

AN AGENT-BASED MODEL TO SIMULATE PATHOGEN TRANSMISSION BETWEEN AQUACULTURE SITES IN THE ROMSDALSFJORD

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ABSTRACT

Fish farming is an important industry along the Norwegian west coast. This industry provides labor opportunities and financial income in areas that are often thinly populated. Fish are subject to diseases carried by pathogens. The value of the fish that are lost due to disease is worrisome, and emergent diseases continue to pose a severe challenge to the aquaculture industry. We have built an agent-based model to simulate the emergence of a hypothetical fish pathogen in an aquaculture facility in the Romsdalsfjord¹ to observe how this pathogen possibly spreads to multiple facilities within the fjord. This model enables us to observe how key parameters such as current speed, current direction, pathogen life span, contagiousness and fish density affect the disease dynamics. The model is implemented in NetLogo, and we have included three aquafarms at the Romsdalsfjord in the experiment.

INTRODUCTION

Aquaculture is about to revolutionize the way we consume fish and other marine food products as agriculture already did on land. During the past few decades world capture fisheries have stabilized or decreased, whereas aquaculture production has increased massively (FAO 2012). In 2010, aquaculture stood for 47% of global food fish production, and in Norway the export value of farmed seafood now exceeds that of wild caught species (FKD 2013). Today, fish is the third most important export product after oil/gas and metal, and accounts for 5.7 per cent of the total Norwegian export value according to Statistics Norway (SSB 2013). Norway is the largest exporter of aquaculture products in Europe, and number six globally, after Asian nations such as China, India and Indonesia (FAO 2012). It is thus clearly of high importance for Norwegian economy to ensure a sustainable aquaculture industry.

However, emergent diseases continue to be a serious challenge to the aquaculture industry and set constraints to its expansion (Murray & Peeler 2005). Diseases both induce large economic costs to the industry (Werkman

et al. 2011) and might threaten wild populations (Murray & Peeler, 2005). A major problem in many areas has been the uncontrolled use of antibiotics leading to resistant bacteria strains (Defoirdt *et al.* 2011). Although the antibiotics use in Norway today is restricted, and some important pathogens have been reduced through vaccination programs, new and/or resistant pathogens still emerge (Olsen & Hellberg 2011). Combatting these diseases is therefore an important research field (Johansen *et al.* 2011) and a hot topic in public debates (e.g. NTB 2011).

Atlantic salmon is by far the most important species in Norwegian aquaculture. The most troublesome diseases for the salmon aquaculture are caused by viruses (Olsen & Hellberg 2011). All major viruses affecting Norwegian aquaculture are thought to spread between fish through sea water (Johansen *et al.* 2011), as infected fish shed pathogens to the surrounding waters. To keep fish farms at appropriate distances is therefore a potential measure to combat this horizontal transmission.

This model aims to simulate the pathogen transmission between aquaculture sites in a Norwegian fjord and observe how this pathogen possibly spreads to multiple facilities within the fjord. This model uses many key parameters such as current speed, direction, pathogen life span, contagiousness and fish density to find possible pathogen transmission patterns. Before describing the model in detail; a more complete background on the dynamics of fish disease transmission and previous studies on this issue will be given in the next section.

Diseases in aquaculture

Knowledge of pathogens in wild fish stocks is generally poor, and it is therefore difficult to predict which diseases might occur once an aquaculture facility is established in an area (Bergh 2007). A wide range of pathogens exists, from viruses and bacteria to crustacean parasites (Olsen & Hellberg 2011). These might be introduced to an aquaculture system through various pathways: movement of infected stocks, equipment or fish products from other areas; or by exposition to wild fish pathogens (Murray & Peeler 2005). Once introduced, pathogens can benefit from the aquaculture environment and pose a graver risk to farmed fish than wild stocks. This is both because of factors such as poor environment, stress and pollution that might reduce individual fish resistance (Murray &

¹ Romsdalsfjorden is 88 km Long and located in the Romsdal district of Møre og Romsdal county.

Peeler 2005), but moreover because the artificial high density of fish, and thus potential hosts for the pathogen, in a fish farm can induce outbreaks (Bergh 2007; Rimstad 2011). Pathogens that benefit from higher host densities follow so-called *density-dependent transmission* (Murray 2009). The rate of transmission is the product of the densities of susceptible and infected individuals.

As previously mentioned, disease transmission can happen with currents, depending on the survival time of the pathogen in water masses, but also through vectors such as wild fish or escaped farmed fish (Murray & Peeler 2005). Hydrodynamic spreading will usually be a local-scale problem, whereas wild fish can become infected nearby a farm and transmit the pathogen over larger distances (Werkman *et al.* 2011). An example of a waterborne virus is the Salmonid alphavirus causing Pancreas disease (PD), an increasing problem in Norwegian aquaculture (Kristoffersen *et al.* 2009). Stochastic models have emphasized the importance of the distance between farms for disease transmission of both this and other diseases affecting farmed salmon, such as heart and skeletal muscle inflammation (HSMI) and infectious salmon anaemia (ISA) (Aldrin *et al.* 2010).

Using an Agent-based model (ABM) to simulate disease transmission in aquaculture

Previous modeling studies on the transmission of pathogens within and between farmed fish populations have either used classical SIR disease transmission models (Susceptible, Infected, Recovered) that focus on the population as a whole (e.g. Murray 2009; Green 2010), or such population models coupled with simple hydrodynamic models or distance measures of transmission between separate populations (Viljugrein *et al.* 2009; Aldrin *et al.* 2010; Werkman *et al.* 2011; Salama & Murray 2011). To our knowledge no studies have previously applied ABMs to assess the transmission of diseases within and/or between aquaculture fish populations. On the other hand, ABMs have been applied to simulate transmission of human viral diseases such as influenza (e.g. Ciofi degli Atti *et al.* 2008; Milne *et al.* 2008). ABMs can be valuable for analyses focusing on individual interactions, and also to incorporate the spatial aspect of the system. Whereas classical SIR-models used in disease transmission modeling represent total populations, we here apply an ABM to simulate individual fish becoming infected, and how pathogens spread spatially by also representing these as agents. By applying an ABM instead of e.g. differential equations more complexity can be added and analysed through simulations. Another reason for applying an ABM is that empirical data regarding fish pathogens are often lacking. It is therefore difficult to predict the threshold for density-dependent outbreaks of diseases in classical disease transmission models (Krkosek 2010). In an ABM, the parameters regarding disease transmission can easily be varied.

Modelling a real system

Since there are presently research activities regarding aqua farms in the Romsdalsfjord, we decided to look for both inspiration for research problems and potential parameter values that have emerged from these studies. From these studies we pursued the effect of the fact that pathogens may survive in water for days without a host. Furthermore, since there are many aqua farms in the Romsdalsfjord (DF 2013), about 35 (*see figure 1*), the close proximity between the farms becomes an important factor in disease transmission. It is a relevant issue in current research efforts to study under which conditions one infected aqua farm may spread disease to other farms by pathogens “jumping” from one aqua farm to the next, creating a domino effect.



Figure 1: Map, which shows the aqua farms in Romsdalsfjorden.

THE MODEL

This section describes how our model works. The model is implemented in NetLogo (*Figure 2*). The idea behind the model is to simulate three aquafarms which reside in the same fjord area, and that we can suspect are subject to cross contamination between each other. All parameters used in the model are listed in *Table 1*.

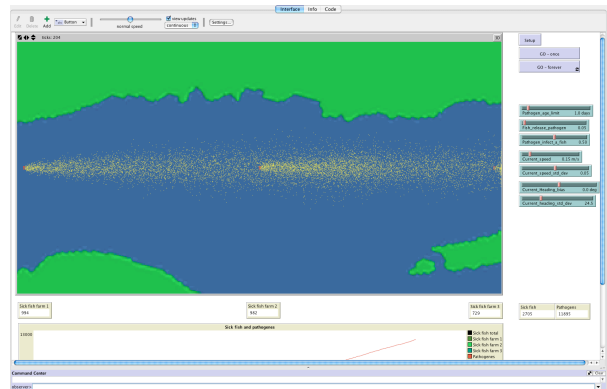


Figure 2: NetLogo model

We do not specifically model the background of introduction of a disease, and assume that the pathogen

is a new variant for which no vaccination is yet in place. The model presented is a general model, which can be adjusted to various pathogens and environmental scenarios. Salmon aquaculture is the most common form of aquaculture in Norway, and since the most important mode of transmission of salmon disease is by water currents we chose to model this process.

Table 1: Parameters used in the model

Parameter	Description	Estimate	Source
Current speed	Current speed in m/s	0.02 – 0.2	4,6,7
Current direction	Current direction in degrees		6,7
Transmission parameter ⁺	Probability that a fish is infected by a pathogen in its surrounding water	5.416×10^{-4} – 8.912×10^{-4} /hour (minimum values)	3
Shedding parameter ⁺	Number of pathogens shed by infected fish	High*	1,2,5
Pathogen lifespan ⁺	Lifespan of pathogen in seawater in days	8.33 – 62.5 hours Depends on pathogen and environment	3

⁺Pathogen values for Infectious salmon anaemia virus (ISAV), Infectious pancreatic necrosis virus (IPNV) and Salmonid alphavirus (SAV).

* Different sources use different units and values are ranging from $10^{6.5}$ PFU/fish/h (PFU= plaque forming units) (1), 10^5 – 10^8 CFU/fish/h (CFU=colony forming units) (2) and 6.8×10^3 TCID₅₀/ml/ kg fish/ h/ (maximum rates) (TCID₅₀=the amount of virus required to kill 50% of infected hosts) (5). The units are not single pathogens but units that are measurable in the lab. Since the numbers are very high, and computationally difficult to implement in the model we set a probability between 0-1 (adjustable) that an infected fish sheds a pathogen, but this pathogen represents a large number. The probability value is likely to be high (close to 1) as observed shedding rates are on these ranges.

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Scaling the model in space and time

As a real-world basis for our model we use the aqua farms at Midsund (Figure 3), in the Romsdalsfjord. This part of the fjord currently hosts three aqua farms, with a

distance of about 8 km between each site. The farms have a size of around 10,000 square meters and host around a million fish each (DF 2013). The total area of interest is about 16 km wide. We constructed the model in a rectangular space of 16 x 8 km. In our model all three aqua farms have the same size, 80 m x 120 m. The farm sizes are thus close to some of the real fish farms in the Romsdalsfjord (DF 2013). Each fish farm is populated with 1000 fish, while in reality you would expect to see about 1000 times more fish in an actual fish farm at this size. This simplification was made in order to save computer resources while running the model.

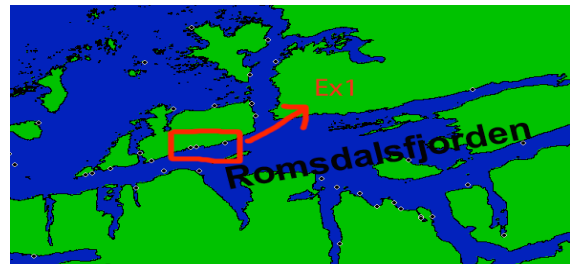


Figure 3: Midsund

According to MODS (2012) the average current speed in the Romsdalsfjord is typically 0.2 m/s. This equals 12 m/min or 120 m/tick. Tick is the time step in the model and its representing 10 minutes. The model enables the current speed to be varied around the default value of 0.2 m/s, and in order to move the pathogens at the correct speed in the simulations the conversion factor was incorporated.

The fish agents

The fish are created during the setup procedure of the model. In each of the three farms; 1000 fish are distributed across the farm’s area. When the simulation starts all fish are healthy, i.e. belonging to the susceptible group. The exception is one infected fish in Farm 1. Healthy fish may become infected if there is a pathogen present at the same place as the fish. Once a fish is infected they start to produce pathogens at a rate given by a certain parameter (*Fish_release_pathogen*). This parameter represents the probability that one fish produces one pathogen in one time step. The probability that a healthy fish gets infected while being on same place as a pathogen during one time step is given by the parameter *pathogen_infect_a_fish*. The two probabilities for pathogen production and infection are flexible (between 0 and 1).

Pathogen agents and current simulations

Pathogens are also represented as agents. It is important to note that one pathogen agent does not represent one pathogen, but a batch of a high number of pathogens. Each pathogen is moved by the currents, with the current speed and direction given by the place the pathogen is at by the start of the time step.

In order to create a model that incorporates some of the variation present in nature we used normally distributed random numbers for both the current speed and current direction. By introducing this randomness we avoid to some extent to end up with a very specific case scenario that would be less valuable for generalizations. In theory, the model can be built with very complex current patterns (see Figure 4): pathogens inherit the current direction of the place they are presently on, and by moving to a new place they change the direction to the direction inherited from that place. In our Nelog model, current angle was set directly inwards in the fjord. During each time step a random deviance is added to this current angle. This randomness is given by two parameters: A current standard deviation (*Current_heading_std_dev*) which can be set from 0 to 90 degrees and a bias term (*Current_heading_bias*) used to offset the direction given by the patch², which can be set between -5 and 5 degrees. When moving the pathogen might hit dry land. In this case the pathogen is removed from the model (dies).

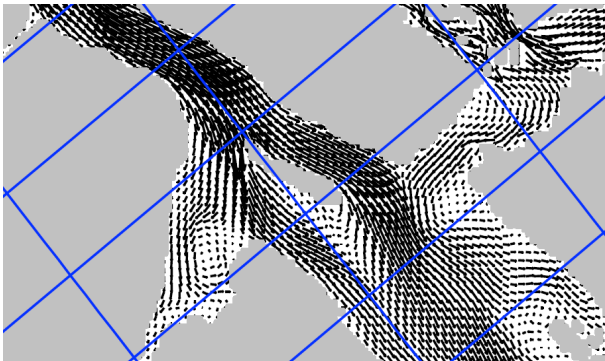


Figure 4: Currents direction in Romsdalsfjorden, MODS (2012).

The current speed is decided by the parameter *Current_speed*, and can be set from 0 to 2 m/s, with a default of 0.2 m/s. The current speed it is also accompanied by a random variation term, given by the parameter *Current_std_dev*, which can be set between 0 - 0.1 m/s. As we expect that current speed is not constant through time and space, this random term is used to make the model more realistic. Likewise the current speed can also depend on the individual patch. This is useful in order to model how current speed varies with the geometry of the fjord (e.g. changes in the

width of the fjord, the presence of islands and peninsulas etc.). This aspect is incorporated by adding a constant to each patch, *the relative speed*. Relative speed is a number by which the global current speed is multiplied for each patch. Hence, a relative speed below one indicates a speed below the average in that particular patch. Likewise, a relative speed greater than one indicates a speed higher than the average.

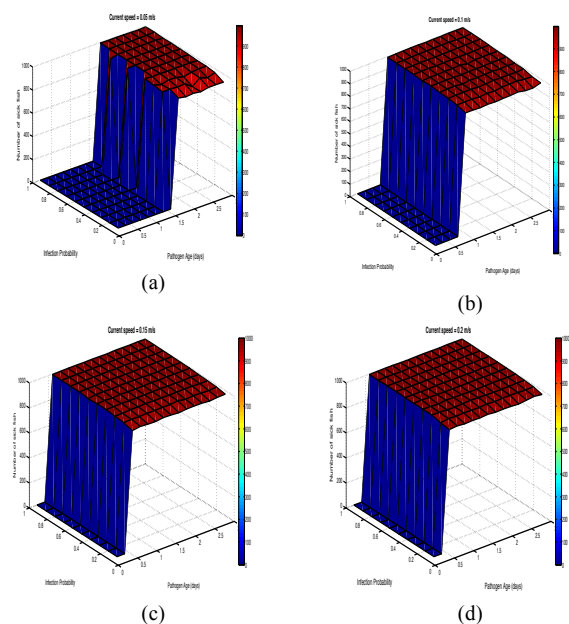
The pathogens are given a fixed life span in the range of the values we found in literature. In the model this parameter can be set between one and ten days, with a resolution of 0.1 day. At each tick all individual pathogens' ages are updated by adding 10 minutes (corresponding to $10/24/6 = 0.069$ days) to their cumulative age. When a pathogen's age exceeds the value given by the age limit it dies.

RESULTS OF THE EXPERIMENT

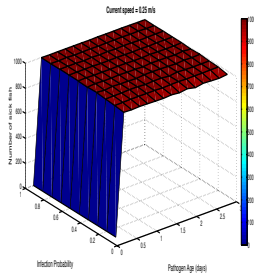
A simulation experiment was set up for the model. The model was run for 400 time steps, and three of the model parameters were varied in the following manner:

- Current speed: 0.05 to 0.25 m/s, at steps of 0.5 (5 values)
- Infection rate (Pathogen infect a fish): 0.1 – 1, at steps of 0.1 (10 values)
- Pathogen age: 0.2 – 3, at steps of 0.2 (15 values)

The simulation experiment output was the number of infected fish in each of the three fish farms (Farm 1, Farm 2 and Farm 3) at each time step. Running the experiment by using normal machine took around 15 hours, but to run it by using super-computer with 6 processors took around 5 hours. Figure 5 (a,b,c,d and e) shows the number of infected fish in Farm 2 after 400 time steps. The interpretations of the figures follow in the next section.



² Patches represent the grids in the landscape in NetLogo.



(e)

Figure 5: Number of sick fish (z-axis) in Farm 2 after 400 time steps, with current speed set at five different values (0.05, 0.1, 0.15, 0.2 and 0.25 m/s), and infection probability (x-axis) and pathogen (y-axis) varied.

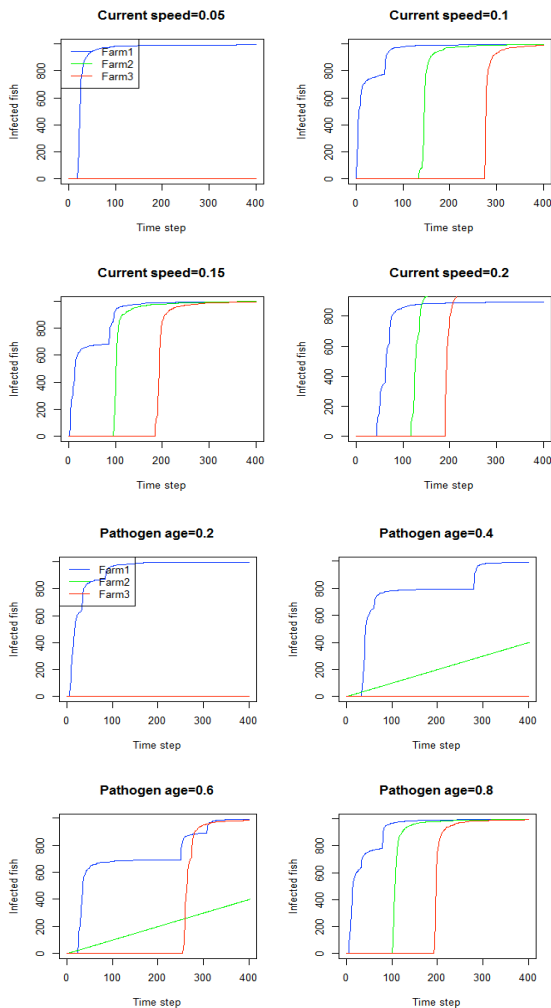


Figure 6: Number of infected fish per time step in the three fish farms (blue line=Farm 1, green line=Farm 2, red line= Farm 3). For the four graphs on the top infection rate and pathogen life span were held constant at 0.5 and 2, respectively and current speed was varied at four different levels. For the four graphs down pathogen age was varied, while infection rate was set at 0.5 and current speed=0.15 m/s.

DISCUSSION

As described above, the aquaculture industry is increasingly important for both the Norwegian economy and people's marine food availability worldwide. A major challenge for aquaculture development is the emergence of diseases, which can be due to a large variety of pathogens. It is therefore highly relevant to study how pathogen transmission between aquaculture sites can vary due to factors such as currents and pathogen virulence. Using an ABM we were able to simulate both fish and pathogens as individual agents and specifically study the movement of pathogens in a simplified fjord system. ABMs have to our knowledge never been employed in this type of study before. Although the model is simplified, we believe that it provides some insight into the transmission of pathogens between fish farm sites.

The simulation output

Current speed was the most important parameter controlling the number of infected fish in the aquaculture sites. The age limit of the pathogens was also important for the number of sick fish in Farm 2 and Farm 3. In fact, as long as the pathogen age was above a certain threshold, all fish in the fish farms would ultimately become infected. When current speed was held constant, the infection rate also had a significant impact on the number of sick fish in Farm 2 and 3, and for Farm 1 for low current speeds (<0.2).

The minimum pathogen age required to get infection in Farm 2 within 400 time steps, and at the lowest current speed we tested (0.05 m/s), was from 1.4 to 1.6 days, depending on infection probability. For Farm 3, the minimum current speed needed in order to get infection within 400 time steps was 0.1 m/s. At this current speed, the minimum pathogen age limit was 0.8 days. With this combination of current speed and pathogen life span the infection process occurs in Farm 2, which is subsequently sending new pathogens to Farm 3. If the current speed maximized to 0.25 m/s, only a lifespan of 0.2 days is needed in order to get infection in Farm 2, and subsequently in Farm 3. I.e., at current speeds above 0.1 m/s the dynamics of Farm 2 and 3 are similar, as Farm 2 acts as a new source of pathogens for Farm 3, and the distances are the same.

Scaling the model

If we were going to represent a realistic number of agents (fish and pathogens) the model simulation would be very computationally demanding. In Norway today, aqua farms can host fish in the order of millions (DF 2013) and for pathogens the numbers would be uncountable, in the order of trillions or higher, depending if we are studying bacteria or virus. In order to be able to run the model we therefore had to scale it down significantly. Even so, the relative magnitude between the number of fish and pathogens is likely

incorrect, with the number of pathogens underestimated. To compensate for this, the probabilities of infection and pathogen release should be adjusted accordingly, meaning that they should be set higher than what might be expected. A challenge in this approach is that these parameters are largely unknown. Another issue arising from scaling down the model is that the pathogens might less easily “hit” the aqua farms, since there are likely fewer pathogens in the system than what would be realistic. It is harder to compensate for this issue by adjusting the probability of infection, since infection requires the actual presence of the pathogen. Increasing the probability of pathogen release would, on the other hand, compensate for this issue by creating more pathogens in the system. Another solution could be to make the aqua farms bigger (scaling up their size) in order to increase the probability of an encounter between the pathogens and the sites.

The matter of scaling thus leads to many challenges for creating a model that represents the real world. We still lack good solutions to many of these challenges, but we are aware of their existence and the results of the model simulations should accordingly be interpreted with this in mind. In essence, our simplified model of the Romsdalsfjord is aimed at studying effects that may occur, but it is not appropriate to make any numerical predictions.

Simulating pathogen transmission and infection

The most challenging part of the modeling process was to simulate the pathogen transmission process, due to lacking empirical data on these issues in the literature. There are several uncertain factors regarding the process of transferring illness between fishes. To create a realistic model for the infection process is therefore difficult, and our approach was to model a simplified scenario that can later be tuned to reproduce results observed in fish farms. Moreover, the few studies that have quantified the rates by which pathogens are shed by infected fish use different units which are not single pathogens, but units that are measureable in the lab or practical for disease monitoring (Salama & Murray, 2011). It was not straightforward to convert these values into probabilities, and we therefore had to experiment to find appropriate values. The probability of pathogen infection was varied during the simulations, while the shedding rate (Fish release pathogen probability) was kept constant (0.05). This was done in order to facilitate the interpretation of the results, and because we lacked any references as to what would be realistic values for the latter parameter.

CONCLUSIONS AND FUTURE WORK

In this ABM, we have simulated three fish farms in the Romsdalsfjord. The number of infected fish in each farm is predicted by factors such as current speed and pathogen life span. The results provide a good base for exploring the relationship between these variables and others (infection rate, pathogen release rate current bias etc.), and platform on which more complexity can be added to the model at a later stage. Such possible complexities are described hereunder.

Pathogen transmission

In reality, the proliferation of a pathogen can be highly dependent on environmental conditions such as temperature, and thus vary with seasonal and inter-annual environmental changes (Krkosek 2010). Also, the virulence of the pathogen and the conditions of the host (health, age, stress etc.) will affect the likelihood of an outbreak (Rimstad 2011). At this stage, we have only modeled a short disease proliferation period, and therefore assumed temperature to be constant. A next step could be to incorporate a dynamic temperature variable in our modeled fjord which would affect infection probability and pathogen release.

In this model, we focused on the distance between aquaculture sites, but not their individual sizes. But water-transmitted pathogens can also benefit from the size of the aquaculture site. A next step for our model could be to vary both distances and sizes of the fish farms. This could be interesting for managers deciding upon which areas would be appropriate in order to avoid disease transmission between fish farms.

Currents modeling

In our model, we have assumed the currents speed and direction act according to MODS (2012), and depend on what is written in the literature. We have assumed that current direction is inwards in the fjord and changes between 0.05 to 0.25 m/s in our experiment (at Midsund, *see figure 3*). We have selected these values by analyzing the geographic of the fjord (at Midsund) and results from MODS (2012). But in the reality, the current speed and direction are more variable and depend on many other factors, as season, snow melting rate, and the geographic of the fjord. The model would become more realistic if it was connected to a current model of the fjord.

Fish movement

Although the transmission through water is the most important route for marine pathogens, a further step could be to incorporate the movement of the farmed fish, with a certain probability of escaping (and which could be infected or not). Fish escapees can in fact pose a risk to the wild population through spreading of diseases (Naylor *et al.* 2005), a risk that increases when farmed fish are in the vicinity of wild populations of the same species and the farmed population contributes a large quantity compared to the wild fish.

Another interesting, but challenging, possibility would be to include wild fish agents. A study on the distribution of saithe within a fjord with salmon aquaculture showed aggregations around aquaculture facilities and a high proportion of fish moving between different farms, indicating that the wild fish might constitute an important connection between fish farms (Uglem et al. 2009). The fish might aggregate around the farms to feed on waste pellets under or around the nets. A range of pathogens is thought to be common to salmon and saithe, but the likelihood of transmission between caged and wild fish is unknown.

Altogether, the model presented here offers an interesting first step towards more complex models of disease transmission between aquaculture sites, an important research issue for the aquaculture industry.

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