

The Role of Fish Density in Infectious Disease Outbreaks

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Introduction

In food animal production, crowding of animals in the necessarily intensive culture environment is well-known to increase the risk of infectious disease outbreaks. In aquaculture, estimation of carrying capacity on a loading rate (kg/L/min) basis accounts for consumption of oxygen and feed and production of carbon dioxide and ammonia (Westers, 1981; Colt and Watten, 1988). The effect of density (kg/m³) on fish behavior and product quality (e.g., fin erosion and skin abrasion) are also considered (Bosakowski and Wagner, 1994; Wagner *et al.*, 1996; Wagner *et al.*, 1997). Acute and chronic losses due to infectious disease outbreaks in aquaculture can impose a significant cost to productivity and, therefore, to profit. A few field studies have demonstrated that mortality during infectious disease outbreaks is higher at higher fish densities (Fagerlund *et al.*, 1984; Mazur *et al.*, 1993; Banks, 1994; LaPatra *et al.*, 1996). Also, some general “rules of thumb” for suggested densities to avoid infectious disease outbreaks have been published for salmonid culture (Wedemeyer and Wood, 1974; Piper *et al.*, 1982). However, details about the relationship between fish density/loading rates, pathogen concentration and the risk of infectious disease outbreaks have not been studied. This relationship should be quantified so that it can be included in estimates of system carrying capacity and in the development of risk analyses for aquaculture production.

We used a disease model to explore three questions about the relationship between fish density, pathogen concentration and characteristics of infectious disease outbreaks. 1) As fish density and pathogen concentration increase, how does this change affect the probability of survival for an individual fish? 2) How does the maximum population death rate change as pathogen load changes? 3) As pathogen load changes, what is the effect on the time at which the maximum death rate is reached?

The model system

The design requirements for studies that will answer these questions make it practically impossible for them to be done in a commercial aquaculture facility. Large numbers of fish and tanks are needed. The requirements can be restrictive even for a well-equipped

aquaculture research laboratory. After considering these requirements, we chose infectious pancreatic necrosis (IPN) as our disease model. IPN, which is a viral disease, is an appropriate model because it affects small salmonid fish. Therefore, small tanks containing large numbers of fish can be used in an investigation.

Young rainbow trout (34 days old) were used for the study. Conditions were set up to mimic rainbow trout fry culture in flow-through conditions. The study consisted of two experiments. The first lasted 59 days and the second lasted 53 days. The day before each experiment began, all of the fish in a single tank were infected with the IPN virus. These fish will be referred to as infectious, or INF, fish. The next day, these INF fish were added to tanks containing various densities of uninfected, susceptible fish (Table 1). Either 1, 2 or 3 of the infectious fish were added. Additional tanks were set up that contained no INF fish or all INF fish. Tank densities ranged from about 23 to 162 kg/m³. The total densities were chosen to be near to, or greater than, the density of 0.5 lbs/ft³ recommended for culturing one inch rainbow trout (Piper *et al.*, 1982) without any outbreaks of infectious disease. Tank volume was 1 liter of spring water at 54°F. Tank turnover rate was 15 times per hour. This high flow rate was chosen to maintain good water quality at high fish densities. Each day dead fish were removed and counted. All remaining fish were counted at the end of each experiment.

Table 1. INF = Number of fish that were exposed to IPN virus (INF) and added to the tanks. Total density = total density of fish in the tank. X is a tank that was lost when water flow to the tank was disrupted.

INF	Total density
1	15,15,25,25,26,30,30,34,37,45,48,50,59,60,74,75,75,75,76,87,90,90,98,100,104,105
2	14,15,27,28,45,45,60,60,75,75,89,92,105,105
3	14,15,25,25,25,30,30,45,49,50,52,X,57,60,64,75,75,75,76,81,90,90,95,100,105,105
All	15,15,30,30,45,45,60,60,69,75,75,86,90,105,105
0	14,15,25,26,44,45,48,49,72,73,74,75,96,97,103,104

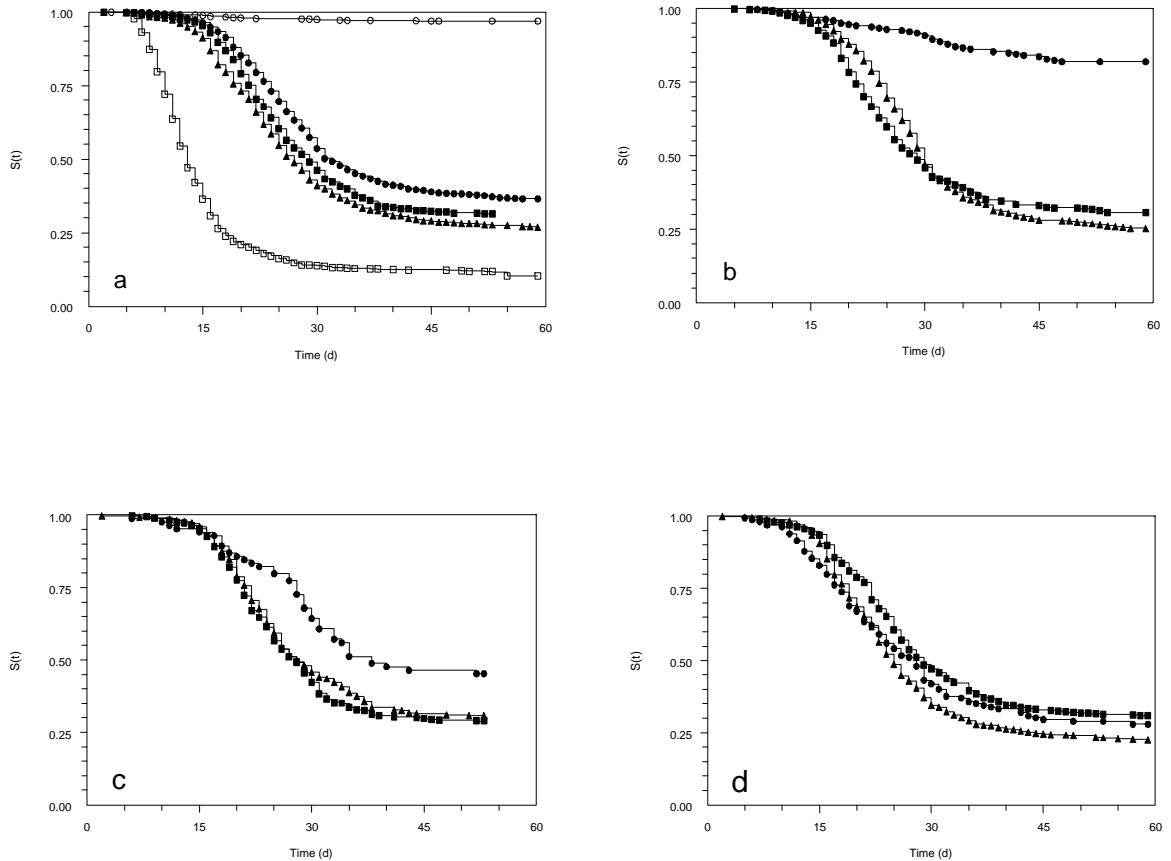
Results

Effect on probability of survival for individual fish

The probability of survival to the end of the experiment was >0.98 for tanks where no INF fish were added (Fig 1A). As the number of INF fish added to the tanks increases, the probability of survival to the end of the experiment (S(t)) decreases (Fig. 1A). When each group of infectious fish is considered separately, the effect of density can be seen (Fig. 1b – Fig. 1d). For the tanks where INF = 1, the probability of survival is higher at densities <40 fish/L than it is at densities ≥40 fish/L (Fig. 1b). As the number of INF fish increases (Fig. 1c and Fig. 1d), the advantage of lower densities disappears until the probability of survival at given time point is about the same regardless of the culture density (Fig. 1d).

This apparent interaction between total density and number of INF fish has been confirmed with regression models.

Figure 1a – 1d. Survival curves. Fig. 1A: Each curve represents number of infectious (INF) fish added (**l** =1 INF fish; **h**=2 INF fish; **c**=3 INF fish; **y**=All INF fish; **~**=0 INF fish). Figure 1B: Survival curves for tanks where 1 INF fish added. Figure 1C: Survival curves for tanks where 2 INF fish. Figure 1D: Survival curves for 3 infectious fish added. For figures 1B – 1D, **l** =density<40; **g**=40≤density<80; **c**=density≥80).



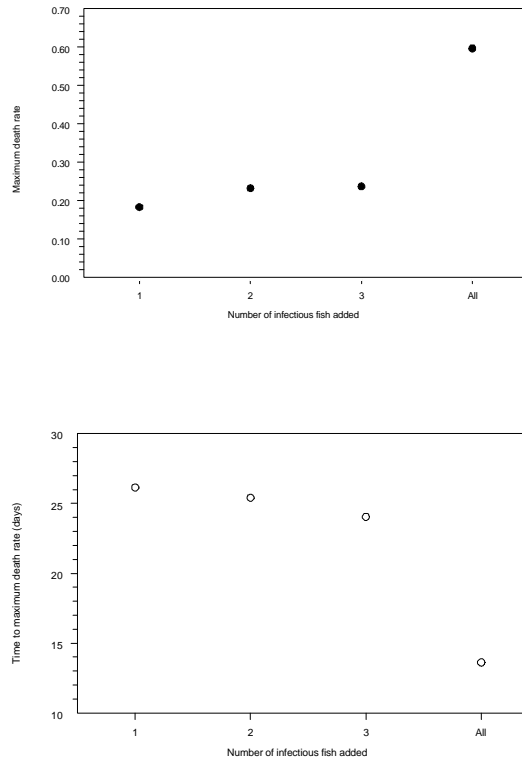
Effect on the maximum death rate and time to the maximum death rate

When one infectious fish was added to tanks of susceptible fish, the maximum death rate was 0.18 fish/(fish-day) (Fig. 2a). As the number of infectious fish increased, the maximum death rate increased, until it reached 0.60 fish/(fish-day) in tanks where all fish were challenged by the IPN virus.

When one infectious fish was added to tanks of susceptible fish, the time taken to reach the maximum death rate was 26 days (Fig. 2b). As the number of infectious fish added

increased, the time to the maximum death rate decreased, until it reached 13.5 days for tanks where all fish were infectious.

Figure 2a - 2b. Fig 2a. Maximum death rate (fish/(fish-day)) vs. number of infectious fish added. Fig. 2b. Time to maximum death rate (fish/(fish-day)) vs. number of infectious fish added.



Discussion

More quantification is needed before fish density/loading, pathogen concentration and the risk of infectious disease outbreaks can be used in carrying capacity and risk analysis estimates. However, this work contributes new information that describes how fish density and pathogen load will interact to affect characteristics of disease outbreaks in an aquaculture system. It also emphasizes the importance of effective biosecurity measures. The effect of fish density on the probability of survival for an individual fish will depend on the pathogen load. For example, when the pathogen concentration is lower (e.g., INF=1), fish in tanks containing lower densities have a greater probability of survival than fish in tanks containing higher densities. However, as the pathogen load increases (e.g., INF=2, INF=3), the advantage of lower density disappears, that is, at a given point in time, the probability of survival is similar regardless of density. Pathogen load also affects the maximum death rate and the time to the maximum death rate. The lower the pathogen concentration, the lower the maximum death rate and the longer the time taken to reach the maximum death rate.

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