IMPACTS OF STORMWATER RUNOFF ON COHO SALMON IN RESTORED URBAN STREAMS

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INTRODUCTION

Beginning in the late 1990's, agencies in the greater Seattle area began conducting fall spawner surveys to evaluate the effectiveness of local stream restoration efforts. These surveys detected a surprisingly high rate of mortality among migratory coho females that were still ocean bright and had not yet spawned. In addition, adult coho from several different streams showed similar symptoms (disorientation, lethargy, loss of equilibrium, gaping, fin splaying) that eventually led to death. In recent years, pre-spawn mortality (PSM) has been observed in many lowland urban streams, with overall rates ranging from ~20-90% of the fall runs. Although the precise cause of PSM in urban streams is unknown, conventional water quality parameters (i.e., temperature and dissolved oxygen) and disease do not appear to be causal. Rather, the weight of evidence suggests that adult coho, which enter small urban streams following fall storm events, are acutely sensitive to non-point source stormwater runoff containing pollutants that typically originate from urban and residential land use activities.

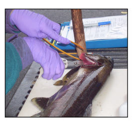
Coho salmon pre-spawn mortality in Des Moines Creek, WA. This female had 75-100% egg retention.

OBJECTIVE

Our ongoing objective is to discover the cause(s) and geographical extent of acute coho salmon die-offs in Puget Sound streams.

METHODS

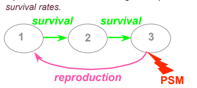
Daily stream surveys were conducted during fall 2002-05 to determine presence or absence of symptomatic fish (gaping, loss of equilibrium, fins splayed, spasming) and pre-spawn mortalities. For each affected fish, we recorded the location, species, gender, fork length, weight (with and without gonads), presence or absence of adipose fin, spawning condition, and percent egg retention in females. We collected tissue (brain, blood, gills, bile from live symptomatic fish, liver, pyloric caeca, intestine, heart, kidney) from symptomatic males and females and freshly dead females. Samples were screened for evidence of disease, vitamin deficiency, pesticide exposure, and hydrocarbon exposure.



Models of established coho populations were constructed to simulate constant and fluctuating levels of PSM that may reflect changes in land use, runoff management or rain events. Supplementation models estimated how many hatchery juveniles may be needed after completing habitat restoration projects to counter ongoing levels of pre-spawn mortality and maintain a designated number of returning adults. All models were constructed with literature data from Puget Sound populations of coho salmon.

Models were based upon a 3 year life history graph (Fig 1), with reproduction occurring at age 3. Models were run for 200 years to assess changes in adult abundance and time to extinction. Initial conditions simulated a stream supporting a population of 300 adults with a stable age distribution. Control conditions exhibited an intrinsic population growth rate of 1.03. The primary PSM scenarios included: a constant level of PSM each year; variable PSM annually selected from a range with a uniform distribution; and gradually increasing levels of prespawn mortality over time. Supplementation models started with introduction of hatchery smolt over 3 years sufficient to establish a naturally spawning population with 300 returning adults. The PSM models assessed the number of hatchery smolt needed to compensate for constant PSM losses and maintain 300 adults.

Figure 1. Life history graph and age structured transition matrix for coho salmon. Pink represents reproductive contribution and green represents survival rates.



Projection Matrix

$$A = \begin{bmatrix} 0 & 0 & 3\rightarrow1 \\ 1\rightarrow2 & 0 & 0 \\ 0 & 2\rightarrow3 & 0 \end{bmatrix}$$

Abundance Vector

$$\vec{n}(t) = \begin{bmatrix} n_1 \\ n_2 \\ n_3 \end{bmatrix}$$

Projection Equation

$$\vec{n}(t+1) = A\,\vec{n}(t)$$

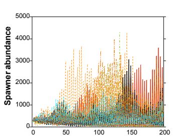
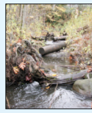
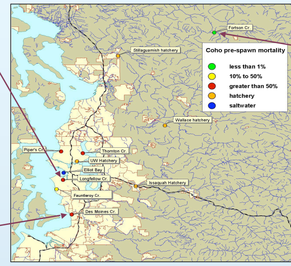
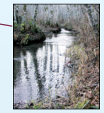


Figure 2. Examples of model runs with variable vital rates over 200 years under control conditions.







Potential Causes for Pre-Spawn Mortality

- High temperature/Low dissolved oxygen
- Hydrocarbon exposure
- Pathology and pathogens
- Vitamin deficiency
- Stress and poor condition
- Pesticide exposure
- Other/combination of factors?

RESULTS

PSM in Longfellow Creek

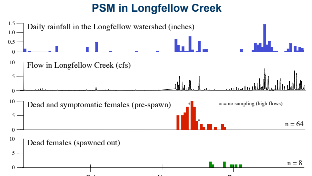


Figure 3. Rate of pre-spawn mortality among adult female coho salmon returning to spawn in Longfellow Creek, West Seattle, in the fall of 2002. After a protracted dry period in the early fall, adult coho salmon began returning with the first major rains in early November. In the 2002 sampling season, all of the females returning to the stream in the first few days died before spawning. Successful spawners were only observed after several significant rain events. The overall rate of female PSM for 2002 was ~90% across the entire fall run. Peaks in flow that did not correspond with storm events were related to tidal influence.

Pre-spawn (top) vs. post-spawn mortality female coho

Stream temperatures in Longfellow Creek during fall 2003 averaged 9°C (range: 3-17°C) and dissolved oxygen averaged 8.1 mg/L (range: 5.4-12.7 mg/L). These values fell within the ranges of tolerable temperature and dissolved oxygen levels for salmonids.

Bile Analysis for Hydrocarbon Exposure

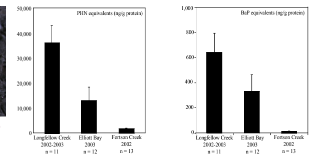


Figure 4. Results of coho salmon bile analysis for hydrocarbon exposure at three sites: Longfellow Creek (2002-03), Elliot Bay (2003), and Fortson Creek (2002). Phenanthrene equivalents (PHN) show exposure to petroleum-like hydrocarbons and benzo(a)pyrene equivalents (BaP) indicate exposure to pyrogenic hydrocarbons (fossil-fuel burning, etc).

High Temperature/ Low Dissolved O_2

Hydrocarbon exposure
Preliminary bile analyses for phenanthrene equivalents and benzo(a)pyrene equivalents indicate higher hydrocarbon exposure in Longfellow Creek than in Elliot Bay; and higher exposure in Elliot Bay than in Fortson Creek (Fig 4).

Pesticide Exposure
We tested acetylcholinesterase activity in coho brain and muscle and found no significant reduction in AChE activity between reference and urban streams, indicating low exposure to AChE-inhibiting pesticides.

?
Other stressors/ combination of factors

Pathology and Pathogens
We assessed for prevalences of tissue lesions of both noninfectious and infectious etiology by histopathology, and for pathogen infection prevalences with species-specific PCR. Certain lesions and pathogens were detected (Tables 1 and 2); however, the impacts of these lesions and pathogens on the metabolic or physiological condition of spawning fish are currently unknown.

Poor "Condition"
We compared fish condition (Fulton's Condition Factor) among sample streams and hatcheries (Fig 5). No consistently significant differences were seen among sites and years or between pre- and post-spawn mortalities.

Vitamin Deficiency
We examined thiamine levels in the eggs, liver, and muscle of female coho salmon, but found no significant difference between symptomatic fish in Longfellow Creek and non-symptomatic fish in Fortson Creek or Issaquah hatchery.

Condition Factor

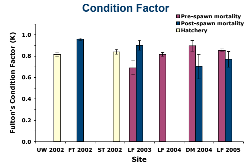


*Figure 5. Fulton's condition factor (K=(weight/length³)*10)) for female pre- and post-spawn mortalities in Longfellow Ck (LF), Des Moines Ck (DM), Fortson Ck (FT), and University of Washington (UW) and Stillaguamish (ST) hatcheries. Ovaries were removed from all fish prior to weighing. Error bars represent standard error.*

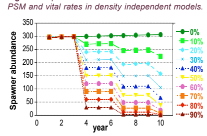
Table 1. Preliminary histopathology results for lesions/pathogens potentially associated with coho pre-spawn mortality, 2003-04.

Organ	Longfellow	Des Moines	Elliott Bay	Issaquah Hatchery	Wallace R. Hatchery
Gill	N=21	N=22	N=27	N=24	N=19
Loma spp.	24%	9%	7%	13%	5%
Heart	N=21	N=22	N=26	N=24	N=20
Loma spp.	19%	5%	0%	13%	10%
Any Organ	N=21	N=22	N=26	N=24	N=20
Loma spp.	29% (but 0% in 2004)	9%	9%	17%	10%
Trunk kidney	N=21	N=21	N=27	N=23	N=20
Nanophyetus (digenetic trematode, metacercaria)	10%	19%	19%	0%	0%
Tubular protein casts, nephrosis	5%	52%	0%	4%	0%
Liver	N=20	N=22	N=27	N=21	N=18
Hydropic degen., coagulative necrosis hepatocytes	25%	36%	0%	19%	17%
Hepatocellular megalocytosis, nuclear pleomorphism (assoc. w/ PAH exposure in other fish species)	15%	9%	0%	0%	6%

Table 2. Prevalences of fish infected with indicated pathogen and number of fish examined.

		C. shasta	P. minibicornis		L. salmonae	T. bryosalmonae	R. salmoninarum	N. salmincola
	N	posterior GI	kidney	gill	gill	kidney	kidney	kidney
East Waterway, Duwamish	27	18.5	100.0	7.4	14.8	7.4	22.2	48.1
Longfellow Creek	14	0.0	100.0	50.0	85.7	14.3	35.7	35.7
Issaquah Hatchery	24	4.2	87.5	79.2	4.2	0.0	33.3	0.0
Wallace River Hatchery	20	0.0	95.0	100.0	5.0	40.0	30.0	0.0

Figure 6. Spawner abundance under constant levels of PSM and vital rates in density independent models.



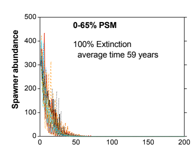


Figure 7. Examples of model runs with variable vital rates over 200 years under variable PSM conditions.

Abundance Models

Constant levels of PSM above 60% caused a very rapid population decline (Figs 6 - 8, Table 3), when vital rates varied within their normal distributions. Gradually increasing the levels of PSM allowed the populations to persist longer than in the constant impact scenarios. The rapid increase to 80% prespawn mortality resulted in extinction at 27 years and the slow increase to 35% prespawn mortality model was extinct in 64 years (Table 3). Varying PSM within a range between 0 and 20% resulted in 75% of the 500 runs conducted ending in local extinction, on average at 154 years (Table 3). Allowing PSM to range from 0 to 90% resulted in 100% local extinction at an average time of 32 years (Table 3).

The restoration models provided an estimate of the number of year old juveniles that would need to be released to provide 300 spawners, after the effects of PSM have been incorporated. An area estimated to have 30% prespawn mortality would need approximately 30% of the smolt needed to establish the population planted each year to maintain the target spawning population abundance (Fig 9).

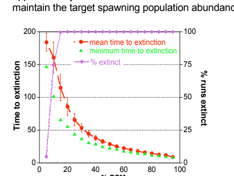


Figure 8. Figure depicts the percent of 500 runs extinct within the 200 year projection as well as the mean and minimum times to extinction for constant levels of PSM and variable vital rates.

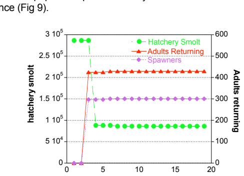


Figure 9. Figure depicts the number of hatchery smolt needed to compensate for 30% PSM to maintain 300 spawners.

Table 3. Percent of runs extinct, and mean and minimum time to extinction under constant PSM, annually varying PSM and gradually increasing PSM.

% PSM	% of Runs Extinct within 200 y	Time to Extinction (Std Dev)	Minimum Time to Extinction
Constant			
0	0	NA	NA
5	7	176 (20.5)	130
10	79	155 (23.7)	99
15	99.8	116 (21.6)	73
20	100	84 (14.7)	47
25	100	66 (10.0)	40
30	100	53 (6.7)	40
35	100	44 (5.4)	31
40	100	38 (3.9)	29
45	100	33 (3.2)	26
50	100	29 (2.7)	23
55	100	25 (2.3)	20
60	100	23 (1.8)	18
65	100	20 (1.6)	17
70	100	18 (1.3)	15
75	100	16 (1.3)	14
80	100	15 (0.6)	12
85	100	13 (1.2)	12
90	100	12 (0.3)	10
95	100	9 (0.5)	9
Uniform distribution			
0-20	79	154(26.8)	78
20-50	100	44 (5.8)	30
0-90	100	28 (5.2)	15
70-90	100	14 (1.4)	12
Gradual Increase			
80% over 20 y	100	27 (1.2)	24
35% over 35 y	100	64 (6.5)	46

OTHER LIFE STAGES?

To investigate the effects of urban stormwater runoff on coho embryos, we constructed a constant-flow bypass system allowing the incubation of embryos in untreated stream water and water filtered using sand, activated charcoal, and UV light. Embryos exposed to untreated stream water had consistently higher rates of developmental defects and mortality when compared to embryos raised in filtered stream water.

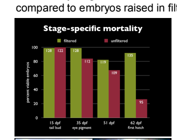
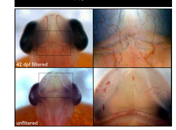


Figure 10. Whitlock-Vibert boxes containing about 150 embryos each were subsampled weekly from each shed. Data represents the percentage of viable embryos from each time-point (N at top of each bar). No differences were observed during early developmental stages, but mortality dramatically increased in the unfiltered water during the eyed stages after 35 days post-fertilization (35 dpf). There were no differences in temperature or dissolved oxygen between filtered and unfiltered water.



A high percentage of embryos with vascular defects in the brain were observed in the unfiltered water at the point where mortality ensued. Surviving alevins at later stages were significantly smaller or developmentally delayed.

Experimental tanks for the embryo studies were housed in a shed adjacent to Longfellow Creek.

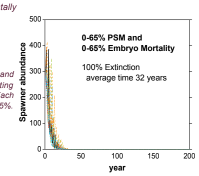


Figure 11. Spawner abundance and extinction time for models incorporating both PSM and embryo mortality. Each impact varied annually between 0 and 65%.

CONCLUSIONS

- Approximately **75-89%** of the female coho returning to Longfellow Creek in 2002-04 and **63%** of the coho returning to Des Moines Creek in 2004 died prior to spawning. The rate of pre-spawn mortality at the reference site (Fortson Creek) in 2002 was **less than 1%.**
- Affected fish showed a consistent suite of symptoms, including loss of orientation, gaping, lethargy, and loss of equilibrium.
- Embryos exposed to unfiltered stream water had higher rates of developmental defects and mortality than embryos exposed to filtered stream water.
- Water quality parameters (i.e. temperature, dissolved oxygen, etc.) were within a reasonable range for salmonid survival.
- Although prevalences of certain pathogens and tissue lesions were higher at urban sites displaying high rates of PSM, these conditions were not consistently found in dead or symptomatic pre-spawn coho, and the typically low severity of these lesions or infections strongly suggests that they are unlikely to be causally related to the rapid death of coho in urban creeks.
- Basic coho models incorporating PSM demonstrate the potential for rapid loss of localized populations in urban streams from nonpoint and stormwater sources.
- **At this time, we do not know the precise cause(s) of coho pre-spawn mortality.**

ONGOING INVESTIGATIONS

Studies are underway to address the following questions:

- What are the cumulative effects of contaminated stormwater runoff on salmonids that spawn and rear in urban streams?
- What land use attributes (i.e. % impervious surface, traffic volume, stormwater outfalls, etc., as analyzed by GIS) correlate most closely with inter-annual observations of pre-spawn mortality?
- What mixtures of contaminants are in stormwater, and at what concentrations, during observed mortality events?
- What are the precise causes of death in adult migrants and the observed developmental defects in embryos (forensic analyses)?
- What are the relative vulnerabilities of Pacific salmonids and steelhead, as well as cutthroat and bull trout, to urban runoff?
- What are the relative rates of salmonid die-offs along an urbanization gradient, from forested watersheds to highly developed watersheds such as Longfellow Creek?

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