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The epidemiological and economic effects from systematic depopulation of Norwegian marine salmon farms infected with pancreas disease virus

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ABSTRACT

Pancreas disease (PD) is a viral disease associated with significant economic losses in Scottish, Irish, and Norwegian marine salmon aquaculture. In this paper, we investigate how disease-triggered harvest strategies (systematic depopulation of infected marine salmon farms) towards PD can affect disease dynamics and salmon producer profits in an endemic area in the southwestern part of Norway. Four different types of disease-triggered harvest strategies were evaluated over a four-year period (2011–2014), each scenario with different disease-screening procedures, timing for initiating the harvest interventions on infected cohorts, and levels of farmer compliance to the strategy. Our approach applies a spatio-temporal stochastic model for simulating the spread of PD in the separate scenarios. Results from these simulations were then used in cost-benefit analyses to estimate the net benefits of different harvest strategies over time. We find that the most aggressive strategy, in which infected farms are harvested without delay, was most efficient in terms of reducing infection pressure in the area and providing economic benefits for the studied group of salmon producers. On the other hand, lower farm compliance leads to higher infection pressure and less economic benefits. Model results further highlight trade-offs in strategies between those that primarily benefit individual producers and those that have collective benefits, suggesting a need for institutional mechanisms that address these potential tensions.

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1. Introduction

Pancreas disease (PD) is an emerging infectious disease associated with significant economic losses in Scottish, Irish, and Norwegian marine salmon farms (Jansen et al., 2016). The causative agent for PD is Salmonid alphavirus (SAV), which belong to the genus Alphavirus, family Togaviridae, a spherical, enveloped, and single-stranded RNA virus (Weston et al., 1999). PD-virus has been found in wild- marine flat fish and Atlantic salmon (*Salmo salar*) (Snow et al., 2010; Biering et al., 2013), but farmed salmon is recognized as the main reservoir (Stene et al., 2014; Jansen et al., 2015a; Svåsand et al., 2015). The most important infection route for PD-virus is horizontal transmission via sea water (Kristoffersen et al., 2009; Kongtorp et al., 2010; Jansen et al., 2010a; Graham et al., 2011), where infected salmon populations may stay asymptomatic for several months before a clinical outbreak becomes evident (Graham et al., 2006; Jansen et al., 2010a, 2010b; Stene et al., 2014). Two subtypes of pancreas disease virus, SAV2 and SAV3, are responsible for present outbreaks in Norway. SAV3 is endemic along the coast from Hustadvika in mid-Norway and southwards, while SAV2 is endemic (from about 2012) from mid-Norway and northwards up to the southern border of Nord-Trøndelag (per January 2016).

Vaccination, functional feed, improved genetics, and biosecurity measures are used to reduce losses and prevent virus transmission between farms. However, these measures have proven insufficient to control PD (Pettersen et al., 2015a). Pettersen et al. (2015b) demonstrated that using prescheduled harvests to avoid losses from a PD (SAV3) outbreak can be economically beneficial for a
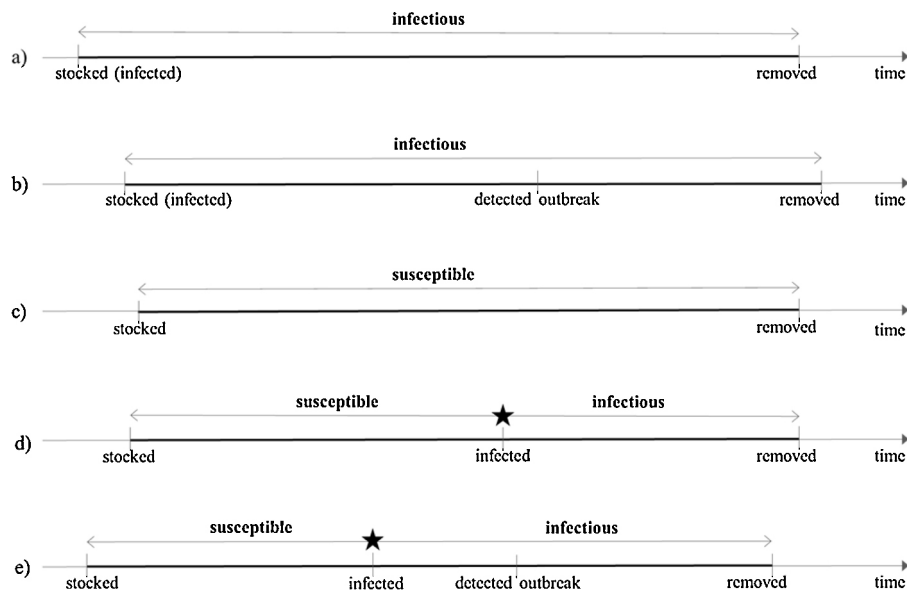
Fig. 1. Illustration of the five possible pattern of infection between stocking and removal (harvest): a) stocked with infection, but no detected outbreak, b) stocked with infection and detected with outbreak, c) stocked and not infected, d) stocked and infected, but no detected outbreak, and e) stocked and infected with detected outbreak (modified from Aldrin et al. (2015)).

farm in the late stages of the production cycle, referred to as a 'disease-triggered harvest strategy'. This strategy requires surveillance to detect virus levels in the salmon stock, which can serve as an early warning of an impending outbreak. Harvesting infected farms prior to outbreaks also reduces the disease spillover to neighboring farms, thus acting as a positive externality by breaking the transmission pathway and reducing the virus reservoir. The coordinated use of disease-triggered harvest strategies in a region may thus decrease the infection pressure in the area and make it economically rational to harvest farms with economic losses to benefit the larger group of salmon producers.

In this study, we investigate how disease-triggered harvest strategies affect the PD dynamics and producer profits when used as a shared strategy in a PD-endemic area (SAV3). We apply a recently developed spatio-temporal stochastic model for simulating the spread of PD in separate scenarios (Aldrin et al., 2015), and combine this model with a bio-economic model in a cost-benefit analysis to estimate the net benefits from using such harvest strategies.

2. Materials and methods

Marine salmon production in the SAV3 endemic area takes place in management areas that consist of several farms, and where smolt transfer and fallowing periods are synchronized. The fish are typically kept at the farm for growth for about 1.5 years before a harvest. After a harvest, it is mandatory to fallow the farm for minimum 2 months before a new production cycle can be initiated. A farm generally contains only fish of the same year class. Occasionally, the fish is moved from one farm to another empty farm during the growth period.

2.1. Disease spread simulations

2.1.1. The epidemiological model

The model used for simulating the spread of PD was previously presented in detail in Aldrin et al. (2015), but the model parameters has been re-estimated based on additional data. The model is a spatio-temporal stochastic model for the spread of PD in marine fish farms producing salmonids, i.e. Atlantic salmon and rainbow

trout (*Oncorhynchus mykiss*), in Norway. It is a model constructed to account for known, likely, or potential transmission pathways between farms and for factors that may affect susceptibility or infectiousness (e.g. the number of farm animals).

The fish population in a salmon farm within a production period (from stocking to removal) is termed a cohort, and this is the basic data unit in the model. In the model, a cohort is either susceptible to infection, or it is infected and then also infectious to other cohorts. Furthermore, each cohort can either i) already be infected when it is stocked, ii) become infected later in the production period, or iii) remain uninfected until it is removed (mostly harvested, but occasionally moved to another farm). The majority of infected cohorts become infected after stocking. Once a cohort becomes infected it stays infected until removed, but its relative infectiousness varies over time. This relative infectiousness is proportional with the proportion of infected fish, and is modelled by an internal seasonal SIR (susceptible–infected–recovered) model, which typically reaches its maximum a few months after infection (see examples in Supplementary Fig. 1b) and 1c)). Furthermore, an infected cohort may develop a clinical disease outbreak after the infection. However, some cohorts may remain infected without the infection ever proceeding to an outbreak, but they may still infect other cohorts (Fig. 1).

Fig. 2 provides an overview of the model. A cohort is stocked at a fixed time and treated as a fixed quantity in our model, with a probability ρ for already being infected when stocked. A cohort that is uninfected at stocking is susceptible and can be infected with a rate $\lambda(t)$ which varies over time. The infection rate includes the following four transmission pathways for infection after stocking: i) infection from infectious cohorts in the neighbourhood, depending on, among others, sea distance to infected cohorts and the proportion of fish infected at these cohorts; ii) infection from infectious cohorts in the same local contact network (defined as farms within the same concession, typically with shared ownership and proximity within farms); iii) infection from previously infected cohorts at the same salmon farm; and iv) infection via other, non-specified, pathways. The first of these pathways, infection from neighbouring farms, is by far the most important. An infected cohort may further develop a detected clinical outbreak with a time varying rate $\kappa(t)$, which is proportional to the proportion of fish infected