Modeling Pathogen Dispersal in Marine Fish and Shellfish

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Abstract

In marine ecosystems, oceanographic processes often govern host contacts with infectious agents. Consequently, many approaches developed to quantify pathogen dispersal in terrestrial ecosystems have limited use in the marine context. Recent applications in marine disease modeling demonstrate that physical oceanographic models coupled with biological models of infectious agents can characterize dispersal networks of pathogens in marine ecosystems. Bio-physical modeling has been used over the past two decades to model larval dispersion, but has only recently been utilized in marine epidemiology. In this review, we describe how bio-physical models function and how they can be used to measure connectivity of infectious agents between sites, test hypotheses regarding pathogen dispersal, and quantify patterns of pathogen spread, focusing on fish and shellfish pathogens.

Marine ecosystems are open but connected

Within terrestrial systems, infected hosts or vectors enter a susceptible population to initiate infection through relatively close or direct contact. In marine settings, water can transmit pathogenic agents between hydrodynamically connected populations [1] and infection can be initiated without the presence of an infected host within a naïve population. Seawater provides an ideal medium for the transportation of parasites, viruses, bacteria, and spores. While analogous to airborne transmission routes, transport distances of waterborne pathogens are typically further and mechanisms of dispersal are arguably more complex. Marine pathogens can often survive for a considerable time outside of the host depending on oceanic conditions such as temperature, salinity, pH, suspended organic material and distance travelled [2,3]. Depending upon oceanographic conditions and pathogen viability outside of the host, dispersal distances can be substantial and disease can spread quickly [4]. Passive dispersal through the water column can carry viable pathogens 50 km or more [5], while additional dispersal can occur where infected marine hosts travel vast distances, or human activity (such as the dumping of ballast water) spreads contaminated water [6,7]. For example, in the case of the herpesvirus spread through pilchard in Australia [8] marine pathogens were shown to be able to travel more than 10,000 km yr^{-1}. A strain of *Vibrio* (a species commonly associated with human gastroenteritis and found in many marine food species) has been shown with DNA sequencing to exist on both sides of the Pacific Ocean, in both Asia and South America [9,10]. In contrast, the fastest documented spread of wildlife diseases in a terrestrial setting are diseases in Australian rabbits, which spread at a rate of about 1000 km yr^{-1}, an order of magnitude slower than in marine environments [4]. All of these factors combine to make marine ecosystems typically more conducive to rapid or widespread outbreaks [4,8-12].

Due to fundamental differences between transmission processes in marine and terrestrial environments, unique tools for modeling marine transmission are required. Reviews have called for “epidemic models capable of generating these high rates of spread”, to “obtain…. estimates of disease spread” [4]. In order to adequately capture connectivity patterns, researchers need tools that can simulate regional circulation patterns, physical ocean conditions such as temperature, salinity and pH, together with the biology of the pathogen. Coupled **bio-physical models** (see Glossary) can capture these factors and are increasingly being used across a variety of marine systems. A common source of confusion is in the numerous terms used to describe these models, which vary across disciplines. Box 1 lists common terms for bio-physical models used in the literature. We describe how these models work, considerations for model **validation**, and applications of these models to quantify pathogen dispersal in marine environments.

Bio-physical modeling

Bio-physical models are a product of sequential runs of a hydrodynamic model, a **particle-tracking model**, and a biological model. Fundamentally a type of **agent-based model**, they track individual particles within the simulation. Particles are assigned behaviors that dictate interactions with their environment and with each other. They are used extensively in fisheries and conservation for modeling larval dispersal, but have only recently been adapted to address questions in marine epidemiology [5,13-15]. In this setting, emergent behaviors of particles simulate pathogen dispersal within and among populations. These simulations can capture connectivity of infectious particles between sites [5,13-20], identify natural firebreaks or breakpoints in pathogen movement [16], characterize seasonal patterns in the connectivity of vulnerable metapopulations [19], optimize surveillance and detection methods [21], and infer mechanisms of pathogen spread [13, 22].

Part 1: The hydrodynamic model

Hydrodynamic circulation models are used to describe physical oceanic properties within a specific geographic domain over a specific time period (Figure 1, Part 1). Hydrodynamic models for large areas are generally process-based, deterministic models.

Input data from weather monitoring stations and/or atmospheric models are used to initiate and force the hydrodynamic model. These data include tidal forcing, freshwater discharge (i.e. measurements at river mouths into the ocean), wind stress (from atmospheric models, or wind measurements), heat flux, precipitation and evaporation. Outputs from hydrodynamic models may include 3-D currents (and normally involve a time dimension), as well as salinity and temperature fields, water elevation, and particle mixing parameters. While hydrodynamic models are the “gold standard” to provide detailed water movement information for any given region, they are time intensive to build and run (though becoming less computationally expensive as technology continues to improve), and are limited in terms of scale [23].

Generally, hydrodynamic models use inputs that are limited to a certain time period, using input from weather stations to measure river runoff, winds, etc. If the time period modeled does not coincide with unusual events (e.g. excessive rain or wind), the model may be assumed to represent typical conditions during that time of the year for that region (given the relevant multi-decadal regime, e.g. North Atlantic Oscillation, Pacific Decadal Oscillation, etc.) [19]. Depending on the geographic area of interest there may be several hydrodynamic circulation models with varying levels of detail [24, 25] or none

at all, in which case building one is the first step. In many coastal areas, detailed hydrodynamic models exist, as in the case of the validated Finite Volume Coastal Oceanographic Model (FVCOM) for the Broughton Archipelago, in British Columbia [23, 24].

The appropriate level of model resolution depends upon the biology of the organisms being modeled. For short-lived viral particles, 2-D models that capture the upper layers of the water may be adequate, though aspects of the circulation are inevitably lost. For longer-lived pathogens, particularly those that have complex behavior, diel migration, or several stages of development, a 3-D model will be necessary. The resolution of the model grid varies, with some frameworks (such as FVCOM), able to implement a variable grid size. This allows for smaller grid sizes (and thus a more accurate model) in complex areas of the coast, with a much larger grid size in wide channels. Other frameworks, however, require a fixed grid size (such as Regional Oceanographic Modeling System (ROMS)).

The time step and overall duration of the hydrodynamic model should balance computational constraints and reflect the biology of the organisms being modeled. The shorter the time step, the more accurately models can track movement of particles. For small scale models (e.g. one fjord or loch), a typical time step is on the scale of minutes [14,15]. Models of large areas (e.g. the entire coastline of Norway), or extensive periods (e.g. on the scale of years) may have longer time steps in order to manage computational demands [16]. One of the earliest bio-physical models, used to define bay management areas for infectious salmon anemia virus (ISAv) in New Brunswick, Canada, simulated only a single tidal cycle [26]. This was considered long enough to establish baseline connectivity where the infectious agent particle, such as this virus, is short-lived.

Part 2: The particle-tracking model

Particle-tracking models are a type of agent-based simulation where **nodes** release particles into the environment, which are then transported by the currents simulated in the hydrodynamic model (Figure 1, Part 2). For many epidemiological models, nodes represent meta-populations of interest. In addition to being moved by simulated currents, particles typically have a “random walk” component; for each step a particle takes, there is a probabilistic component to where it will move next. Particles can disperse passively or be assigned model parameters that confer simple behaviors (discussed in the next section). Particle-tracking models are typically run offline, with the movement of particles being driven according to the saved output from the oceanographic model.

Many models are designed so that the emitting nodes release a predetermined number of particles at each time step. These particles are then followed for a predetermined length of time, dependent on the biology of the disease particle. For example, for a virus that can quickly degrade under UV light, less than 24 hours may suffice [27]. In contrast, for parasitic crustaceans, three or more weeks may be necessary to fully capture their survival as free-living organisms, particularly if the simulation is in cold water which can slow development [2,28,29].

The location, amount and frequency of particle emissions depend upon the research question. To quantify the relative risk among emitting nodes, equally sized releases of particles from each node can be simulated and the connectivity between sites compared. If the relative contribution of nodes can be estimated (e.g. in the case of salmon farms, where stocking densities of host salmonids may be known), the number of particles emitted can be scaled accordingly. Additionally, the infection pressure needed to initiate an outbreak varies with life stage of the host organism [30, 31]. If the life stage and vulnerability of the host is known (e.g. during wild salmon outmigration periods, the smolts are more vulnerable than during return migrations of adult salmon), this can be accounted for in the risk mapping stage.

The model output provides snapshots in time, recording particle location and other parameters of interest, such as particle age, velocity, depth, or the salinity the particle was exposed to during that time step. With current computational capabilities, 20-30 minute time steps are frequently used and the pathways of particles between time steps are interpolated [13-17, 25].

Part 3: The biological model

Biological models typically run in tandem with particle-tracking models [15,32]. These models can be used to add information regarding the lifespan, development and environmental niche of the pathogen being modeled (Figure 1, Part 3). Equations describing the impacts of physical conditions on the pathogen biology are usually parameterized from lab experiments [2,33]. These could include impacts of temperature on maturity [2], impacts of salinity on mortality [29,33], or the amount of UV radiation necessary to degrade a virus [27]. Pathogen behaviors can also be modeled. For example, biophysical models demonstrate that vertical migration of sea lice with diel cycles and to avoid low salinity surface waters impacts their dispersal in fjordic systems [34]. However, it is important to note that much of the current understanding of the biology and behaviors of pathogens comes from laboratory studies, because, for many pathogens, finding the free-living stages can be very difficult [14]. For example, it is difficult to capture density-dependent dynamics seen on salmon farms or schooling

behavior in a laboratory study [22]. Therefore, a biological model may be more likely to miss key aspects compared to a physical model.

Connectivity is the result of the complex interactions of four key processes. In larval biology these are referred to as: i) Initiation of emigration (referring to the reproductive output of source population); ii) Transport (referring to physical movement); iii) Settlement; and iv) Recruitment/post-settlement survival [35]. In epidemiological models, these would typically be referred to as: I) Replication/reproduction rates [36]; II) Dispersal [36]; III) Host contact/density thresholds [37]; and IV) Host infection [36].

Most epidemiological bio-physical models incorporate the first two of these stages, but host contact and infection (III-IV) are often ignored [37], In contrast, transport, settlement and recruitment (stages II-IV) are often explicitly modeled within planktonic dispersal simulations [35]. In epidemiological models, the four stages taken together are often referred to simply as transmission probability, and are encapsulated in a single parameter β within traditional epidemiological frameworks, such as SIR (Susceptible-Infected-Recovered) models [38]. This parameter is the result of many complex biological processes in both the host and pathogen, and there is recent interest in decomposing it into distinct mechanisms [38].

Steps III and IV of transmission depend upon contact rates between the pathogen and host (which may be moving targets) [39], the pathogen successfully evading the host immune response (which varies with host age and condition as well as abiotic factors such as salinity and temperature) [40], and, in some cases, exceeding enough pathogen contacts for infection to occur (i.e. the **infectious dose**) [41,42]. The infectious dose is unknown for most marine diseases [42]. All of these steps can be environmentally dependent. For example, the stability of viral hemorrhagic septicemia virus (VHSv) in the water column increases with the concentration of protein in the water (such as fish spawning products) and decreases with increasing salinity, seawater temperature and UV radiation. [43–46]. The susceptibility of Pacific herring to VHSv, is inversely correlated with the ambient seawater temperature [46]. Thus, transmission is impacted by environmental conditions, which can alter factors in both the host and pathogen.

Model complexity

Complexity of biological models varies for a number of reasons; from passive particles being dispersed by circulation models over short time periods [47], to complex behaviors and life histories [5,15,18]. The length of simulations can vary from one tidal

cycle [47], to several months over multiple years [19]. A study simulating a single tidal cycle demonstrated predictive improvement of ISAv dispersal compared to a statistical model using seaway distance [48]. While viruses are generally considered the "simplest" to model, dispersal can still be complex; inclusion of sediment disruption processes such as storms and trawling can improve simulations of viruses that can be stored in sediment, as can including a slow sinking rate for viruses [47]. Viruses can also be transferred by asymptomatic host species (e.g. wild salmonids), or vectors such as sea lice [49-51].

Most bio-physical dispersal models classify particles as infectious or not [15]. However, Adams and colleagues [17] quantify life stages of the infectious agent (in this case, the sea louse) by using outputs from a bio-physical model [14] within a set of differential equations describing population parameters [17]. Where knowledge of population dynamics aids in management decisions, such detail is important, but may be unnecessary for other purposes [14,17].

Quantifying connectivity

Defining what constitutes a successful "connection" between emitting and receiving nodes is challenging [52], whether those nodes are aquaculture sites in Norwegian or Canadian fjords [15,16], reefs in the Caribbean [13], or beaches surrounding a storm-water runoff pipe in California [53]. Connectivity between two nodes is frequently quantified as the probability of a particle emitted from node A making contact with node B [5,18] (Figure 1, Part 4), though other connectivity indices have been used, such as the transfer rate between nodes [54]. A more epidemiologically meaningful measurement would capture the amount of time a particle spends in the receiving node. For example, in a study of sea lice transmission between salmon farms in Scotland, connectivity was defined as the number of particle time steps required for a particle to pass through grid cells [55]. Another study of sea lice in British Columbia, defined connectivity using kernel density estimation weighted on time on particle pathways [15]. In a study of *Panulirus argus* Virus 1 in spiny lobsters, connectivity was defined by the number of time steps required to reach a reef of interest [13].

These measures of connectivity provide a measure that quantifies the relative risk that each emitting node exerts on receiving nodes (and on itself, if self-infection is occurring). However, because studies define connectivity differently, direct comparisons of **infectious pressures** across studies are rarely possible. Additionally, interpreting these absolute values of "residence time" or "particles km^{-2}" in terms of actual infections is problematic, even when the density of hosts in each receiving node is known, as the

necessary infectious pressure to result in disease is also often unknown. This is analogous to the “settlement” values often included in larval dispersal/connectivity models, though here it may be complicated by host immune responses. Unfortunately, infectious dose and host density can often not be estimated. Therefore, absolute values produced by bio-physical models are best interpreted in terms of relative risk among nodes, rather than as real world infectious pressure values.

Capturing temporal variation in connectivity levels is complex [20], and depends on the management goals of the simulation (Figure 1, Part 4). In outbreak scenario dynamics, even short pulses of increased infectious/infestation pressure from other farms or wild reservoirs are important [56], and averaging across time may mask important spikes in connectivity. This could be important in sensitive scenarios, such as the impact of sea lice on very young juvenile wild salmon [57], or for virulent notifiable diseases that require immediate culls on aquaculture sites, such as IHNv [48]. However, smoothing or averaging connectivity across time may be a perfectly reasonable approach for connectivity across wild meta-populations where the ability to take management actions that curtail the spread of the disease may be more limited [13].

Validation

Technologies such as satellite altimetry and gliders, combined with ships-supported suites of sensors, have led to a growing wealth of observational data to evaluate and validate ocean circulation forecasts. More challenging, and less developed, is validating the emergent behavior of coupled bio-physical models, as these results reflect assigned behaviors and biology of tracked particles. Nevertheless, larval dispersal studies have benefitted from additional data sources, notably genetic parentage datasets which provide evidence of individual larval dispersal events that are spatially and temporally limited. Integrating genetic data with bio-physical models serves the dual role of validating the high-resolution predictions from the latter while providing a more complete picture of larval dispersal patterns at regional scales [58]. Genetic parentage studies of infecting parasite are often confounded by competing parasite strains infecting individual hosts [59], but spatial data describing the intensity of parasitic infection are becoming available [15,34]. Dynamically integrating bio-physical models with spatial epidemiological data provides opportunities for major breakthrough in improving the predictive capabilities of such models if the validity of the biological model can be ensured.

Concluding remarks

While epidemiological biophysical models have already proven valuable for spatial planning [16], disease control [60], coastal management [61], designing surveillance programs for notifiable diseases [21], and establishing hypotheses about disease transmission [13], this is the tip of the iceberg (see Outstanding Questions). Marine disease outbreaks in wild populations around the globe are occurring with increasing frequency, (as are human marine-associated disease outbreaks), as the oceans warm and seawater chemistry shifts, influencing the potential and realized niches of hosts and pathogens [11, 62-64]. In aquaculture, the importance of disease control is also vital, particularly as the distance between aquaculture sites decreases and production density increases [16]. Biophysical models will be key for identifying susceptible host populations, predicting disease transmission pathways across larger areas, exploring the impacts of future climatic scenarios on transmission processes, quantifying the vulnerability of regions to future outbreaks, and developing intervention strategies, such as where to implement biosecurity steps, when to cull species, or when and how to treat populations. Table 1 summarizes important marine diseases and how they would benefit from bio-physical model studies. These models will be used to identify rate-limiting processes that should be targeted for intervention. They have untapped potential to test competing hypotheses, for example comparing different modes of transmission, to determine which results in outcomes observed in nature [13]. Limitations in what we know about marine pathogens and their hosts could potentially be filled in using these models.

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Glossary

Agent-based model: computational models that simulate the actions and interactions of autonomous agents, to better understand the behavior of the whole system. Bio-physical models are a type of agent-based model.

Bio-physical modeling: coupling of several simulations to capture emergent behavior of system composed of both biological and physical components. Typically, a bio-physical model is comprised of an underlying circulation model, an offline particle-tracking model, and the particles are also assigned a biological model to simulate the organism being modeled (e.g. viruses, larvae, bacteria).

Hydrodynamic circulation model: numerical models that take inputs such as tidal forcing, freshwater discharge, wind stress, heat flux, precipitation, and evaporation. Outputs include current models, as well as salinity and temperature fields.

Infectious pressure/dose: combination of concentration of infectious agents and exposure time resulting in host infection.

Management/Treatment areas in aquaculture: groups of farms that are required to coordinate treatments in order to limit re-infection from untreated, hydrodynamically connected farms.

Node (emitting/receiving): in network theory, a node exists where two edges connect. In the context of bio-physical modeling, it is the point that emits and/or receives particles. These could represent aquaculture sites, or other susceptible meta-populations.

Particle-tracking model: simulation in which nodes emit particles that are passively carried by an offline circulation model. Particles can be tracked for period of interest, to see how these move.

Validation: in the context of bio-physical modeling, this means finding “real world” data and comparing these to simulated outcomes, to evaluate how accurately the simulation was able to replicate reality.

Table 1. Economically and/or ecologically important marine diseases and how they would benefit from a bio-physical model study.

Disease / Causative agent	Host	Region	Rationale for model	Key aspects to simulate	References
Numerous viruses including infectious hypodermal and hematopoietic necrosis virus (IHHNV), yellow head virus (YHV), Taura syndrome virus (TSV), white spot syndrome virus (WSSV), infectious myonecrosis virus (IMNV)	Penaeid shrimp in aquaculture	Areas where shrimp farming occurs	Numerous disease outbreaks have decimated shrimp aquaculture, predicting the connectivity of farms will help in siting farms, identifying firewalls for disease and identifying source and sink farms for pathogens with varying lifespans	Dispersal of pathogens with different 'lifespans' in planktonic stages over space and time	[65]
White plague disease/There is debate over if this is caused by an unknown virus or bacteria. There are three types of the disease, with varying rates in how quickly they can kill a coral colony.	Various species of coral	Caribbean	Causative agent(s) remains unknown and has killed 70-80% of the coral in the Caribbean. This could be a way to test different causative agents in a simulation, and see which one results in the most similar transmission patterns. Simulating different possible climate change scenarios could provide key information for reef managers in the Caribbean.	Water temperature, as heat stress plays a key component in coral diseases. There are at least three know mechanisms of transmission (contact with contaminated water, contact with macroalgae, and predation by a snail).	[66]
Vibrio	Oysters	worldwide	*Vibrios* sp. can cause illness in humans, particularly when they consume shellfish that have bioaccumulated these bacteria. Understanding causes of increased *Vibrio* levels in oysters could aid regulatory bodies in establishing targeted surveillance methods.	The impact of changing temperature on transmission and replication should be investigated	[67]

Mycobacteriosis/ *Mycobacteria spp.*	Striped Bass (*Morone saxitalis)* in temperate waters, numerous fish species in tropical and subtropical water	worldwide	Mycbacterial infections are slow-progressing and eventually lethal in fish species and some species can cause infections in humans. Mycobacteria is temperature sensitive and may have an increased range with warming seawaters and changing host ranges.	Evaluate how changing host range and seawater temperatures can impact dispersal of *Mycobacteria* sp.	[68]
Viral hemorrhagic septicemia (VHS)/Viral hemorrhagic septicemia virus (VHSv)	Salmonids, Pacific Herring and many other wild and farmed fish	Europe and North America	Many countries carry out routine surveillance of aquaculture sites for this disease. A bio-physical model could highlight which sites and times of the year should have elevated surveillance and/or biosecurity efforts.	The impact of changing temperatures on pathogen dispersal over time and space	[69]
Terebrasabella heterouncinata Sabellid polychaete	Abalone/*Haliotis sp.* and their gastropod hosts	Southern Africa-introduced in 1993 to California [57]	Understanding how this invasive polychaete is carried through the southern California region can aid in developing management methods	Dispersal of larval stages: characterization of connectivity between sites and identification of critical source sites	[70]
Oyster herpes virus (OSHV1)	Pacific oyster/ *Crassostrea gigas*	Many areas where Pacific oysters are cultured around the world	Virulent strains cause high mortality in juveniles oysters and moderate mortality in adults	Dispersal of virions: characterization of connectivity between sites and identification of critical source sites, impact of temperature on transmission and virulence	[71]
Sea Lice (*Lepeoptheirus salmonis* and *Caligus rogercressyi)*	Salmonids (though *C. rogercressyi* is less host specific and can infect other fish hosts, such as arctic charr)	*L. salmonis* is the main disease issue for salmon aquaculture in the northern hemisphere, and *C. rogercressyi* in the southern hemisphere.)	This costs the salmon industry millions annually in treatment costs and lost salmon stock growth. Sea lice also has the possibility of impacting wild salmon stocks, as small smolts (<1g) can be killed by sea lice infestations.	Biology of the sea lice larvae, which is impacted by temperature and salinity, and perhaps some behaviors, such as diel vertical migration.	[16]

Bitter crab disease/ *Hematodinium* sp. infections (dinoflagellate)	Numerous crustaceans, particularly, blue crab, snow crab and tanner crab	Europe, North America, Australia; range expanded to China and Russia in early 2000s	Understanding transmission of this pathogen will be helpful for predicting transitions between enzootic and epizootic states and predicting global spread of this parasite	Dispersal of infectious stages: characterization of connectivity between sites and identification of critical source sites, impact of temperature and salinity on transmission and virulence	[72]
Harmful algal blooms (HAB)*	Do not infect one particular host, but can have a wide variety of impacts on coastal species.	Global distribution	HABs can use oxygen, leading to widespread fish kills due to suffocation. HABs can produce neurotoxins, making shellfish or fish species toxic for humans to consume, among other possible impacts. Thus accurate predictions of HABs can lead to better coastal and fisheries management.	Sea surface temperatures, nutrient loads, upwelling, storm water runoff and/or eutrophication	[73,74]
Cholera*	Humans	Global distribution, wherever crowded living conditions and poor clean water access exist.	The causative agent of cholera is often associated with algal blooms. Models to predict impacts of warming coastal water temperatures and predict the timing of algal blooms could help aid workers better mobilize responses to cholera outbreaks.	Sea surface temperatures, eutrophication, upwelling, identification of susceptible areas.	[75,76]

* While human diseases are not the focus of this paper, one entry for an important human water-borne pathogen (cholera), and one entry about harmful algal blooms (HABs) are included to help guide those interested.

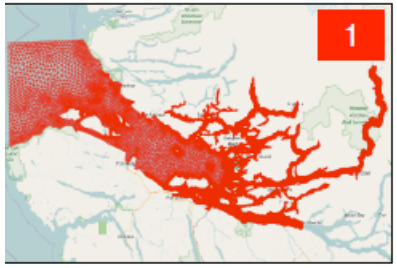
Box 1. Alternative names used in the literature to describe bio-physical models

- Numerical and dispersion/dispersal model [60]
- Hydrology/hydrodynamic and dispersal model [77]
- Hydrodynamic and particle-tracking model [15]
- Hydrodynamic and individual based model [78]
- Hydrodynamic and agent-based model [7]
- Specific name of the circulation model (i.e. FVCOM or ROMS) and particle-tracking model [5]

Figure caption:

Figure1. Schematic illustrating key steps involved in building bio-physical models. Each of the 4 steps lists the inputs and outputs for that step. For parts 1-3, the outputs of each step is then the inputs for the next step. All figures relate to a set of simulations reported in [15]. The illustrative equation in part 3 was used in [15], to describe the biological model.

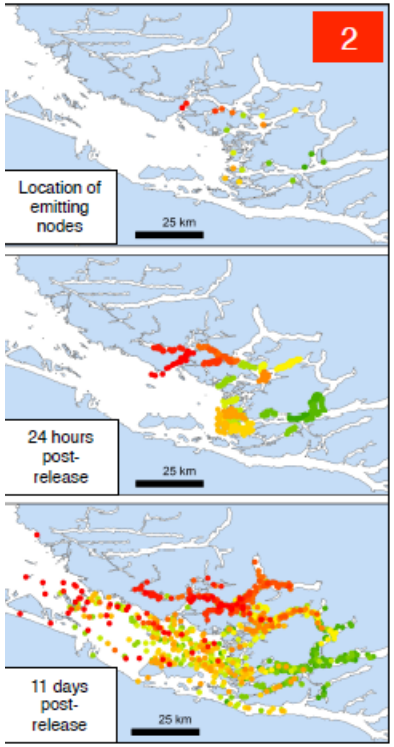
Bio-physical Modeling



Part 1. Hydrodynamic model

INPUTS
Tidal forcing, freshwater discharge, wind stress, heat flux, precipitation, evaporation

OUTPUTS
3-D currents, temperature, salinity, water elevation, mixing parameters



Part 2. Particle-tracking model

Particles are simulated to be emitted from meta-population locations. The frequency of particle releases, amount of particles released, and time the particle movements are tracked for will depend on the study goals.

INPUTS
3-D currents, temperature, salinity, water elevation, mixing parameters

OUTPUTS
Tracking of particles locations. Can also extract and save information about the particles, such as temperature or salinity the particle encounters during each time step.

Part 3. Biological model

Part 3 often occurs in tandem with Part 2

INPUTS
- The particle pathways
- Relevant physical conditions the particles encounter (e.g. salinity or temperature)
- Equations to govern the biology (i.e. how salinity or temperature impact mortality or maturity)

3

$$\tau(T) = \left[\frac{\beta_1}{T-10+\beta_1\beta_2}\right]^2$$

$$\mu_{pl}(S \leq 30) = 0.16 \cdot S - 5.11$$

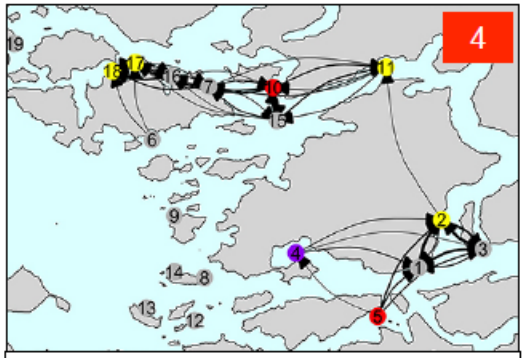
Illustrative equations

OUTPUTS
Particle pathways, together with associated biological factors, such as mortality and/or maturity, etc.

Part 4. Additional analysis

INPUTS
Particle pathways, together with the associated biological information (can be maturity, mortality, etc.)

OUTPUTS
- Networks of emitting/receiving nodes
- Source/sink dynamics of connected meta-populations
- Temporal trends in dispersal
- Impacts of hypothetical management actions, warming oceans, etc.



Infectious agent network. Color of nodes indicate varying levels of inward and outward connection.