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California-Nevada Fish Health Center FY2010 Technical Report: Health, energy reserves and smolt development of juvenile Stanislaus River Chinook Salmon, 2010

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Summary

This study examined the role of fish health and condition contributing to mortality among juvenile Stanislaus River Chinook Salmon. Fish were collected from two existing out-migrant monitoring sites once each month during March, April and May, 2010. Overall health, condition and smolt development appeared good at the time of sampling. No obvious insult or tissue abnormalities due to environmental toxicity were detected. Parasitic and bacterial infections were noted in a few fish. While not a threat to fish health at the early stages observed during this study, infections with the kidney parasite *Tetracapsuloides bryosalmonae*, the causative agent of Proliferative Kidney Disease, will progress and could be a significant cause of mortality during migration and transition to sea water.

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Notice

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Objectives

This study examined the role of fish health and condition in contributing to mortality among juvenile Chinook salmon in the Stanislaus River, California. To determine if disease or fish condition was limiting survival in this system, fish were screened for pathogens, while condition (triglyceride level) and smolt development (gill Na^+ , K^+ -ATPase) were compared between fish captured in the upper and lower river. This work was performed as a pilot study under the USFWS National Wild Fish Health Survey with cooperative assistance by The Anadromous Fish Restoration Program, FISHBIO and Cramer Fish Scientists.

Methods

Juvenile fall-run Chinook salmon were collected at two rotary screw trap sites on the Stanislaus River. The upper trap (Oakdale, RM 40, operated by FISHBIO) and lower trap (Caswell, RM 6, operated by Cramer Fish Sciences) were sampled three times during the spring of 2010 to target the fry, parr and smolt life stages. Target sample size was 68 fish which were systematically assigned to a 30 fish general pathogen, 20 fish histopathology, and 18 fish energy reserve and smolt development assessment.

- General pathology assessment included: bacteriology by culture of individual kidney samples on BHIA media and identification by standard biochemical methods, virology by tissue culture of up to 5 pooled kidney and spleen tissues on EPC and CHSE cell lines, and direct florescent antibody test of individual kidney tissues for *Renibacterium salmoninarum* using polyclonal antibody (USFWS and AFS-FHS 2007).
- Histopathology assessment inspected for tissue abnormalities causes by parasites or environmental conditions was performed on whole sagittal sections of smaller fish or multiple individual tissues of larger fish.
- Fish condition was assessed by measurement of triglyceride stores in whole fish homogenates (Weber et al. 2003).
- Smolt development was assessed by measurement of Na^+ , K^+ -ATPase activity in gill tissue (McCormick and Bern 1989).

Results

A total of 259 juvenile Chinook salmon were collected from the Oakdale (n=163) and Caswell (n=96) traps (Table 1).

No obligate bacterial or viral fish pathogens were detected in the 109 fish sample. Several bacterial and parasite infections were detected by histopathological examination (Table 2). Early infections of *Tetracapsuloides bryosalmonae*, the causative agent of Proliferative Kidney Disease, were detected in 22-33% of smolts collected in May. Intestinal trematodes and gill parasites (including *Ichthyophthirius multifiliis*, amoebas and *Trichodina*) were detected in fish from both the Oakdale and Caswell trap sites. A bacterial infection with associated gill hyperplasia (presumptive *Flavobacterium columnare*) was observed in a single fish captured in March.

Table 1. Number of juvenile Chinook salmon collected on the Stanislaus River. Fish were systematically assigned to pathogen screening, histopathology or condition assessments.

Sample Date	Site	Pathogen	Histopathology	Condition	Total
March 10	Oakdale	30	20	18	68
	Caswell	3	3	3	9
April 1	Oakdale	30	18	20	68
	Caswell	30	18	20	68
May 12	Oakdale	9	9	9	27
	Caswell	7	6	6	19
Total		109	74	76	259

The most serious tissue abnormality observed in the histopathological examination was the single fish with gill hyperplasia associated with the *F. columnare* bacterial infection. Most of the various pathologies observed in the intestine, visceral fat, acinar cells or liver were not associated with any obvious causes and were not likely to be affecting fish performance at the time of sampling (Table 2). The *T. bryosalmonae* infections were in an early stage and no kidney pathology was associated with the infections.

No differences in energy reserves (Figure 1) or smolt development (Figure 2) were detected between sites. Energy stores (mg triglyceride/g fish) ranged from 0.13 to 1.44 with a median value of 0.65 over the whole study. Significant energy reserves were also noted in the histopathological examination, with high amounts of hepatic glycogen storage noted in 5/46 fish over the whole study. Smolt development was measured during the May (smolt stage) sample, and ATPase activity ($\mu\text{mol ADP/mg protein/hr}$) over both sites ranged from 5.2 to 13.8 with a median of 8.5.

Table 2. Abnormalities observed in histopathological examination of juvenile Chinook salmon collected on the Stanislaus River. The number of abnormalities over total samples (%) examined by histopathology is given for each tissue. Footnotes (letters) describe type and severity of abnormalities.

	Kidney	Intestine	Fat	Acinar	Liver	Gill
March 10						
Oakdale	0/12	0/12	0/12	0/13	2/14 (14) a	0/18
Caswell	0/3	0/3	0/3	1/3 (33) b	1/3 (33) a	1/3 (33) c
April 1						
Oakdale	0/20	9/20 (45) d	5/18 (28) e	0/18	1/20 (5) a 5/20 (25) f	3/20 (15) g
Caswell	0/6	0/6	0/6	0/6	1/6 (17) f	1/6 (17) g
May 12						
Oakdale	2/9 (22) h	2/9 (22) d	6/9 (67) e	0/9	1/9 (11) a	0/9
Caswell	2/6 (33) h	1/6 (17) d	0/5	0/6	1/6 (17) f	0/6
		1/6 (17) i				

- a) High amount of hepatic glycogen vacuoles. Suggests high energy reserves in this fish.
- b) Inclusions observed in acinar cells between pyloric ceca. Cause was not apparent.
- c) Bacterial infection (likely *Flavobacterium columnare*) observed with associated hyperplasia. This is a common bacterial infection and can cause significant mortality in salmonids at warm water temperatures.
- d) Trematode observed in intestine. These parasites are common and do not cause problems for healthy fish.
- e) Inflammation observed in the visceral fat. No cause was apparent.
- f) Multifocal inflammation involving macrophages was observed in the liver. No cause was apparent
- g) External parasites (including *Ichthyophthirius multifiliis*, amoebas and *Trichodina*) were observed on the gill. These are common infections in freshwater salmonids and can cause mortality at warm water temperatures. No significant inflammation or tissue lesions were associated with these infections.
- h) Early infections with the parasite *Tetracapsuloides bryosalmonae* (causative agent of Proliferative Kidney Disease) were observed. This infection was likely not causing problems at this early stage of infection.
- i) A granuloma was observed in the viscera. No parasite or other cause was apparent.

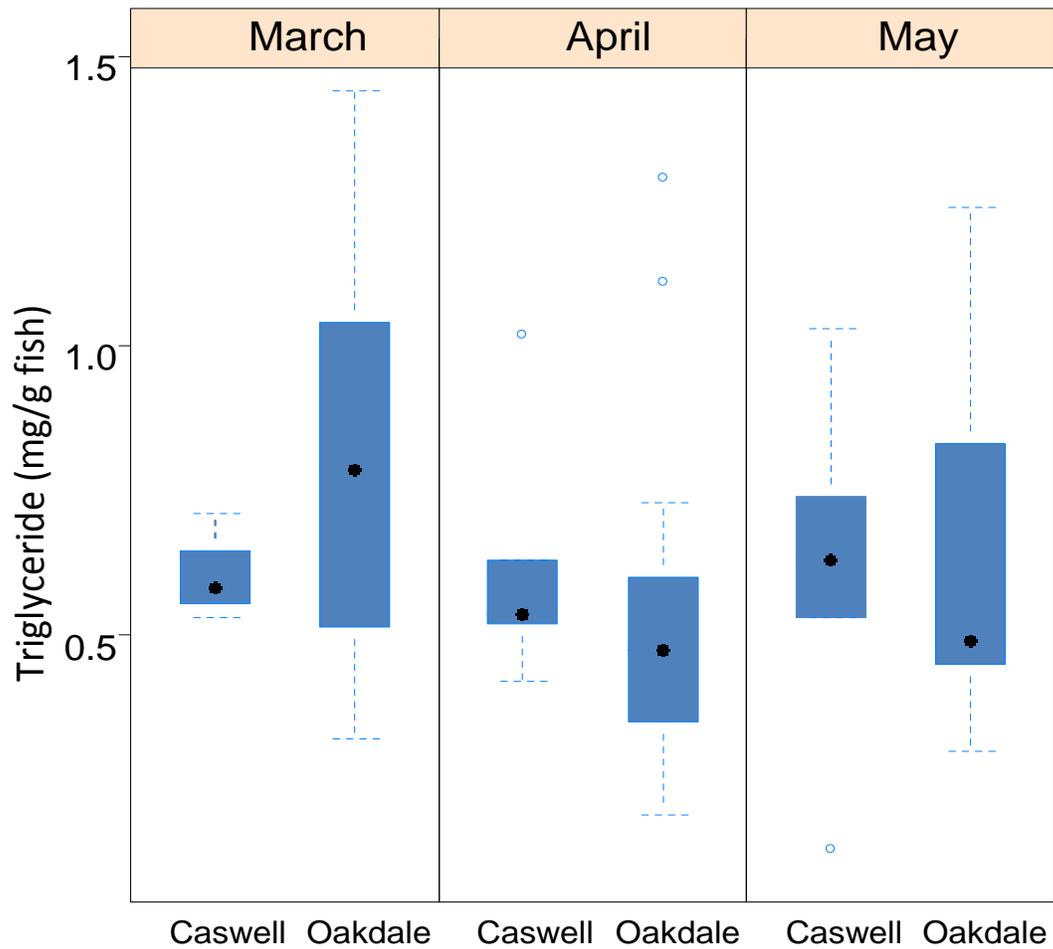


Figure 1. Boxplot (median, 25-75%, range and outliers) of fish condition measured by whole body triglyceride levels (mg/g of fish) in juvenile Chinook salmon captured on the Stanislaus River. No significant differences were detected between sites in any of the 3 months ($P > 0.05$, Wilcoxon rank-sum test)

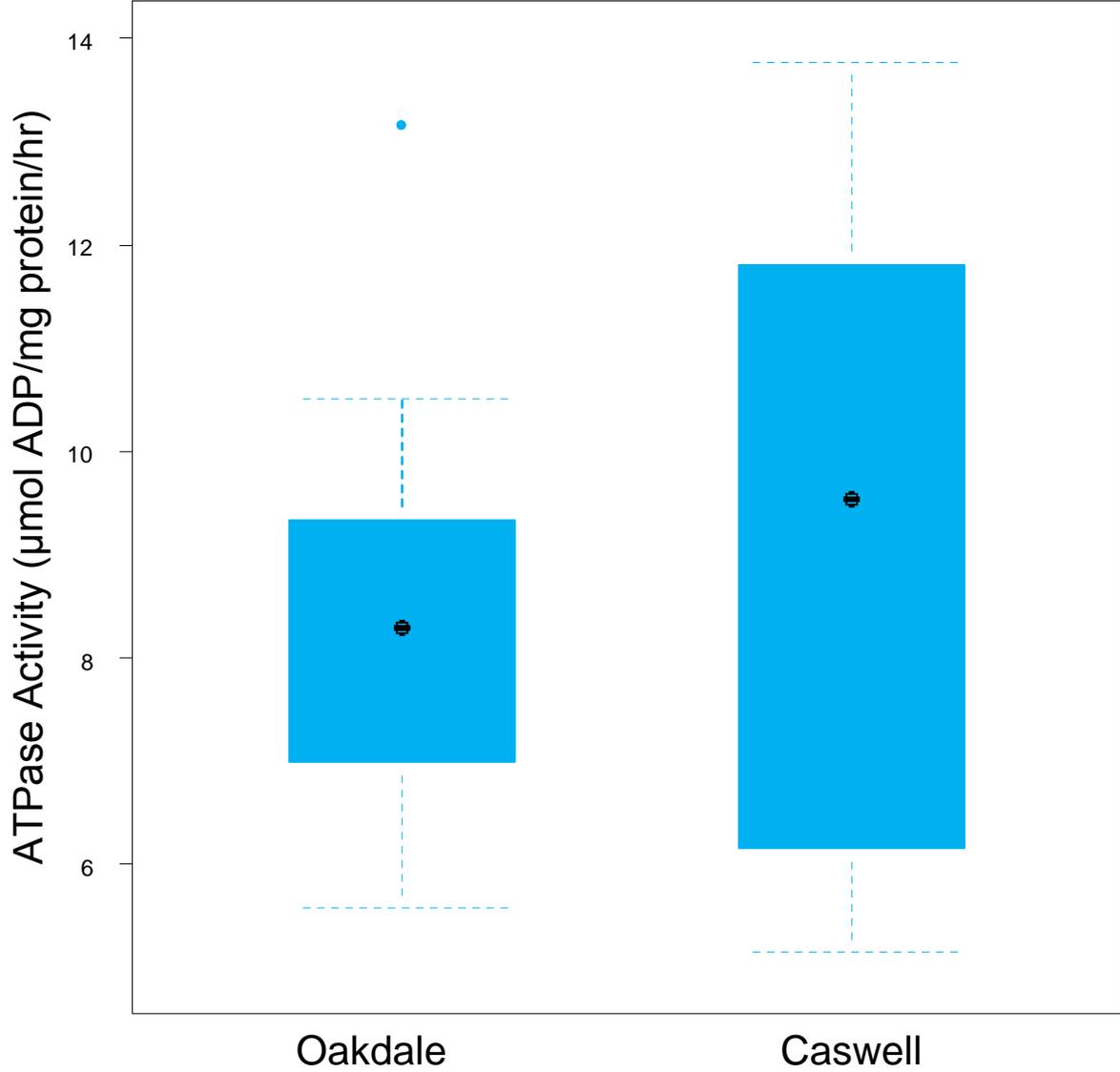


Figure 2. Boxplot (median, 25-75%, range and outliers) of smolt development measured by Gill Na^+ , K^+ -ATPase activity levels in Stanislaus River juvenile Chinook salmon during May 2010. No significant differences were detected between sites ($P > 0.05$, Wilcoxon rank-sum test).

Discussion

No pervasive health issues were identified in the sampled population. Infections with the kidney parasite, *Tetracapsuloides bryosalmonae*, were at early stages when observed in fish captured during May, but this progressive disease is known to cause mortality in Chinook smolts from the nearby Merced River (Foott, Stone and Nichols 2007). The parasites and bacterial pathogen

(*Flavobacterium columnare*) observed were ubiquitous in the aquatic environment and not unexpected at low levels; however, the one *F. columnare* infection observed was likely lethal, and incidence would increase at warmer water temperatures. Inflammation in the liver and visceral fat of fish sampled in April and May was likely not affecting performance or survival, but investigation into the causes if these tissue abnormalities may merit further study. Chinook at both samples sites had adequate energy reserves suggesting food was not a limiting factor for fish survival. Smolt development was not significantly different between sites and also did not suggest any problem. Based on the health and physiological factors evaluated in this study, juvenile Stanislaus River Chinook in 2010 appeared healthy at the time of sampling, but *T. bryosalmonae* infections will progress, and these fish could be experiencing Proliferative Kidney Disease during migration and transition to sea water.

Acknowledgements

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