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Farm size as a factor in hydrodynamic transmission of pathogens in aquaculture fish production

Nabeil K. G. Salama*, Alexander G. Murray

Marine Scotland Science, Marine Laboratory, 375 Victoria Road, Aberdeen AB11 9DB, UK

ABSTRACT: Global aquaculture production has rapidly increased over recent decades, primarily through the increase in production per farm unit. However, size (biomass) may be a factor in the transmission of infectious diseases between hydrodynamically linked fish farms. A combined epidemiological–simplified hydrodynamic model is used to demonstrate that as farm units increase they experience higher numbers of infections caused by a range of pathogen characteristics. The model demonstrates that as farm size increases in areas where faster currents prevail, there is a need to increase the separation distance between farms to prevent pathogen transmission. A comparison of production regimes demonstrates, however, that fewer, highly separated, larger farms reduce overall losses compared to numerous smaller farms in close proximity to each other.

KEY WORDS: Farm size · Pathogen transmission · Hydrodynamics

INTRODUCTION

Infectious disease dynamics in aquaculture are regulated by host population size (Ögüt 2001, Krkošek 2010) because larger populations have increased contact between infectious and susceptible individuals. Herd size has been identified as a disease transmission risk factor for terrestrial animal production (Sørensen et al. 2000, Humblet et al. 2009). In aquaculture, host density has been shown to be a factor in transmission of infectious pancreatic necrosis virus (IPNV) (Bebak-Williams et al. 2002) and infectious hematopoietic necrosis virus (IHNV) (Ögüt & Reno 2004) in salmonids. Sensitivity analysis of susceptible-infected-recovered (SIR) models have demonstrated that host density alters the transmission rate of disease within a host fish population (Ögüt et al. 2005), indicating that population size could also have an impact on the transmission of disease between host populations. Pathogen concentration can increase in the surrounding environment with increased numbers of infected fish on a farm, irrespective of density (Murray 2009).

Aquaculture production continues to increase globally (Food and Agriculture Organisation of the United Nations [FAO] 2008) and is predicted to surpass fisheries as the major source of fish for human consumption (FAO 2010). Global production is forecast to increase by 6% per annum to 130 million t by 2020 (FAO 2009), and to fulfil this demand aquaculture sites need to become more abundant and increase the level of production per site. The aquaculture industry in China accounts for 2/3 of global production (FAO 2007) and has doubled the area used for aquaculture whilst having a 6-fold increase in production during 1979 to 1996 (FAO 1997), indicating that on average size per production unit has increased. For production to further increase, industries must become more efficient by minimising stock loss due to infectious disease, as it is estimated that this costs 3 billion USD globally (Subasingh et al. 2001).

Salmon accounts for approximately a tenth of global aquaculture production by value, and the size of the industry continues to grow (FAO 2008). Within the European Union, Scotland is the largest aquaculture producer of Atlantic salmon *Salmo salar*. Since 1988

*Email: salaman@marlab.ac.uk

production has increased to the current level of 144 000 t and is expected to continue to grow (Walker 2010). However, since 1998, the number of farm sites has decreased (Walker 2010); thus, production per farm is increasing. The largest Scottish farms currently have biomass consents to stock up to 2500 t (available from the Scottish Environment Protection Agency [SEPA] production database upon request), while salmon farms over 5000 t exist in Norway (Fiskeridirektoratet 2011); therefore, it is possible that Scottish production could increase production through greater stocking densities. The economic value of current production is reported to be over £500 million yr^{-1} (Scottish Salmon Producers' Organisation 2010) and the industy provides valuable employment in rural communities; therefore, aquaculture is an important contributor to the economy of Scotland. However, pathogenic diseases have significant impacts due to production losses through mortality (Murray & Peeler 2005), and this subsequently has an economic effect, as demonstrated by the estimated £20 million cost of an infectious salmon anaemia (ISA) outbreak in Scotland in 1998/1999 (Hastings et al. 1999), whilst the costs of the recent 2008/2009 outbreak (Murray et al. 2010) are still to be established. Due to the negative impact of pathogenic disease transmission, it is important to ascertain methods of assessing pathogen transmission pathways in order to reduce the impact on farmed fish production.

An important concept in disease progression is that of a dose threshold that initiates the disease symptoms and is commonly termed the minimum infective dose (Ward & Akin 1984). This threshold is often obtained by measuring the minimum detectable levels of pathogens that cause an infection response. Below this threshold no symptoms occur because the innate immune system is able to eliminate or mitigate against low levels of pathogen exposure (Watts et al. 2001); above the threshold an individual has a certainty of becoming infected. Such minimum infectious doses have been identified for salmonid pathogens causing ISA (Raynard et al. 2001, Gregory et al. 2009), IPN (Urquhart et al. 2008) and amoebic gill disease (Morrison et al. 2004).

Infectious disease dynamics are controlled by characteristics associated with host and pathogen biology. Host population size often plays a role in the transmission, persistence and also the dynamic trajectories of the epidemic (Anderson & May 1979, May & Anderson 1979, Grenfell & Dobson 1995) due to increased contact between infectious and susceptible individuals. Within terrestrial systems it is often the case that a diseased individual enters a susceptible population, possibly initiating an epidemic. However, in aquaculture systems, water is able to transmit free-moving pathogenic agents between hydrodynamically connected discrete fish farms (Amundrud & Murray 2009, Frazer 2009) and infection can be initiated without the presence of an infected host within a naïve population. Hydrodynamic transmission between farms has been demonstrated for ISA (Gustafson et al. 2007) and pancreas disease (PD) (Viljugrein et al. 2009) and has been attributed to the spread of ISA (e.g. McClure et al. 2005, Aldrin et al. 2010), sea lice (e.g. Amundrud & Murray 2009) and PD (e.g. Kristoffersen et al. 2009, Aldrin et al. 2010). The importance of hydrodynamic disease movements has been demonstrated by the introduction of disease management areas (DMAs) to prevent the spread of ISA (Scottish Executive 2000) based on tidal excursions surrounding producing farms (Marine Scotland 2010). The recent (2008/2009) outbreak of ISA in the southwest Shetland mainland demonstrates how hydrodynamically linked farms led to infection in 6 nearby farms (Murray et al. 2010). In Norway, proximity is described as a risk factor in the spread of ISA (Lyngstad et al. 2008); likewise, the Chilean ISA outbreak (2007/2008) demonstrated spatial clustering of infectious farms surrounding an initial outbreak farm (Mardones et al. 2009).

This paper presents the use of an adapted discrete-time susceptible-exposed-infected-recovered (SEIR)-type model (Diekmann & Heesterbeek 2000), representing farm units linked by a simplified hydrodynamic model which allows for between-farm transmission. An example use of the model is provided by identifying the characteristics of example salmonid pathogens and then deriving the separation distances between farm units that are necessary in order to prevent the transmission of disease between sites, for farms of varying sizes.

METHODS

Fig. 1 shows a schematic of the model framework used within this paper. It is similar to the standard SEIR frameworks but with an additional reservoir compartment for waterborne pathogens (W) which expose other farms. It is assumed that, due to tidal movements, these particles pose no additional risk to the source farm as direct transmission dominates infection risk within a farm. A constant farm population size (N) and a closed system without population size change are assumed. The parameters used in the model are described in Table 1.

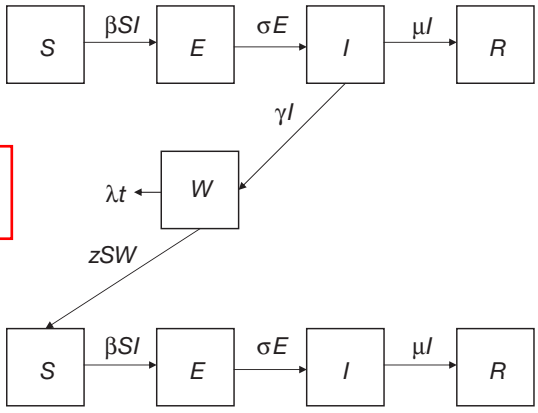

Fig. 1 corrected after online publication

Fig. 1. Schematic of a compartmentalised susceptible (*S*)-exposed (*E*)-infected (*I*)-recovered (*R*) model representing 2 farms connected by a waterborne pathogen phase (*W*). See Table 1 for description of parameters

In this study, a discrete form of an epidemiological model is constructed to remain consistent with the modelling approach of Murray et al. (2005), who use time units of 15 min intervals, assuming that rate of change was uniform between each quarter hour time period within each hour. Furthermore, disease progression in challenge experiments is reported in daily discrete-time periods (e.g. Urquhart et al. 2008, Gregory et al. 2009) and therefore aggregates the observable change in disease status of individuals over time. Similarly, dose and shedding is reported in hourly units (e.g. Urquhart et al. 2008, Gregory et al. 2009), as is the reporting of aspects of pathogen biology (Toranzo & Hetrick 1982, Løvdal & Enger 2002). This, therefore, lends itself to the development of epidemiological models based on difference equations representing discrete 15 min time-steps (*t*). Discrep-

Table 1. Parameter descriptions. Note that subscripts relate to farm position in a sequence in the model equations

Pathogen parameter	Description	ISAV–t (Source)	IPNV–t (Source)	AS–t (Source)
β (z)	Pathogen transmission probability	0.015 d^{-1} (Gregory et al. 2009)	0.013 d^{-1} (Smith et al. 2000)	0.0214 d^{-1} (Ögüt & Bishop 2007)
σ	Infectious individual expression probability	0.14 d^{-1} (Gregory et al. 2009)	0.36 d^{-1} (Smith et al. 2000)	0.29 d^{-1} (Ögüt & Bishop 2007)
μ	Infected individual epidemic removal probability	0.04 d^{-1} (Gregory et al. 2009)	0.062 d^{-1} (Smith et al. 2000)	0.33 d^{-1} (Ögüt & Bishop 2007)
γ	Pathogen particle shedding rate	7.2×10^{-1} ml^{-1} h^{-1} kg^{-1} (Gregory et al. 2009)	6.8×10^{-2} ml^{-1} h^{-1} kg^{-1} (Urquhart et al. 2008)	1.75×10^{6} cfu ml^{-1} h^{-1} (Rose et al. 1990)
λ	Pathogen decay rate	0.12 h^{-1} (Løvdal & Enger 2002)	0.016 h^{-1} (Toranzo & Hetrick 1982)	0.12 h^{-1} (Rose et al. 1989
φ	Minimum infectious dose	10^{-1} $TCID_{50}$ ml^{-1} kg^{-1} (Gregory et al. 2009)	10^{-4} $TCID_{50}$ ml^{-1} kg^{-1} (Urquhart et al. 2008)	10^{8}cfu ml^{-1} (Pérez et al. 1996)

	Description	Parameter value (where applicable)	Source (where applicable)
Population parameter			
N	Total biomass on a farm	10^{2}–10^{5} t	SEPA[a], Fiskeridirektoratet (2011)
S	Susceptible biomass on a farm		
E	Exposed biomass on a farm		
I	Infectious biomass on a farm		
R	Removed from an epidemic on a farm		
W	Number of pathogen particles in the environment		
t	Time-step		
ε	Dose-related infection probability between farms		
Transport parameter			
a	Minimum transport step-length		
b	Maximum transport step-length		
s	Mean transport step-length		
U	Tidal current amplitude	51 cm s^{-1}	Murray et al. (2005)
T	Tidal period	12.42 h	Murray et al. (2005)
C	Constant residual advection current	1–8 cm s^{-1}	Murray et al. (2005)
D	Diffusion coefficient	10^{4} cm^{2} s^{-1}	Murray et al. (2005)
x	*x*-coordinate location		
y	*y*-coordinate location		
d	Euclidian distance between farm sites and predicted particle location		

[a]Scottish Environmental Protection Agency; available upon request

ancies between continual and discrete forms of SEIR are minimised, as the scale of the time-step is relatively small compared to the disease progression observed over some 3 mo in challenge experiments (Urquhart et al. 2008, Gregory et al. 2009).

An initial infected fish farm (Farm 1) is described as a closed population which has no mortality or recruitment and the transition with discrete-time-steps between: susceptible (S); exposed (E); infected (I) and recovered (R) hosts and can be expressed by an adapted discrete-time SEIR model (Eqs. 1 to 4):

$$S_{1,t+1} = S_{1,t} - \frac{\beta S_{1,t} I_{1,t}}{N_1} \quad (1)$$

$$E_{1,t+1} = E_{1,t}(1-\sigma) + \frac{\beta S_{1,t} I_{1,t}}{N_1} \quad (2)$$

$$I_{1,t+1} = I_{1,t}(1-\mu) + \sigma E_{1,t} \quad (3)$$

$$R_{1,t+1} = R_{1,t} + \mu I_{1,t} \quad (4)$$

where β is the transmission rate, σ is the infective rate and μ the removal rate. N_1 is the total farm biomass ($S_1+E_1+I_1+R_1$) of Farm 1.

In order for disease to persist within an infected population, R_0, the net rate of secondary infection caused by an infected individual (Anderson & May 1979), is represented in Eq. (5) as:

$$R_0 = N_1 \frac{\beta}{\mu\sigma} \quad (5)$$

For infection to persist in Farm 1, R_0 must be greater than or equal to one. For this system Eq. (6) represents the critical population size (N_c) and is:

$$N_{1c} = \frac{\mu\sigma}{\beta} \quad (6)$$

When considering the spread of infective particles to a secondary, susceptible farm, the initiation of the infection is caused by free-moving infective particles which pose a risk probability (ε) to the secondary farm. This risk value is a summation of factors such as distance between farms, pathogen decay, pathogen tidal-movement speeds and the number or dose of infective agents the susceptible farm is exposed to.

The SEIR model for the secondary farm is represented by Eqs. (7) to (10), and the remaining number of the particles in the water surrounding the secondary farm is expressed by Eq. (11), demonstrating the decay of particles shed from infective individuals.

$$S_{2,t+1} = S_{2,t}\left(1 - \varepsilon_t - \frac{I_{2,t}\beta(1-\varepsilon_t)}{N_2}\right) \quad (7)$$

$$E_{2,t+1} = E_{2,t}(1-\sigma) + S_{2,t}\left[\varepsilon_t + \frac{I_{2,t}\beta(1-\varepsilon_t)}{N_2}\right] \quad (8)$$

$$I_{2,t+1} = I_{2,t}(1-\mu) + \sigma E_{2,t} \quad (9)$$

$$R_{2,t+1} = R_{2,t} + \mu I_{2,t} \quad (10)$$

$$W_{2,t} = \gamma I_{1,[t-f(t)]} e^{-\lambda t} \quad (11)$$

$f(t)$ is the time taken for a particle cohort to be transmitted between sites which varies dependent on the current speeds. The risk to a secondary naïve farm at a given time point is related to the number of particles from infected individuals shed at a rate (γ) that survive a decay function related to time, and biological decay rate (λ) from infected individuals in the first farm, at a point in time determined by the separation distances. The infection rate (z) determines the proportional rate of infection for an individual exposed to the minimum infective dose of pathogen within the water.

Each individual requires a minimum dose (ϕ) in order to become infected. Should the remaining dose be sufficient to saturate the population with infection, all individuals in the population then instantaneously become infected.

$$\frac{zW_{2,t}}{N_2\phi} \geq 1, \varepsilon_t = 1 \quad (12)$$

Should the dose be sufficient to infect a proportion of the population, then this proportion becomes infected whilst the proportion $1-\varepsilon_t$ remains susceptible.

$$\phi < zW_{2,t} < N_2\phi,\ \varepsilon_t = \frac{zW_{2,t}}{N_2\phi} \quad (13)$$

When the dose is below the minimum infection threshold, the population remains disease free.

$$zW_{2,t} < \phi,\ \varepsilon_t = 0 \quad (14)$$

The threshold for infection to occur on a second farm can be expressed as:

$$I_{2,t+1} = S_{2,t+1}\frac{z\sigma W_{2,t}}{\phi} \geq 1 \quad (15)$$

where the secondary farm is completely susceptible, i.e. $S_2 = N_2$, the invasion condition is $\frac{z\sigma W_{2,t}}{\phi}$, and therefore independent of secondary farm size. This expression does not account for the ability of infection to persist within a farm, which is dependent on farm size (Eq. 5). The proportion of susceptible individuals within Farm 2 that become infected is the product of the invasion condition and the number of susceptible individuals within the farm, i.e. $\frac{z\sigma W_{2,t}S_{2,t}}{\phi}$, indicating that the resulting number of fish which become infected is greater in larger farms.

Therefore, there are 2 mechanisms to avoid a farm epidemic: being sufficiently separated from an infection source so as to not to become exposed, or being sufficiently separated that the exposure dose is too low to infect and support a self-sustaining infectious population.

There is a disease-free equilibrium when $W_{2,t} < \phi$, $(S_2,E_2,I_2,R_2) = (N2,0,0,0)$ and there is an unstable endemic equilibrium when $R_0 \geq 1$ (not expanded in this paper).

Combining Eqs. (6) and (11) with the invasion condition (Appendix 1) produces a term for the critical maximum number of time-steps (t_c) a particle cohort can survive to cause sustained infection in a secondary farm of varying size:

$$t_c = \frac{-\ln\left[\frac{\mu\phi}{S_2\gamma I_1 z\beta}\right]}{\lambda} \quad (16)$$

In order to provide representative outputs of the dispersal distance of a cohort of particles, $\gamma I_{1,[t-f(t)]}$ is assigned either the peak shed, half of peak shed or a quarter of the peak shed value derived from a source farm.

Within a given time the mean distance travelled by a particle undertaking a random walk, with random-sized steps of uniform distribution of variance, with a minimum step-length of a and a maximum step-length of b, has a mean step-length s where:

$$s = \frac{b-a}{\sqrt{12}} \quad (17)$$

The maximum step length for a particle at each time–step moving without random movements can be approximated to the size of movement due to additive constant movement and sinusoidal tidal current:

$$b = U\sin\left[\frac{2\pi t}{T}\right] + C \quad (18)$$

where U is the tidal current, T is the tidal period and C is the constant residual advection current, and for simplification (and maximisation of variance) it is assumed that a negative movement is equally likely, i.e. $a = -b$; therefore:

$$s = \frac{\left[U\sin\left[\frac{2\pi t}{T}\right] + C\right]}{\sqrt{3}} \quad (19)$$

By manipulating the hydrodynamic expressions for movement in the x- and y-axis presented by Murray et al. (2005), it is possible to provide a discrete expression for the mean distance travelled by a particle in a given time that has the remaining infectious capability of infecting a secondary farm:

$$\bar{x}_t = \bar{x}_{t-1} + \frac{s}{t}\sqrt{\frac{\frac{s}{t}D}{T}} + s\sqrt{3} \quad (20)$$

$$\bar{y}_t = \bar{y}_{t-1} + \frac{s}{t}\sqrt{\frac{\frac{s}{t}D}{T}} \quad (21)$$

With standard deviations for both x and y dimensions of:

$$\pm s\sqrt{t} \quad (22)$$

where x is the position along the x-axis of the horizontal plane, y is the y-axis position in the horizontal plane and D is the diffusion coefficient. An example of the mean distances travelled by a cohort of particles over a 2 wk period is shown in Fig. 2.

Therefore, it is possible to provide an expression for the maximum location reached by an infectious particle (and its standard deviations) as a function of farm size by replacing t with Eq. (16) in Eqs. (20) and (21).

To demonstrate the exposure distances for the example pathogens, characteristic parameters used within the presented model were obtained from the published literature for infectious pancreatic necrosis virus type (IPNV-t), infectious salmon anaemia virus type (ISAV-t) and *Aeromonas salmonicida* type (AS-t) pathogens (Table 1). Physical parameters used are the same as found in Murray et al. (2005) such that: D is 10^4 cm^2 s^{-1}, U is the maximum spring tide current of 51 cm s^{-1}, T is 12.42 h, C ranges between 1 and 8 cm s^{-1} and fish production unit size between 10^2 and 10^5 t of fish (SEPA, Fiskeridirektoratet 2011; see Table 1). It is assumed that $z = \beta$ for simplicity. Biolog-

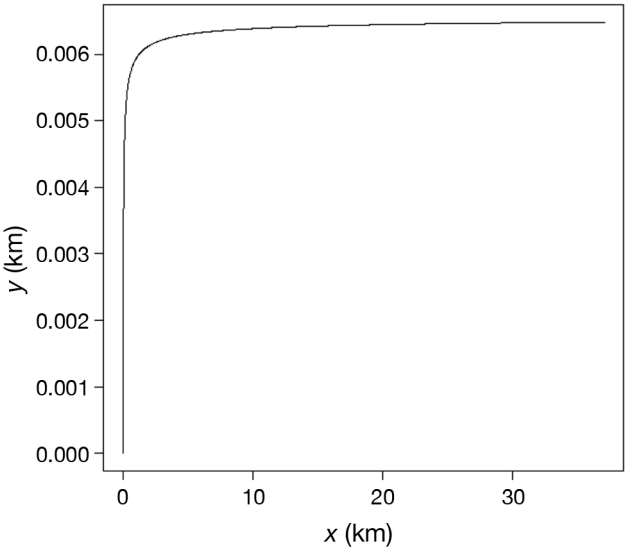

Fig. 2. Mean distances travelled by a cohort of particles over a 2 wk period

ical parameters were obtained from Smith et al. (2000) with the following mean daily rates of IPNV-t per fish: $\beta = 0.013$; $\sigma = 0.36$; and $\mu = 0.062$. For IPNV-t $\lambda = 1.6\%\ h^{-1}$ (Toranzo & Hetrick 1982). The ISAV-t parameter of $\lambda = 12\%\ h^{-1}$ was obtained from Løvdal & Enger (2002); mean daily rates of ISAV-t per fish were derived from Gregory et al. (2009): $\beta = 0.015$ (rate of mean cumulative mortality over the study period for immersion-challenged fish), $\sigma = 0.14$ (mean cumulative daily rate from intraperitoneal [i.p.]-injected fish to express viral particles) and $\mu = 0.04$ (mean cumulative time for i.p.-injected fish who underwent no mortality over the study period). Ögüt & Bishop (2007) provided parameter estimates for AS-t in Chinook salmon *Oncorhynchus tshawytscha*: $\beta = 0.0214$, and $\mu = 0.29$, $\sigma = 0.33$. The parameter estimate of $\lambda = 12\%\ h^{-1}$ for AS-t was derived from Rose et al. (1989).

The exposure risk posed by one infectious particle for a farm (ε) located at (x,y) at a given time point diminishes the further it is located from the mean location reached for that given time point $(\bar{x}_t,\bar{y}_t)$ based on the Euclidean distance (d) between the farm site and the mean resting location such that:

$$d = \sqrt{(|x| - \bar{x})^2 + (|y| - \bar{y})^2} \tag{23}$$

This is scaled by the numbers of particles located at (x_t,y_t) such that γI_t, the particles resting at location (x_t,y_t), are equal to: $\gamma I_{t-(t-1)}$; this allows for the incoming risk for a single cohort of particles at (x_t,y_t) to be defined as:

$$\varepsilon_{(x_t,y_t)} = \frac{\gamma I_{1,t-f(t)} \beta e^{-\lambda(1-d)t}}{\phi} \tag{24}$$

For any farm at a given location in this hypothetical system it is possible to predict the epidemic trajectory within that farm by including ε_t in Eqs. (7) to (10).

For ease and maximum transmission, the maximum risk distance from a farm with the mean Scottish consented biomass of 1400 t (Walker 2010) containing infected fish at reported prevalences is assumed to have the peak shedding rates (γ) of 6.8×10^3 $TCID_{50}$ ml^{-1} h^{-1} kg^{-1} for IPNV-t particles (Urquhart et al. 2008) and 7×10^1 ml^{-1} h^{-1} kg^{-1} for ISAV-t particles (Gregory et al. 2009). The minimum infectious doses (ϕ) are 10^{-1} $TCID_{50}$ ml^{-1} kg^{-1} for IPNV-t (Urquhart et al. 2008) and 10^1 $TCID_{50}$ ml^{-1} kg^{-1} for ISAV-t (Gregory et al. 2009). For rainbow trout *Oncorhynchus mykiss* challenged with *Aeromonas salmonicida* (Pérez et al. 1996) there was an associated minimum infective dose of 10^8 cfu ml^{-1} over 12 h exposure and a shedding rate of 1.75×10^6 cfu ml^{-1} h^{-1} (Rose et al. 1990). The prevalence for each pathogen is reported as 12.5% for IPNV-t in Scottish salmon (Bruno 2004) and 75% for AS-t from experimental infection of Chinook salmon (Ögüt & Reno 2005). For ISAV-t a prevalence of 30% is determined because it has been reported as ranging between 28 and 40% for Canadian farmed Atlantic salmon (McClure et al. 2005) whilst the recent ISA occurrence in the Shetland Isles demonstrated a prevalence of 30% (M. Hall pers. comm.). To demonstrate the change in the safe separation distances with variable dose responses simulations are produced with peak, half peak and quarter peak shedding. As it would be too time consuming to consider all time steps throughout a disease outbreak, these shedding points provide a general description of the influence of shedding intensity on transmission with peak being a worse case scenario. These shedding values are also akin to varying the size of the farm or the proportion of infected individuals on the farm. Although the number of particles shed would be similar under these scenarios, to avoid inconstancies due to internal disease transmission within farms of varying size (Krkošek 2010) it is beneficial to consider a range of conditions in farms of equal size. Likewise, considering many permutations of farm sizes and separation distances becomes intensive. Therefore, units are used representing smaller sized (500 t) farms and moderately sized (1250 t) farms currently present in Scotland and Norway, and larger farms of 2500 and 5000 t, which are currently found in Norway.

The separation distance scale used is for descriptive purposes and is based on the assumption that scaling of farm size produces the same scaled increase in shed particle cohort number. Thus, for the cohort to exhibit the same dose it requires the same scaled increase in time. For example, a doubling of farm size is assumed to produce double the number of particles shed, and thus a secondary farm is assumed to require double the separation distance in order to be exposed to the same dose as a source farm half its size.

Shed particles are moved by persistent currents at speeds, experienced in Scottish inshore waters (Lee & Ramster 1981), ranging from 1 to 8 cm s^{-1} at 1 cm s^{-1} intervals. The production units used represent the smaller farms (10^2 t) in Scotland, increasing to moderate–sized farms ($10^{2.5}$ t) and to larger farms (10^3 t). In recent years production has been administered at a DMA level where farms are clustered together and considered as one production unit; it is, therefore, possible to consider these DMA units as large farms. Hence, DMA production scenarios with 'farm units' greater than single farms are considered to be those from $10^{3.5}$ up to 10^5 t, therefore providing possible

separation distances required to avoid transmission between DMAs.

Simulations were conducted in R 2.8.1 (R Development Core Team 2008) in order to ascertain separation distances in a range of simulations representing varying tidal speeds, farm sizes and pathogen characteristics.

RESULTS

The mean distances covered by all particles (Fig. 2) over a 2 wk period demonstrate that there is considerably more movement in the *x*-dimension compared to the *y*-dimension within the simulation hydrodynamic model. Therefore, safe distance calculations can be made based on the movement in the *x*-dimension.

The mean simulated safe distances needed to avoid persistent infection for farms exposed to the peak, half peak and one-quarter peak shed of IPNV-t, ISAV-t and AS-t pathogens from an infected 1400 t farm are shown in Fig. 3. As secondary farm size, shedding dose from a source farm, and residual tidal current speed increase for each of the pathogen types, the separation distance required to avoid infection also increases.

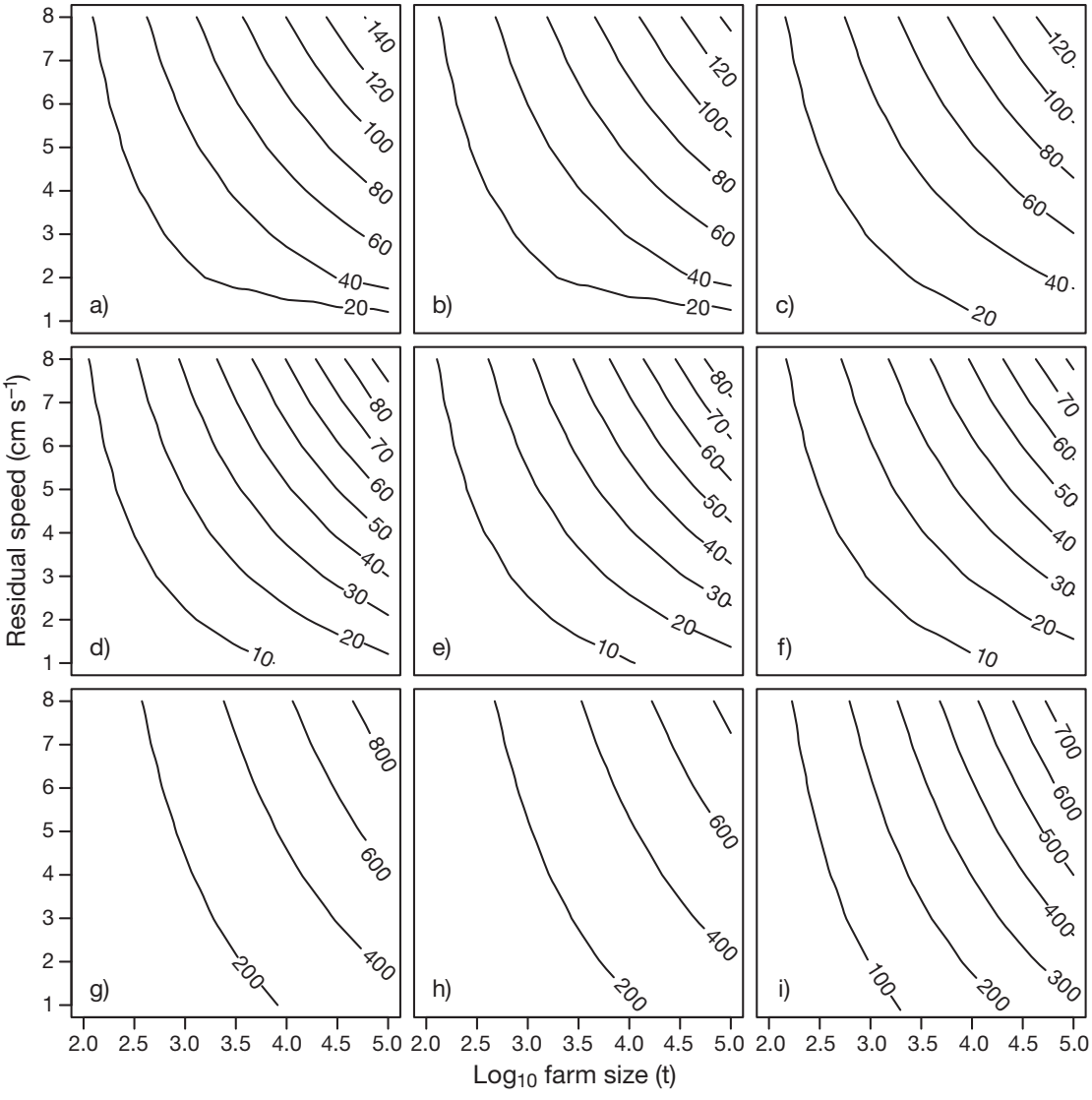

Fig. 3. Mean distances (km) at which a farm of a given size in varying current speeds avoids having a persisting epidemic when exposed to a 1400 t farm infected with reported prevalence of: *Aeromonas salmonicida* type (AS-t) at (a) peak shed, (b) half peak shed, (c) a quarter peak shed; infectious salmon anaemia type virus (ISAV-t) at (d) peak shed, (e) half peak shed, (f) a quarter peak shed; or infectious pancreatic necrosis virus type (IPNV-t) at (g) peak shed, (h) half peak shed, (i) a quarter peak shed

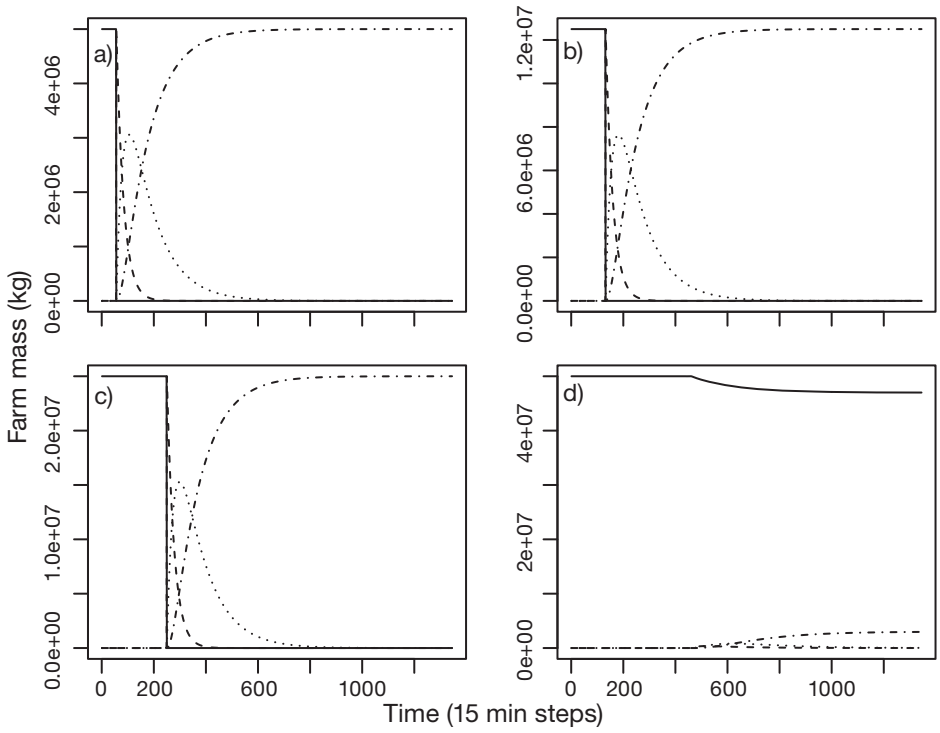

Fig. 4. Epidemic trajectory over a 2 wk period of varying farms infected by the peak shed of infectious salmon anaemia type virus (ISAV-t) particles from a 5 t infected farm located at varying distances: (a) a 5×10^2 t farm separated by 1 km, (b) a 12.5×10^2 t farm separated by 2.5 km, (c) a 2.5×10^3 t farm located 5 km from the origin, and (d) a 5×10^3 t farm located 10 km away. Solid line: susceptible, *S*; dashed: exposed, *E*; dotted: infected, *I*; dot-dash: recovered, *R*

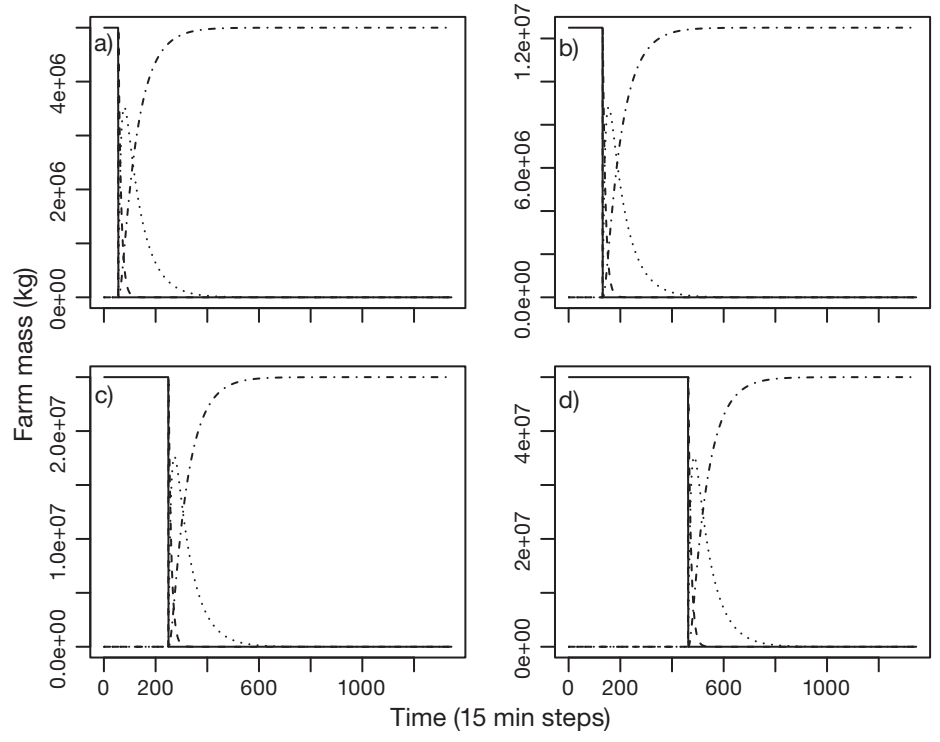

Fig. 5. Epidemic trajectory over a 2 wk period of varying farms infected by the peak shed of infectious pancreatic necrosis virus type (IPNV-t) particles from a 5 t infected farm located at varying distances: (a) a 5×10^2 t farm separated by 1 km, (b) a 12.5×10^2 t farm separated by 2.5 km, (c) a 2.5×10^3 t farm located 5 km from the origin, and (d) a 5×10^3 t farm located 10 km away. Solid line: susceptible, *S*; dashed: exposed, *E*; dotted: infected, *I*; dot-dash: recovered, *R*

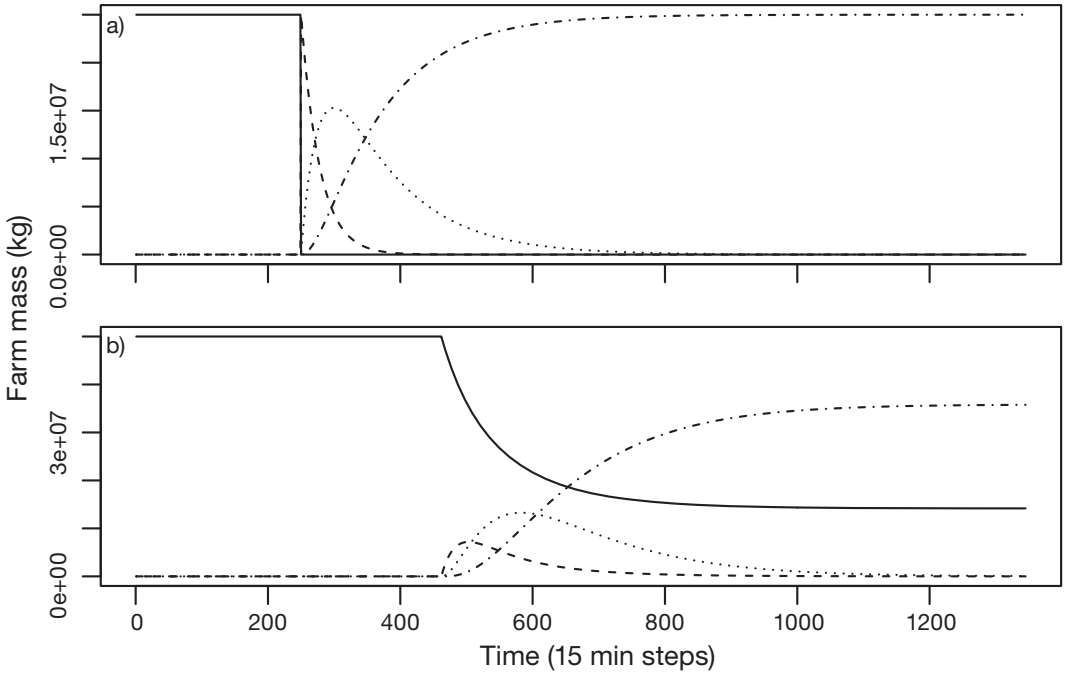

Fig. 6. Epidemic trajectory of (a) a 2.5×10^3 t farm separated by 5 km from a same size farm with 5% prevalence ISAV-t pathogen (infection causing complete infection) and (b) a 5×10^3 t farm separated by 10 km from another farm of the same size with 5% infection of an ISAV-t pathogen causing ~88% infection. Solid line: susceptible, *S*; dashed: exposed, *E*; dotted: infected, *I*; dot-dash: recovered, *R*

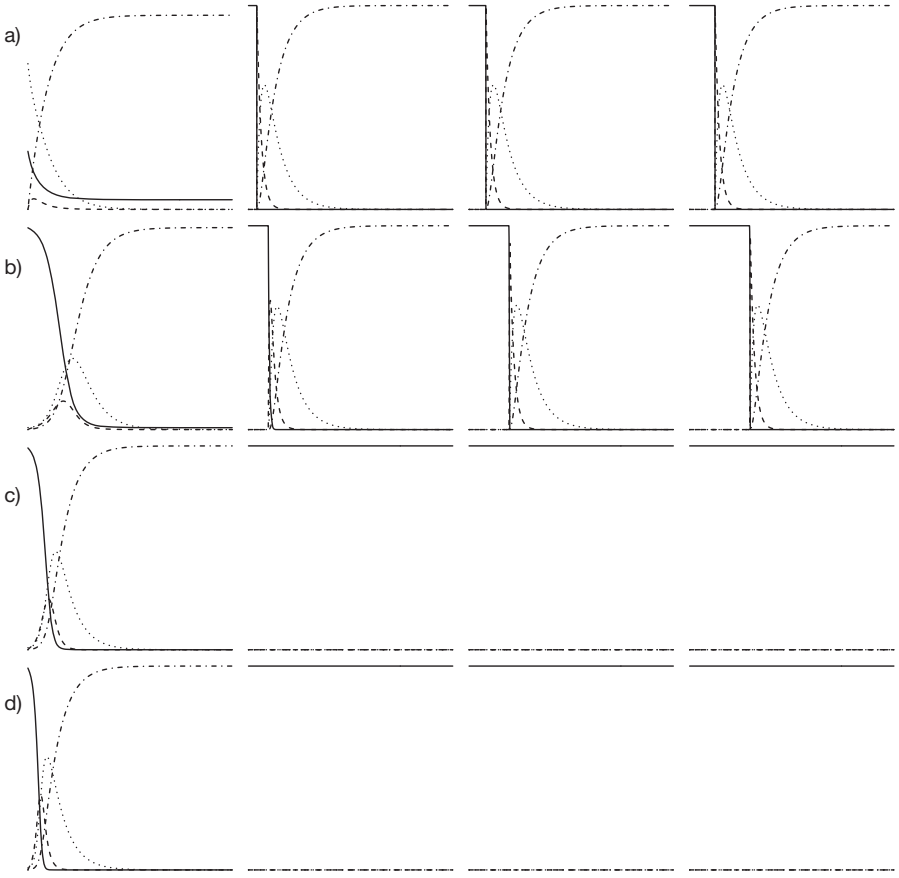

Fig. 7. Disease progression over a 2 wk period from a 1% prevalence infectious salmon anaemia type virus (ISAV-t) initially infected site between farms in sequence: (a) 5×10^6 t at 1 km intervals; (b) 1.25×10^7 t at 2.5 km intervals; (c) 2.5×10^7 t at 5 km intervals; (d) 5×10^7 t at 10 km intervals. Axis scale is removed so that comparison in total disease incidence across sites can be compared. Solid line: susceptible, *S*; dashed: exposed, *E*; dotted: infected, *I*; dot-dash: recovered, *R*

The simulation model provides an example (Fig. 4) of smaller farms close to the source of infection which have larger infection numbers than larger farms which are further away when exposed to ISAV-t. A 5×10^2 t farm located 1 km downstream experiences complete infection (Fig. 4a), a 12.5×10^2 t farm located 2.5 km downstream experiences ~95% infection (Fig. 4b), a 2.5×10^3 t farm located 5 km from the origin experiences some 36% infection (Fig. 4c), and Fig. 4d demonstrates a 5×10^3 t farm located 10 km away causing some 393 kg of infected production. Fig. 5 demonstrates that, irrespective of separation distance within the simulation and of farm production, a highly conserved pathogen such as IPNV-t will lead to complete infection for farms even when separated by 10 km.

An example comparison of production strategies is considered in order to produce 5×10^3 t fish. The epidemic trajectory of two 2.5×10^3 t farms separated by 5 km from a same size farm with 5% ISAV-t pathogen (Fig. 6a) causes complete infection whereas a 5×10^3 t farm separated by 10 km from another farm of the same size with 5% prevalence of an ISAV-t pathogen causes ~88% infection (Fig. 6b).

Fig. 7 shows a sequence of 4 equal-sized farms separated by equal distances allowing for pathogens to be transmitted from a source farm to the fourth farm via intermediate farms. More of the total production becomes infected when smaller farms are located closer together compared to when larger farms are highly separated.

DISCUSSION

This paper demonstrates a model which combines a SEIR model, a reservoir-SEIR model and a simplified hydrodynamic transport model in order to produce a framework to assess the impact of farm size on the transmission of pathogens within aquaculture.

A SEIR model in discrete-time represents the initially infected farm. Similar discrete forms of epidemic models have been used in a variety of studies, such as the description of rabies in predator populations (Allen et al. 2002) and gene frequency and disease spread in plant populations (Kesinger et al. 2001). By modelling in a discrete time format, it is possible to replicate systems where there are non-continuous generations or production cohorts, such as the case in salmonid aquaculture. This aspect of the model acts as a source of infectious particles which are then transported towards a secondary naïve farm through hydrodynamic pathways.

The hydrodynamic model is a modified version of that presented by Murray et al. (2005) which accounts for the mean location reached by each cohort of particles derived from a source farm. This process removes the need for multiple hydrodynamic simulations in order to capture stochastic variability. The model is essentially a 2D dispersion model which is dominated by horizontal movements away from farms along the *x*-axis.

As the cohort of particles are transported predominantly along the *x*-axis with little movement on the *y*-axis (Fig. 2), they decay as a function of time and biological decay and therefore the effective number of particles for each cohort of particle reservoir is diminished. Infection occurs when the number of particles in the reservoir and the infection probability is greater than the minimum infective dose. The rate of infection is assumed to spread equally amongst the susceptible individuals within the naïve farm. The invasion condition and persistence condition used within this paper indicate that farm size is not a factor in a farms' susceptibility to infection. However, persistence is conditional on a critical population size, as is the rate of epidemic development. For simplicity, the model assumes that, should the dose exceed the minimum infective dose, then infection will occur. This infectious class will then propagate the disease through internal contact with susceptible individuals. The secondary SEIR phase has unstable endemic states in this model as shown for the basic SEIR model (Li & Muldowney 1995), but this is further complicated by the external influx of disease-inducing particles. The analysis of such states is not considered here. Due to this complication, assessment of separation distances is conducted using the peak shedding rate, as this will cause the largest number of pathogen particles. Here, we demonstrate that safe separation distances increase for IPNV-t, ISAV-t and AS-t pathogens exposing farms that increase in size in increasing residual current flows. The more rapidly the pathogens decay, the less separation distance is required. Likewise, when farms are exposed to half and quarter maximum doses (representing a reduction in infected individuals in the initial farm), the separation distance required to prevent risk of infection is reduced. Reducing farm size decreases the number of shed pathogens; therefore, farms could be located closer together. However, when farms are closer together, they obtain a larger incoming particle reservoir and therefore experience greater infection numbers.

As production is concentrated in larger farms, there are fewer of these and therefore separation dis-

tances can be increased. In order to assess the disease dynamics of differing production systems the model is considered with farms producing 5×10^3 t in 4 production methods:

(1) Ten farms stocked with 5×10^2 t, located 1 km from an initial source,

(2) Four farms stocked with 1.25×10^3 t located 2.5 km from an initial source,

(3) Two farms stocked with 2.5×10^3 t located 5 km from an initial source,

(4) One farm stocked with 5×10^3 t located 10 km from the initial source.

The closer the farms are together, the higher the percentage infection per farm (Fig. 4) by labile pathogens. However, when exposed to highly conserved pathogens, a complete infection of the farm occurs (Fig. 5). This demonstrates that the optimal aquaculture production strategy to avoid infection of individuals on a farm is to have larger separation distances and larger farms, compared to many small farms in close proximity. Fig. 6 presents infection from an initial 5 t infected source. When considering the effect of many small and nearby farms versus a few separated and larger farms on additional farms downstream, simulations indicate that 2 farms separated by 5 km and stocked with 2.5×10^3 t leads to more infected individuals compared to a situation where a larger 5×10^3 t stocked farm is separated by 10 km. Furthermore, the rate of infection within the larger, more separated farms is decreased, whereas in smaller nearby farms the spread of infection throughout the individuals within the farm is almost instantaneous. This is an important factor in monitoring and mitigation strategies which can be implemented by farm operators. With lower rates of infection the farm operators may be able to act before the epidemic peaks, thus preventing spread to downstream farms, whereas infection in a smaller farm system requires immediate response and may still lead to substantial infection in downstream farms. When considering farms in sequence (Fig. 7), smaller farms clustered together experience disease incidence, whereas larger farms separated further apart do not demonstrate infection. For smaller farms in sequence there is a time lag before incidence of disease based on distance away from the source, with those further away becoming infected later than those nearby. This is a similar pattern to the recent ISA outbreak in Shetland, where incidence reporting occurred later with increased distance from the initially infected site (Murray et al. 2010).

Separation distances for large farms may lead to a requirement for greater separation distances for MA-based production. Here, production unit size varies representing moderate- to large-sized farms up to and through the DMA scale. Current ISAV DMAs are based on tidal excursion distances of 7.2 km for mainland Scotland and 3.6 km for the Shetland Islands. These were developed using simple yet robust tidal models (Scottish Executive 2000); DMAs are continuous over the area in which adjacent farms overlap, so a separation distance of greater than 14.4 (or 7.2 km in Shetland) is required for a DMA boundary. Fig. 3d–f indicate that these DMAs are likely to be appropriate for current production levels in mainland Scotland and the Shetland Islands for ISAV-type pathogens when farms are located in low residual current areas. However, they become unsuitable for more robust pathogens. Should farms be increased in size or be situated in faster current locations, such as offshore, it is possible that the DMAs will need to be reconsidered. However, even DMAs with imperfect boundaries can be useful for the management of disease (Werkman et al. 2011).

For this work a simplified discrete model that provides uniform transmission from source farm to naïve site was used in order to assess the role of farm size in the transmission of pathogenic diseases between hydrodynamically connected farms. Previous work by Scheel et al. (2007) developed a stochastic probabilistic model of ISA transmission based on empirical information relating to individual farms, including a biomass parameter, seaways separation distance, as well as local contact networks. Aldrin et al. (2010) amended Scheel et al.'s model to include a time-constrained measure of cohort size and previous farm infection status and applied parameter estimates for heart and skeletal muscle inflammation (HSMI) and PD in addition to ISA. Although this model can not be directly validated, as it would be unethical to allow pathogenic disease to progress within a farm, nor does this study include estimations of network-based risk, it is demonstrated that risk avoidance distances are comparable to previous work. The relative infection rate from ISAV is radically reduced for farms separated by more than 11 km (Aldrin et al. 2010), which is consistent with earlier assertions that much of the ISA risk is concentrated within 5 km (Scheel et al. 2007) of a farm and that no significant risk exists beyond 10 km (Scottish Executive 2000). Additionally, Green (2010) argues that localised clusters of farms (akin to a large farm unit in the present study) separated by increased distances slow the spread of a disease within a production system.

For robust pathogens, risk may exist over very large hydrographic distances similar to transmission

distances for airborne pathogens in terrestrial systems of 300 km (Sørensen et al. 2000) with recorded transmission up to 200 km (Schley et al. 2009). However, the risk in aquaculture could be considered as a worse case scenario as it is dependent on persistent, reasonably high velocity currents for prolonged periods without deposition or non-linear dispersion, and without sufficient turbulent mixing. An example where potential and observed long-distance dispersal differs is provided by the invasive Mediterranean mussel *Mytilus galloprovincialis* in South Africa (McQuaid & Phillips 2000). Although it was expected that larval dispersal could occur up to 220 km, 90% of the larvae were sampled within 5 km, and due to changing wind directions, maximum dispersal distances ranged between 54 and 165 km. Likewise, although pathogens could disperse over long distances, it is probable that existing separation distances between DMAs, while far smaller than potential transfer distance, could still be reasonably helpful for disease control of even robust pathogens. Furthermore, it must also be noted that the existence of the pathogens near farms does not necessarily mean infection will occur (Murray 2009).

The framework in the present study is limited by the use of simplified assumptions for both the biological and hydrodynamic components. The conditional expression for transmission based on minimum infectious doses is likely to have a functional response (e.g. Joh et al. 2009) as opposed to simply saturation, proportional or disease avoidance. However, the minimum infectious doses presented in the literature describe discrete thresholds as opposed to dose responses. In the environment, pathogens have variable decay rates when exposed to different conditions in e.g. salinity, pH and temperature (e.g. Toranzo & Hetrick 1982, Rose et al. 1990). In our study, the physical environment is assumed to be homogenous, allowing extended pathogen survival for possible further transmission. Our model takes no account of bathymetry, topography or the presence of obstacles that prevent long-distance transmission. Persistent currents are unlikely to occur over such distances demonstrated in our model, whilst at the same time infrequent, stochastic transmission events could still occur beyond distances presented here. Therefore, the simplified structure allows for a trade-off between transmission events. In order to provide accurate dispersal models, 3D oceanographic circulation models are required (e.g. Venayagamoorthy et al. 2011). This would greatly increase the complexity and limit the general applications of the model; as they are site specific, this makes them inapplicable for assessing issues regarding dispersal scale between farms of varying size. Amundrud & Murray (2009) combined fish disease agent characteristics with system-specific particle dispersal models for sea lice dispersal in Loch Torridon; however, this required high levels of computational processing and model validation and only provided characteristics for one individual system.

The present study is concerned with the alteration of disease transmission by varying farm size and separation distance and does not consider environmental impacts. However, recent work by Mayor et al. (2010) suggests that overall environmental efficiency may be improved by locating larger farms in faster currents. They demonstrate that a critical threshold exists between 800 and 1000 t where there is no additional rate of benthic biology degradation for farms located in increased currents. Likewise, a similar farm size threshold occurs for minimal changes in benthic chemistry measurements derived from fish-farm waste (Mayor & Solan 2011).

Our paper only addresses the role of farm size in disease transmission through hydrodynamic pathways. It must be noted that farm size may have a role in disease transmission through alternative routes, such as fish movement networks (Green et al. 2009, Munro & Gregory 2009), whereby larger farms may have more sources of smolt inputs. Larger farms may also have altered biosecurity practices, which changes their susceptibility to disease outbreaks. For example, increased wellboat movements between larger sites may transmit pathogens more frequently whilst larger farms may be able to implement more stringent disease monitoring practices. Clearly, the role of farm size in alternative methods of disease transmission needs additional consideration.

Although this is a simple model system, it highlights the fact that, as unit size increases, farms experience higher numbers of infections. In order to improve production efficiency larger farms located further apart experience fewer infections compared to many smaller farms closer together with similar overall stocking levels. Furthermore, not only do larger, more separated farms experience fewer infective individuals, the rate at which the population becomes infected is reduced, thus allowing for disease monitoring and intervention management to take place to prevent further transmission.

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Appendix 1

The derivation of the critical time for a cohort to lead to sustained infection is:

$$N_{1c} = \frac{\mu\sigma}{\beta} \quad (6)$$

$$W_{2,t} = \gamma I_1 e^{-\lambda t} \quad (11)$$

Simplify for time delay between sites and insert Eq. (11) into Eq. (15):

$$I_2 = S_2 \frac{z\sigma\gamma I_1 e^{-\lambda t}}{\phi} \quad (11a)$$

Insert the critical number of infectious individuals needed to sustain an infection without further external inputs into Eq. (6):

$$S_2 \frac{z\sigma\gamma I_1 e^{-\lambda t}}{\phi} = \frac{\mu\sigma}{\beta} \quad (11b)$$

Eq. (11b) corrected after online publication

Rearrange (11b):

$$t_c = \frac{-\ln\left[\frac{\mu\phi}{S_2\gamma I_1 z\beta}\right]}{\lambda} \quad (16)$$

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