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Short communication

Estimation of the reproduction number of salmon pancreas disease virus subtype 3 in homogeneously mixed populations of Norwegian farmed Atlantic salmon

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ABSTRACT

The reproduction number (R) of salmon pancreas disease (PD) was estimated within homogeneously mixing populations (within-cage) of Norwegian farmed Atlantic salmon (*Salmo salar* L.) based on data collected during PD epidemics from 10 cages at 2 farming sites. Two approaches were used: (a) estimation of an overall reproduction number ($R_{(cmd)}$) and a time-dependent reproduction number ($R_{(t)}$) using mortality records during PD epidemics, and (b) estimating the reproduction number during the early stage of infection ($R_{(sd)}$) based on data from a surveillance program for SPDV subtype 3. The $R_{(cmd)}$ estimates based on the mortality data ranged from 1.02 to 1.45, and the $R_{(sd)}$ estimates ranged from 1.0 to 2.9. Plots of the $R_{(t)}$ estimates covering the whole epidemic period yielded an increasing slope prior to SPDV3 detection. This study presents a framework for the quantitative measurement of a PD epidemic that could be useful for the evaluation of prevention methods. The time-dependent $R_{(t)}$ estimate can provide an early warning of PD outbreaks.

1. Introduction

Pancreas disease (PD) is an infectious disease of Atlantic salmon that causes substantial economic losses in major producing countries including Norway, Scotland, and Ireland. The etiological agent is salmon pancreas disease virus (SPDV) of which at least 6 subtypes have been identified. A comprehensive review of PD and SPDV is given by McLoughlin and Graham (2007). In Norway, the first PD epidemic was officially confirmed more than 2 decades ago (Poppe et al., 1989). Due to a considerable difference in PD occurrence along the Norwegian coastline, the country has been divided into 2 areas separating the disease-free area in the North from the PD endemic area in the South. A commercial vaccine against PD was introduced in 2007 and is commonly used in most Atlantic salmon farms in the endemic area. PD continues to be the major cause of losses in the affected area of Norwegian aquaculture despite knowledge concerning appropriate prevention and control strategies.

Knowledge concerning PD epidemics at the smallest and practically manageable unit (cage) is a crucial starting point for the evaluation of methods to reduce losses and prevent further spread. The magnitude of a PD epidemic can be measured by using the reproduction number (R), which is defined as an average number of secondary infections rising from a single infectious individual during its entire infectious period (Anderson and May, 1992). When an infectious individual is introduced into a completely susceptible population, the value of R is maximal and is called

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the basic reproduction number (R_0); however, in a partially protected population the number of secondary infections can be fewer. R_0 is not as effective for estimating disease transmission in endemic situations because the population will not be fully susceptible (Cintron-Arias et al., 2009).

The primary aim of the present study was to estimate R for PD in homogeneously mixing populations (within-cage) of farmed Atlantic salmon using observed data at PD-infected sites. R was estimated based on: (a) mortality records during PD epidemics, and (b) repeated test results from a PD screening program.

2. Materials and methods

2.1. Study population and data collection

Two marine farming sites rearing Atlantic salmon, named site A and site B, were included in the study. Site A consisted of 6 cages with approximately 200,000 fish per cage. Site B consisted of 4 cages each with approximately 190,000 fish per cage. Study populations were located within the PD endemic area and all fish were vaccinated against SPDV3. PD outbreaks were officially confirmed in all cages of both sites during the study period (2011–2012). Daily mortality records from the time of sea-transfer of smolts to the end of production were obtained from farm owners. Study sites also participated in a voluntary screening program for early detection of SPDV3 that consisted of biweekly collection of 10 fish (a combination of moribund and random) per site. Collected fish were tested for SPDV3 using RT-PCR test kit (PatoGen Analyze AS). The number of samples was increased to 10 fish per cage and collected weekly if the virus was detected at the site. Study sites were monitored until SPDV3 was detected in all cages.

2.2. Estimation of R

Overall $R_{(\mathrm{cmd})}$ and time-dependent $R_{(t)}$ reproduction number was estimated using the change in daily mortality over the entire PD epidemic period. The $R_{(\mathrm{cmd})}$ was estimated from the attack rate (final size) of PD using the approach of Dietz (1993). We assumed the epidemic started when SPDV3 was first detected at the site (126 days and 150 days after sea-transfer in sites A and B, respectively) and then calculated the cumulative mortality and the proportion of remaining susceptible fish S_∞ at the end of the epidemic. Then, $R = (\ln S_0 - \ln S_\infty)/(S_0 - S_\infty)$; where S_0 is the initial susceptible population. We also estimated the $R_{(t)}$ for the entire PD epidemic period according to Wallinga and Teunis (2004). The time-dependent $R_{(t)}$ estimation requires knowledge of the generation time (the time lag between onset of clinical signs in a primary and a secondary case). The generation time was assumed to be 30 ± 7 days, which was the incubation periods derived from a cohabitant trial (Graham et al., 2011). For each cage, an estimate of $R_{(\mathrm{cmd})}$ and $R_{(t)}$ along with 95% confidence intervals was calculated using the R0 package in R open-source software (Obadia et al., 2012; R Development Core Team, 2012).

For the second approach, we obtained time-sequential results from the SPDV3 screening program employing RT-PCR. The approach was based on the assumption that during early stages of an epidemic, the number of infected individuals I_t is approximately equal to $I_0 * e^{(R-1)*\gamma*t}$; where γ is a recovery rate or a reciprocal of an infectious period, and I_0 is an initial number of infectious individuals (Drake and Rohani, 2010). Taking logarithms of both sides, the logarithm of I_t is a linear function of time with a slope dependent upon both R and γ. The recovery rate was assumed to be 0.034 per day based on results of a cohabitant trial (Graham et al., 2011). We fitted a cubic spline (i.e. a smooth interpolation of the data) for the number of infected fish and the logarithm of I_t was regressed on time. We performed this analysis using the function "spline" in R open-source software.

3. Results

3.1. Mortality and surveillance

At site A, the cumulative mortality was 37% on average, and the cage-specific cumulative mortality ranged from 2% to 56%. SPDV3 was first detected by the surveillance program in cage number 2 and spread to all cages within 4 months of first detection. The virus was detected in cage number 1, 2, 3, 4, 5, and 6 at Day 235, 126, 193, 193, 193, and 235, respectively after the sea-transfer. At site B, the overall cumulative mortality was 8% and the cage-specific cumulative mortality ranged from 6% to 10%. SPDV3 was first detected in cage number 2 and spread to all 4 cages within 2 months. The virus was detected in cage number 1, 2, 3, and 4 at Day 241, 150, 241, and 225, respectively.

3.2. Estimates of the reproduction number (R)

The R estimates based on the cumulative mortality data, $R_{(\mathrm{cmd})}$, ranged from 1.14 to 1.45 in site A, and ranged from 1.02 to 1.03 in site B. The R estimates based on the surveillance data, $R_{(\mathrm{sd})}$, ranged from 1.8 to 2.9 in site A, and ranged from 1.0 to 2.2 in site B. The $R_{(\mathrm{sd})}$ estimates were higher than those of $R_{(\mathrm{cmd})}$ for each cage but cage-to-cage variation was observed both within and between sites. Plots of time-dependent R estimates, $R_{(t)}$ by cages were bimodal with the higher numbers in the first peak than the second peak of the epidemic (Fig. 1). Prior to the beginning of infection, the $R_{(t)}$ was approximately 1, and the slope increased before the detection of SPDV3 by the surveillance program. Similar plots were observed among cages of the same site.

4. Discussion

The results of this study provide a framework to quantify the magnitude of PD epidemics in Norwegian farmed Atlantic salmon populations. The findings suggest that mortality data can be used to anticipate an outbreak and act as an indicator for measuring the impact of PD. Our study sites were located in the PD-endemic area where viable SPDV3 is likely to circulate in the environment because the virus can survive in seawater for some time (Graham et al., 2007). Vaccination is a common practice for salmon farms located in Norwegian PD endemic area and all cages in our study were vaccinated. The PD outbreak in our study cages provided an excellent opportunity for estimating