# 19 Modeling the Effects of Climate Change on Disease Severity: A Case Study of *Ceratonova* (syn *Ceratomyxa*) *shasta* in the Klamath River

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**Abstract**

Shifts in future temperature and precipitation patterns will have profound effects on host-parasite interactions and the dynamics of disease in freshwater systems. The aims of this chapter are to present an overview of myxozoan disease dynamics in the context of climate change, and to illustrate how these might be predicted over the next several decades by developing a case study of disease dynamics of *Ceratonova* (syn *Ceratomyxa*) *shasta* in the Klamath River, California USA. Our case study introduces a model ensemble for predicting changes in disease dynamics under different climate scenarios (warm/dry, moderate/median, and cool/wet) from 2020 to 2060. The ensemble uses Global Circulation Models (GCMs) and basin scaled models for the Klamath River to generate predictions for future water temperature and river discharge. The environmental data are used as inputs for a predictive model and a degree day model to simulate effects of climate change on polychaete host populations and on *C. shasta* spore viability, respectively. Outputs from these models were then used to parameterize an epidemiological model to predict changes in disease dynamics under each climate scenario. The epidemiological model outputs were measured against baselines established using real data for low (2006), high (2008) and intermediate (2011) disease risk years. In general, the epidemiological model predicts that except for infrequent high discharge years, *C. shasta* dynamics will be

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similar to the high disease risk baseline (2008). This suggests that the recovery and management of Klamath River salmon will continue to be impacted by *C. shasta*.

**Keywords**
Epidemiological models · Disease risk · Disease dynamics · Phenology · Host-parasite interactions · *Manayunkia speciosa*

## 19.1 Overview of Climate Change Effects

Global Circulation Models (GCMs) simulate the circulation of the atmosphere and ocean and are the principal tools used to predict future climate change. Outputs from GCMs depend on the model type (Table 19.1). Most GCMs provide predictions for atmospheric temperatures and precipitation. Atmospheric temperature shifts are the most striking predictions from these models. Globally, air temperatures are predicted to increase by ~2–4 °C over the next century (Solomon et al. 2007), with the magnitude of increase varying spatially as a function of distance from the equator. Increases of ~7 °C are predicted for arctic regions whereas only slight (1–2 °C) increases are predicted for equatorial regions (Solomon et al. 2007). The GCM predictions for atmospheric temperatures also vary seasonally. For example, data for the northern hemisphere predict that winter air temperatures will warm at approximately twice the rate of summers (Solomon et al. 2007). Increasing air temperatures are the most consistent predictions from the group of commonly used GCMs.

The GCMs also predict significant changes in precipitation patterns, but with greater uncertainty than for air temperature. Most GCMs predict shifts in the timing and form of precipitation, rather than net changes in the amount. Thus, winters will be wetter and summers drier (Mote 2003; Frei et al. 2006). These shifts will be linked with changes in precipitation form: as temperatures increase precipitation will increasingly fall as rain instead of snow, especially at lower elevations (<2,000 m) (Regonda et al. 2005). The effects of shifts from snowmelt to rainfall will include earlier onset of spring runoff, higher magnitude discharge over a shorter period of time, and reduced water availability in summer months (Regonda et al. 2005). Because precipitation determines the hydrological regimes of freshwater ecosystems, any change in the type and timing of precipitation is likely to have direct and indirect effects on the characteristics of freshwater ecosystems (Poff 1992).

Aquatic environments will also be susceptible to eutrophication caused by increasing temperatures, decreased precipitation, and rising nutrient inputs (Moss et al. 2011). These effects will vary depending on the characteristics of the water body (Gerten and Adrian 2001). For some aquatic environments this will affect changes in trophic structure due to increased sedimentation of organic matter resulting in less oxygen in bottom waters and sediments (Moss et al. 2011).

Shifts in temperature and precipitation will also alter the phenology (or timing) of biological events, with important consequences for disease dynamics. A recent meta-analysis determined that increased temperature was associated with the earlier occurrence of emergence, migration, or reproduction in 62 % of 677 terrestrial and aquatic species (Parmesan and Yohe 2003). The relationship between precipitation and phenological change/shifts is not as clear as for temperature (e.g. Peñuelas et al. 2004). For example, shifts that result in higher magnitude maximum (peak) flows (in winter/spring) and lower base flows (in summer) may alter the stability of host habitats, possibly reducing the overlap between hosts and parasites. However, the effects that shifts in precipitation are predicted to have on phenology often contrast with those predicted for temperature and both co-vary. Consequently, the types and directions of phenological responses to shifts in precipitation can be difficult to predict *a priori*.

## 19.2 Effects of Climate Change on Myxozoan-Host Interactions

Organismal responses to climate change effects are frequently non-linear and opposing (Altizer et al. 2013). For example, both parasite replication and parasite mortality rates are non-linear and increase with water temperature up to a threshold (Patz et al. 2003). When opposing factors such as replication and mortality both increase with temperature, the net effect can be difficult to predict. Predicting the effects of climate change on myxozoan disease is additionally complicated by the complexity of myxozoan life cycles, as the effects of each parameter must be considered in the context of both the vertebrate and invertebrate hosts and for each free-living spore stage. In this section, we review some of the potential effects of climate change on interactions between aquatic myxozoans and their hosts.

### 19.2.1 Water Temperature Effects

Water temperature influences each phase of the myxozoan life cycle, from interactions within hosts to spore viability (Chaps. 14 and 16). Consequently, temperature is likely to be a primary driver of myxozoan disease dynamics. The effects of increased water temperature on interactions between fish hosts and myxozoan parasites commonly manifest as more rapid disease progression and increased mortality. These effects are well documented for the myxosporeans *Myxobolus cerebralis* (Halliday 1973; Baldwin et al. 2000; Bettge et al. 2009), *Ceratonova shasta* (syn. *Ceratomyxa shasta*, Udey et al. 1975; Ray et al. 2012) and *Henneguya ictaluri* (Griffin et al. 2008), and for the malacosporean *Tetracapsuloides bryosalmonae* (Bettge et al. 2009). The effects likely extend to other pathogenic myxozoans. In addition to the increased rate of parasite replication at higher temperatures, temperature effects on fish host immune function also play a role in determining the rate of disease progression (see Chap. 13).

Water temperature also affects invertebrate hosts and interactions with their myxozoan parasites (see Chaps. 10 and 11). In general, individual and population growth rates of invertebrate hosts increase with temperature (Hogg et al. 1995; Hogg and Williams 1996) up to host thresholds. The relationship between temperature and parasite proliferation in invertebrate hosts has not been widely characterized but likely also increases until the upper thermal tolerance of the parasite or hosts is exceeded. For example, development and release of *M. cerebralis* actinospores in the invertebrate host (*Tubifex tubifex*) increase with temperature up to ~20 °C (El-Matbouli et al. 1999; Blazer et al. 2003; Kerans et al. 2005), but at temperatures ≥25 °C the host appears to clear or purge the parasite (El-Matbouli et al. 1999), suggesting *M. cerebralis* may have an upper thermal tolerance between 20 and 25 °C. A similar relationship between water temperature and spore production was noted for *T. bryosalmonae*, but an upper thermal limit was not identified (Tops et al. 2006).

The longevity of myxozoan stages in the environment is inversely correlated with water temperature (Yokoyama et al. 1995; El-Matbouli et al. 1999; Foott et al. 2007; Kallert and El-Matbouli 2008, and see Chap. 12). However, actinospores may be more negatively affected by temperature than myxospores, which are comparatively stable due to the hardened valves that surround the sporoplasm (Hedrick et al. 2008). For example, *M. cerebralis* actinospores remain viable for ~15 days at 15 °C and only 1 day at 23 °C, whereas myxospores remain viable for >60 days at <10 °C and 7 days at 22 °C (El-Matbouli et al. 1999; Hedrick et al. 2008; Kallert and El-Matbouli 2008). Similar relationships were observed for *C. shasta*: actinospores were viable for ~7 days at 4 °C and for ~4 days at 20 °C and myxospores persisted for >150 days at 4 °C and for 50 days at 20 °C (Foott et al. 2007; Bjork 2010; Chiaramonte 2013).

Increasing water temperatures may affect disease dynamics indirectly through effects on host distribution and on host and parasite

phenology. Shifts in the distributions of host species in response to changing environmental conditions may allow myxozoans to disperse further upstream or into previously uninhabited locations. Shifts in host distributions over larger spatial scales could result in major changes in parasite distributions. For example, Okamura et al. (2011) predicted increased ranges and/or range shifts to more northern latitudes or higher elevations for *T. bryosalmonae* in response to warmer temperatures. Shifts in host distributions may in turn affect the timing or duration of host-parasite interactions. Warmer temperatures will alter life cycle phenology, potentially resulting in longer periods of invertebrate host and parasite reproduction as well as increased numbers of parasite life cycles completed within a year (see Marcogliese 2001).

### 19.2.2 Precipitation and Discharge Effects

The effects of climate driven changes in precipitation (considered as discharge) on phases of the myxozoan life cycle may be just as important as those of temperature. The role of precipitation in these interactions is, however, less clear in the context of future climate scenarios. The predicted shifts in precipitation from snowpack runoff to rain will affect freshwater habitat stability through differences in the timing and magnitude of discharge. Decreased magnitude discharges may increase habitat (e.g. fine sediment) available for invertebrate hosts (Marcogliese 2001). The concomitant lower water levels may also cause vertebrate hosts to aggregate in greater densities. An increased overlap between high densities of hosts (vertebrate and invertebrate) and parasites can lead to greater infection prevalence and disease severity (Izyumova 1987; Holmes 1996).

Several studies have examined the effects of water velocity, which is influenced by discharge, on interactions between myxozoans and their hosts. When transmission and infection dynamics of *M. cerebralis* were examined at low and high velocity in a laboratory experiment, Hallett and Bartholomew (2008) observed that prevalence of infection in *Tubifex tubifex* (invertebrate host) and actinospore densities in water were higher in the low velocity treatment. Prevalence and severity of infection in the fish hosts were also higher in the low velocity treatment. Ray and Bartholomew (2013) similarly observed an inverse relationship between velocity and *C. shasta* transmission to fish hosts in a laboratory challenge, as did Bjork and Bartholomew (2009) for *C. shasta* transmission to its invertebrate host, *Manayunkia speciosa*. Although the mechanism (s) have not been identified, spore attachment to fish hosts (e.g. Ray and Bartholomew 2013) and invertebrate host density and foraging success (and hence ingestion of infectious spores; see Chap. 12) (Bjork and Bartholomew 2009; Jordan 2012) are likely reduced at higher velocities.

### 19.2.3 Nutrient Effects

Climate related changes in water quality (nutrient effects) may also affect disease dynamics through their effects on hosts. The amount of organic material in a stream is positively associated with the occurrence of several invertebrate hosts. For example, abundance of *T. tubifex* is correlated with high organic material (Allen and Bergersen 2002; Kaeser et al. 2006). Similarly, greater abundances of bryozoans occur in rivers with higher nutrient levels (Hartikainen et al. 2009) and increased growth and higher intensity of infection with *T. bryosalmonae* in bryozoans at higher nutrient/food levels (Hartikainen and Okamura 2012). Eutrophication also appears to influence outbreaks of proliferative kidney disease (PKD) caused by *T. bryosalmonae* infection. Following diversion of effluent from sewage treatment, the prevalence of PKD was reduced in hatchery and wild fish sampled downstream (El-Matbouli and Hoffmann 2002). Thus,

increased nutrient input or reduced water quality may exacerbate disease risk through effects on invertebrate hosts.

## 19.3 Modeling Approaches for Predicting the Influence of Climate Change on Disease in Aquatic Systems

Bioclimatic models (also known as envelope models, ecological niche models or species distribution models) are primarily applied to zoonoses (e.g. Olwoch et al. 2003; Ogden et al. 2006; Zhou et al. 2008). Such models can be statistical, predicting range shifts from correlations between climate variables and disease responses, or mechanistic, predicting change in spatial patterns of disease from inferred effects on physiological factors (Jeschke and Strayer 2008). Despite their limitations these models can be useful for predicting trends and relationships between climatic and response variables and to guide management actions. Meta-analysis has been used to examine broad patterns by synthesizing results from numerous small-scale correlative studies or experiments. This approach can provide evidence for responses to climate change by quantitatively summarizing data from multiple studies (Root et al. 2003). Statistical approaches have been used to examine correlations between disease incidence or severity and climate patterns that vary over space or time (e.g. Chaves and Pascual 2006). For aquatic parasites, the most informative modelling approaches will likely include a combination of correlative analyses that describe variation under current conditions, and predictive models that forecast disease dynamics under future conditions. The development of predictive models for most aquatic parasite infections and diseases is, however, constrained by a lack of baseline data and by the complexity of host-parasite interactions.

In this case study, we present an approach consisting of a combination of meta-analysis, and statistical and mathematical models to predict the dynamics of *C. shasta* in the Klamath River basin under several future climate scenarios.

## 19.4 Case Study: *Ceratonova shasta* and the Future Klamath River Basin

*Ceratonova shasta* is endemic to river systems throughout the Pacific Northwest region of North America. The parasite causes enteronecrosis (ceratomyxosis) in salmon and trout. Disease impacts on fish populations vary widely, from largely unnoticeable to highly significant effects (Ching and Munday 1984; Hallett and Bartholomew 2011). The Klamath River, California, is one of the more heavily affected rivers, and despite an intensive hatchery enrichment program (from Iron Gate Hatchery), salmon populations have continued to decline, in part due to high *C. shasta*-related mortality (Stocking et al. 2006; Fujiwara et al. 2011; True et al. 2011). Therefore, managing the parasite is a high priority in this river.

In the following sections we describe a model ensemble for predicting the dynamics of *C. shasta* under different future climatic scenarios (Fig. 19.1). The ensemble includes: (1) GCMs to predict future air temperatures and precipitation patterns (Table 19.1) and a fine-scale climate change model to predict future stream temperatures and discharge in the Klamath River (Perry et al. 2011), (2) polychaete models to predict changes in invertebrate host populations under different discharge scenarios (Wright et al. 2014; Alexander et al. unpub. data), (3) a degree-day model to predict *C. shasta* spore viability and number of annual generations under different temperature scenarios (Chiaramonte 2013), and (4) an epidemiological model to predict how *C. shasta* dynamics may respond to future climate scenarios (Ray 2013; Ray et al. unpub. data). The first model (Perry et al. 2011) used downscaled GCM data to provide water temperature and discharge data for the Klamath River and the latter three models are derived from empirical data. Below, we describe each model, the inputs and outputs for each model, and how these outputs are used in the epidemiological model to predict changes in *C. shasta* dynamics under the different future climate scenarios.

**Table 19.1** Global Circulation Models (GCMs) for warm/dry and cool/wet climate scenarios and the predicted climatic factors for the Klamath River by decade from 2020 to 2060. Current conditions from 2006, 2008, and 2011 are shown as a baseline to compare the future river conditions including number of days <4 °C, max spring temperatures, and maximum discharges by Greimann et al. (2011)

| Model name | Modeled year (disease status/ discharge) | Maximum discharge (CMS) | Maximum discharge (CFS) | Maximum spring temp (°C) | Maximum summer temp (°C) | #Days <4 °C |
|---|---|---|---|---|---|---|
| Baselines (current conditions) | 2006 (low disease) | 339.6 | 12,000 | 17.8 | 22 | 31 |
| | 2008 (high disease) | 100.23 | 3,544 | 21.7 | 24.7 | 34 |
| | 2011 (moderate disease) | 161.31 | 5,700 | 19.6 | 21.8 | 8 |
| MIUB (warm/dry) | 2046 (min) | 32.97 | 1,164 | 23.4 | 25.6 | 0 |
| | 2039 (median) | 52.59 | 1,857 | 20.7 | 24.5 | 0 |
| | 2015 (max) | 474.02 | 16,738 | 22.4 | 24.4 | 0 |
| GFDL (avg/avg) | 2046 (min) | 34.98 | 1,235 | 19.9 | 25.7 | 0 |
| | 2040 (median) | 90.62 | 3,200 | 20.2 | 24.6 | 0 |
| | 2015 (max) | 943.96 | 33,332 | 20.5 | 23.5 | 5 |
| MRI (cool/wet) | 2046 (min) | 34.41 | 1,215 | 21.9 | 24 | 0 |
| | 2061 (median) | 78.47 | 2,771 | 24.6 | 26.8 | 41 |
| | 2048 (max) | 634.93 | 22,420 | 21 | 24.8 | 39 |

*MIUB* Meteorological Institute of the University of Bonn
*GFDL* Geophysical Fluid Dynamics Laboratory
*MRI* Meteorologic Research Institute
*CMS* discharge $m^3\ s^{-1}$
*CFS* discharge $ft^3\ s^{-1}$

### 19.4.1 Environmental Data Models

We selected three GCMs to provide temperature and precipitation predictions: (1) MIUB—warm/dry, (2) GFDL—average temperature and precipitation, and (3) MRI—cool/wet (Table 19.1, Fig. 19.1). The GCMs were selected to represent a wide range of conditions for both temperature and precipitation (based on the quantile rankings for predicted temperature and precipitation). We note that other factors will be affected by climate change (e.g., acidification, UV-radiation), but limit our analyses to temperature and precipitation predictions provided by the different models.

Future (2012–2061) Klamath Basin water temperature and discharge values were estimated from a calibrated one-dimensional water model (RBM10, Perry et al. 2011). This RBM10 model was developed using data from GCMs ($\sim$275 $km^2$) downscaled to provide more refined watershed-specific estimates (270 $m^2$) as described by Flint and Flint (2008).

*Data Inputs*: (1) Temperature and precipitation from GCMs, (2) basin scaled air temperature and discharge from an environmental model for the Klamath Basin (Flint and Flint 2008).

*Data Outputs*: Water temperature and maximum discharge at study location (Fig. 19.2) for each of three climate scenarios (min, median and max) for each GCM (Table 19.1, Perry et al. 2011).

### 19.4.2 Polychaete Predictive Model

To predict the effects of climate change on polychaete hosts, we used two modelling approaches: (1) two-dimensional hydraulic models (2DHMs) and (2) a logistic regression model (Fig. 19.1) (Alexander et al. unpub. data). The purpose of the hydraulic model was to predict depth, velocity, and substrate data. These variables are the inputs needed for the logistic regression model, which predicts the probability

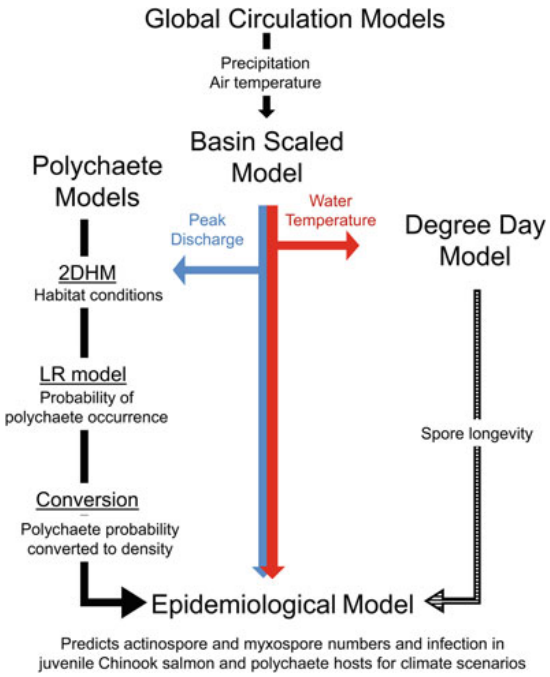


**Fig. 19.1** Conceptual diagram of model ensemble and sources of data and data flow among the different models. Model outputs are represented by *smaller font*. *2DHM* 2 dimensional hydraulic model, *LR* logistic regression. The *dashed arrow* from the degree-day model indicates that this model was not used in this analysis as there was not a significant difference in degree-day accumulations between the current and future temperature scenarios in the Klamath River basin

of polychaetes being present at specific locations. Together, these models predict the probability that polychaetes will occupy specific locations based on maximum winter discharge predicted for each of the modeled future climate scenarios. 2DHMs have been developed for three sections of the Klamath River (Wright et al. 2014) in which prevalence and severity of *C. shasta* infection is high in juvenile salmon (Hallett and Bartholomew 2006; Hallett et al. 2012). The logistic regression model developed by Alexander et al. (unpub. data) uses data outputs from the 2DHMs to predict the probability of polychaete presence at specific x, y locations.

*Data Inputs for 2DHM*: Maximum discharge predicted from the basin scaled model (Table 19.1).

*Data Outputs from 2DHM*: Depth, velocity, and shear stress at 0.5 m grid cells at each site. These data are coupled with a substrate map created in 2011 (Wright et al. 2014).

*Data Inputs for logistic regression model*: Depth, velocity, and substrate at maximum discharge for each of the modeled future climate scenarios.

*Data Outputs from logistic regression model*: The probability of polychaetes at 122,747 x, y data points approximately 0.5 m grid cells apart. The probabilities were weighted by grid cell size and then averaged to obtain an overall probability of occurrence for the entire study reach. To obtain polychaete densities for input into the epidemiological model, we adjusted a baseline density (measured in 2010, Jordan 2012) by the difference in the modeled probability of polychaetes for each climate scenario. This required two steps. First, the probability of polychaete presence was modeled for the baseline (2010) and compared to the modeled probability of polychaetes for each climate scenario. Second, the baseline density was adjusted by the difference between each modeled scenario and the baseline. Thus, a 20 % decrease in the probability of polychaete presence was translated into a 20 % decrease in the baseline density. The baseline density was estimated using data from Beaver Creek (Jordan 2012), a site for which long term data for parasite density and prevalence of infection in sentinel fish are available (Hallett et al. 2012) and for which Wright et al. (2014) developed a 2DHM.

### 19.4.3 Degree Day Model

Chiaramonte (2013) developed a degree day model for the *C. shasta* life cycle that uses the accumulation of thermal units to estimate the time to completion of a single generation and the longevity of each spore stage (Fig. 19.1). Results from a combination of field and laboratory studies identified a temperature threshold of ~4 °C, below which development of *C. shasta* is inhibited. This is significant because if winter temperatures increase, as predicted, there is a potential for year round release of *C. shasta* actinospores. In the salmon host, myxospore

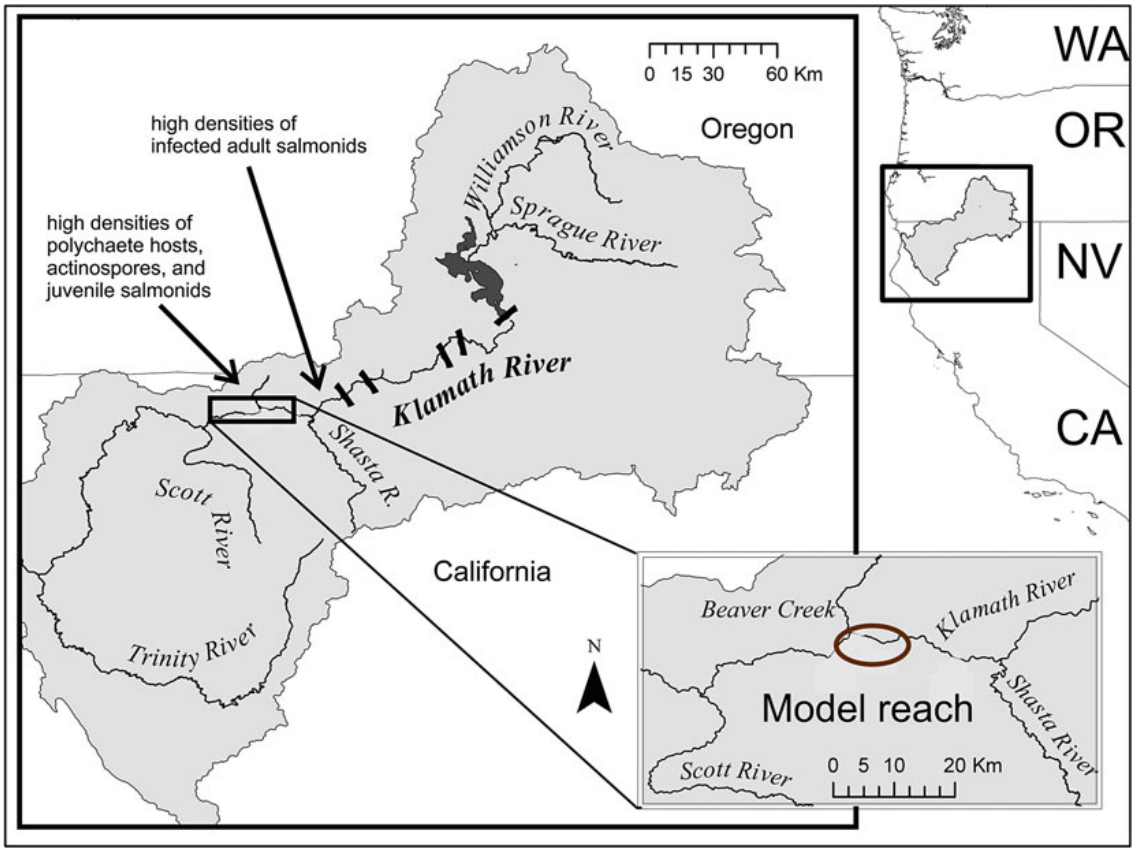


**Fig. 19.2** Location of case study reach on the Klamath River, California. *Black perpendicular lines* indicate location of dams, the lowermost of which is a barrier to anadromous salmonids. High densities of infected adult salmonids are found in the reach immediately downstream from the lowermost dam, and high densities of polychaetes, actinospores, and juvenile salmonids are observed in the reach downstream from the confluence with the shasta River

development occurs in ~282 degree days. About 651 degree days are required for development and release of actinospores from the polychaete host. Thus, one complete generation of *C. shasta* requires ~934 degree days at >4 °C, assuming instantaneous transmission to the next host. This is about the number of degree days required from spawning to emergence of fry.

*Data Inputs*: Water temperatures predicted from the downscaled GCMs were used for each water year type (2006, 2008, 2011) and future climate scenario (MIUB, GDFL, MRI) (Table 19.1). Data included the number of days from November 1st to March 31st where water temperatures were <4 °C and the mean water temperature in spring, summer, and fall.

*Data Outputs*: Longevity of myxospores and actinospore stages.

### 19.4.4 Epidemiological Model

Ray (2013) developed an epidemiological model for the *C. shasta* life cycle that incorporates the rates of spore production from, and transmission to, their respective hosts, and mortality rates of host and parasite stages. This model is comprised of a series of differential equations that mathematically represent phases of the *C. shasta* life cycle. We have further developed the model to include a periodicity function to more accurately capture the timing of life cycle phases (Ray et al. unpub. data, Box 19.1).

**Box 19.1** Epidemiological model periodic equations and descriptions. κ (kappa) is a periodic "switch" function that activates different parts of the model corresponding to the season in which they occur. κ is "switched on" in the spring with a value of 1 and "switched off" (value of 0) in the other seasons. ω denotes a maturation rate term for juvenile salmon that results in a loss of juveniles but a gain in adult salmon (here we simplify and assume instantaneous maturation).

Equation (19.1) Actinospores are produced ($\lambda_a$) from infected polychaetes ($P^*$) in either the spring or fall and are lost due to natural mortality ($\nu_a$; related to water temperature) or transmitted (β) to juvenile ($S_J$) or adult ($S_A$) salmon hosts. Equation (19.2) Myxospores are produced ($\lambda_m$) from infected juvenile ($S_J^*$) and adult ($S_A^*$) salmon hosts and are lost due to natural mortality ($\nu_m$) or are transmitted (β) to polychaete (P) hosts. Equation (19.3) Polychaetes are assumed to have year round production ($\lambda_P$) and natural mortality rates ($\mu_P$). Polychaetes become infected when the myxospore stage is transmitted (β) in either the winter, from infected adult salmon, or summer, from infected juvenile salmon. Equation (19.4) Infected polychaetes are produced in the winter and summer from the transmission (β) of myxospore stage and are lost due to natural mortality ($\mu_P$). Equation (19.5) Adult salmon return in the fall (uninfected, $\lambda_A$) and we assumed no *C. shasta* related pre-spawn mortality. Adult salmon become infected when the actinospore stage is transmitted (β) from infected polychaetes in the fall. In the winter all adult salmon succumb to natural mortality ($\mu_{SA}$). Equation (19.7) In most systems, Juvenile salmon are produced in the spring from returning adults the previous fall ($f_{SA}$) but due to the significant and consistent hatchery production in the Klamath River system we assume this value to be constant. Juvenile salmon are lost due to transmission of the actinospore stage (β), natural mortality (μ), and maturation to the adult stage (ω). Equation (19.8) Juvenile salmon become infected when the actinospore stage is transmitted (β) from infected polychaetes in the spring and are removed from the system due to mortality ($\mu_{SJ}^*$, both natural and parasite induced).

$$da/dt = \kappa_{Sp}(t)(\lambda_a P^* - \nu_a a - \beta a S_J) + \kappa_F(t)\,(\lambda_a P^* - \nu_a a - \beta a S_A) - \kappa_{Su}(t)(\nu_a a) - \kappa_w(t)(\nu_a a) \tag{19.1}$$

$$dm/dt = \kappa_W(t)(\lambda_m S_A{}^* - \nu_m m - \beta m P) + \kappa_{Su}(t)\,(\lambda_m S_J{}^* - \nu_m m - \beta m P) - \kappa_{Sp}(\nu_m m) - \kappa_F(\nu_m m) \tag{19.2}$$

$$dP/dt = \kappa_W(t)(\lambda_P P - \mu_P P - \beta m P) + \kappa_{Su}(t)\,(\lambda_P P - \mu_P P - \beta m P) - \kappa_{Sp}(\mu_P P) - \kappa_F(\mu_P P) \tag{19.3}$$

$$dP^*/dt = \kappa_W(t)(\beta m P - \mu_P P^*) + \kappa_{Su}(t)\,(\beta m P - \mu_P P^*) - \kappa_{Sp}(t)(t)(\mu_P P^*) - \kappa_F(t)(\mu_P P^*) \tag{19.4}$$

$$dS_A/dt = \kappa_F(t)(\lambda_A - \beta a S_A) - \kappa_W(t)\mu_{SA} S_A + \kappa_{Sp}(t)\omega_{S_J} \tag{19.5}$$

$$dS_A{}^*/dt = \kappa_F(t)\beta a S_A - \kappa_W(t)\mu_{SA}{}^* S^*{}_A \tag{19.6}$$

$$dS_J/dt = \kappa_{Sp}(t) f_{SA} - \kappa_{Sp}(t)(\beta a S_J + \mu_{SJ} S_J + \omega_{SJ} S_J) \tag{19.7}$$

$$dS_J{}^*/dt = \kappa_{Sp}(t)(\beta a S_J - \mu S_J{}^*) - \kappa_{Su}(t)(\mu_{S_J}^* S_J^*) \tag{19.8}$$

*Data Inputs*: Predicted (min, mean, max) spring, summer, and fall water temperatures from the GCMs and the basin scaled models were used as inputs into the epidemiological model to parameterize parasite induced mortality and myxospore production in the salmon host (Fig. 19.1, Table 19.2). Predicted maximum winter discharges from GCMs were input into the epidemiological model to parameterize myxospore transmission rates to the polychaete host. Changes in occurrence and availability of polychaete habitat were used as a proxy metric for changes in polychaete population densities for the different climate scenarios. The differing spore longevity rates from the degree-day model were used to parameterize spore survival.

In addition to data provided from models in this ensemble, data from a long-term longitudinal survey were incorporated. Data from sentinel ('caged') fish and water samples collected since 2006 were used to parameterize parasite distribution and density, and the severity of infection in fish at the study site (Ray et al. 2010; Hallett et al. 2012). Data from out-migrating juvenile salmon were used to parameterize prevalence of infection in juveniles (True et al. 2013).

*Data Output*: Changes in the population size (or population growth) of actinospores, myxospores, infected polychaetes and infected juvenile Chinook salmon over a 12 month time frame for each of the different climate scenarios and water years.

### 19.4.5 Predicted Climate Scenarios and Their Effects on the *C. shasta* Life Cycle

The environmental data models predict important differences in temperature and significant differences in discharge in the Klamath River under all future climate scenarios (Flint and Flint 2008; Perry et al. 2011) (Table 19.1). Spring water temperature is predicted to increase by ~1–3 °C between 2012 and 2060, depending on the climate scenario (Perry et al. 2011). In the Klamath River, where salmonids already exist near their upper thermal tolerance limits (<20 °C) this seemingly small temperature increase may be particularly significant. Overall, net precipitation is predicted to remain the same as current conditions, but summer precipitation is predicted to decrease by 1–3 mm, with the balance

**Table 19.2** Epidemiological model assumptions and starting values for density of polychaetes (Poly) and infected polychaetes (Inf Poly), parasite transmission, and mortality rates for actinospores in spring ($A_{sp}$) and fall ($A_f$), myxospores in winter ($M_W$), and infected juvenile salmon (*Inf JS*)

| Scenario | Density | | Transmission | Mortality rates | | | |
|---|---|---|---|---|---|---|---|
| | Poly | Inf Poly | Rate | $A_{sp}$ | $A_f$ | $M_W$ | Inf JS |
| 2008 | 100,000 | 20,000 | 2.00E−06 | 0.20 | 0.20 | 0.067 | 0.05 |
| 2011 | 76,000 | 15,200 | 1.30E−06 | 0.1 | 0.1 | 0.01 | 0.04 |
| 2006 | 36,000 | 7,200 | 5.70E−07 | 0.067 | 0.067 | 0.067 | 0.033 |
| MIUB min | 100,000 | 20,000 | 6.00E−06 | 0.2 | 0.2 | 0.01 | 0.05 |
| MIUB med | 97,000 | 19,400 | 4.00E−06 | 0.2 | 0.2 | | 0.04 |
| MIUB max | 22,000 | 4,400 | 4.20E−07 | 0.2 | 0.2 | | 0.04 |
| GDFL min | 100,000 | 20,000 | 6.00E−06 | 0.125 | 0.1 | | 0.04 |
| GDFL med | 90,000 | 18,000 | 2.20E−06 | | | | |
| GDFL max | 1,000 | 200 | 2.13E−07 | 0.125 | | 0.067 | 0.04 |
| MRI min | 100,000 | 20,000 | 6.67E−06 | 0.125 | | 0.01 | 0.05 |
| MRI med | 92,000 | 18,400 | 2.50E−06 | 0.25 | 0.25 | 0.067 | 0.05 |
| MRI max | 1,000 | 200 | 3.17E−07 | 0.125 | 0.25 | 0.067 | 0.05 |

Starting values for actinospores (1,000), myxospores (2,500), adult salmon (0.01), infected adult salmon (0), juvenile salmon (1), infected juvenile salmon (0), myxospore mortality (0.02), polychaete mortality (0.01), adult salmon mortality (*winter* 1; all die), and juvenile salmon mortality (0.002) were held constant across modeled scenarios

(+1–14 mm) occurring in winter (Barr et al. 2010). Maximum winter discharge is predicted to vary significantly among and within the different climate scenarios and the majority of the differences we detected among modeled response variables (actinospores, myxospores, polychaete populations) appear to be driven by discharge.

The epidemiological model predicts substantial variation in numbers of actinospores, myxospores, infected polychaetes and infected juvenile salmon among the modeled climate scenarios (Fig. 19.3a–d). The outputs are interpreted relative to model outputs for three baselines for the years 2008 (high mortality in salmonids), 2011 (intermediate mortality), and 2006 (low mortality), when inputs were based on real data collected in the Klamath River. In general, many of the future values for actinospores, myxospores, infected polychaetes, and infected juvenile salmon were predicted to be similar to the 2008 baseline values. This provides compelling evidence that *C. shasta*-induced mortality will remain high in the Klamath River. Predicted effects of climate scenarios on life cycle phases and model limitations are discussed below.

#### 19.4.5.1 Actinospores

Under current climate conditions, actinospore release begins in early spring (3rd time step—roughly March or April, Fig. 19.3a) as water temperatures increase above 10 °C. Densities peak in late spring and a second peak, of lower magnitude, generally occurs in the fall. The model predicts that actinospore numbers during juvenile salmon migration will be similar to 2008 (high disease risk) for all scenarios except the

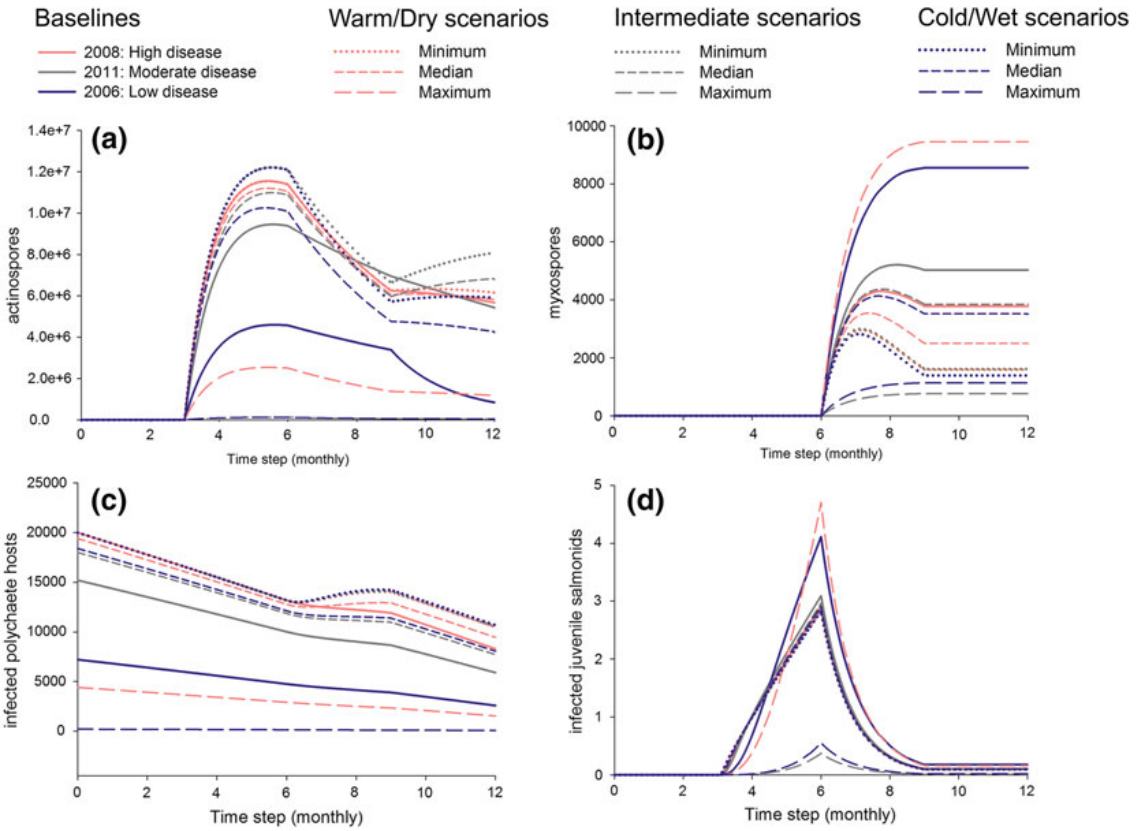


**Fig. 19.3** Epidemiological model predictions for numbers of **a** actinospore **b** myxospore, **c** infected polychaete, **d** infected juvenile salmon populations under nine future climate scenarios, including *minimum*, *median* and *maximum* for MIUB (warm/dry), GDFL (intermediate) and MRI (cool/wet). Baselines (2006, 2008, 2011) are shown as *solid lines*. Time steps are based on monthly periods (from Jan to Dec)

maximum scenarios for all climate models, where actinospore numbers decrease below 2006 (low disease risk) levels. A limitation of the current model is that it does not capture interannual variability in start and peak dates of actinospore release because the periodicity function is based on a monthly time step. Our empirical data demonstrate earlier and longer periods of actinospore detection in warmer years (2008 and 2009) than in cooler years (2010 and 2011, Chiaramonte 2013). Because these effects may counteract or offset the increased spore mortality at higher temperatures, we recognize this may be an important limitation of the model.

#### 19.4.5.2 Myxospores

Under current conditions, myxospore production occurs in late spring (outmigrating juvenile salmon) and in late fall to early winter (upstream migrant adult salmon). The epidemiological model predicts myxospore production in late spring through winter (time steps 6–12; Fig. 19.3b). The model also predicts that myxospore levels will be lower or equal to 2008 baseline level. We would expect 2008 myxospore levels to have been high relative to 2006 and 2011 baselines but the model predicts the opposite. One explanation for this is that under high disease risk conditions juvenile fish may die faster than myxospores develop. A limitation of the current model is that it doesn't reflect that the majority of myxospores are likely derived from the infected spawning adults as their carcasses decompose (late fall to winter). As with the actinospore predictions, the model fails to capture interannual variability in myxospore production, likely as a result of the relatively constant juvenile and adult salmon populations (see starting values for adult returns, Table 19.2).

#### 19.4.5.3 Polychaete Hosts

Under current conditions, polychaete populations expand in late spring, peak in summer and decline in fall (Jordan 2012). Overwintering mature (adult) polychaetes that consume myxospores in late fall or early winter are assumed to be the source of actinospores released in spring. The epidemiological model predicts that under future climate scenarios, numbers of infected polychaetes will be similar to the 2008 baseline (high), except under all maximum scenarios, when densities of infected polychaetes are predicted to be lower than the 2006 (low disease risk) baseline (Alexander et al. 2014). This illustrates the importance of maximum discharge. The model predicts declining numbers of infected polychaetes from time steps 0 to 5, which we attribute to (1) a lack of myxospore input, and (2) natural polychaete mortality in winter to spring (time steps 0–5). During time steps 6–9, the model predicts an increase in numbers of infected polychaetes under all minimum climate scenarios and the warm/dry median scenario, which we attribute to (1) myxospore input from infected juvenile salmon, and (2) polychaete population growth during spring-summer.

For the purpose of examining the effects on juvenile salmon, we focus on model predictions between time steps 5 and 7 (when juvenile salmon are present). During these time steps, water temperatures are >10 °C, and the model predicts high numbers of infected polychaetes (except under maximum discharge scenarios, Fig. 19.3c), which corresponds to the high numbers of actinospores (Fig. 19.3a). As actinospore numbers begin to decline (model time step 6), the increase in numbers of infected polychaetes is likely due to new infections arising from myxospore inputs derived from infected juvenile salmon (see below).

#### 19.4.5.4 Juvenile Salmonid Hosts

Under current conditions the numbers of juvenile salmon released from Iron Gate hatchery are relatively constant and we observe substantial interannual variation in infection rates and numbers of infected juveniles (Ray et al. unpub. data). The epidemiological model predicts that under most climate scenarios, numbers of infected juvenile salmon will be similar to 2008, a year in which high mortality was observed (Fig. 19.3d). However, under cool/wet and intermediate climate scenarios at maximum

discharge, which are predicted to occur at rare and irregular intervals, the numbers of infected juvenile fish will be low relative to present-day conditions (e.g. reduced by one third), indicating the effects of *C. shasta* on juvenile salmon may be less dramatic in those year types. Interestingly, the epidemiological model predicts that numbers of infected juvenile salmon will be highest under the warm/dry maximum discharge and 2006 baseline scenarios. This result may be explained by slower parasite induced mortality at lower temperatures, resulting in greater numbers of juveniles that take longer to be removed from the (modeled) population. This explanation is supported by the high infection prevalence among the outmigrant fish sampled during 2006 (True et al. 2011). Alternatively, this may represent another model limitation that could be resolved by separately modeling juvenile mortality rates and prevalence of infection.

## 19.5 Conclusions

The most striking prediction from the epidemiological model is that only two scenarios predict that the numbers of infected juvenile salmon will be lower than most present-day conditions. For the majority of the scenarios, the numbers of infected salmon will be similar to the 2008 baseline, a year in which high mortality was observed. One important limitation of our epidemiological model is that *C. shasta* life cycle parameters (e.g. actinospore production, polychaete birth/death, transmission rates, etc.) cannot yet be modeled on a daily time step. Future iterations of this model should begin to address these limitations as data become available.

In our case study high winter discharge had the greatest effect on disease dynamics. Smaller polychaete host populations predicted under high discharge scenarios likely produce fewer actinospores, which in turn should result in lower numbers of infected juvenile salmon, while the number of infected juvenile fish was predicted to decrease under cooler temperature regimes. Surprisingly, we did not see a dramatic effect of increasing temperature, which is likely a result of the Klamath system already being near the upper thermal limit for both the parasite and salmon host. This is evidenced by the summer maximum temperatures ranging between 22 and 27 °C, regardless of climate scenario. Another effect of temperature that was not detected in our model is the increase in numbers of infected juvenile salmon compared to the 2008 baseline (a warm/dry year with high disease-related mortality) (True et al. 2011). One explanation for this result is that the thermal tolerance of juvenile salmon is not incorporated in our model. If it had been, the abundance of infected individuals would decrease in the summer months once temperatures exceed 23–24 °C (e.g. Udey et al. 1975; Richter and Kolmes 2005).

Climate change will alter host-parasite interactions, however the magnitude and direction of overall effects are difficult to predict and may be context specific. Our case study exemplifies this complexity in examining the potential effects of temperature and precipitation-driven climate change on the *C. shasta* life cycle in the Klamath River. The study demonstrates the types of data required to develop and parameterize models that can be used to predict *C. shasta* dynamics. We hope that our case study exemplifies how ensemble modelling may be applied for predicting disease dynamics in other pathogenic myxozoans.

## 19.6 Key Questions for Future Research

- Will increasing temperatures cause changes in parasite virulence that affect model predictions?
- Will changes in precipitation be more influential than temperature changes at host or parasite range limits?
- Will the effects of other anthropogenic factors (e.g. dams, pollution) mask the predicted effects of climate change on disease?
- Can conceptual models be developed for myxozoans for which there are limited data on the effects of climate-related parameters on life cycle stages?

- Is this model ensemble approach applicable to other river systems and to other myxozoans?
- How will myxozoans infecting terrestrial and avian hosts be affected by climate change?

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