63				21-Sep	dropout	Fraser - Thompson Confluence
6165	17-Sep			16.37	14.97	15.63	72	216.75	530.25	3-Nov	spawn	Adams River
6166	17-Sep			16.37	14.97	15.63	75.25	237	284.25	7-Nov	spawn	Mobile Adams below receiver site #11
6167	17-Sep			16.37	14.97	15.63	42.25			23-Sep	casualty	Thompson - Above Kamloops Lake
6168	17-Sep			16.37	14.97	15.63	46			21-Sep	casualty	Thompson - North Thompson
6169	19-Sep			16.81	15.69	16.21	74.25			22-Sep	casualty	Thompson - Below Ashcroft
6170	19-Sep			16.81	15.69	16.21				22-Sep	dropout	Fraser - Thompson Confluence
6171	24-Sep									24-Sep	dropout	Never detected
6172	24-Sep						109	250.5		5-Oct	spawn	Little River
6173	30-Sep						86.25			4-Oct	casualty	Thompson - Below Ashcroft
6174	1-Oct						56.5	198	235.75	12-Oct	spawn	Adams River
6175	1-Oct						66.25			4-Oct	casualty	Thompson - Below Ashcroft
6176	1-Oct									6-Oct	dropout	Fraser - Thompson Confluence
6177	1-Oct									21-Oct	dropout	Canyon - Below Hells Gate
6178	1-Oct									1-Oct	dropout	Never detected
6179	1-Oct									4-Oct	dropout	Fraser - Thompson Confluence
6180	1-Oct						70.25			4-Oct	casualty	Thompson - Below Ashcroft
6181	1-Oct						76.5			5-Oct	casualty	Thompson - Below Ashcroft

Appendix 3 - Data for sockeye salmon captured for electromyogram radio transmitter telemetry

Fish #	Date	Capture time	Surgery start time	Surgery end time	Time of release	Transmitter frequency	Surgeon	Bleed during surgery?	Notes
6002	28-Aug	7:00	20:53		6:00	149.556	JLY		
6003	29-Aug	13:40	20:25		n/a	151.205	SJC	heavy	
6004	29-Aug	19:00	20:50		n/a	151.126	JLY		heavy bleeding
6005	29-Aug	19:50	n/a	n/a	n/a	n/a	n/a		
6006	30-Aug	19:30	22:42	22:52	7:00:06	151.265	JLY		
6007	30-Aug	19:57	21:35	21:57	n/a	151.126	JLY		
6008	30-Aug	20:28	n/a	n/a	n/a	n/a	n/a		
6009	30-Aug	20:43	22:07	22:29	n/a	151.104	JLY		
6010	30-Aug	21:05	n/a	n/a	n/a	n/a	n/a		
6011	2-Sep	18:45	n/a	n/a	n/a	n/a	n/a		
6012	3-Sep	19:45	n/a	n/a	n/a	n/a	n/a		
6013	2-Sep	19:04	20:51	21:05	12:45	151.066	JLY	heavy	
6014	2-Sep	19:25	10:31	10:46	n/a	149.625	JLY	no	heavy bleeding
6015	2-Sep	19:45	12:20	12:36	12:45	151.225	JLY	no	heavy bleeding
6016	2-Sep	20:30	21:21	21:37	n/a	149.206	JLY	no	heavy bleeding
6017	5-Sep	19:20	7:40	7:54		150.167	JLY		0 heavy bleeding
6018	6-Sep	6:30	n/a	n/a	n/a	n/a	n/a		
6019	11-Sep	6:40			n/a	149.625	SJC		0 heavy bleeding
6020	11-Sep	6:55	n/a	n/a	n/a	n/a	n/a		
6021	11-Sep	7:13	n/a	n/a	n/a	n/a	n/a		
6022	16-Sep	19:20	9:57	10:11	10:30	149.206	JLY		0.5 moderate bleeding
6023	17-Sep	7:12							
6024	21-Sep						JLY		0 light bleeding
6025	21-Sep	9:10	9:18	9:26	9:31	149.785	JLY		1 little bleeding
6026	24-Sep	19:17	9:26	9:35	9:38	149.605	JLY		0.5 moderate bleeding
6027	25-Sep	7:59	8:04	8:12	8:20		JLY		0 heavy bleeding
6028	25-Sep	9:10	9:11	9:17			JLY		0 heavy bleeding
6029	1-Oct	8:20	8:24	8:33	8:35	149.726	JLY		0.5 moderate bleeding