

Connecting managed river conditions with salmon disease/pathogens in the Central Valley

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Funding:

CDFW: Q1996036

MWDSC: 193912

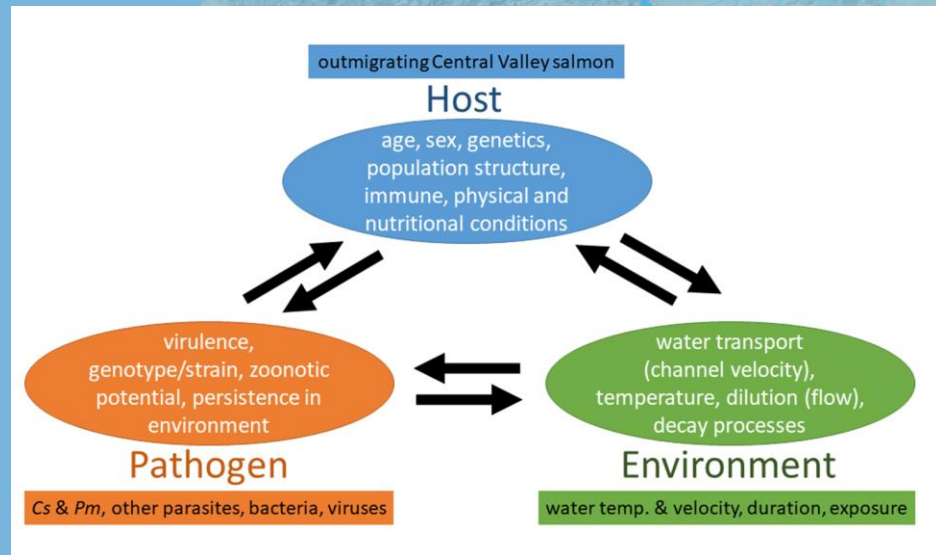
CA DWR: 4600014526



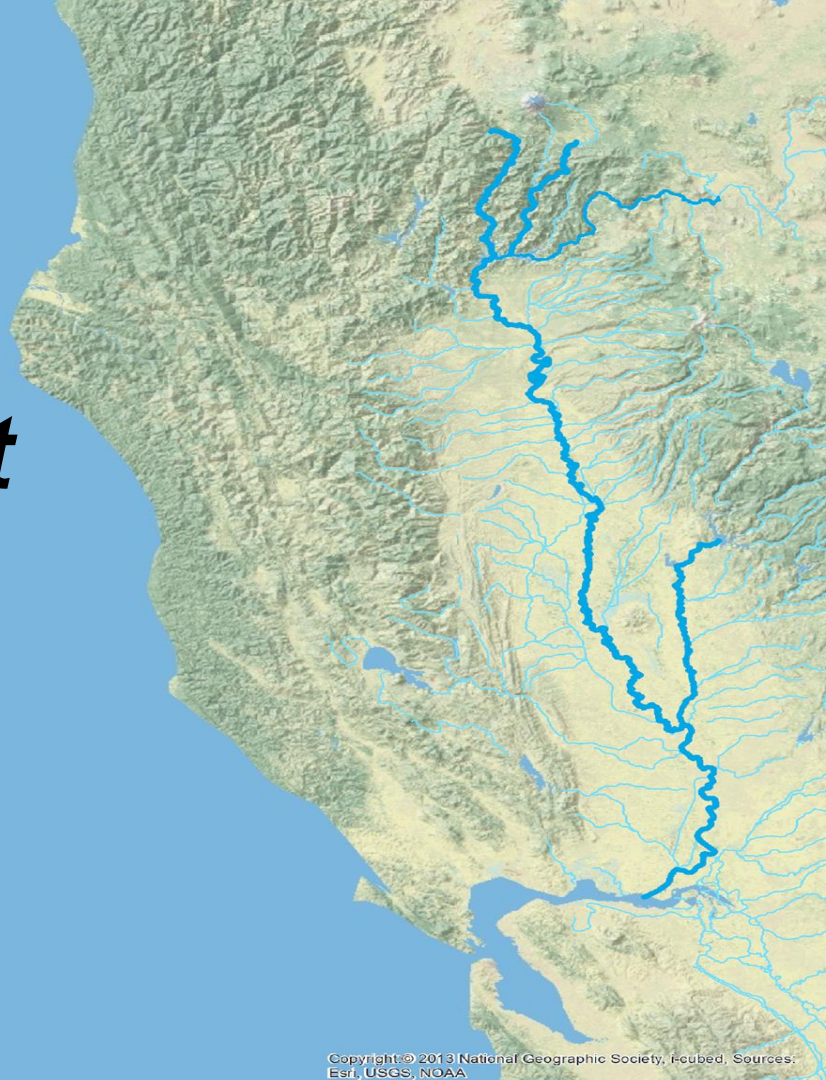
Big Picture

Our goal is to fill gaps of how exposure to pathogens and interacting stressors affect outmigration survival of Chinook salmon

We are doing this by monitoring salmon pathogens and developing a disease transmission model

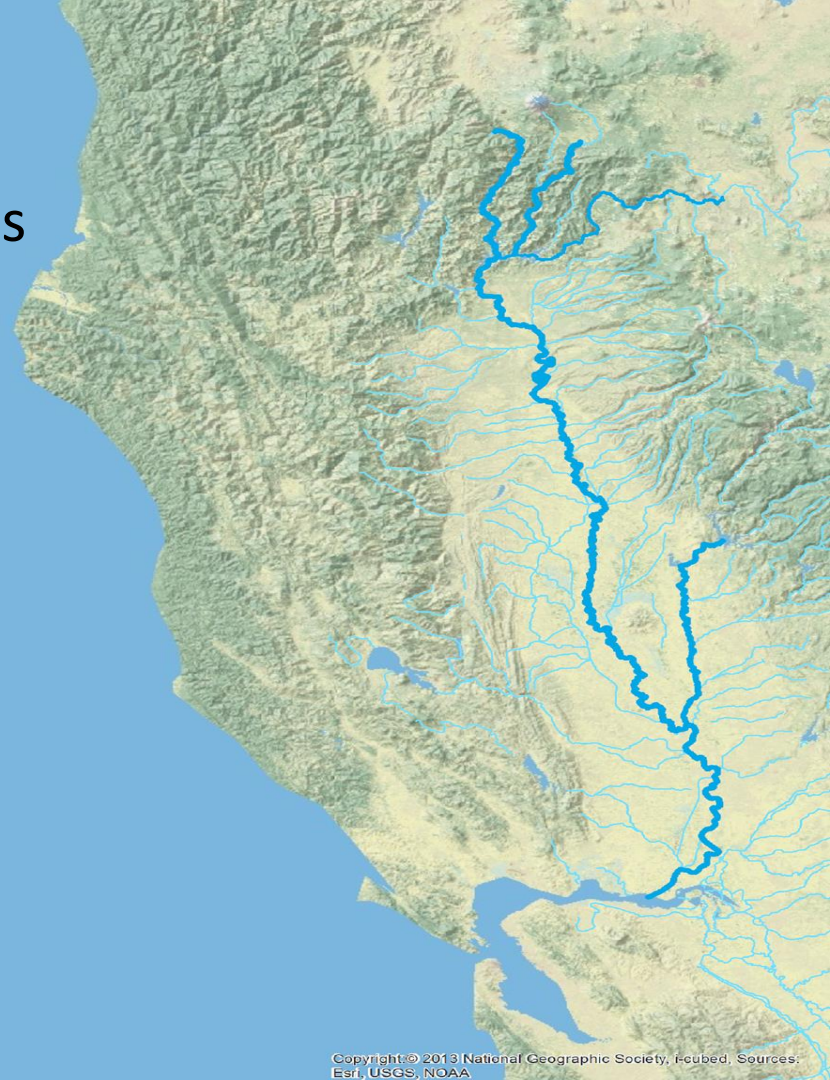


Summary of project objective



Main Objectives

- 1) Screen environment for pathogens
- 1) Screen fish for pathogens
- 1) Build a model to simulate pathogen exposure and health outcome
- 1) Use the above to better understand the connection to management



● **Screening Environment** (water)

- Samples collected bi-weekly
- Screen for:
 - *C. shasta* *Known pathogen of concern
 - eDNA panel 36 pathogens



● **Screening Environment** *(sentinel fish)*

- Hatchery fall-run exposed to ambient river conditions
- Tissue sampled
 - gill, kidney, intestine
- Screened for same pathogens as water



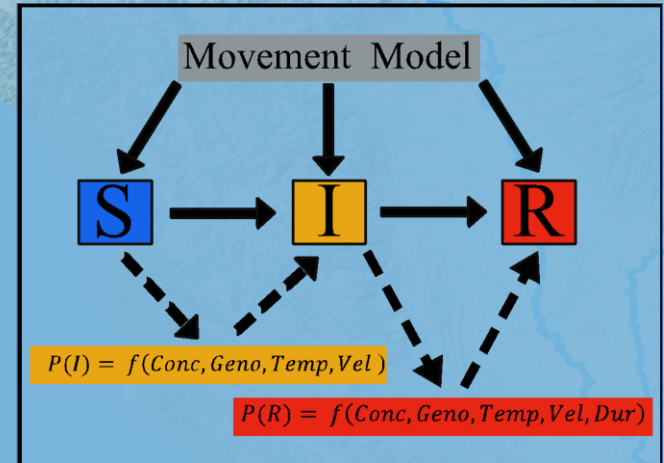
● **Screening Wild Salmon** *(rotary-screw trap samples)*

- 80 outmigrating winter run sampled
- Gill tissue processed for pathogens
- Additional tissues processed via histology



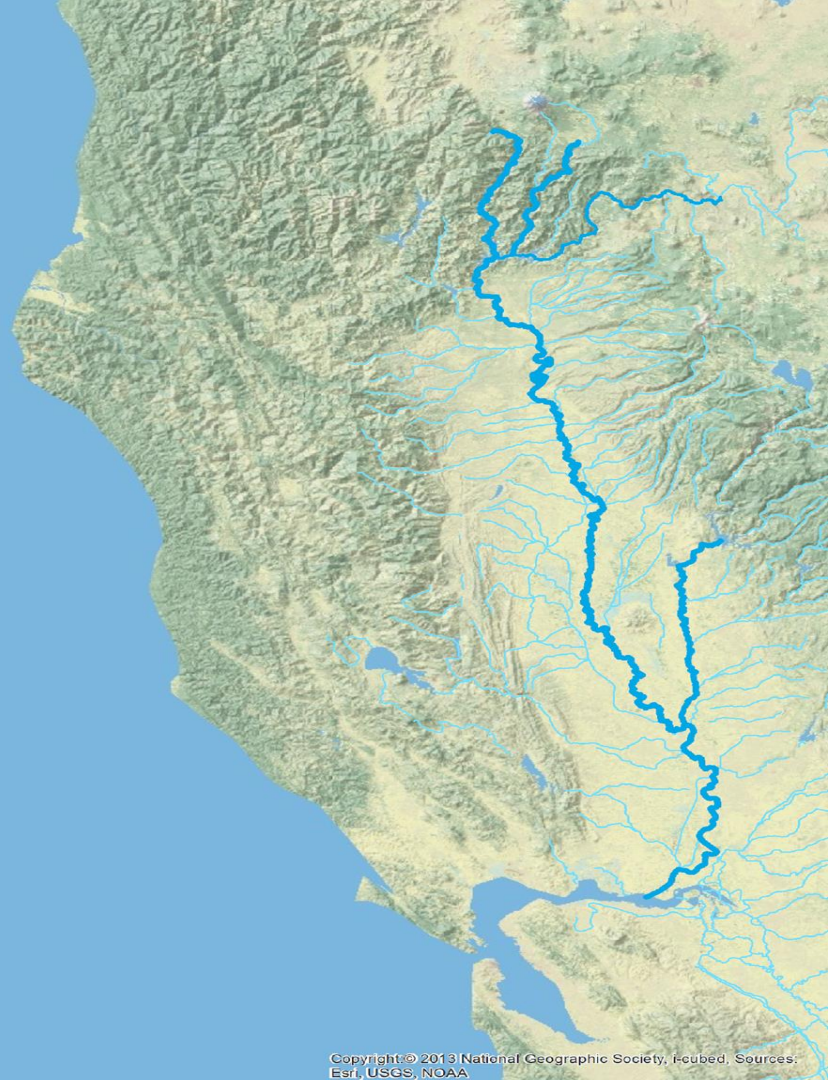
Disease Transmission Model

Link a movement and exposure model to simulate prevalence and probability of mortality associated with *C. shasta* exposure

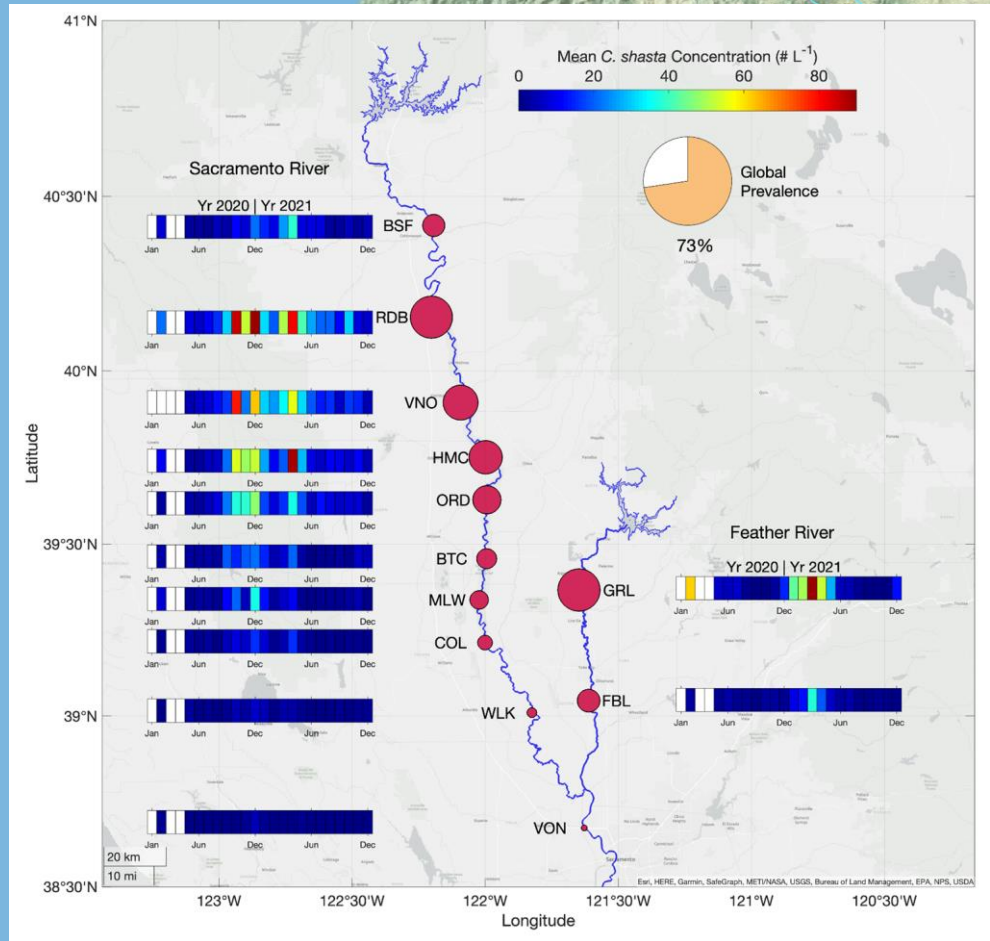


*Note methods can be applied to other pathogens of concern.

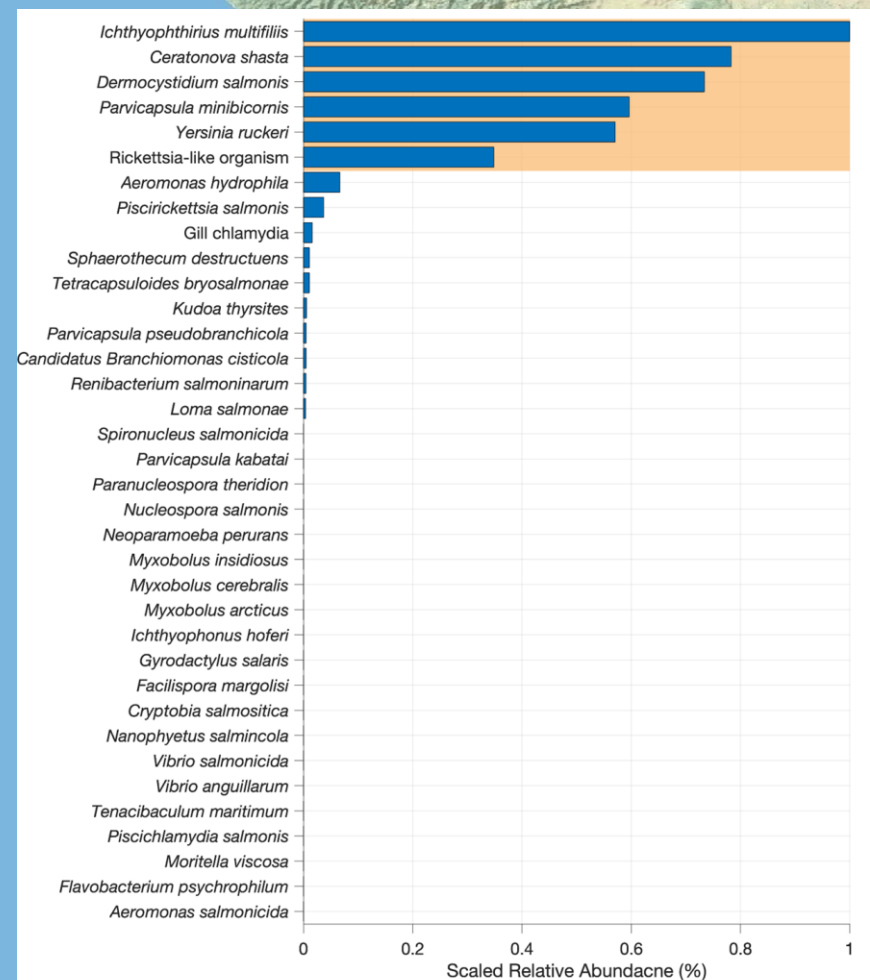
Results



Screening Environment (*C. shasta* water)



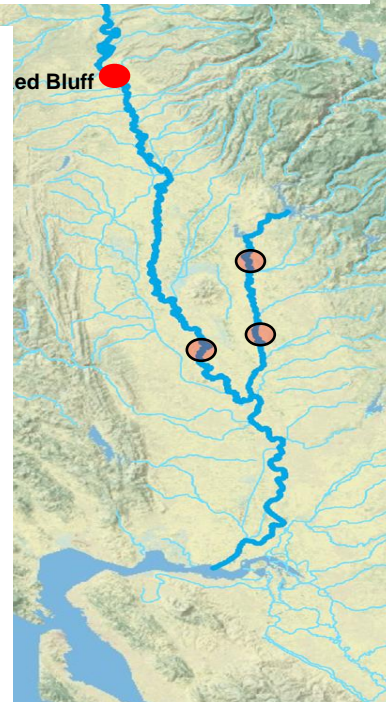
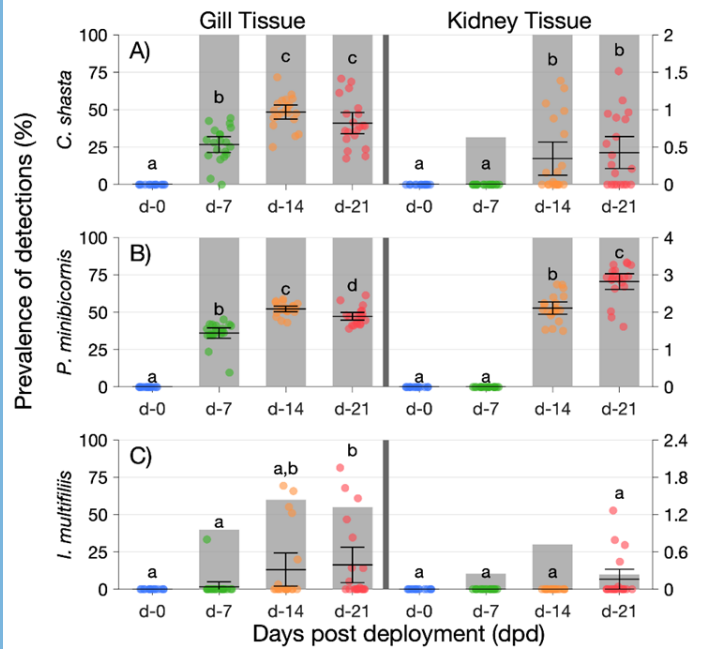
Screening Environment (additional pathogens)



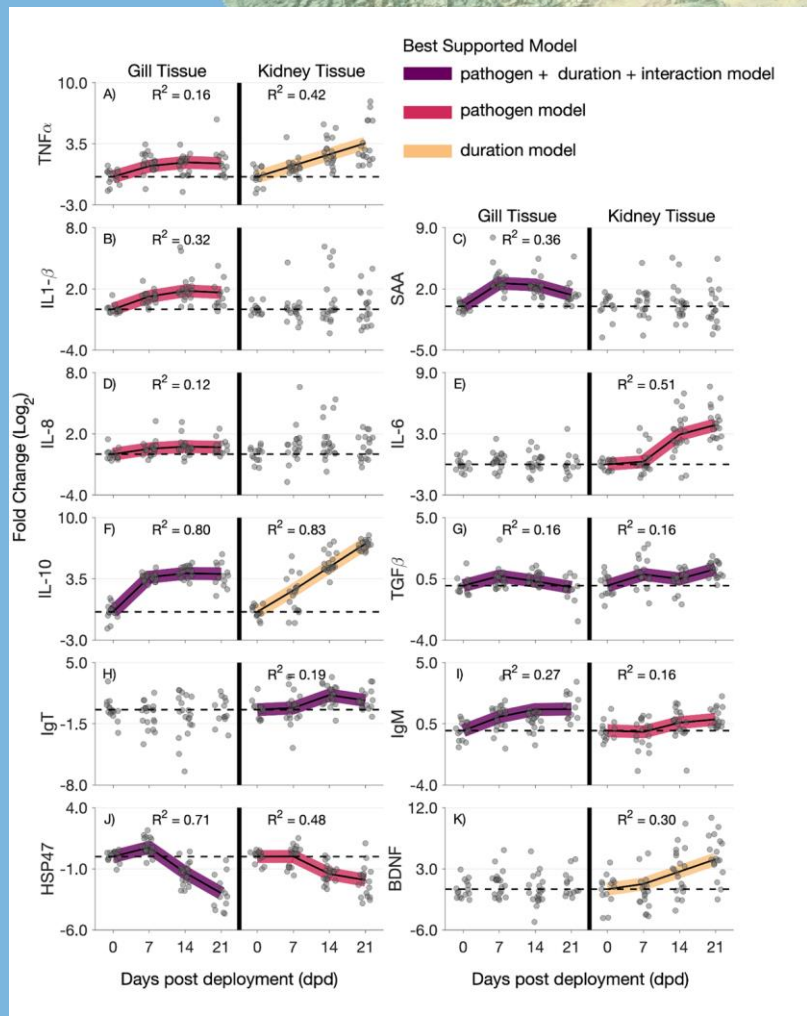
*Note: relative abundance does not account for differences between single and multi-cellular organisms (i.e., bacteria vs protozoa) and molecular amplification

Screening Environment (sentinel fish)

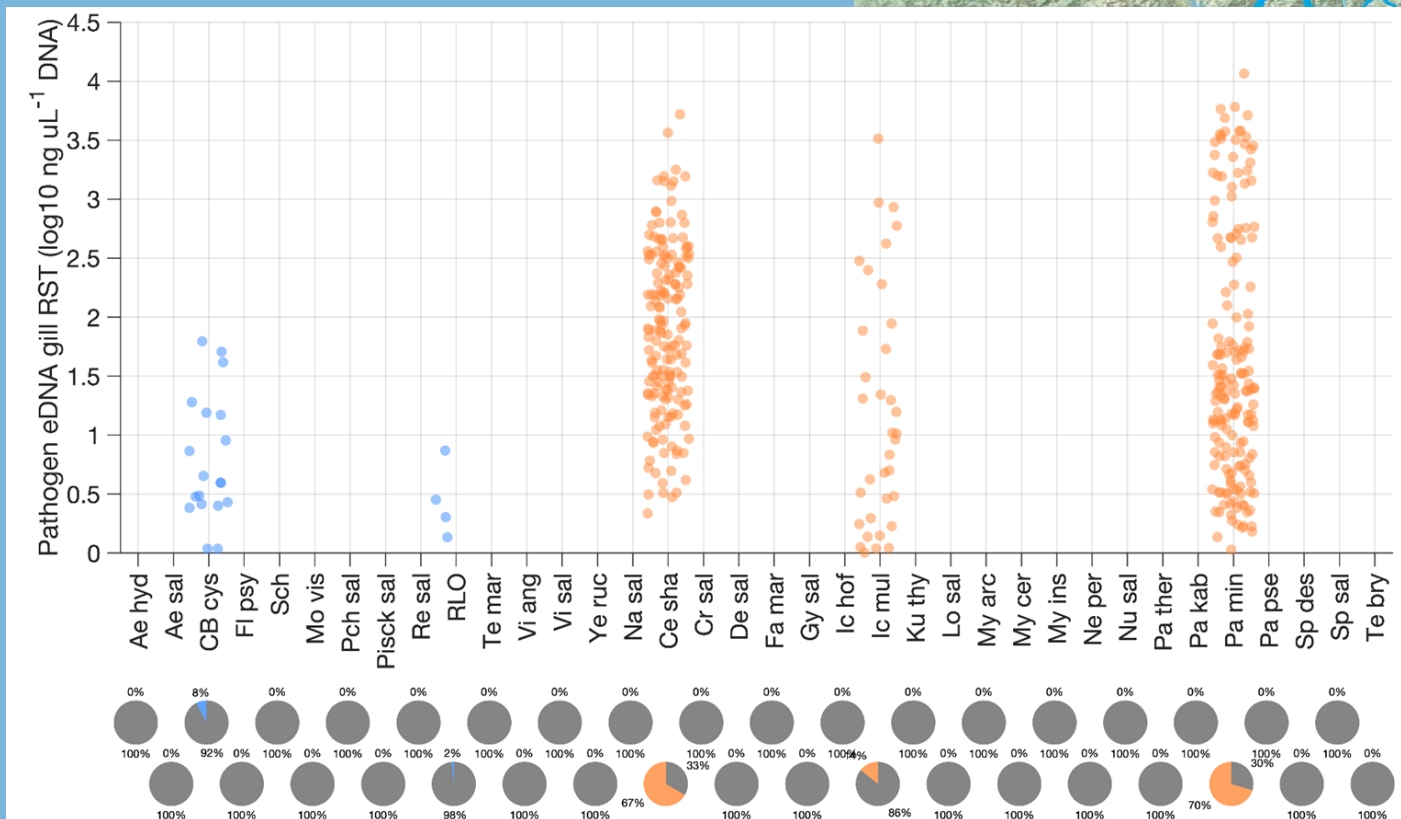
Responses to pathogen exposure in sentinel juvenile fall-run Chinook salmon in the Sacramento River, CA



Screening Environment (sentinel fish)



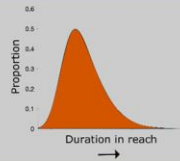
Screening Wild Salmon (rotary-screw trap samples)



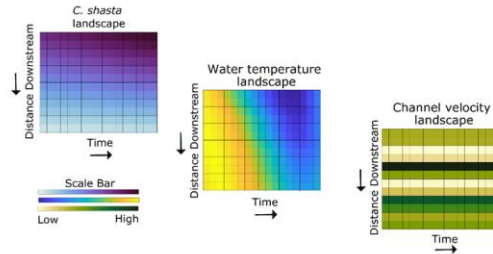
***Integrating data
and disease
transmission model***



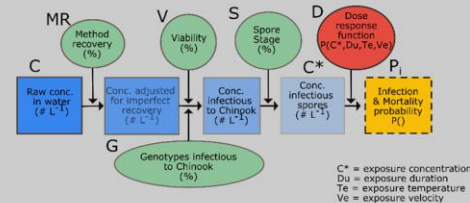
Outline of models to estimate *C. shasta* infection and mortality



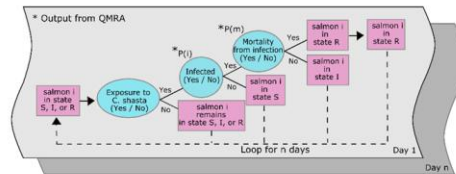
(A) Movement Model:
Based on empirical tagging studies
to estimate outmigrating timing



(B) Exposure Model:
Based on empirical water samples
and hydraulic and water temperature
models to estimate spatially and
temporally explicit exposure



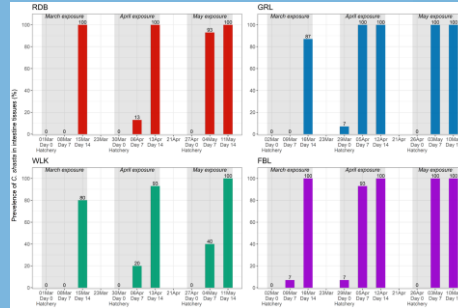
(C) QMRA Model:
Based on A and B models and used
to estimate probability of infection
and mortality for individuals



(D) SIR Model:
Based on A, B, and C models and used
to estimate probability of infection
and mortality for population

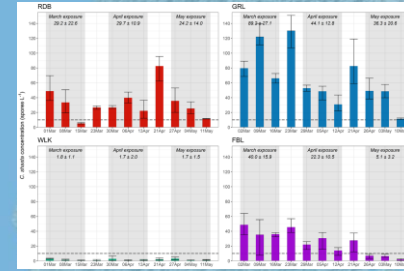
Dose-Response Model

Infection prevalence



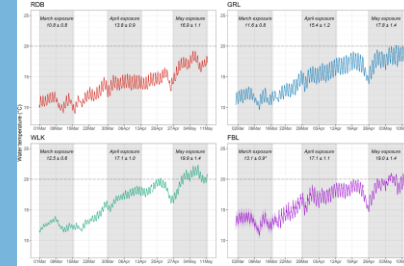
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Mixed Effects Logistic regression
(cluster by exposure and site, constant intercept, slope ~ Cs)



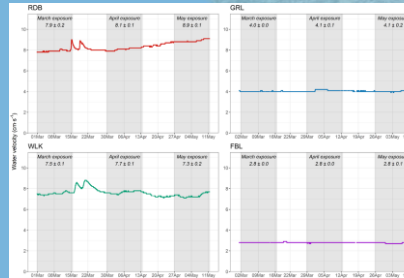
Cs concentration

+



Water temperature

+



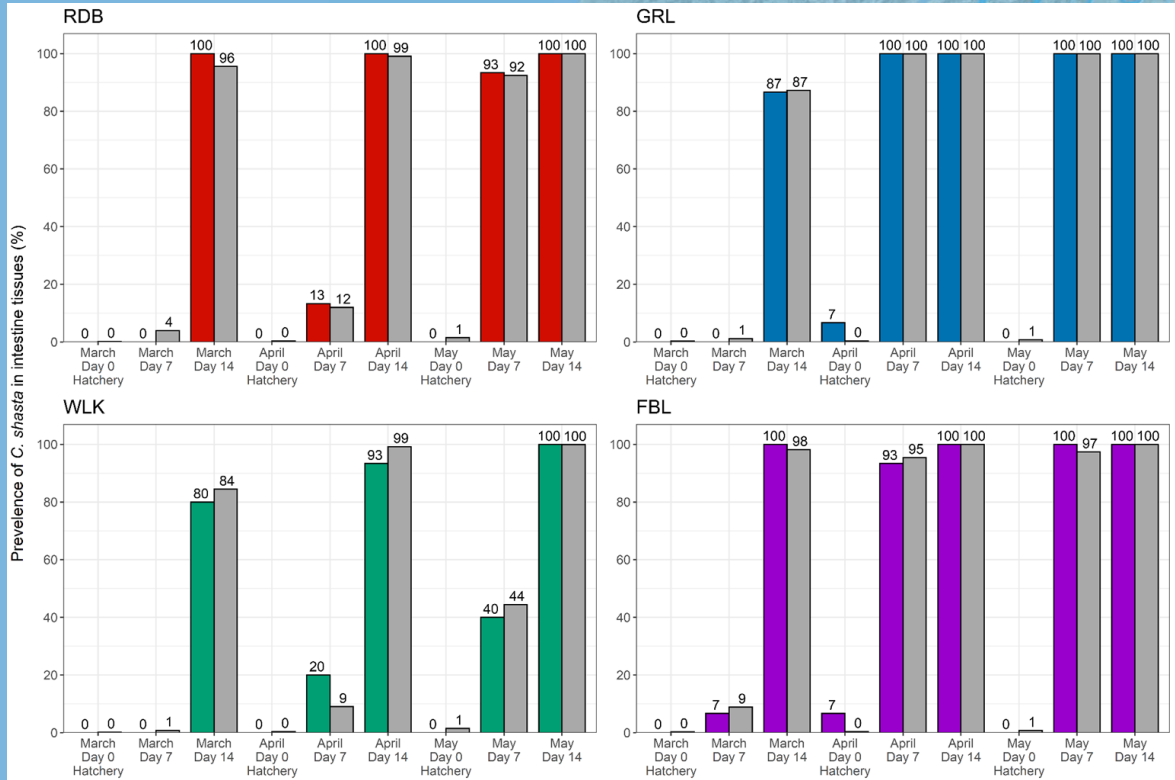
Channel velocity

+

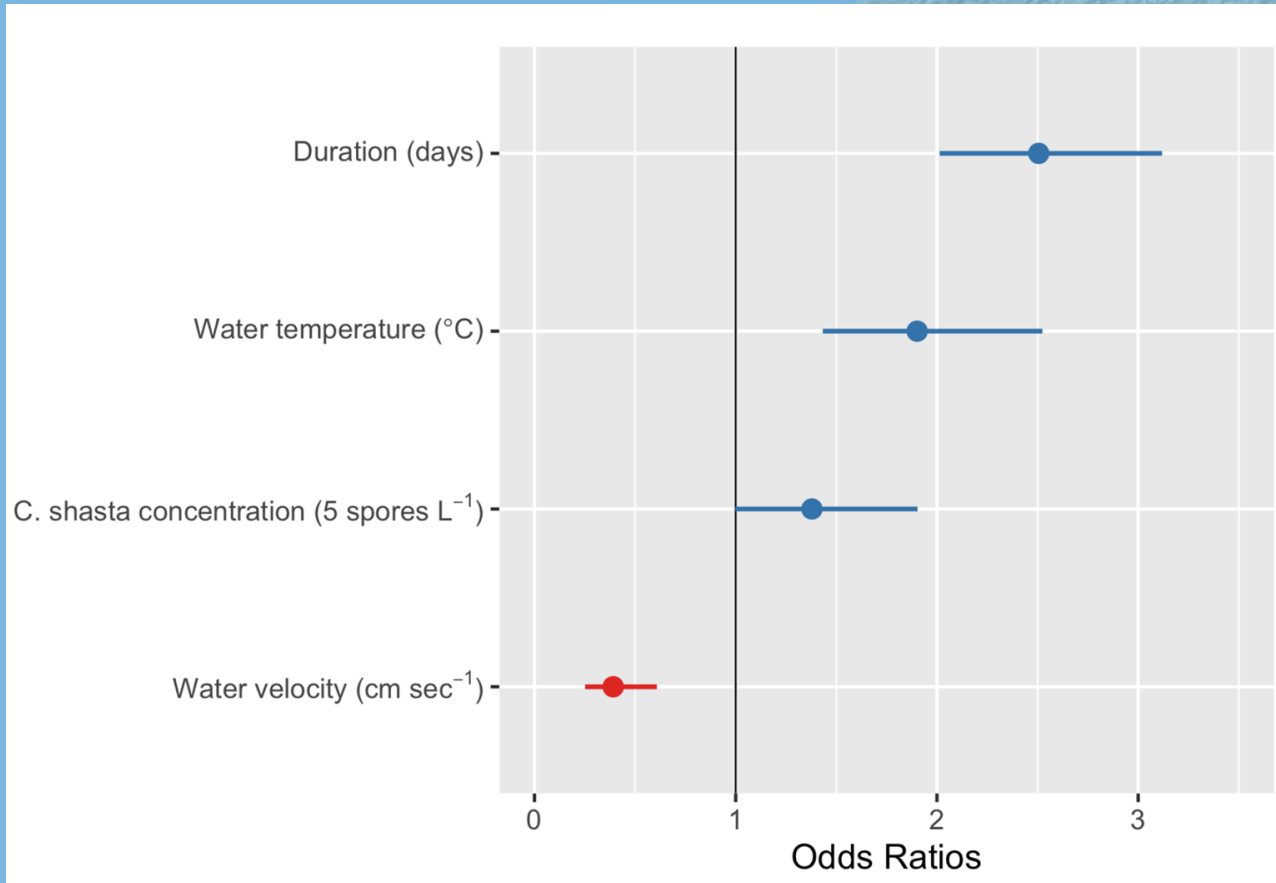
Duration

Infection model skill

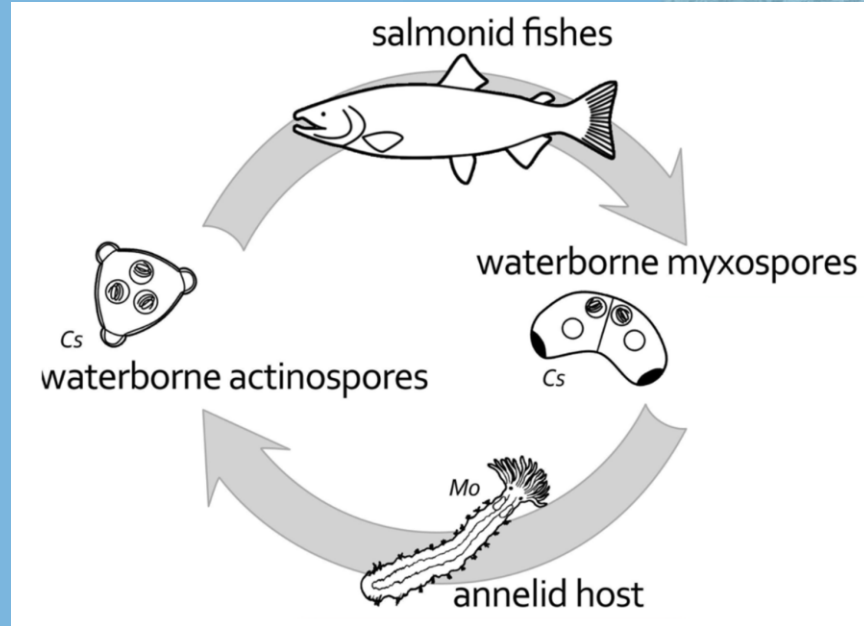
Prediction



Infection model inference

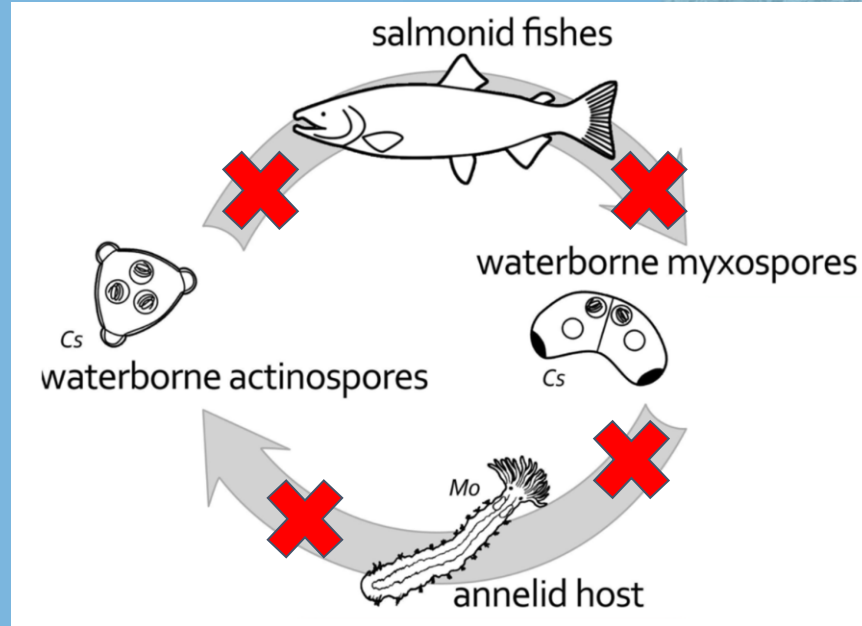


Ongoing Research for Central Valley



Better understand pathogen lifecycle
dynamics and relation to water
management

Ongoing Research for Central Valley



Better understand pathogen lifecycle dynamics and relation to water management

Conclusions

- Multiple lines of evidence (water, sentinel, RSTs) point to consistent detection of some pathogens
- Gene expression and histology indicates host response
- Understating ultimate fate of infection and side effect is critical in relation to management knobs

Investigators & Collaborators:

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Scott Foott (USFSW),
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Samah Adbelrazek (UCD)
Benjamin Atencio (UCSC/NOAA)
Camilo Sanchez (UCD)

and others...

Thanks for listening!

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