Aquaculture 505 (2019) 98–111

Contents lists available at ScienceDirect

Aquaculture

journal homepage: www.elsevier.com/locate/aquaculture

An agent-based approach for predicting patterns of pathogen transmission between aquaculture sites in the Norwegian fjords

Saleh Alaliyat[a,b,*], Harald Yndestad[a], Pål I. Davidsen[b]

[a] Norwegian University of Science and Technology, Aalesund, Norway
[b] University of Bergen, Bergen, Norway

ARTICLE INFO

Keywords:
Agent-based method
Aquaculture
Fish disease dynamics
Pathogen transmission
Netlogo

ABSTRACT

The aquaculture industry is a main industry in Norway, and it must be sustainable, i.e. experience long-term growth and development: It is necessary to build an environmentally sustainable aquaculture industry that minimizes risks to the marine environment and biological diversity, – including the transmission of fish diseases. The process of fish disease transmission in aquaculture systems is influenced by many factors, including individual (fish and pathogen) conditions, movement behavior and environmental conditions. Fish disease dynamics originates from a complex system, and the transmission of viruses is an unstable process, making it difficult to predict and analyze. In preparation for this paper, we built an agent-based model to predict patterns of pathogen transmission with the purpose of identifying risks and hazards in the space and time domains. The model presented explores the potential effects of different factors, such as the conditions of agents, movement behavior and environmental conditions, on the simulated spread of a fish disease.

We applied the model developed to different case studies in the Norwegian fjords. The results demonstrated how the infection risk at any point around the infected site is dependent on both the pathogen and the fish density at that point, and the infection risk increases when the pathogen or fish densities increase. The pathogen density decreases exponentially as a function of an increase in the water temperature, and the pathogen density increases with the velocity of the current or the fish density at the infected site. The pathogens are moved faster by higher current velocity, so this will slow the infection process at the local infected site. Nevertheless, the current will carry the pathogens to nearby places faster. The direction of the current is very important since the pathogens are predominantly moved by the currents.

The agent-based method helps us advance our understanding of pathogen transmission and builds risk maps to help us reduce the spread of infectious fish diseases. By using this method, we may study the spatial and dynamic aspects of the spread of infections and address the stochastic nature of the infection process.

1. Introduction

Fish farming in Norway has increased steadily in recent years and is expected to continue to increase for years to come (www.ssb.no). The continued growth of Norwegian aquaculture production has presented the industry with a range of challenges. One of the main challenges is to understand fish disease dynamics within and between the aquaculture sites in the Norwegian fjords, characterized by a rich marine life and considerable human activities. Fish are subjected to diseases caused by different types of pathogens.[1] Pathogens are transmitted in space and time by sea currents at an irregular velocity (speed and direction). The sea currents in the Norwegian fjords exhibit a complex pattern of behavior, as shown in Fig. 2. Pathogen transmission is dependent on many different biological, environmental, and physical factors. Due to the complex relationships that exist between these different factors and the way they change in time and space, (e.g. fluctuating sea-water temperature alter pathogen's lifespan and its ability to cause a disease), other approaches previously applied to study this issue, that are not including active parts (agents) to model this complex dynamics relationships, they have not successfully reduced the ambiguity in our understanding of how pathogens spread in the Norwegian aquaculture system. Therefore, we need a method that allows us to address this ambiguity so as to limit the risk of fish disease spreading. In this study, we will use an agent-based approach in building models that predict patterns of pathogen transmission for the purpose of identifying the risks and hazards in the space and time domains. It is expected that this

* Corresponding author at: Norwegian University of Science and Technology, Aalesund, Norway.
E-mail address: alaliyat.a.saleh@ntnu.no (S. Alaliyat).

[1] A pathogen is anything that causes a disease.

https://doi.org/10.1016/j.aquaculture.2019.02.044
Received 8 December 2018; Received in revised form 11 February 2019; Accepted 18 February 2019
Available online 19 February 2019

risk assessment will inform the fish industry management in Norway in their fight against infectious fish diseases.

1.1. Aquaculture in Norway

In recent decades, the aquaculture industry has probably been the fastest growing food-production sector in the world, and it provides a significant supplement to, and substitute for, the catch of wild aquatic organisms. Norway has a long and jagged coastline that is bordered by cold, fresh seawater endowed with a rich marine life. This environment provides excellent conditions for aquaculture activities. Today, Norway is the second largest seafood exporter in the world and the world's leading producer of Atlantic salmon (FKD, 2018). Since the advent of commercial salmon farming in Norway around 1970, the aquaculture industry has grown to become an industry of major importance. Not only is aquaculture important to the Norwegian economy as a whole, it is also very important to the many local communities along the coast where other economic opportunities are sometimes limited. Today, farming of salmon and rainbow trout takes place in nearly 160 municipalities along the Norwegian coast. Approximately 5900 people are directly employed in aquaculture production, and 21,000 people are employed in aquaculture-related activities (FKD, 2018; SSB, 2018).

Emerging diseases pose a serious challenge to the aquaculture industry, and the value of the fish that are lost due to disease is worrisome (Grefsrud et al., 2018). Ten years ago, Iversen et al., 2005 assessed the general cost of such diseases imposed on the Norwegian fish farming industry to be US$ 150 million annually (Iversen et al., 2005). Fish are subjected to diseases caused by pathogens, including viruses, and these pose particular challenges to the salmon aquaculture (Olsen and Hellberg, 2011). Our knowledge of pathogens and their effect on wild fish stocks is generally poor, and it is, consequently, difficult to predict which diseases might occur once an aquaculture facility is established in an area (Bergh, 2007). A wide range of pathogens exist, from viruses and bacteria to crustacean parasites (Olsen and Hellberg, 2011). These pathogens might be introduced to an aquaculture system through various pathways, e.g.; through the relocation of infected stocks, by the use of equipment or fish products from other areas, or by exposure to wild fish pathogens (Murray and Peeler, 2005). Once introduced, pathogens may benefit from the aquaculture environment and pose a graver risk to farmed fish than they do to wild stocks. This is partially because of factors such as unfavorable environmental conditions, stress and pollution, which might reduce the resistance of individual fish (Murray and Peeler, 2005). Moreover, pathogens may benefit from the artificially high density of fish, and thus, the numerous potential hosts of the pathogen that are present in a fish farm and thus cause frequent and massive disease outbreaks (Bergh, 2007; Rimstad, 2011). Pathogens that benefit from higher host densities cause a so-called *density-dependent transmission* (Murray, 2009). The rate of transmission is the product of the densities of susceptible and infected individuals. Disease transmission may also occur with currents as carriers, − depending on the survival time of the pathogen in the water masses, and also through vectors such as wild fish or escaped farmed fish (Murray and Peeler, 2005). Hydrodynamic spreading will usually be a local-scale problem, whereas wild fish can become infected near a single farm and transmit the pathogen over larger distances to other farms (Werkman et al., 2011). An example of a waterborne virus is the salmonid alphavirus that causes salmon pancreas disease (PD), that has turned out to be an increasing problem in Norwegian aquaculture (Kristoffersen et al., 2009).

All major viruses affecting Norwegian aquaculture are thought to spread between fish through seawater (Johansen et al., 2011), as a result of infected fish shedding pathogens into the surrounding waters. To retain a sustainable fish industry in Norway, we need tools for effective risk analyses and consequence assessments. In this paper, we aim at developing models to help identify the pathogen transmission patterns between fish populations so as to support such analyses and assessment in the combat against fish diseases.

Previous Norwegian studies on fish diseases in aquaculture have used classical SIR (susceptible, infected, recovered) disease transmission models that have focused on the population as a whole (Reno, 1998; Ögüt, 2001; Murray, 2009; Green, 2010) or such population models coupled with either simple hydrodynamic models or distance measures of transmission between separate populations (Stene et al., 2014). These models are inherently limited in their ability to predict the dynamics of diseases because they are based on structural assumptions and historical data that do not offer a valid description of the system at hand. They, consequently, do not offer an adequate explanation for the complex dynamics observed. In particular, they do not capture the phenomenon of emerging diseases, i.e. the onset of a disease in an aquaculture farm.

Fish disease dynamics are affected by many variables that modeling techniques, applied so far, cannot address; however, the agent-based modeling (ABM) technique can include all necessary variables to build a valid model even if there is a lack of available empirical data. By using ABM, we move to the individual's level and how the individuals' characteristics and their behavior are connected to the overall system behavior.

1.2. Related work

Fish disease dynamics are affected by many different biological, environmental and physical characteristics, such as fish density and stress, water temperature and salinity, as well as current speed and direction. This constitutes some of the complexity to be addressed when modeling these processes. Fish disease dynamics within an aquatic site or between many such sites is itself a part of such a complex environment in which it evolves. Earlier modeling studies on the transmission of pathogens within and between aquaculture farms have mainly been based on mathematical models that focused on the population as a whole (e.g., Murray, 2009; Green, 2010). Kermack and McKendrick were pioneers in establishing the mathematical modeling of disease epidemics in 1927 (Kermack and McKendrick, 1927). They created the mathematical SIR (susceptible, infectious, recovered) model, based on ordinary differential equations. That model includes the assumptions that all fish are homogeneous, initially equally susceptible to the disease, and completely immune after having been infected. The SIR models do not treat the pathogens as separate individuals who may survive without a host, and they do not include the environmental conditions that may change over time. SIR models are simple and, typically, deterministic and do not validly represent some important aspects of disease spread, including the variety in properties across in the individuals, the spatial aspect of the spread of disease and the characteristics, including causes of delays, of the environment in which this spread takes place. Many researchers have, over the years, applied a variety of such mathematical models to simulate disease dynamics. Some studies have coupled such models to simple hydrodynamic models and to distance measures of transmission between separate populations (Viljugrein et al., 2009; Aldrin et al., 2010; Werkman et al., 2011; Salama and Murray, 2011).

Hydrodynamic models, combined with particle tracking and statistical analyses, have been widely used in Norway to identify the salmon louse and pancreas disease (PD) transmission dynamics in Norwegian fjords (MODS, 2012; Stene et al., 2014). SINMOD is the most famous hydrodynamic model in Norway (www.sinmod.no), and it couples physical and biological processes in the ocean. Hydrodynamic models are based on the assumption that the pathogen agents drift passively with the sea currents. Hydrodynamic models do not incorporate the effects of the surrounding nature (e.g. sea-water temperature change) on the pathogens, and the heterogeneity among the pathogens is being ignored. Also, the statistical analyses are based on the assumption that the fish populations are homogeneous.

Cellular automata (CA) theory has also been used for modeling the

dynamics of infectious disease spread (Sirakoulis et al., 2000; Zhen and Quan-Xing, 2006; Kocabas and Dragicevica, 2006), but the individuals movements and interactions across space over time have not been represented in such models.

Agent-based methods have been applied to simulate the transmission of human viral diseases such as influenza (Ciofi degli Atti et al., 2008; Milne et al., 2008; Khalil et al., 2010; Arduin et al., 2017; Venkatramanan et al., 2018; Yang, 2019). In this project, we have applied the agent-based method to simulate disease dynamics in a fish population (Alaliyat and Yndestad, 2015b), but we did not extend the model to simulate how pathogens spread between aquaculture sites in the fjord. Agent-based models (ABMs) can be valuable in analyses focusing on the effects of individual interactions, and they may incorporate the spatial aspect of a system. Whereas the classical SIR models, used in classical disease transmission modeling, represent total populations, in this study, an ABM approach is applied to simulate the infection process of the individual fish, the movement of fish in the cages, and the way that pathogens spread spatially, in the form of individuals, by representing fish and pathogens as agents. ABMs are computationally costly compared to other models, and the costs increase exponentially with the number of individuals included in the model.

1.3. The modeling approach

The main aim of this study was to develop an agent-based modeling approach for studying the dynamics of fish diseases within and across aquaculture sites in the Norwegian fjords. This approach considers the interactions between individuals' (fish and pathogens) and with their environment in a space-time context and is expected to advance our understanding of the disease dynamics process and help combat such a development. The process of disease transmission is influenced by many factors, including the conditions of the individuals (fish and pathogen), movement behavior and environmental conditions. The model presented explores the potential effects of these factors on the spread of a simulated fish disease.

ABM provides a realistic representation of the system by including the interactions of individuals. In addition, ABM offers more flexibility in the modeling and allows for more complexity to be added and analyzed by way of simulation. Another reason for applying ABM is to compensate for the lack of empirical data regarding fish disease transmission. By using ABM for predicting pathogen transmission, a simulation of future disease transmission scenarios could provide a means to compensate for this lack of empirical data. In ABMs, the values of the parameters governing the disease transmission may easily be varied (Alaliyat et al., 2013; Alaliyat and Yndestad, 2015a; Alaliyat and Yndestad, 2015b).

ABM has been suggested in different fields as one of the most appropriate approaches to modeling and simulation when addressing complex, dynamic system. ABM captures the complex network of interactions and interconnections that comprise real systems and makes it possible to derive emerging dynamic patterns, unexpected changes in those patterns and events characterizing such patterns. This makes such a bottom-up approach advantageous in simulating the spread of pathogens in aquaculture systems. ABM provides insights into the structural origin of emerging phenomena that are caused by the interactions among individuals (pathogens and fish). Using ABM, one may describe how fish and pathogens behave rather than develop equations that we believe govern the overall dynamics of the densities and infection rates of system entities. Reality is transparet in the model by using ABM. ABM is flexible in that it allows for the addition (and elimination) of agents and for adjustments in the agent behavior. ABM provides a framework for analysis and testing of the emergent dynamics. ABM provides a flexible framework for answering questions, such as what is happening, what will occur next, or identifying what the best/worst outcome might be. Thus ABM may serve well as modeling technique with the purpose to predict pathogen transmission between aquaculture sites.

In this study, we focus on the Romsdalsfjord.[2] This fjord has been selected because of the extensive empirical research that has been undertaken at this site, Thus, we had access to the data we needed for our model building, validation and simulation. The Romsdalsfjord is a semi-closed fjord in mid-Norway that has a massive fish industry with > 35 aquaculture sites throughout the fjord (Fig. 1). The aquaculture industry data, including biological, physical and environmental data, reflects the aquaculture system in the Romsdalsfjord, and our proposed model either used this data or was inspired by this data.

The close proximity between aquaculture sites in the Romsdalsfjord is an important factor in disease transmission. Consequently, it becomes very important to study the environmental, biological and physical conditions of an infected aquaculture site because from that site pathogens may spread to other aquaculture sites by sea currents, − creating a domino effect. The sea currents in the Romsdalsfjord exhibit a very complex pattern (Fig. 2). The currents in fjords are the strongest and most varying in the upper 20 m (closest to the surface), i.e. where the aquaculture farms are located. The currents are driven by topographical distinctions, river runoffs, winds, tides and water exchanges caused by offshore density differences (Urke et al., 2011; Stene et al., 2009). In this work, we built models to predict the patterns of the spread of pathogens from infected sites. This has enabled us to build risk maps that depicted the hazardous areas around infected sites in which diseases may be transmitted to neighboring sites.

We have built three different simulation scenarios to explore the potential effects of fish, pathogen and environmental factors on the spread of a simulated fish disease in the Romsdalsfjord. The first simulation experiment had only one hypothetical infected fish farm in an open area, and the sea current moved from the west to the east (i.e., left to right). In this scenario, we ignored the topography of the fjord, but we focused our investigation on the risk-values and -maps that resulted from the pathogen's density in space, over time, and on the largest distance that pathogens could spread. In the second scenario, we added a second hypothetical fish farm to the previous simulation map and tested the effects of various parameters (e.g., sea temperature, current speed, current direction, biomass) on the spread of the infectious disease from the source to the destination. Moreover, we simulated the on-site disease dynamics in this scenario. In the third scenario, we simulated real aquaculture systems with three sites, one of them is assumed infected site. We included the topography of the Romsdalsfjord and the aquaculture industry data in the simulations. The purpose of this scenario was to test the effects of various parameters on the spread of an infectious disease from a source to several destinations, to simulate the disease dynamics in the destinations and identify how the destination sites will become source sites as well (nested).

2. Materials and methods

2.1. Materials

The data used in this study can be categorized into four main types: aquaculture, geospatial, oceanographic and disease data. The aquaculture data included the aquaculture site name, location, operator, maximum allowed capacity, type of production and the farm's current production state.

The aquaculture data are available online on the Norwegian Directorate of Fisheries' webpage, http://www.fiskeridir.no/. The geospatial data utilized consists of three-dimensional (3D) maps of the Romsdalsfjord. We obtained 3D maps that included terrain and bathymetry data from the Norwegian Mapping Authority (NMA, 2016).

[2] Romsdalsfjord is 88 km long and located in the Romsdal district of Møre og Romsdal county in mid-Norway.

Fig. 1. Map of the aquaculture farms in the Romsdalsfjord.

The terrain had a resolution of 10 m × 10 m, while the bathymetry had a resolution of 50 m × 50 m. The oceanographic data included data on the sea currents (i.e., speed and direction), sea temperature and salinity. In our simulations, we used the monthly average sea current data from the SINMOD model (MODS, 2012) with 800 m resolutions, and we added some noise to emulate the natural variability. The seawater temperature and salinity data are available online on the Institute of Marine Research webpage, http://www.imr.no/en/. Similar to the data on currents, we used the monthly average data and adding some noise. The water temperature was varied in the water column as well (Alaliyat and Yndestad, 2015b).

The fish disease data utilized includes fish health, pathogen biology data and disease transmission factors. Wide ranges of pathogens exist, from viruses and bacteria to crustacean parasites. The infection and shedding parameters and the pathogen life span are dependent on the type of pathogen and the type of host. These data are characterized by uncertainty and have, for the most, been derived from laboratory experiments (Salama and Murray, 2011; Stene et al., 2014). In our simulations, these values varied between different values.

To implement the models using agent-based methods, NetLogo 3D was used. NetLogo is a multi-agent programmable modeling environment. The NetLogo toolkit allows for simulations within a geographic information system environment, and it is easy to include physical and environmental data (Wilensky, 1999). We used MATLAB to analyze the simulation results and create figures that were easy to interrupt (MATLAB, 2015). We used GlobalMapper (Bluemarblegeo.com) to build 3D maps of the Romsdalsfjord by combining the terrain and bathymetry data, and we removed the noise from the data and rescaled the maps to fit in NetLogo.

2.2. Methods

2.2.1. The system model

In this study, we simulated fish disease dynamics and pathogen transmissions in a Norwegian fjord aquaculture system. The aquaculture system has a set of fish farms, a swarm of pathogens and the landscape. This system $S(t)$ can be formalized as shown in Eq. (1).

$$S(t) = \{FF(t), P(t), L(t)\} \quad (1)$$

where $FF(t)$ is a set of fish farms, $P(t)$ is a swarm of pathogens, and $L(t)$ is the landscape or the environment where the previous components are located (Yndestad, 2010). The purpose of this study has been to investigate how a swarm of pathogen $P(t)$ that was produced by a hypothetical, infected fish farm (initial producer farm) will flow with the current and spread in a given landscape $L(t)$. The fish are producer-consumer agents; they produce pathogens, and at the same time they consume pathogens in the fish disease process (Yndestad, 2010).

2.2.1.1. The landscape L(t). The landscape $L(t)$ is divided into four overlaying sub-landscapes and can be formalized as shown in Eq. (2).

$$L(t) = \{L_{tr}(t), L_{cu}(t), L_{sa}(t), L_{tm}(t)\} \quad (2)$$

where $L_{tr}(t)$ represents the terrain, $L_{cu}(t)$ represents the map of the sea currents, $L_{sa}(t)$ represents the map of the sea salinity, and $L_{tm}(t)$ represents the map of the sea temperature.

In this study, the terrain $L_{tr}(t)$ covers part of the Romsdalsfjord area. The terrain is divided into many 3D grids with pixels of 13 × 13 × 13 points. The sea current landscape, $L_{cu}(t)$, represents the speed and direction of the sea currents. The Romsdalsfjord has very complex current patterns (*see* Fig. 2). The sea currents are driven by a variety of factors that are changing massively in time and space. Therefore, in order to create a model that incorporates some of the variation present in nature, we use normally distributed random numbers for both the current speed and the current direction. The user can set the average current angle and speed at the beginning of the simulation, and then, at each time step, a random deviance is added to these values for current angle and speed. For the current angle, this randomness is characterized by two parameters: a current direction standard deviation, which can be set from 0 to 90°, and a bias term used to offset the direction given by the grid, which can be set between −5 and 5°. For the current speed, this randomness is also characterized by two parameters; a current speed standard deviation, which can be set between 0 and 0.1 m/s, and a relative speed, which is associated with each grid and depends on the geometry of the fjord (e.g., changes in the width of the fjord, the presence of islands, and peninsulas). The relative speed is the number by

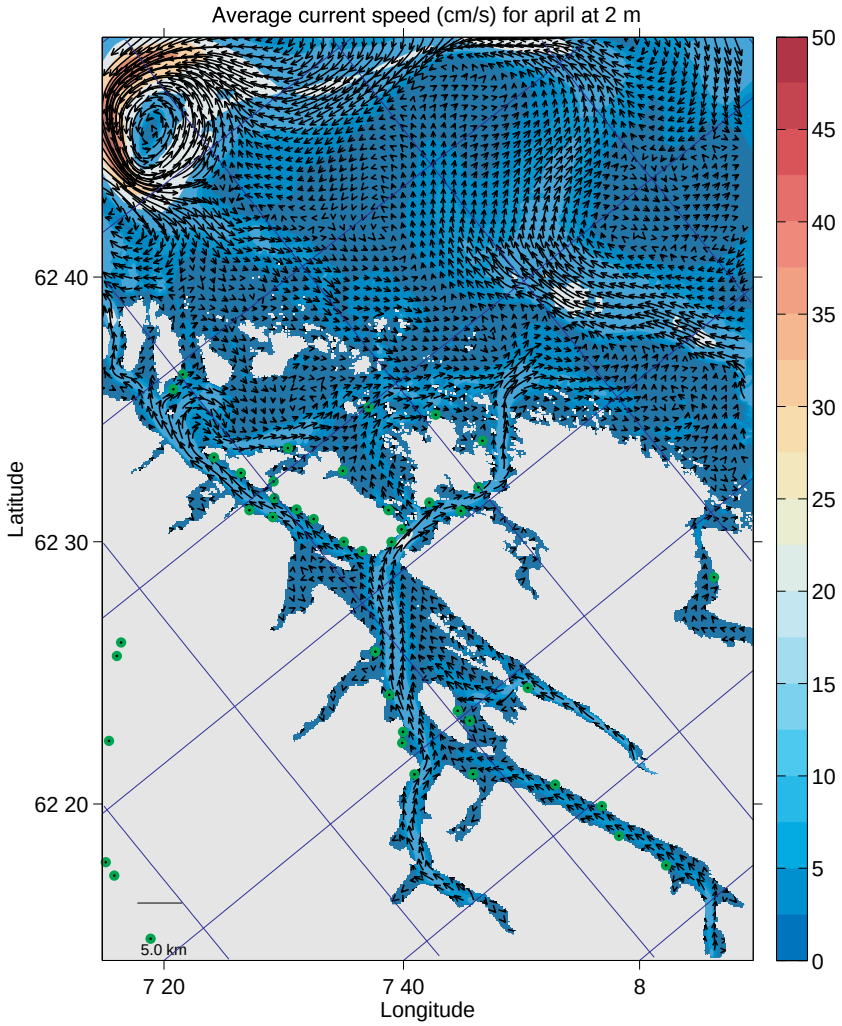

Fig. 2. Current speeds and directions in the Romsdalsfjord, MODS (2012).

which the global current speed is multiplied to obtain the speed for each grid.

The sea salinity, $L_{sa}(t)$, and sea temperature, $L_{tm}(t)$, landscapes are changing in time and space. The chosen temperature profile from January to December at the surface level is $L_{tm}(t)$ = {5.7, 5, 5.1, 6.1, 8.1, 11.3, 12.7, 15.5, 14.0, 11.2, 9.5, 8.0 (°C)} (www.imr.no). The user can set the average water temperature and salinity at the surface level, and then we add some noise to include the variation that is present in nature. The water temperature also varies in the deep levels, as shown in Eq. (3).

$$\overrightarrow{temp}(x,y,z) = \overrightarrow{temp_0}(x,y,z) - C * \overrightarrow{L}(y) \quad (3)$$

where $\overrightarrow{temp}(x,y,z)$ is the water temperature at *x,y,z* grid; $\overrightarrow{temp_0}(x,y,z)$ is the water temperature at the surface level ($y = 0$); C is a constant; and $\overrightarrow{L}(y)$ is the water depth level.

2.2.1.2. The fish farm $FF_k(t)$. Each fish farm, $FF_k(t)$, has a swarm of fish agents, $FA(t)$, and is represented by $3 \times 3 \times 3$ grids that are all assumed equal to 3*20 m × 3*20 m × 3*20 m. Additionally, the grid outside the aquaculture sites measures 200 m × 200 m × 200 m. In this study, the farms' positions are set hypothetically in the first two simulation scenarios, while they are based on real aquaculture data in the third scenario. The swarm of fish agents has some social rules that manage the individual movements in the swarm, consumes pathogens and produces pathogens.

2.2.1.3. The pathogens swarm P(t). The pathogens swarm consists of many individual pathogens, as shown in Eq. (4).

$$P(t) = \{PA_1(t), PA_2(t), ..., PA_n(t)\} \quad (4)$$

where $PA_j(t)$ is the pathogen agent j, and n is the total number of pathogens at time t.

In reality, the swarm of pathogens also has social rules (e.g., move together and align with one another), and the swarm relates to the landscapes to facilitate the individual pathogens' movements (Reynolds, 1999). In this study, however, we have ignored the social rules, while, as we will see in the next section, the pathogens' dependence on the landscape steer the movements of individuals.

Table 1
Agents in the model.

Agent type	Attributes	Behavioral rules
Fish	• Position • Health status • Energy • Vaccinated	• Update position • Update health status • Update energy (resistance factor) • Shed pathogens
Pathogen	• Position • Life span • Ability	• Update position • Update life span • Update ability

2.2.2. Agent-based model

The agent-based approach is applied to simulate fish disease dynamics and pathogen transmission in a fjord aquaculture system. Table 1 shows the agent-based model's agents. We have two types of agents; fish and pathogen. Each agent has many attributes and behavioral rules that update the values of these attributes in the context of time and space.

We designed our model in a rectangular shape with 501 × 201 × 3 patches (a patch is a grid in NetLogo). Each fish farm has a maximum population of 1000 fish; however, you would expect to see approximately 1000 times more fish in an actual fish farm of this size. This simplification was made to save computer resources while running the model. A tick is the time step in the model, and it can represent 10 min, one hour, or one day (it can be selected by the user, *time-step*).

2.2.2.1. The fish agent FA(t). The fish are located in the farms, and each fish farm $FF_k(t)$ has a swarm of fish that is composed of many fish agents, *FA(t)*, as is shown in Eq. (5).

$$FF_k(t) = \{FA_1(t), FA_2(t), \ldots, FA_n(t)\} \tag{5}$$

1: **For each** susceptible fish agent i…**then**
2: **If** $(RF_i * \sum_{p_j\, in\, r}(p_j * ab_j) \geq T_0)$,
Where
RF_i is a fish i resistance factor, p_j is any pathogen j in r distance from fish i, ab_j is the infection ability of pathogen j, and T_0 is a selected threshold.
3: get infected
4: **End if**
5: **End for each**

Unlabelled Image

Fish agent $FA_i(t)$ has several attributes and behavioral rules that update these attributes (see Table 1). In this context, we are interested in the position, energy, and health status attributes, as well as the behavioral rules that governs the values taken by these attributes and that produce pathogens.

2.2.2.2. Fish swimming rules. Fish agents swim within cages, and since we use a large time step in our simulations in this study (i.e., 10 min or one hour), the fish's positions are updated randomly at each time step, as shown in Eq. (6).

$$\overrightarrow{FA_i}(x,y,z,t+\Delta t) = \overrightarrow{R_i} * \left(\frac{max_{x,y,z} - min_{x,y,z}}{2}\right) + \overrightarrow{FF_k}(x,y,z,t) \tag{6}$$

where $\overrightarrow{FA_i}(x,y,z,t+\Delta t)$ is the fish position vector, $\overrightarrow{R_i}$ is a unit random vector in 3D, $(max_{x,\,y,\,z} - min_{x,\,y,\,z})$ is the fish farm dimensions, and $\overrightarrow{FF_k}(x,y,z,t)$ is the farm position vector in the simulation space. Fish can swim in different formations, and they can socialize to form a school. We have previously investigated the effects of different swimming behaviors on infectious fish diseases (Alaliyat and Yndestad, 2015b). Since, in this study, we used a large time step, we have chosen to ignore the social rules, and assumes that the fish is distributed randomly in the fish farm.

2.2.2.3. Fish energy attribute. Each fish has an epidemic resistance factor (i.e. immunity to the disease), which is a value between 0 and 1. Fish immune system depends on different factors such as the vaccination process and doses of vaccine (Madonia et al., 2017). Out model is a fixable model which can includes computational models in fish vaccination such as the one introduced by Madonia et al. (2017) and other fish stress models to measure the immunity to the disease in fish agents.

In our simulations we assign reference values of 0.8 with some noise (to simplify the model), as shown in Eq. (7).

$$RF_i = RF_{ref} \pm R_i * N_{rf} \tag{7}$$

where RF_i is the resistance factor of fish i, RF_{ref} is the reference resistance factor value that can be set by the user, R is a random number in the range of [0,1], and N_{rf} is the noise value.

2.2.2.4. Infection rules. Fish agents are categorized into four main health states as in the SEIR (susceptible, exposed, infected and removed) model (Bjørnstad, 2005). In the following text, we will explain how the fish health state of individuals will be updated over time. A susceptible fish becomes infected if there are many pathogens around it, the pathogens have a good ability to infect, and the fish has a week *RF*, as determined by the *Algorithm I* procedure. Salama tried to quantify the infection probability, but his results depended on laboratory data (Salama and Murray, 2011). In our model, this probability can vary between different values, and it is also related to the densities of fish and pathogens.

Once the fish has been infected, it will leave the susceptible category and enter the exposed category. All fish agents transfer between the four health states. Therefore, the population of agents is divided into four groups or compartments consisting of individuals that are susceptible, exposed, infected and removed. The fish agents are heterogeneous, and each agent has its own individual discrete SEIR model. The contact rate in the SEIR model is equivalent to the individual fish infection rules in ABM. The fish agent health state in ABM is dynamically updated. The number of fish with the same health state provides the number of fish agents in the four groups.

The process for updating the fish health states at each time step is achieved by applying the health-state update method, shown in *Algorithm II*.

2.2.2.5. Pathogens production process. Each time step the infected fish, *i*, may shed a pathogen, *j*, at a certain probability where that fish is located. Different sources refer to different units, and values range from $10^{6.5}$ PFU/fish/h (PFU = plaque forming units) (Gregory, 2008), 10^5–10^8 CFU/fish/h (CFU = colony forming units) (Rose et al., 1989) and $6.8*10^3$ $TCID_{50}$ /ml/ kg fish/ h/ (maximum rates) ($TCID_{50}$ = the amount of virus required to kill 50% of infected hosts) (Urquhart et al., 2008). The units are not single pathogens; rather, they are 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 and 1 (adjustable) that an infected fish sheds a pathogen, but this pathogen represents a large number of pathogens, i.e. effectively a rate of pathogens per time unit.

```
For each fish agent…then
  Check health status
  If fish is susceptible then
    If there are a number of pathogens around the fish then
    1. Change fish state to exposed
    2. Die by normal death rate
    Else
      Die by normal death rate
    End If
  End If
  If fish is exposed then
    If fish passed exposed period then
    1. Change fish state to infected
    2. Die by illness death rate
    Else
      Die by normal death rate
    End If
  End If
  If fish is infected then
    Produce a pathogen by a given probability "shedding rate"
    If fish passed infected period then
    1. Change fish state to recovered
    2. Die by normal death rate
    Else
      Die by illness death rate
    End If
  End If
  If fish is recovered then
    If fish passed immune period then
    1. Change fish state to susceptible
    2. Die by normal death rate
    Else
      Die by normal death rate
    End If
  End If
End for each
```

2.2.2.6. The pathogen agent PA(t). The pathogens swarm *P(t)* consists of many individual pathogens, as shown in Eq. (4). The pathogen agent *PA*(*t*) has three main attributes (see Table 1): position in the space, ability to infect fish, and life span.

2.2.2.7. Moving rules. The pathogens are moved by sea currents. Each pathogen moves based on the current speed and direction, which is based on the location of the pathogen at the start of each time step. Pathogens inherit the current direction of the place they are presently located, and by moving to a new place, they inherit the direction of that new place. When moving, the pathogen might hit dry land. In that case, the pathogen is removed from the model (dies).

The pathogen *j* updates its position, as shown in Eqs. (8) & (9).

$$\overrightarrow{PA_j}(x,y,z,t+\Delta t) = \overrightarrow{PA_j}(x,y,z,t) + \overrightarrow{v(t)} * \Delta t \tag{8}$$

$$\| \overrightarrow{v(t)} \| = C_{sr} * R_n(C_s, std) \tag{9}$$

where $\overrightarrow{PA_j}(x,y,z,t+\Delta t)$ is the new pathogen agent *j* position, $\overrightarrow{PA_j}(x,y,z,t)$is the current pathogen agent *j* position, $\overrightarrow{v(t)}$ is the pathogen velocity, Δt is the time step, $\| \overrightarrow{v(t)} \|$is the magnitude of the velocity, C_{sr} is the relative current speed that is inherited from the grid where the pathogen *j* is, and R_n is a normally distributed random floating point with a mean of C_s (average current speed in this area) and a standard deviation *std*.

The velocity direction is related to the pathogen's orientation. The pathogen's orientation is defined by two variables: heading ($PA_j(t)_{hed}$) and pitch ($PA_j(t)_{pit}$). Heading is the angle between the forward vector of the pathogen projected onto the xy-plane and the vector [010], and pitch is the angle between the forward vector of the pathogen and the xy-plane. We calculated these variables using Eqs. (10) & (11).

$$PA_j(t)_{hed} = C_d + R_n(C_{bias}, std) \tag{10}$$

$$PA_j(t)_{pit} = pit_{in} - R_j * pit_v \tag{11}$$

where C_d is the currents' direction angle, R_n is a normally distributed random floating point with a mean of C_{bias} (current heading bias variable) and a standard deviation *std*, pit_{in} is the initial pitch value, R_j is a random number in the range of [0,1], and pit_v is the pitch value.

Table 2
Model parameters.

Parameter	Min value	Max value	Default value
Fish number	1	1000	100
Shedding rate (%)	0	100	50
Infection period (days)	0	100	2
Immune period (days)	0	100	5
Infectious radius (patches)	0	20	0.5
Initial infected (%)	0	100	5
Prior immunity (%)	0	100	0
Mortality (%)	0	100	3
Mortality_normal (%)	0	100	0.00001
Pathogen-ability	0	1	0.8
RF	0	1	0.8
Weight (kg)	0	10	4
Current speed (m/s)	0	1	0.15
Current speed std.	0	0.1	0.03
Current heading (degree)	0	360	90
Current heading bias (degree)	−5	5	0
Current heading std.	0	90	30
Sea temperature (°C)	0	20	10
Vaccinated			Off[a]
Time step			10 min[a]

[a] Parameters that can select between many values or be turned on/off.

2.2.2.8. Life cycle. Pathogen life span is a function of seawater condition (temperature and salinity). Salama estimated the life span for infectious salmon anemia virus (ISAV), infectious pancreatic necrosis virus (IPNV) and salmonid alphavirus (SAV) to be between 8.33 and 62.5 h (Salama and Murray, 2011). The life span's relation to the sea temperature can be modeled using the following equation (Stene et al., 2014).

$$PA_j(t)_{LC} = \mathrm{a} * \exp.(-\mathrm{x}/\mathrm{b}) \tag{12}$$

where *x* is the water temperature, *a* is the pathogen life span at a water temperature of 0 °C, and *b* is the decay rate.

2.2.2.9. Ability to infect. Each pathogen has an attribute that represents the ability to infect, that takes values between 0 and 1. We use 0.8 as the initial value and added some noise, as is shown in Eq. (13).

$$PA_j(t)_{ab} = PA_j(t)_{ab0} \pm R_j * N_{ab} \tag{13}$$

where $PA_j(t)_{ab}$ is the ability of pathogen *j* to infect, $PA_j(t)_{ab0}$ is the initial ability value that can be set by the user, R_j is a random number in the range of [0,1], and N_{ab} is the noise value.

Once the pathogens arrive at the neighboring sites (i.e., susceptible farms), they will try to infect the susceptible healthy individual fish in that farm. We apply the same infection rules as in *Algorithms I & II*.

The density of pathogens is directly related to the risk value in both space and time. A disease outbreak occurs only if there is a high density of pathogens and a high density of fish, as indicated by the following equation (Reno, 1998; Krkošek, 2010; Alaliyat and Yndestad, 2015c; Salama and Murray, 2011):

$$Risk\ (t,s) = K * I_v(t,s) * I_f(t,s) \tag{14}$$

where *Risk (t,s)* is the infection risk value in time and space, $I_v(t,s)$ is the

Table 3
Infection risk in an open area experiment.

Parameters of susceptible farm	Values
Fish number	100, 250,500,750,1000
Current speed (m/s)	0.05, 0.1, 0.15, 0.2, 0.25
Sea temperature (°C)	5, 7.5, 10, 12.5, 15

pathogen density at time t and in space s, $I_f(t,s)$ is fish density at time t and space s, and K is a constant.

2.2.3. Investigations

Fish disease dynamics and pathogen transmission depend on many different factors, such as fish density, farm location, fish and pathogen conditions and environmental conditions. In this study, we have built an agent-based model and simulated a variety of scenarios to investigate the effects of different combinations of parameter values on the fish disease dynamics. First, we investigated the minimum safe distance from the infected site under a variety of environmental conditions. Then, we investigated the effects of fish density, sea currents and temperature on the spread of an infectious disease from a source (producer) facility to a destination (consumer) facility, and we simulated the disease dynamics across time. Finally, we built scenarios based on empirical data to test the effects of the fjord's topography and the domino effect (producer-consumer facilities) on the spread of the infectious disease. Table 2 shows the model parameters that the user of our model may change.

2.2.4. Verification and validation

In general, and in aquaculture industry specifically, it is often very difficult to validate epidemiological simulation models due to the lack of reliable field data. The logical choice of validation techniques in such situations is to use cross-validation (i.e. to run a validated model for some simplified scenarios where the results are known or obvious) or to compare the model output with other available models that have been validated (so-called model alignment) (Chen et al., 2004).

We have done both (Alaliyat and Yndestad, 2015c): We ran our model for a simple scenario where the results were as expected, and we aligned it with well-known models, such as the SIR model (Skvortsov et al., 2007). We validated our results of simulation scenarios in parts of the Romsdalsfjord with the results from the infection spread model developed by SINTIF (midtnorge.sinmod.no). Internal validation or verification is very important also in ABMs. When the model is implemented by using NetLogo tool, the model must be verified by investigating whether the model behaves as expected. The purpose of the verification process is to build confidence in the behavioral characteristics that we assign to the agents and their interactions. We have tested the model under extreme conditions where the outcome is easily predictable to assess the validity of the agent descriptions used to ensure model consistency (avoid logical errors) as well as model coherence.

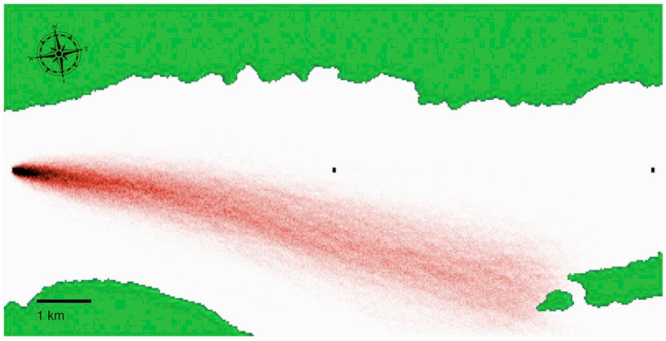

Fig. 4. Pathogen concentration in two dimensions (2-D) after a period of time.

3. Results

3.1. Infection risk in an open area

In this experiment we designed a simulation space to identify the risk of becoming infected in the vicinity of an infected aquaculture site: We assumed that the risk of a fish becoming infected is related to the concentration of pathogens in the space. The individual fish becomes infected by the procedure described in *Algorithm I*, so that the risk of any aquaculture site becoming infected in the area in the vicinity of the infected farm depends on the densities of the fish and pathogens at this site, as shown in Eq. (14). Table 3 shows the selected parameters from Table 2 to which we assigned the values listed during the simulation experiment.

Fig. 3 shows the farthest distance that live pathogens can reach alive (i.e. Risk Distance) based on the sea currents and water temperature during a simulation time of 8 days (cold water favors survival). The infection risk, which results from the presence of pathogens in a space unit (I_v), is a function of current speed and water temperature. This risk decreases as the distance increases from the infected site. Fig. 3 allows us to estimate the threshold (D_T) (between blue and other colors) of the

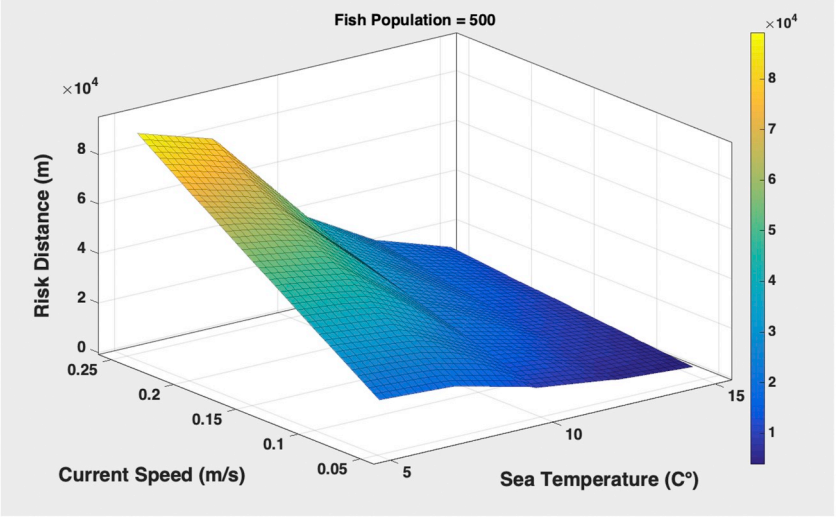

Fig. 3. Risk distance from the infected site.

Table 4
Infection risk between two fish farms.

Parameter	Values
Fish producer-farm number	100, 1000
Fish consumer-farm number	100,1000
Current speed (m/s)	0.05, 0.25
Sea temperature (°C)	5,15

farthest distance (Far_d ($temp, Cs$)) that the pathogens can spread; under normal sea-water and currents conditions. This threshold can change significantly depending on the current speed and the sea-water temperature. In this case, the threshold is determined by a current speed of 15 cm/s and a sea-water temperature of 10°.

$$Far_d(temp, Cs) > D_T \quad \text{if} \quad temp < 10^{\circ} \quad \text{and} \quad Cs > 15\ cm/s \tag{15}$$

where $Far_d(temp, Cs)$ is the maximum distance where pathogens can travel alive, D_T is a derived threshold from Fig. 3, $temp$ is a sea-water temperature, and Cs is the current speed.

The results (see Fig. 3) show that the farthest distance that the pathogens spread to does not exceed 12 km (D_T) given the current speed and water temperature threshold. This distance could be considerably larger, however, if the current speed was higher and/or if the water was colder.

Using ABM, we are able to track the pathogens in time and space. That enables us to identify the spatial characteristics of the spread of a disease. Fig. 4 shows the pathogen concentration (density) in each spatial cell resulting from running the simulation for a period of time. Thus, if the susceptible fish farm is located in the more risky (more red) area, then the greater is the probability that a fish in the farm becomes infected.

The pathogen density as described in Eq. (14) is dependent on four main variables: water temperature, current speed, distance from the infected site, and fish density at the infected site. From our simulation results, the pathogen density I_v in cell (x, y, z) after the period of time t can be modeled using the following exponential decay equation:

$$I_v(x, y, z, t) = a_0 * \exp\left(-\frac{(\text{temp}(x, y, z, t))}{b_0}\right) * \text{Cs}(x, y, z, t) * I_f / dis(x, y, z, t) \tag{16}$$

where temp(x, y, z, t) is the water temperature, Cs(x, y, z, t) is the current speed, dis(x, y, z, t) is the distance from the infected site, a_0 is the pathogen density at the infected site, b_0 is the decay rate, and I_f is the fish density at the infected site. We have derived the previous equation by estimating the relation between each input variable (i.e. water temperature) and the simulating results (pathogen density), then we combined these relations in one equation and validated this equation.

3.2. Infection risk between two fish farms in fjord area

In the next scenario, we extended the previous scenario by locating a susceptible fish farm at the edge of the risk area associated with the infected farm, i.e. at a distance, $dis = 9.6\ km < D_T$ from that farm. We simulated the fish disease dynamics in the infected farm that constituted the source of the pathogens to be transferred to the susceptible farm. We designed the simulation space so as to test the effects of changes in parameter values on the spread of the infectious disease from the source to the destination, and then we simulated the disease dynamics in the susceptible farm as well. We used the same scaling for time and space as in the previous scenario, and we used the default values shown in Table 2 for this model. Table 4 shows the parameter values in Table 2 that we varied in this simulation experiment.

Fig. 5 shows the percentage of the infected individuals (prevalence) in the two fish farms (infected and susceptible) under a variety of current speeds, sea temperatures and fish population parameter values.

With regard to the infection risk Eq. (14), the infection risk at cell (x,y,z) must be greater than a risk threshold R_0 that can be derived from the simulation results, in order for an infection to occur in this cell. The

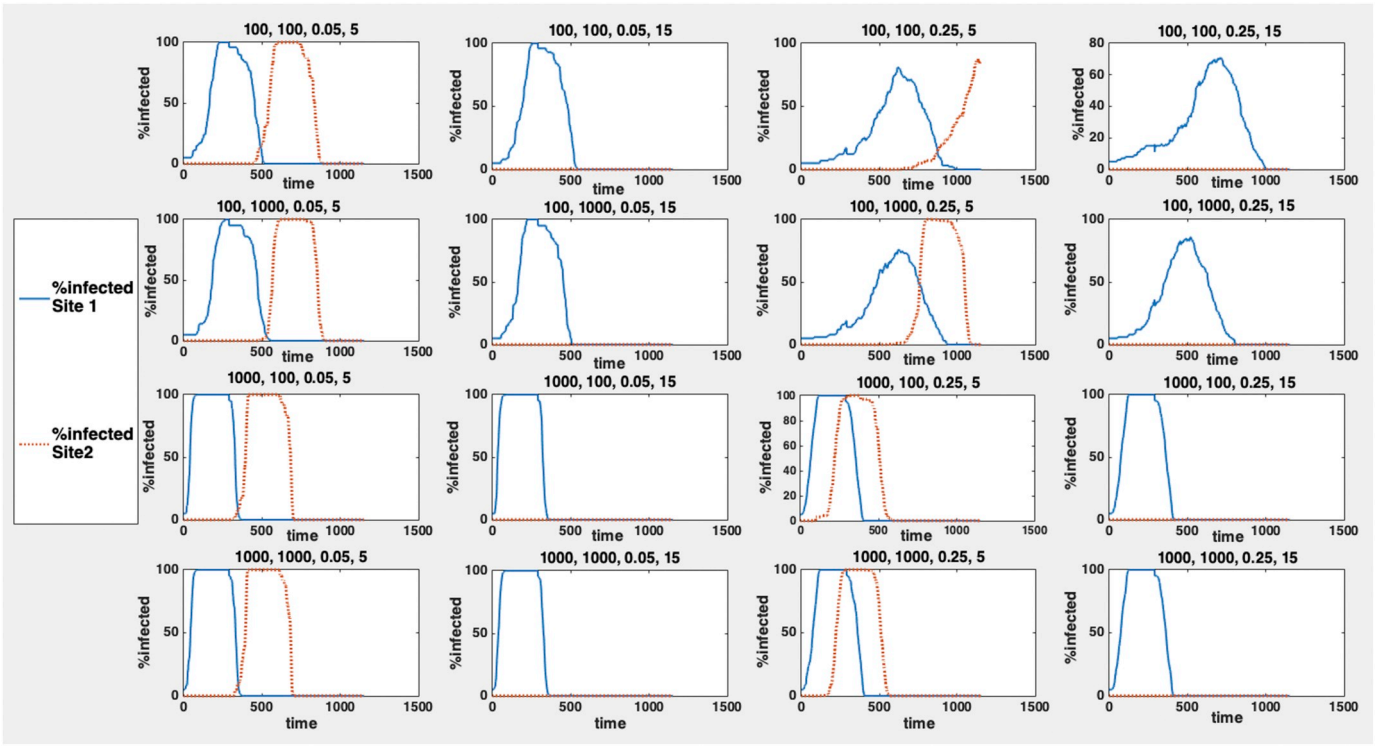

Fig. 5. Percentage of infected fish at site one, an infected farm (blue), and site two, a susceptible farm (red), as a result of water temperatures, current speeds and fish population values. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Fig. 6. Aquaculture system in part of the Romsdalsfjord (TN = tons): (a) Midsund area. (b) Vestness area.

Table 5
Scenario A parameters: midsund area.

Parameter	Bogen MD	Juvika	Myrane
Fish number	400	100	200
Current speed (m)	0.15	0.15	0.15
Sea temperature (°C)	8	8	8
Current direction (degree)	270	270	270

Table 6
Scenario B parameters: vestness area.

Parameter	Gjermundnes	Gjermundnesholmene	Furneset
Fish number	150	100	200
Current speed (m)	0.15	0.15	0.15
Sea temperature (°C)	7	7	7
Current direction (degree)	270	270	270

$I_v(x,y,z,t)$ in Eq. (16) is dependent only on sea-water temperature, current speed and producer farm pathogen density, since the distance is fixed at 9.6 km. The results show that the susceptible fish farm, located at (9.6 km, 0, 0), will become infected only if the sea-water temperature is low ($< 5^{\circ}$); if this is the case, then all fish become infected at the site. In the case when the water temperature is 15°, most of the pathogens die before arriving at the susceptible fish farm, so the pathogens will not infect that site. The fish densities in both the infected and susceptible farms play major roles, as described in Eqs. (14) and (16), and shown in Fig. 5. The disease is spreading faster when the population is higher as shown in the lower two rows in Fig. 5.

The spread of a disease inside a facility is faster when the current speed is low, as we see illustrated by the infected farm in the left two columns of Fig. 5. The infection at the susceptible fish farm starts only

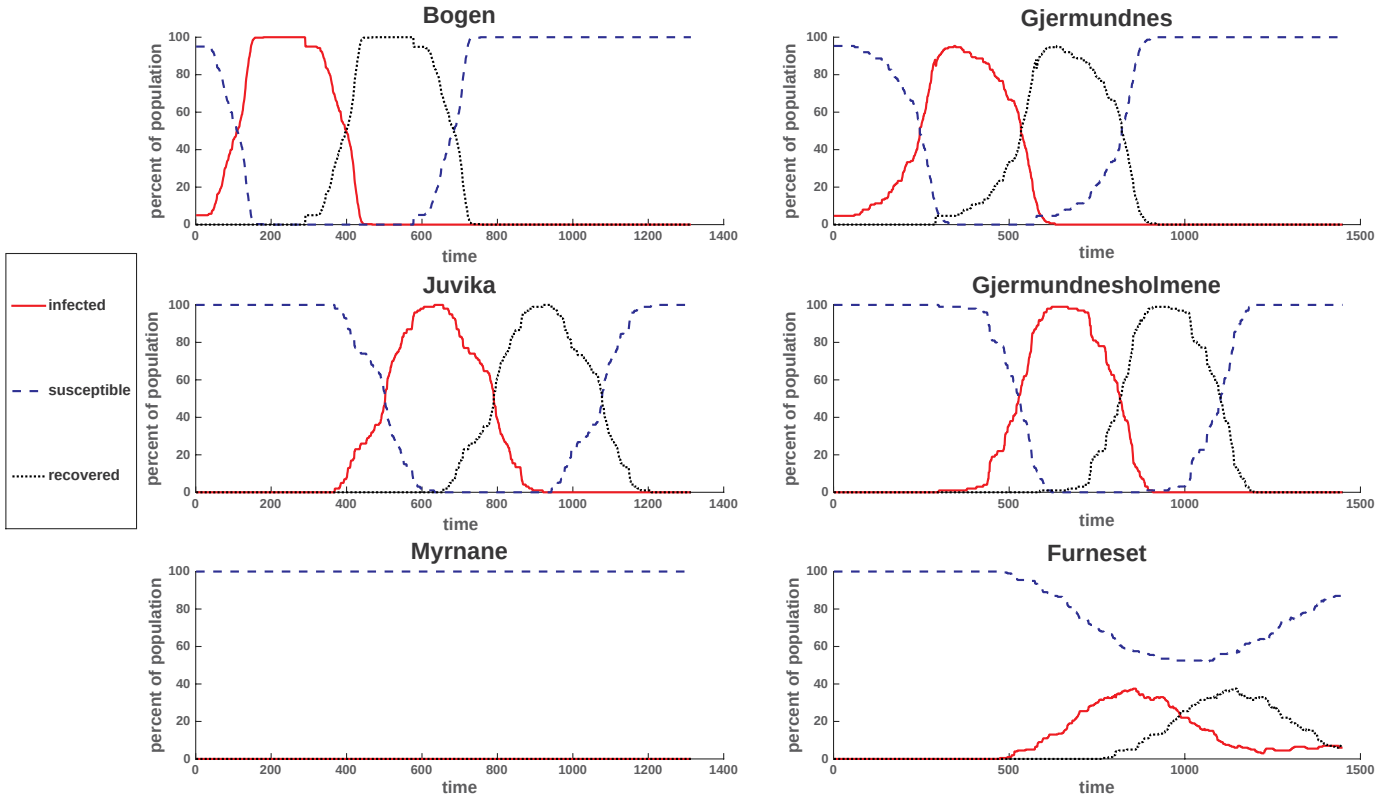

Fig. 7. Epidemic curves (results after 10 days).

after the arrival of a sufficient number of pathogens at the site (Eq. (16)); our results show that this happens after an average of two days across the simulation experiments in cases of higher fish density (e.g. 1000 individuals) in the susceptible farm, and the disease will spread to most of the individuals in the susceptible farm in approximately the same amount of time. In a case of lower fish density (e.g. 100 individuals), this time is approximately doubled.

3.3. Infection risk in a multi-farm system in fjord area (domino-effect)

In this scenario, we designed the simulation space so as to test the effects of a number of parameter values on the spread of the infectious disease across many fish farms, including the disease dynamics in each of them. In this scenario, the farms may, in principle, take the role as infected and susceptible and do so simultaneously; any farm can simultaneously shed pathogens to the others and receive pathogens from the others. We used the same scaling for time and space as in the previous scenarios, and we used the default values shown in Table 2 in the model. Fig. 6 shows the experimental setup of the simulation. We have run two different scenarios in the Romsdalsfjord. We have selected these areas because they are different in terms of their geospatial and aquaculture nature.

3.3.1. Scenario A (Midsund)

In this scenario, we included three farms (see Fig. 6(a)), and we used the average current speed and direction recorded in May 2008. We scaled the fish population, so that each fish in reality represents 15 tons of fish. In Table 5 we summarize the parameter values applied. These facilities are located within $\boldsymbol{D_T}$, − < 12 km apart. We assume that 5% of the fish population in the Bogen facility is initially infected. Fig. 7*(left hand side)* shows the disease dynamics in each site at Midsund.

3.3.2. Scenario B (Vestness)

In this scenario, we included three farms (see Fig. 6(b)), and we used the average current speed and direction recorded in April 2008. Again, each fish represents 15 tons of fish in reality. In Table 6 we summarize the parameter values applied. These facilities are also located within $\boldsymbol{D_T}$, < 12 km apart. We assume that 5% of the fish population in Gjermundnes facility is initially infected. Fig. 7*(right hand side)* shows the disease dynamics in each site at Vestness.

The simulation results from both scenarios, presented in Fig. 7, show the effects of current patterns and the geometry of the fjord on the spread of fish disease in parts of Romsdalsfjord, Midsund and Vestnes. In scenario A, the Myrane facility did not get infected because it was not located along the path of the sea currents that passed the infected sites during this period, − even though it was located within the risk distance ($\boldsymbol{D_T}$). As a result, the $\boldsymbol{I_v(x,y,z,t)}$ is very low, so the infection risk (Eq. (14)) for Myrane is less than $\boldsymbol{R_0}$. While Juvika got infected after almost three days. These results are reasonable compared to the results from MODS model (midtnorge.sinmod.no). Results from MODS model show that 34% of the pathogens from Bogen will hit Juvika in May, while only 1% of the pathogens will hit Myrane. However, in scenario B, the Gjermundnesholmene facility became infected and produced pathogens that infected the Furneset facility, which is located along the path of the currents that pass Gjermundnesholmene. Consequently, the $\boldsymbol{I_v(x,y,z,t)}$ is sufficiently high to cause an infection risk (Eq. (14)) above the threshold $\boldsymbol{R_0}$. Thus, the infection took place.

The infection of the Juvika and Gjermundnesholmene farms started after a sufficient amount of pathogens had arrived at each location. This took place after an average of four days. The Furneset facility was infected after 6 days because the distances from the two infected source sites were longer (see Eq. (16)).

The simulation results show that the sea current patterns play major roles in the spread of fish disease in Norwegian fjords. The pathogens are moved by sea currents, so if the fish farms are not in the path of the sea currents carrying pathogens from the infected sites, or they are located sufficiently far apart (beyond D_T), then the chance of infection is very low (as in the Myrnane case).

4. Discussion

4.1. Infection risk in an open area

It is very important to evaluate the infection risk in space and time when we want to assess the probability that a fish farm could act as the origin of an epidemic or as an intermediator. This evaluation is particularly important when we want to build and locate a new fish farm (Taranger et al., 2015) so as to prevent the spread of fish diseases in an aquaculture system. E.g. how will the surrounding area be affected if the fish in this new farm become infected? The first simulation scenario helps answer this question by exhibiting the risk values in space and time in the vicinity of such a new, hypothetically infected farm.

In this scenario, we studied the risk resulting from the pathogens that are predominantly relocated by sea currents (Murray and Peeler, 2005). The pathogens could, however, also move by way of fish boats or any other ships, or by way of escaped, infected fish. In our simulations, we focused on movement caused by the sea currents and sink effect. It is, however, easy to adjust our model to include other such factors.

Sea current speed and direction may vary significantly in the time and space domain considered, and we can expect values to remain steady for only a few days -or even less (MODS, 2012). In reality, we utilized empirical material reflecting average values, limited value ranges under which our model ran, and simulated the worst-case scenarios when the infection pressures are at most. The results demonstrate how the risk patterns are determined by the sea current patterns, − affected by the geometry of the fjord (see Fig. 4).

The pathogen's life span is associated with the sea temperature and salinity (Groner et al., 2016) and is influenced by significant changes in these values (Stene et al., 2014). In our model, it is easy to include such changes by way of modifications in parameter values.

The sea currents and sea-water temperature impact the distance that the pathogens can travel (spread) and thus the associated risk. A high water temperature decreases the distance since the pathogen's life span is shorter in hot than in cold water, while strong currents can move the pathogens farther away from its point of origin. The results demonstrate the current speed and water temperature thresholds (15 m/s and 10°, respectively) at which the risk distance increased considerably ($> \boldsymbol{D_T = 12\ k}$).

As shown in Eq. (14), the infection risk in space and time is dependent on the densities of the pathogens and fish. In this scenario, we investigated the factors that affect the pathogen density in each space cell in the vicinity of the infected site over a period of time. We derived from our theory and discussion Eq. (16), which shows the effects of water temperature, current speed, distance from the infected site, and fish density at the infected site on the pathogen's density. The pathogen's density exponentially decreases near the infected site as the water temperature increases. Moreover, the pathogen's density decreases as the distance from the infected site increases. The pathogen life span exponentially decreases as the water temperature increases. Conversely, if the water gets colder, then the pathogens spread to a larger area. The pathogens spread more with high current speeds. The pathogen density at a fixed position in the vicinity of the infected site $\boldsymbol{I_v(x,y,z,t)}$ is dependent only on water temperature, current speed and fish density at the infected site.

Building risk maps around the fish farm facilities in different environmental conditions helps inform management of the fish industry and helps prevent the spread of fish diseases that cause serious losses in Norwegian aquaculture.

4.2. Infection risk between two fish farms in a fjord area

If we assume there is a disease outbreak in an aquaculture facility

and there is another aquaculture facility located within the infection risk of the infected fish farm (< 12 km), then what will happen to the susceptible fish farm that is located in the risk area in the course of time? To answer this question, we simulated the fish disease dynamics in an aquaculture system that consisted of two fish farms located within a risk distance (9.6 km). We simulated the disease dynamics in the infected farm, the pathogen transmission time from the source to the destination, and the disease dynamics in susceptible farm.

The fish disease dynamics in fish populations are affected by many key factors, such as fish density, fish swimming behavior, water temperature, sea currents and other environmental factors (Alaliyat and Yndestad, 2015b). The infection process occurs only when there is a sufficient number of pathogens in the vicinity of the susceptible fish (see *Algorithm I*). The fish disease dynamics in a single fish population is faster with higher fish densities and lower sea current speeds (Alaliyat and Yndestad, 2015a). The pathogens are moved by sea currents, so with high-speed sea currents, the pathogens spread across larger distances and move faster to nearby sites. The number of pathogens released from the infected site increases as the fish population increases (Alaliyat and Yndestad, 2015a).

Once the pathogens arrive at a susceptible farm, the infection process will start as soon as a sufficient number of pathogens arrive the susceptible fish (*see Algorithm I*). The rules of disease transmission that applies to an infected farm, also applies to the susceptible farm.

The results demonstrate that a susceptible fish farm, located 9.6 km from the infected farm, will become infected after two to four days, depending on the fish density, provided the average sea-water temperature is 5°. The pathogens need < 12 h to cross the 9.6 km distance from the infected site when the current speed is 0.25 m/s. Yet, based on the diffusion factor that spread the pathogens to a wider area, the probability of reaching the susceptible site is considerably reduced in only 12 h. In addition, even if the pathogens arrive at the susceptible farm, the pathogen density and the fish density in that farm will have to be sufficiently high to initiate an infection (Eq. (14)).

4.3. Infection risk in a multi-farm system in fjord area (domino-effect)

It is important to test our agent-based model on real case studies with more than two fish sites, to simulate cases where the fish farms are infected and susceptible sites, and to include the effects of the topography of the fjord on the simulated fish disease dynamics. In these scenarios, we hypothetically assume that there is an infection in the Romsdalsfjord during the particular period of the year. The Romsdalsfjord is an area that hosts many fish farms that are located close to each other (Fig. 1), and the dynamics of the sea currents in the fjord exhibit a very complex pattern (Fig. 2).

Our results show the effects of the topography of the Romsdalsfjord on the spread of fish disease. In scenario A, the Myrane facility was not infected even though it was close to the infected site Bogen (the distance between them is 8.4 km, which is < 12 km). This is because it was not located along the path of the sea currents that passed the infected sites during this period. However, in scenario B, the Gjermundnesholmene facility was infected and produced pathogens that, subsequently, infected the Furneset facility, located along the path of the sea currents that passed Gjermundnesholmene. In this scenario, the Gjermundnesholmene facility was initially a susceptible site, and then an infected site that shed pathogens. These simulation experiments support the idea that vicinity is a not a sufficient cause for infection; in fact, the current patterns are just as important (Stene et al., 2014). The pathogens need a transport vehicle to bring them from the infected site to the susceptible farm.

We used monthly average sea current speed and direction in the Romsdalsfjord. Note that the currents in the fjords change significantly due to wind and other factors. E.g. the current direction in the Romsdalsfjord changes in the spring as a result of the melting snow causing fresh water to be transported along the fjord towards the sea. Tides can cause the water to move back and forth within a range of a few kilometers, and this supports the results from other studies that have demonstrated the impact of the close sea distance between the farms (Aldrin et al., 2010; Tavornpanich et al., 2012).

There are some limitations in applying the model to real cases in order to analyze the risk and provide advice to the fish industry. Scaling the model in space and time is one of these limitations. We introduced a portion of the individuals (fish and pathogens) in our simulation, since representing a huge number of agents is computationally very demanding. This, however, limits the contact between agents. In addition, there are several factors of uncertainty pertaining to the process of transmitting illness between fish. We are not in the possession of data on this process, and the available data are based on the lab experiments that have limited relevance in empirical settings (Salama and Murray, 2011).

4.4. Using agent-based methods

Pathogen transmission within and between aquaculture farms is dependent on a variety of interrelated biological, physical and environmental factors, such as fish density, shedding rate, infection radius, current speed, current direction, pathogen life span, seawater temperature, and seawater salinity. ABM simulates the overall dynamics based on how individuals (fish and pathogens) interact and adapt to such factors. ABM provides insights into the underlying structural causes of emerging dynamic phenomena resulting from the interactions between individuals. Models developed previously have predominantly been relying on differential equations in describing the overall population dynamics of fish diseases (e.g., Murray, 2009; Green, 2010) or they have been hydrodynamic models combined with particle tracking and statistical analyses (MODS, 2012; Stene et al., 2014). Mathematical models, such as the SIR model, do not cover the individual variety of the biological and physical characteristics of fish as well as other animals; fish vary in their resistance to pathogens, and the pathogens themselves vary in their ability to infect fish. In non-ABM models this variability in fish and pathogens is being ignored. The analysis is based on the assumption that the populations are homogenous. By using ABM, we are able to address specific spatial aspects of the spread of infections and address the stochastic nature of the infection process of fish diseases.

ABM provides flexibility in modeling, implying that additional complexity may be introduced to form the basis for simulation-based analyzes. Agents may be added to or removed from the model, and the attributes and behavioral rules can be modified as well. For an example, the swimming rules have been included in previous model to create new ones (Alaliyat and Yndestad, 2015b).

Another reason for applying ABM is the lack of empirical data we experience regarding fish disease transmission. In ABMs, the parameters characterizing disease transmission, such as fish infection rules and the shedding rates, may easily be varied, to assess how sensitive the results are to the assumptions we introduce in the models. We may assess sensitivities at a high level of resolution rather than merely address the sensitivity aggregate characteristics such as the contact rate in an SIR model.

There are limitations in an ABM approach to the modeling and simulation of fish disease dynamics. Typically, the magnitude of the population (fish and pathogens) is too large for a full-scale simulation, even on massive computers. By scaling down, the contact frequency may be affected and suffer from not being sufficiently representative. Moreover, as reliable data on pathogen are hard to come by, the pathogen model component is based on a theoretical foundation. The validation of dynamic fish disease models is inherently challenging, not the least because of the lack of reliable empirical data.

Previous models have typically focused on the analysis of historical data to support future predictions in similar cases (Stene, 2013). The pathogen transmission and fish disease dynamics vary significantly,

depending on a variety of factors such as sea currents and water temperature. In a model such variations may be included as a basis for sensitivity analyses. The results are generally applicable, but may not be used for point prediction due to the uncertainties characterizing the underlying structural assumptions. The results of the sensitivity analyses will guide us in our evaluation of the validity of any prediction made based on the model, − i.e. tell us whether we can trust the predictions produced by such a model.

ABMs are stochastic models that can capture the randomness in natural systems. Therefore, it may well be advantageous to apply ABM in the modeling and simulation of fish infectious disease dynamics compared to the application of simple deterministic models (such as the SIR model), often characterized by static estimates of parameters that, in reality, vary across the populations and are sensitive to the varying, environmental conditions.

5. Conclusions and future work

The aquaculture industry is one of the main industries in Norway. Emergent diseases continue to pose a serious challenge to the industry and constrains its development. Predicting fish disease dynamics is important when preventing and combating fish diseases. The results of such predictions may facilitate the management process and inform the aquaculture industry, and it also helps combat the spread of diseases by applying actions to stop the spread of the disease, such as through vaccinations or removal of the pathogens. Fish diseases typically cause large economic losses to the aquaculture industry and might threaten wild populations of species as well. To manage our natural resources, such as fish, in a sustainable way, we rely on analyses based on validated models in which we have confidence.

Models, such as the SIR model, predict the spread of fish diseases based on the assumption that the fish population is homogeneous, and focus on the population as a whole. In this paper, we assume that the fish and pathogens are heterogeneous. To do so, we applied an agent-based approach in our modeling of the dynamics of fish diseases within and between aquaculture facilities in a Norwegian fjord. Using this approach, we consider the interaction of each individual with others and with the natural environment in the context of space and time. The agents in our model are fish and pathogens.

We predict the dynamics of the pathogen transmission patterns so to identify the infection risk in a space-time domain. Our results show (see Fig. 4) that the pathogen density decreases as the distance from the infected site increases. Pathogen density at a fixed position around the infected site decreases exponentially by increasing sea-water temperature, while pathogen density increases when current speed and fish density at the infected site increase. Because the pathogens are predominantly relocated by sea currents, such currents play a major rule in the pathogen transmission process, the infection risk is a function of fish and pathogen density, and that risk increases when these densities increase.

Adding fish swimming behavior or additional social behavior rules to the swarm of pathogens constitutes a potential extension of the model. Also, we may include additional key parameters associated with the fish industry process (i.e., stocking and harvesting process) or its interaction with the outside environment, including escaped fish, wild fish, feed, and the working environment.

ABMs are inherently flexible and represent the systems structure at the level of individuals (high resolution). The interaction unfolding between individuals may be analyzed in the context of a wide variety of scenarios, in particular pertaining to the environment in which the interaction takes place. Fish infectious disease dynamics is a complex process that results from the extensive network of interactions existing between large variety of characteristics of fish, pathogens and seawater. The high resolution facilitated by an agent-based approach offers an opportunity to describe the system in relatively realistic terms, − provided we know the processes taking place at that detailed level and the variety in the parameter values governing these processes.

NetLogo is the software applied in this work. It offers a simple user interface that facilitates changes in parameter values and immediate responses to such changes. This way, the models developed may easily be shared among a variety of stakeholders that may benefit from experimentation, simulation and analysis of the results.

References

Alaliyat, S., Yndestad, H., 2015a. An agent-based model to simulate infectious disease dynamics in an aquaculture facility. In: Proceedings of UKSIM-AMSS 17th International Conference on Modelling and Simulation, Cambridge, 25–27 March 2015, pp. 131–136.

Alaliyat, S., Yndestad, H., 2015b. An aqua agent-based model to simulate fish disease dynamics with reference to Norwegian aquaculture. In: Proceedings of 11th International Conference on Innovations in Information Technology (IIT'15), Dubai, UAE, 01–03 November 2015, pp. 350–355.

Alaliyat, S., Yndestad, H., 2015c. An agent-based model to simulate contagious disease dynamics in fish populations. Int. J. Simul. 16 (3).

Alaliyat, S., Osen, O.L., Kvile, K.O., 2013. An agent-based model to simulate pathogen transmission between aquaculture sites in the Romsdalsfjord. ECMS 46–52.

Aldrin, M., Storvik, B., Frigessi, A., Viljugrein, H., Jansen, P.a., 2010. A stochastic model for the assessment of the transmission pathways of heart and skeleton muscle inflammation, pancreas disease and infectious salmon anaemia in marine fish farms in Norway. Prevent. Vet. Med. 93, 51–61.

Arduin, H., Domenech de Celles, M., Guillemot, D., Watier, L., Opatowski, L., 2017. An agent-based model simulation of influenza interactions at the host level: insight into the influenza-related burden of pneumococcal infections. BMC Infect. Dis. 17, 382 (pmid:28577533).

Bergh, O., 2007. The dual myths of the healthy wild fish and the unhealthy farmed fish. Dis. Aquat. Org. 75, 159–164.

Bjørnstad, O. (2005), SEIR Model, Retrieved from http://www.stat.colostate.edu/~rdavis/ey680/sir.pdf

Chen, L.C., Kaminsky, B., Tummino, T., Carley, K.M., Casman, E., Fridsma, D., Yahja, A., 2004. Aligning Simulation Models of Smallpox Outbreaks. Lecture Notes in Computer Science: Intelligence and Security Informatics. Springer, Berlin.

Ciofi degli Atti, M.L., Merler, S., Rizzo, C., Ajelli, M., Massari, M., Manfredi, P., Furlanello, C., Scalia Tomba, G., Iannelli, M., 2008. Mitigation measures for pandemic influenza in Italy: an individual based model considering different scenarios. PLoS One 3, e1790.

FKD, 2018. Fiskeridirektoratet. www.fiskeridir.no (accessed 08.12.2018).

Green, D.M., 2010. A strategic model for epidemic control in aquaculture. Prevent. Vet. Med. 94, 119–127.

Grefsrud, E.S., Glover, K., Grøsvik, B.E., Husa, V., Karlsen, Ø., Kristiansen, T., Kvamme, B.O., Mortensen, S., Samuelsen, O.B., Stien, L.H., Svåsand, T., 2018. Risikorapport norsk fiskeoppdrett 2018. Fisken og havet, særnr. 1-2018.

Gregory, A., 2008. A Qualitative Assessment of the Risk of Introduction of Viral Haemorrhagic Septicaemia Virus into the Rainbow Trout Industry Scotland. Aberdeen, UK.

Groner, M.L., McEwan, G.F., Rees, E.E., Gettinby, G., Revie, C.W., 2016. Quantifying the influence of salinity and temperature on the population dynamics of a marine ectoparasite. Can. J. Fish. Aquat. Sci. 2016, 1–11 (Web).

Iversen, et al., 2005. Stress responses in Atlantic salmon (Salmo salar L.) smolts during commercial well boat transports, and effects on survival after transfer to sea. Aquaculture 243 (1–4), 373–382.

Johansen, L.-H., Jensen, I., Mikkelsen, H., Bjørn, P.-A., Jansen, P.A., Bergh, Ø., 2011. Disease interaction and pathogens exchange between wild and farmed fish populations with special reference to Norway. Aquaculture 315, 167–186.

Kermack, W.O., McKendrick, A.G., 1927. Contribution to the mathematical theory of epidemics, part I. Proc. R. Soc. Lond. A 53, 33–55.

Khalil, K.M., Abdel-Aziz, M., Nazmy, T.T., Salem, A.-B.M., 2010. An agent-based modeling for andemic influenza in Egypt, Informatics and Systems (INFOS). In: The 7th International Conference on, pp. 1–7 28–30 March.

Kocabas, V., Dragicevica, S., 2006. Assessing cellular automata model behaviour using a sensitivity analysis approach. Computers. Environ. Urban Syst. 2006 (30), 921–953.

Kristoffersen, A.B., Viljugrein, H., Kongtorp, R.T., Brun, E., Jansen, P.A., 2009. Risk factors for pancreas disease (PD) outbreaks in farmed Atlantic salmon and rainbow trout in Norway during 2003-2007. Prevent. Vet. Med. 90, 127–136.

Krkošek, M., 2010. Host density thresholds and disease control for fisheries and aquaculture. Aquacult. Environ. Int. 1 (1), 21–32. https://doi.org/10.3354/aei00004.

Madonia, A., Melchiorri, C., Bonamano, S., Marcelli, M., Bulfon, C., Castiglione, F., et al., 2017. Computational modeling of immune system of the fish for a more effective vaccination in aquaculture. Bioinformatics 33 (19), 3065–3071. https://doi.org/10.1093/bioinformatics/btx341.

MATLAB version R2015a, 2015. Computer Software. TheMathWorks Inc., Natick, Massachusetts 05.Mar.2015. URL. http://www.mathworks.com.

Milne, G.J., Kelso, J.K., Kelly, H.A., Huband, S.T., McVernon, J., 2008. A small community model for the transmission of infectious diseases: comparison of school closure as an intervention in individual-based models of an influenza pandemic. PLoS One 3, e4005.

MODS, 2012. Strømmodellering og smitte-spredning. http://mods.sinmod.no/.

Murray, A.G., 2009. Using simple models to review the application and implications of different approaches used to simulate transmission of pathogens among aquatic

animals. Prevent. Vet. Med. 88, 167–177.
Murray, A.G., Peeler, E.J., 2005. A framework for understanding the potential for emerging diseases in aquaculture. Prevent. Vet. Med. 67, 223–235.
NMA, 2016. The Norwegian Mapping Authority. http://www.kartverket.no/, Accessed date: 5 May 2016.
Ögüt, H., 2001. Modeling of fish disease dynamics: a new approach to an old problem. Turk. J. Fish. Aquat. Sci. 1, 67–74.
Olsen, A.B., Hellberg, H., 2011. The Health Situation in Norwegian Aquaculture 2011. Norwegian Veterinary Institute, pp. 39.
Reno, P:.W., 1998. Factors involved in the dissemination of disease in fish populations. J. Aquat. Anim. Health 10 (2), 160–171.
Reynolds, C., 1999. Steering Behaviour for Autonomous Characters. http://www.red3d.com/cwr/steer/ (first version from 1999).
Rimstad, E., 2011. Examples of emerging virus diseases in salmonid aquaculture. Aquac. Res. 42, 86–89.
Rose, A.S., Ellis, A.E., Munro, A.L.S., 1989. The infectivity by different routes of exposure and shedding rates of Aeromonas salmonicida subsp. salmonicida in Atlantic salmon, Salmo salar L., held in sea water. J. Fish Dis. 12, 573–578.
Salama, N., Murray, A., 2011. Farm size as a factor in hydrodynamic transmission of pathogens in aquaculture fish production. Aquacult. Environ. Int. 2, 61–74.
Sirakoulis, G.C., Karafyllidis, I., Thanailakis, A., 2000. A cellular automaton model for the effects of population movement and vaccination on epidemic propagation. Ecol. Model. 133, 209–233.
Skvortsov, R., Connell, B., Dawson, P., Gailis, R., 2007. Epidemic modeling: validation of agent-based simulation by using simple mathematical models. In: Proceedings of Land Warfare Conference, pp. 221–227.
SSB, 2018. Statistics Norway, Fishing and Fish Farming. http://www.ssb.no/fiskeri_havbruk_en/, Accessed date: 8 December 2018.
Stene, A., 2013. Transmission of Pancreas Disease in Marine Salmon Farming in Norway. Norwegian School of Veterinary Sciences, Oslo, Norway (Doctoral dissertation, PhD thesis).
Stene, A., Hansen, G.A., Yttredal, E.R., Solevåg, S.E., 2009. Integrert Akvakultur - Effektiv utnyttelse av kystsonen – Effekt på arealbinding og interessekonflikter. Ålesund 03, 2009.
Stene, A., Bang Jensen, B., Knutsen, Ø., Olsen, A., Viljugrein, H., 2014. Seasonal increase in sea temperature triggers pancreas disease outbreaks in Norwegian salmon farms. J. Fish Dis. 37, 739–751. https://doi.org/10.1111/jfd.12165.
Taranger, G.L., Karlsen, Ø., Bannister, R.J., Glover, K.A., Husa, V., Karlsbakk, E., Kvamme, B.O., et al., 2015. Risk assessment of the environmental impact of Norwegian Atlantic salmon farming. ICES J. Mar. Sci. 72, 997–1021.
Tavornpanich, S., Paul, M., Viljugrein, H., Abrial, D., Jimenez, D., Brun, E., 2012. Risk map and spatial determinants of pancreas disease in the marine phase of Norwegian Atlantic salmon farming sites. BMC Vet. Res. 8 (1), 172.
Urke, H.A., Kristensen, T., Daae, K.L., Bergan, M.A., Ulvund, J.B., Alfredsen, J.A., 2011. Konsekvenser Av Sjødeponi i Repparfjorden for Anadrom Laksefisk. Delutredning i KU Program for Planlagt Gruvedrift i Nussir Og Ulveryggen i Kvalsund Kommune.
Urquhart, K., Murray, A.G., Gregory, A., O'Dea, M., Munro, L.A., Smail, D.A., Shanks, A.M., Raynard, R.S., 2008. Estimation of infectious dose and viral shedding rates for infectious pancreatic necrosis virus in Atlantic salmon, *Salmo salar* L., post-smolts. J. Fish Dis. 31, 879–887.
Venkatramanan, S., Lewis, B., Chen, J., Higdon, D., Vullikanti, A., Marathe, M., 2018. Using data-driven agent-based models for forecasting emerging infectious diseases. Epidemics 22, 43–49. https://doi.org/10.1016/j.epidem.2017.02.010.
Viljugrein, H., Staalstrøm, A., Molvaelr, J., Urke, H.A., Jansen, P.A., 2009. Integration of hydrodynamics into a statistical model on the spread of pancreas disease (PD) in salmon farming. Dis. Aquat. Org. 88, 35–44.
Werkman, M., Green, D.M., Murray, A.G., Turnbull, J.F., 2011. The effectiveness of fallowing strategies in disease control in salmon aquaculture assessed with an SIS model. Prevent. Vet. Med. 98, 64–73.
Wilensky, Uri, 1999. Netlogo. The Center for Connected Learning (CCL). https://ccl.northwestern.edu/netlogo/, Accessed date: 29 May 2016.
Yang, Y., 2019. A narrative review of the use of agent-based modeling in health behavior and behavior intervention. Transl. Behav. Med. https://doi.org/10.1093/tbm/iby132.
Yndestad, H., 2010. Agents and Landscapes in Complex Systems. Aalesund University College, Aalesund, Norway.
Zhen, J., Quan-Xing, L., 2006. A cellular automata model of epidemics of a heterogeneous susceptibility. Chin. Phys. 15, 1248–1256.