BIOSECURITY CONSIDERATIONS FOR OFFSHORE FINFISH AQUACULTURE IN NEW ZEALAND

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BIOSECURITY CONSIDERATIONS FOR OFFSHORE FINFISH AQUACULTURE IN NEW ZEALAND

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Abbreviations and Acronyms

AGD	Amoebic gill disease
ERM	Enteric redmouth disease
EUS	Epizootic ulcerative syndrome
IHN	Infectious haematopoietic necrosis
IHNV	Infectious haematopoietic necrosis Virus
IPN	Infectious pancreatic necrosis
IPNV	Infectious pancreatic necrosis virus
ISA	Infectious salmon anaemia
ISAV	Infectious salmon anaemia virus
NZ-RLO	New Zealand rickettsia-like agent, 3 known genotypes of PLB including one closely related to *Piscirickettsia salmonis*
OIE	World Organisation for Animal Health (formerly Office International des Epizooties).
PLB	*Piscirickettsia*-like bacteria
RLO	Rickettsia-like organism
RSIVD	Red seabream iridoviral disease
VER	Viral encephalopathy and retinopathy
VHS	Viral haemorrhagic septicaemia
VHSV	Viral haemorrhagic septicaemia virus
YAV	Yellowtail ascites virus

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Executive Summary

This document was developed for Aquaculture Direct to provide an overview of biosecurity-related issues associated with offshore aquaculture of marine finfish in New Zealand. The cultured species of interest included chinook salmon (*Oncorhynchus tshawytscha*), sockeye salmon (*O. nerka*), rainbow trout (*Oncorhynchus mykiss*), Atlantic salmon (*Salmo salar*), kingfish (*Seriola lalandi*) and hapuku (*Polyprion oxygeneios*). The introduction of Atlantic salmon from overseas was not recommended due to the inherent exotic disease risk (especially for VHS, ISA). The potential size of the offshore finfish farming industry in New Zealand will ultimately depend on the availability of suitable sites and ability to control biosecurity issues/disease outbreaks.

Experience overseas has found that management arrangements that allow spatial separation of different year classes of fish into independent farm management areas separated by ideal buffer zones represents worlds best biosecurity practice, as this allows integrated pathogen management as well as regular synchronised fallowing of each farming area. Planning in this manner is recommended in New Zealand as it would provide added protection if/when biosecurity leaks allow exotic diseases to be introduced, and/or if/when new endemic diseases emerge. Regarding disease threats to industry development, viral and bacterial diseases have caused significant disruption of the culture of salmonid and non-salmonid fishes in several overseas countries. Data from high intensity farming of salmonids in Chile suggest buffer zones of 10-15 km are required in order to effectively manage risks from outbreaks of viral diseases (e.g. ISAV, IPNV) and also bacterial infections (e.g. *Piscirickettsia salmonis*). For non-salmonid fishes, infection pressure from monogeneans (e.g. *Benedenia seriolae* and *Zeuxapta seriolae* infecting kingfish) may reduce to background levels between 8-18 km from the source farm. For sealice, which can infect both salmonids and non-salmonid marine fishes, modelling has suggested that their infective stages can be transported large distances (90-100 km) by currents, however in these extreme cases the viability of the infective stages is greatly reduced. The distance at which sealice infection pressure remains significantly higher depends on various factors, but appears to be over 8-12 km and less than 30-45 km from a source farm.

Literature review therefore suggests the minimum width of an ideal on-water buffer zone ("as the fish swims", not "as the crow flies") to ensure true independence of marine finfish farming management areas in New Zealand would be somewhere around 15 km. However, if sealice outbreaks became problematic in New Zealand in the future, the width of an ideal buffer zone may need to be increased to between 18 and 45 km, with the actual minimum distance depending on detailed modelling. When the biosecurity risks associated with the culture of chinook salmon and Atlantic salmon were compared, the qualitative risk estimations suggest that chinook salmon has the lowest disease risk profile (total risk score of 53), due mainly to their resistance to amoebic gill disease (AGD), sealice infections.

The greater isolation from the coast, together with increased water depths, will inherently provide offshore aquaculture protection against many diseases of concern (by dilution and disruption of multi-host parasite lifecycles). Nevertheless, biosecurity planning for offshore aquaculture in NZ should emphasise prevention through use of vaccination for microbial diseases, and to reduce reliance on chemical treatments, seacage barrier/submerged cage technology combined with integrated pathogen management should be adopted to reduce impacts of parasitic infections from sealice and monogeneans.

1.0 Objectives of this document

This document was developed by DigsFish Services for Aquaculture Direct to provide an overview of biosecurity-related issues associated with offshore aquaculture of marine finfish in New Zealand. Aquaculture Direct indicated that the cultured species of interest in New Zealand include various salmonids that already occur in New Zealand (McDowall 1994), including chinook salmon (*Oncorhynchus tshawytscha*), sockeye salmon (*O. nerka*), rainbow trout (*Oncorhynchus mykiss*) and Atlantic salmon (*Salmo salar*). Furthermore, certain species of marine fishes were also considered, including kingfish (*Seriola lalandi*) and hapuku (*Polyprion oxygeneios*) which have been identified as promising aquaculture candidates (Symonds et al. 2014). The objective of this document was to review and update information on diseases of these marine finfish species, and to summarise the currently available information on potential dispersal distances for these and other infectious agents which may be relevant to offshore farming of these species in New Zealand. This information may be useful for defining the potential size and economic value of the offshore finfish farming industry in New Zealand, and may help determine the location and extent of farm management areas if they are to be truly independent (i.e. to ensure they are separated by ideal on-water buffer zones which prevent cross-infection between different farm management areas, allowing integrated pathogen management and effective site fallowing). Provision of appropriate buffer zones between farming areas is a critical biosecurity management consideration, given that new endemic diseases could emerge in finfish aquaculture in New Zealand at some time in the future, as well as the ever present, but unquantifiable, risk of biosecurity leaks that could allow exotic disease incursions to occur (Diggles 2011, 2016, 2018).

Aquaculture Direct have asked for specific consideration of the following questions

1. Ranking of biosecurity risks for each group of fishes (salmon, other marine finfish and sea reared rainbow trout)

2. Minimum distances for buffer zones between stock owned in common and competively owned stock

3. Differences in biosecurity risk between chinook and Atlantic salmon

4. Use of treatment agents for farmed fin fish, NZ vs other countries especially Australia and Norway

2.0 Method

The peer reviewed scientific literature on marine finfish (salmon and other marine fish) farming in New Zealand and elsewhere was searched for keywords including disease; risk factors; connectivity; dispersal; infection; infection pressure; disease transfer and integrated pathogen management using Google Scholar. Citations of all the papers referenced in Diggles (2011), Diggles (2016) and Diggles (2018) relating to dispersal of infective stages of disease agents from finfish farmed in seacages were also cross checked through cross-referencing engines in Google Scholar and Scopus. Those papers which were considered relevant as primary sources of information on dispersal and transmission distances of relevant pathogens were then examined and the relevant information was collated into tabular form presenting the relevant variables for the different pathogen groups (viruses, bacteria,

monogeneans and sealice). The identification of diseases of concern was undertaken using the process outlined in Figure 1 and with due consideration of New Zealand's national list of notifiable diseases of finfish (Table 1). For each disease of concern, a qualitative estimation of the relative risk to marine finfish culture in New Zealand was provided using the criteria outlined in Appendix 1, based on the scientific literature as well as the professional judgment of the author.

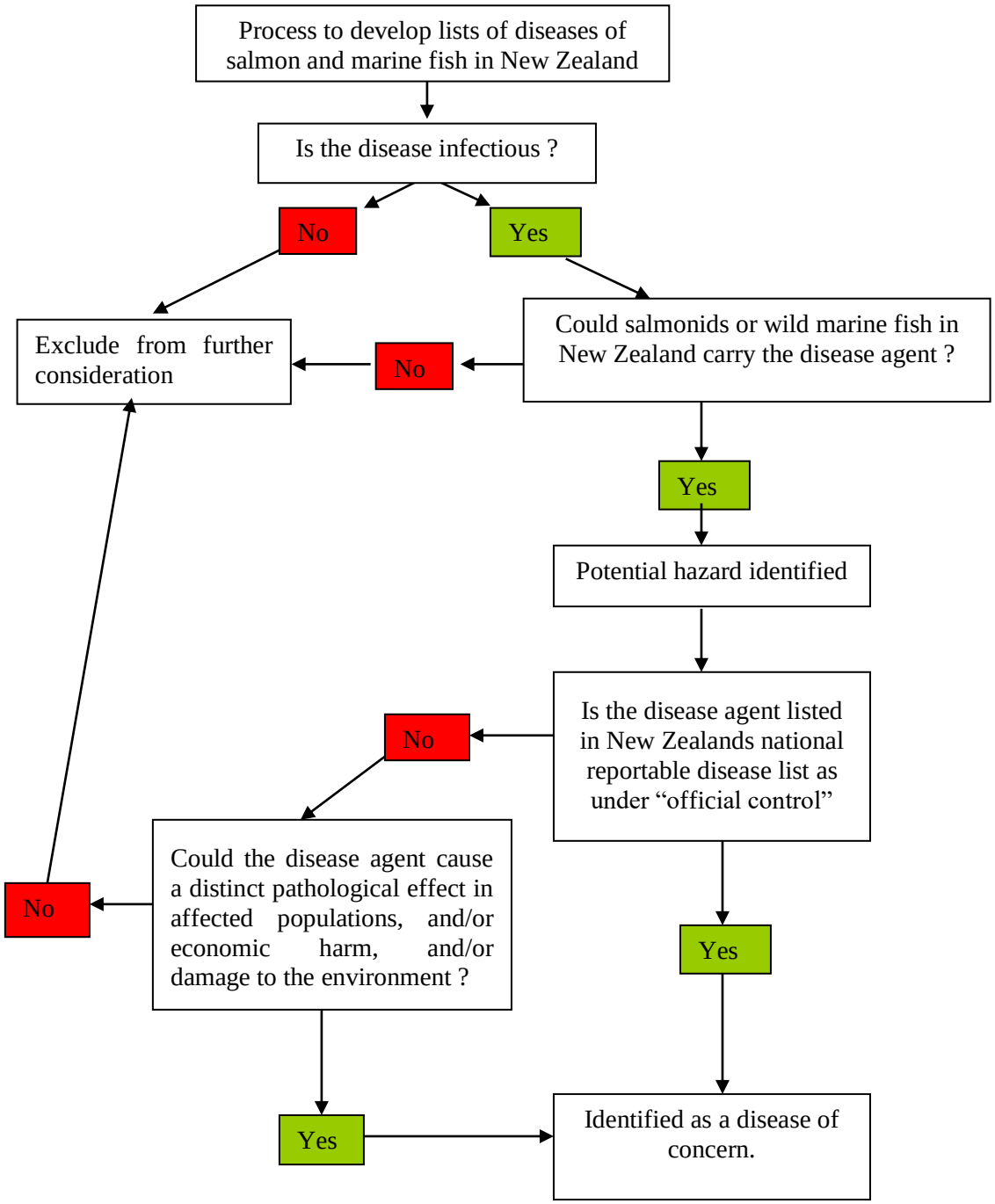

Figure 1. Flow chart showing the decision making process used to identify diseases of concern.

Table 1. New Zealand's national list of notifiable diseases of finfish (i.e. diseases under official control). (https://www.mpi.govt.nz/protection-and-response/finding-and-reporting-pests-and-diseases/registers-and-lists/)

New Zealand's National List of Notifiable Diseases of Finfish (as of January 2020)	**Listed in the OIE *Aquatic Animal Health Code (2019)****	**Exotic to New Zealand**	**Found in salmonids in New Zealand**	**Found in kingfish or grouper in New Zealand**
1. Bacterial kidney disease (*Renibacterium salmoninarum*)		✓		
2. *Yersinia ruckeri* (Exotic strains)		✓		
3. Epizootic haematopoietic necrosis – EHN virus	✓	✓		
4. Infection with *Aphanomyces invadans* (EUS)	✓	✓		?
5. Furunculosis (*Aeromonas salmonicida* subsp. *salmonicida*)		✓		
6. Gyrodactylosis (*Gyrodactylus salaris*)	✓	✓		
7. Infectious haematopoietic necrosis – IHN virus	✓	✓		
8. Infectious pancreatic necrosis virus (IPN virus, exotic strains)			✓**	?
9. Infection with infectious salmon anaemia virus (ISA)	✓	✓		
10. Infection with Koi herpesvirus (KHV)	✓	✓		
11. *Oncorhynchus masou* virus		✓		
12. Red sea bream iridoviral disease	✓	✓		?
13. Spring viraemia of carp – SVC virus	✓	✓		
14. Viral haemorrhagic septicaemia – VHS virus	✓	✓		
15. Infection with *Myxobolus cerebralis* (Whirling Disease)			✓	
16. Infection with New Zealand *Rickettsiaceae* species			✓***	?

* (see OIE 2019).

** A birnavirus (IPNV Genogroup 5) occurs in returning sea run salmon in New Zealand (Davies et al. 2010).

*** declared unwanted organisms under section 131 of the Biosecurity Act 1993 on 20 April 2016.

? denotes disease not recorded from kingfish or hapuku in New Zealand, though both are likely to be susceptible.

3.0 Results

3.1 Ranking of biosecurity risks for different fish groups

Salmonids

Extensive salmon farming experience in Europe (Norway, Scotland, Ireland, Iceland), North America (Maine USA, Canada), South America (Chile) and Australia (Tasmania) has identified a range of disease threats which from time to time have caused significant disruption of the culture of salmonids (mainly Atlantic salmon, *Salmo salar*) in several overseas countries (Diggles 2011, 2016, 2018). These include viral diseases such as infectious salmon anaemia (ISA), which caused major epizootics and economic losses in Atlantic salmon culture in Norway, Scotland, Canada, and Chile. Other viruses causing significant outbreaks of disease cultured salmonids overseas include infectious pancreatic necrosis (IPN), viral haemorrhagic septicaemia (VHS), infectious haematopoietic necrosis (IHN), and sleeping disease (SD) or pancreas disease (PD) caused by salmonid alphavirus (Table 2). Economically important bacterial diseases of cultured salmonids include *Piscirickettsia*-like bacteria (PLB), furunculosis, bacterial kidney disease (BKD), and enteric redmouth, amongst others (Table 2). Important diseases caused by protozoan parasites include white spot disease and amoebic gill disease (AGD), while the major metazoan parasites of importance to salmon farming are sea lice (Table 2).

Table 2. Risk scores for infectious diseases of relevance to culture of salmonids (*Oncorhynchus tshawytscha, O. nerka, O. mykiss, S. salar*) in New Zealand [a]

Diseases of salmon	Under official control	Occurs in salmonids in NZ	Risk estimation for disease in chinook salmon	Risk estimation for disease in Atlantic salmon	Risk estimation for disease in Sockeye salmon	Risk estimation for disease in rainbow trout
VIRUSES			15	21	15	22
Epizootic haematopoietic necrosis (EHNV)*	Yes	No	Low	Low	Low	High
Infectious haematopoietic necrosis (IHNV)	Yes	No	Moderate	Moderate	Moderate	Moderate
Infectious pancreatic necrosis virus (IPNV)	Yes	No	Low	Moderate	Low	Moderate
Infectious salmon anaemia (ISAV)	Yes	No	Very low	High	Very low	Very low
Oncorhynchus masou virus (OMV)	Yes	No	Very low	Very low	Moderate	Moderate
Salmonid alphavirus (PD, SD)	No	No	Low	High	Low	High
Viral haemorrhagic septicaemia (VHS)	Yes	No	High	High	Low	High
BACTERIA			21	21	21	21
Flexibacter spp./ *Tenacibaculum* spp.	No	Yes	Low	Low	Low	Low
Bacterial gill disease (BKD)	No	Yes	Very low	Very low	Very low	Very low
Bacterial kidney disease	Yes	No	Moderate	Moderate	Moderate	Moderate
Enteric redmouth disease (*Yersinia ruckeri*)*	Yes	No	Low	Low	Low	Low
Furunculosis (*Aeromonas salmonicida*)	Yes	No	High	High	High	High
Mycobacterium spp.	No	Yes	Low	Low	Low	Low
Nocardia spp. (Nocardiosis)	No	Yes	Low	Low	Low	Low
Piscirickettsia-like bacteria (PLB, incl. NZ-RLO)	Yes	Yes	Moderate	Moderate	Moderate	Moderate
Vibrio spp.	No	Yes	Low	Low	Low	Low
FUNGI			2	2	2	2
Aphanomyces invadans (EUS)*	Yes	No	Negligible	Negligible	Negligible	Negligible
Saprolegnia spp.*	No	Yes	Low	Low	Low	Low
PROTOZOA			9	10	9	10
Amoebic gill disease (AGD)	No	Yes	Low	High	Moderate	Moderate
Chilodonella spp., *Trichodina* spp.	No	Yes	Low	Low	Low	Low
Ichthyophthirius multifiliis (White spot disease)*	No	Yes	Low	Low	Low	Low
Microsporidia (*Loma*, *Nucleospora*, *Desmozoon*)	No	Yes	Moderate	Low	Low	Moderate
METAZOA			6	10	8	10
Caligus spp., *Lepeophtheirus* spp. (sea lice)	No	Yes	Low	High	High	Moderate
*Gyrodactylus salaris**	Yes	No	Low	High	Low	Moderate
Myxobolus cerebralis (Whirling disease)*	Yes	Yes	Low	Low	Low	High
TOTAL RISK SCORE			53	64	55	65

*** Denotes problematic in freshwater only.** **[a] See Appendix 1 for definitions for risk estimations and risk scoring method. A lower risk score indicates a lower disease risk.**

When the qualitative risk estimations for the various diseases listed in Table 2 are compared between the various species of salmonids, it is evident that viral and bacterial disease agents generally pose the greatest risk, while chinook salmon has the lowest disease risk profile (virus risk = 15 points, bacteria risk = 21 points, fungi risk = 2 points, protozoan risk = 9 points, metazoan risk = 6 points, giving a total risk score of 53). Sockeye salmon had the next lowest disease risk profile (total risk score = 55, due to their higher risk of infection by sealice compared to chinook salmon), while Atlantic salmon (total risk score 64) and rainbow trout (total risk score 65) had much greater disease risk scores due to increased risk of infection by viral diseases, amoebic gill disease and metazoans such as sealice and whirling disease (Table 2).

Marine finfish

A wide range of pathogens have been recorded in a wide range of marine fish species, especially in warm water aquaculture in Asia (Sheppard 2004, Tak-Seng 2014), but also in New Zealand (Diggles et al. 2002). For disease agents infecting kingfish (*Seriola lalandi*), key references include Arimoto et al (1993), Sharp et al. (2001, 2003), Sheppard (2004), Diggles and Hutson (2005), Hutson and Whittington (2006), Hutson et al. (2007, 2011), Stephens and Savage (2010), Stride et al. (2013), Sicuro and Luzzana (2016), Stephens (2016) and Garcia-Mendoza et al. (2019). The list of known disease agents of kingfish at this time include three significant viral agents, namely the IPNV- like birnavirus that causes yellowtail ascites viral disease (YAV), and the OIE listed red seabream iridoviral disease (RSIVD) and viral encephalopathy and retinopathy (VER). Bacterial diseases of kingfish include epitheliocystis, vibriosis, nocardiosis, lactococcosis, and *Tenacibaculum*-like agents, and for the purposes of this report, it is assumed that kingfish will also be susceptible to *Piscirickettsia*-like bacteria. Kingfish are also susceptible to protozoan parasites such as scuticociliates, and most marine fish species are susceptible to *Trichodina* spp. and white spot disease caused by *Cryptocaryon irritans*. Kingfish are also known to host a number of metazoan disease agents, several of which cause significant diseases in captivity, including blood feeding (Polyopisthocotylean) monogeneans on the gills like *Zeuxapta seriolae*, mucous feeding (Capsalid) monogeneans on the skin (*Benedenia seriolae*), sanguinicolid blood flukes (*Paradeontacylix* spp.), sealice (*Caligus* spp., *Lepeophtheirus* spp.) and various myxozoa, including *Kudoa neurophila*, *Ceratomyxa* and *Unicapsula* in Australia, and *Myxidium* spp. in New Zealand (Table 3).

Relatively little is known about diseases of hapuku (*Polyprion oxygeneios*), however some information is available from Hewitt (1963), Johnston (1983), Hine et al. (2000), Smith et al. (2009), Hutson et al. (2011) and Salinas et al. (2012). For the purposes of this report, it is assumed that hapuku will be susceptible to the viral, bacterial and myxozoan diseases that affect kingfish (Table 3). It is known that wild hapuku are infected with blood feeding (Polyopisthocotylean) monogeneans on the gills (*Allocotylophora polyprionum*) as well as mucous feeding (Capsalid) monogeneans on the skin (*Calicobenedenia polyprioni*). Wild hapuku in some areas of New Zealand (e.g. Cook Strait) can also be naturally infected by high numbers of sealice (*Lepeophtheirus polyprioni*). For example, Hewitt (1963) recorded 227 female, 14 male, and 26 juvenile *L. polyprioni* from 18 hapuku, with another 4 uninfected (prevalence 81.8%, mean intensity 14.8 copepods/fish), while Johnston (1983) in his study of hapuku from Cook Strait, found prevalences of *L. polyprioni* of up to 30%, and intensities of up to 20 copepods on one fish, usually observed on the flanks, but sometimes also on head and tail. Hutson et al. (2011) found that wild hapuku in Australia are also infected with capsalid (*Calicobenedenia* spp.,

probably *C. polyprioni*) and polyopisthocotylid monogeneans (*Allocotylophora polyprionum*) on the skin and gills, respectively. Furthermore, when hapuku were bought into captivity for breeding, juveniles appeared particularly vulnerable to systemic infection and mortalities caused by scuticociliates, which were identified as *Miamiensis avidus* and *Uronema marinum* (see Smith et al. 2009, Salinas et al. 2012).

Table 3. Risk scores for infectious diseases of relevance to culture of kingfish (*Seriola lalandi*) and hapuku (*Polyprion oxygeneios*) in New Zealand [a]

Diseases of kingfish	Under official control	Occurs in kingfish or hapuku in NZ	Risk estimation for disease in kingfish	Risk estimation for disease in hapuku
VIRUSES			10	10
Aquatic Birnavirus (IPNV Genogroup 5)	Yes	?	Moderate	Moderate
Red sea bream iridoviral disease (RSIVD)	Yes	No	High	High
Viral encephalopathy and retinopathy (VER)	No	No	Moderate	Moderate
BACTERIA			12	11
Epitheliocystis (Chlamydiae, *Parilichlamydia*)	No	Yes	Moderate	Moderate
Flexibacter spp./ *Tenacibaculum* spp.	No	Yes	Low	Low
Lactococcus garviae	No	No	Low	Low
Nocardia spp. (Nocardiosis)	No	?	Moderate	Low
Piscirickettsia-like bacteria (NZ-RLO)	Yes	Yes	Very low	Very low
Vibrio harveyi, *Vibrio* spp.	No	Yes	Moderate	Moderate
PROTOZOA			7	8
Cryptocaryon irritans (white spot disease)	No	?	Moderate	Moderate
Trichodina spp.	No	Yes	Very low	Very low
Scuticociliates (*Uronema*, *Miamiensis*)	No	Yes	Moderate	High
METAZOA			35	22
Monogenea			14	6
Benedenia seriolae	No	Yes	High	Negligible
Zeuxapta seriolae	No	Yes	High	Negligible
Other Capsalidae	No	Yes	Moderate	Moderate
Other Polyopisthocotylea	No	Yes	Moderate	Moderate
Digenea			4	3
Sanguinicolid blood flukes (*Paradeontacylix* spp.)	No	Yes	High	Moderate
Crustacea			5	5
Caligus epidemicus, *C. lalandei*, *Caligus* spp., *Lepeophtheirus* spp. (sea lice)	No	Yes	High	High
Lernanthropus spp.	No	Yes	Very low	Very low
Myxozoa			12	8
Ceratomyxa spp.	No	?	Low	Low
Kudoa neurophila, *Kudoa* spp.	No	?	Moderate	Moderate
Myxidium spp.	No	Yes	Moderate	Moderate
Unicapsula seriolae	No	No	High	Negligible
TOTAL RISK SCORE			64	51

[a] See Appendix 1 for definitions for risk estimations and risk scoring method. A lower risk score indicates a lower disease risk. ? = disease agent known to occur in New Zealand, but infections of kingfish/hapuku not recorded at this time.

When the qualitative risk estimations for the various diseases listed in Table 3 are compared between kingfish and hapuku, it is evident that for kingfish, metazoan diseases (particularly monogeneans and myxosporeans, but also blood flukes and sea lice) pose the greatest disease risk, contributing 35 points towards a total risk score of 64 points. For hapuku, metazoan diseases also contribute the highest risk,

however due to the relatively limited knowledge of their disease status, it is difficult to accurately assess the relative disease risk for hapuku, which may explain the relatively low total risk score of 51 points for this species.

3.2 Minimum distances for buffer zones

Experience overseas has found that management arrangements that allow spatial separation of different year classes of fish into independent farm management areas separated by ideal buffer zones represents worlds best biosecurity practice, as this allows integrated pathogen management as well as regular synchronised fallowing of each farming area (Murray et al. 2005, Gustafson et al. 2007, Chang et al. 2007, Jones et al. 2015, Sitjá-Bobadilla and Oidtmann 2017). Review of scientific literature relating to disease control in marine fish farms worldwide found that increasingly sophisticated epidemiological models are being used which take into account many variables including disease agent life cycle, hydrography, currents, winds, fish population sizes (both cultured and wild), water temperature, salinity, river flows, coriolis forces and other factors.

Empirical evidence based on surveys of wild marine fish showed that the risk of infection with IPNV in coastal waters of Scotland was increased slightly above background levels (from 0.15% prevalence to 0.58% prevalence) in wild fishes within 5 km of salmon farms that contained fish clinically diseased with IPN (Wallace et al. 2008), while Murray et al. (2004) and Murray and Gubbins (2016) found that increased risk of IPNV infection no longer occurred at a distance of 8-10 km from an infected farm. In farms rearing *S. salar* in Chile, however, Mardones et al. (2016) found that having one other marine salmon farm within a 15 km radius increased the risk of clinical outbreaks due to IPNV between 22 to 34% (Table 4). Another study from Chile suggested that increased infection pressure for IPNV in *S. salar* may extend as far as 10 km from a source farm in instances of high fish stock intensity (Escobar – Dodero et al. 2018).

For ISAV, earlier studies in Norwegian fjords showed that infection risk for farmed *S. salar* increases significantly when non-infected salmon farms are less than 5 km from a processing facility or infected salmon farm (Jarp and Karlsen 1997, Scheel et al. 2007). McLure et al. (2005) found risk of infection with ISAV in Canadian (New Brunswick) bays was increased within 0.5 km of cages with clinically diseased *S. salar*, however in Chile, Mardones et al. (2009) found that in some cases salmon farms within a 15 km radius of an ISA index case had up to 12 times increased risk of infection (Table 4).

For bacteria, an epidemiological study in Chile found that the infection pressure for the pathogen *Piscirickettsia salmonis* in farmed *S. salar*, rainbow trout *Oncorhynchus mykiss*, and Coho salmon (*Oncorhynchus kisutch*) was elevated above background levels within a distance of 7.5–10-km of an infected source farm (Rees et al. 2014). Indeed, they found that the probability that a farm will report disease due to *P. salmonis* was directly associated with whether their neighboring farms within 10 km had the disease (Table 4). For monogeneans a study by Chambers and Ernst (2005) examined infections of the capsalid monogenean *Benedenia seriolae* on kingfish (*Seriola lalandi*) cultured in Spencer Gulf, South Australia. Their data was based on empirical results from sentinel fish held in experimental cages at varying distances from a farm holding a population of *B. seriolae* infected kingfish. They found that infection pressure was highest nearest the infected farm, reducing logarithmically with distance from the

Table 4. Dispersal/transmission distances for infectious agents infecting finfish in seacages as determined by empirical measurements of wild or sentinel fish, or by hydrographic and/or epidemiological modelling.

Disease agent	Location	Environment	Host species	Current speed (m/s)	Dispersal distance	Transmission distance	Reference
Viruses							
IPNV	Scotland	Coastal	*Salmo salar*	-	8-10 km (model)		Murray et al. 2004
IPNV	Scotland	Coastal	*Salmo salar*	-	5 km (empirical)	5 km	Wallace et al. 2008
IPNV	Chile	Coastal	*Salmo salar*	-	15 km (model)		Mardones et al. 2016
IPNV	Scotland	Coastal	*Salmo salar*	0.51	10 km (model)		Murray and Gubbins 2016
IPNV	Chile	Coastal	*Salmo salar*	-	10 km (model)		Escobar – Dodero et al. 2018
ISAV	Norway	Fjord	*Salmo salar*	-	5 km (model)		Jarp and Karlsen 1997
ISAV	Canada (New Brunswick)	Bay	*Salmo salar*	-	0.5 km (empirical)	0.5 km	McClure et al. 2005
ISAV	Norway	Fjord	*Salmo salar*	-	5 km (empirical)	5 km	Scheel et al. 2007
ISAV	Chile	Inland sea (bay)	*Salmo salar*	0.3	15 km (model)		Mardones et al. 2009 Sobarzo et al. 2018
Bacteria							
Piscirickettsia salmonis	Chile	Coastal	*Oncorhynchus kisutch, O. mykiss, S. salar*	-	10 km (model) at 10-13°C		Rees et al. 2014.
Monogenea							
Benedenia seriolae	South Australia (Spencer Gulf)	Inverse estuary gulf	*Seriola lalandi*	n/a	>8 km (empirical)	>8 km	Chambers and Ernst 2005
Sealice							
Lepeophtherius salmonis	Norway (Sognefjord Fjord system)	Fjord	*Salmo salar*	0.5	c. 100 km at 7.5-8°C (model)		Asplin et al. 2004
Lepeophtherius salmonis	Canada (Broughton Archipelago)	Coastal islands	*Oncorhynchus gorbuscha, Salmo salar*	-	30 km (model)		Krkosek et al. 2005
Lepeophtherius salmonis Caligus spp.	Canada (Broughton Archipelago)	Coastal islands	*Oncorhynchus gorbuscha, Salmo salar*	0.14	10-40 km (model) at 7.5-9°C		Brooks 2005
Lepeophtherius salmonis Caligus spp.	Northern Hemisphere	Coastal	Salmonids	0.05 0.2	5-17 km (model) 23-70 km (model)		Costello 2006

Disease agent	Location	Environment	Host species	Current speed (m/s)	Dispersal distance	Transmission distance	Reference
Lepeophtherius salmonis	Scotland (Loch Torridon, Loch Shieldaig)	Sea loch	*Salmo salar*	1.39 peak 0.05 mean*	6 km at 10°C (model)		Gillibrand and Amundrud (2007), Amundrud and Murray (2009)
Lepeophtherius salmonis	Canada (Broughton Archipelago)	Coastal islands	*Oncorhynchus* spp., *Salmo salar*	0.3-0.5	4-40 km (model)		Brooks 2009
Lepeophtherius salmonis *Caligus elongatus*	Scotland (Loch Torridon, Loch Shieldaig)	Sea loch	*Salmo salar*	-	>5-8 km (empirical)	>5-8 km	Penston et al. 2011
Lepeophtherius salmonis	Norway (Hardangerfjord)	Fjord	*Salmo salar*	0.7- 1.0	most 20-40 km few 90-100 km		Asplin et al. 2014
Lepeophtherius salmonis	Scotland (Loch Shieldaig)	Sea loch	*Salmo salar*	1.39 *	>5 km at 7.8-13.7°C(empirical)	>5 km	Pert et al. 2014
Lepeophtherius salmonis	Scotland (Loch Linnhe)	Sea loch	*Salmo salar*	-	median 6.1 km max 36 km (model)		Salama et al. 2016
Lepeophtherius salmonis	Scotland (Loch Linnhe)	Sea loch	*Salmo salar*	-	median 6.1 km (model)		Murray and Gubbins 2016
Lepeophtherius salmonis	Norway (Folda Fjord system)	Fjord	*Salmo salar*	0.05-0.3	20-45 km at 5-12°C (model)		Johnsen et al. 2016
Lepeophtherius salmonis	Canada (Bay of Fundy)	Bay	*Salmo salar*	0.1	4.4- 7.7 km (empirical)	7.7 km	Nelson et al. 2018
Lepeophtherius salmonis	Scotland (Loch Linnhe)	Sea loch	*Salmo salar*	-	20-30 km (model)		Salama et al. 2018
Lepeophtherius salmonis	Norway (Altafjorden)	Fjord	*Salmo salar*	0.25	>100 km (model) at 8-12°C		Skarohamar et al. 2018
Lepeophtherius salmonis *Caligus elongatus*	Faroe Islands	Offshore islands	*Salmo salar*	3.6	50% >50 km 10% >80 km (model) at 6-10°C		Kragesteen et al. 2018
Lepeophtherius salmonis *Caligus* spp.	Canada (Muchalat Inlet, British Columbia)	Inlet	*Oncorhynchus keta*, *Salmo salar*	-	30 km (model)		Nekouei et al. 2018

* wind forcing probably the dominant influence in lice dispersion patterns in Lochs Torrindon and Shieldaig.

farm. Kingfish within an 8 km radius of the farm still had increased levels of infection (3.7% of that at the source), but infection pressure had returned to background levels (0.7% of that at the source) by 18 km down current (Table 4). Monogeneans have a simple direct lifecycle and it was presumed that the current was the main factor responsible for transport of the non-infective eggs as well as the short-lived (24 hours) oncomiricidium infective larval stage (Chambers and Ernst 2005). Entrapment of eggs on cage infrastructure was likely to be a major factor responsible for high infection pressure at the farm site. Unfortunately, no current data was presented in this paper (except observations that the "strong" current from the 2.4 m spring tides had dragged one sentinel cage from 16 km to 18 km in 24 hours), so it is difficult to compare these results to those from other metazoan parasites such as sealice.

Several studies have shown that peak sealice (*Lepeophtheirus salmonis*, *Caligus* spp.) infection pressure for wild fish is likely to be located away from salmon farms under any residual current scenarios, because the newly hatched sealice nauplii are not infective (Table 4). Indeed, sealice larval stages only become infective after several days in the plankton (up to 2 weeks at 10°C, see Johnson and Albright 1991) after they moult into the infective copepodid stage (Brooks 2005, Murray and Gillibrand 2006, Amundrud and Murray 2009, Molinet et al. 2011). Asplin et al. (2004) used a numerical model to estimate that *L. salmonis* infective stages could spread as far as 100 km from salmon (*S. salar*) farms in Norwegian fjords. However, they assumed the salmon lice infective stages were immortal, which resulted in overestimation of the dispersal capabilities of viable infective stages (Brooker et al. 2018). Similarly, an early model for the Pacific coast of Canada suggested that sea lice infection pressure near infected marine farms can be 2 to 4 orders of magnitude higher than ambient background levels, and can exceed background levels at least 30 km from infected farms (Krkosek et al. 2005). However, the model by Krkosek et al. (2005) had several errors and was likely to be unreliable (Brooks 2005). A particle tracking model used by Brooks (2005) predicted that most sea lice nauplii would be transported by currents 7.3–10.0 km downcurrent (out of the archipelago) before they become infective, and that they may be transported up to 40 km from an infected farm before becoming infective (Table 4).

Based on data from Johnson and Albright (1991), copepodids suffer mortality at an average rate of 1.0 - 2.9% per hour in seawater, depending on temperature and salinity (Stein et al. 2005, Bricknell et al. 2006), so while some infective stages can survive for long periods under optimal conditions, infection pressure still decreases rather rapidly with increasing distance from an infected marine farm (Amundrud and Murray 2009). Dispersion distance depends greatly on prevailing currents, and thus also varies with many other hydrodynhamic and geographic variables (Salama et al. 2013, Johnsen et al. 2016, Sandvik et al. 2020). When these are taken into consideration (usually using complex modelling, but sometimes using empirical data from plankton samples or caged sentinel fish), the risk of sealice infection tends to be increased above background levels at least 8 to 18 km from lice infected salmon farms (Brooks 2009, Penston et al. 2011), and the maximum distance where increased infection pressure has been observed empirically or modelled is around 30-45 km (Amundrud and Murray 2009, Penston et al. 2011, Johnsen et al. 2016, Table 4), with individual particles (not all of which are likely to be viable) being modelled up to 90-100 km away in some of the larger Norwegian fjords (Asplin et al. 2004, 2014, Skarohamar et al. 2018) and the high current environment of the Faroe Islands (Kragesteen et al. 2018, Table 4). However, in smaller systems, such as Scottish lochs, sealice tend to travel shorter distances, with the

highest accumulation of infective stages tending to occur between 6 km (Amundrud and Murray 2009) and up to 12 km away from individual source seafarms (Gillibrand and Willis 2007).

The markedly reduced infection pressure with distance for viruses and bacteria is probably because they require a minimum infectious dose before they are successfully transmitted. In contrast, monogeneans have an intermediate transmission distance due to the robust non infective egg stage that can be transported away from the farm before it hatches, but the resulting oncomiricidium larvae is relatively fragile and short lived. Sealice are the most persistent because they have a non-infective nauplius stage which moults after a week or two (depending on water temperature) into a reasonably robust infective planktonic copepodid, and theoretically a single copepodid is sufficient to cause reinfection. Literature review therefore suggests the minimum width of an ideal on-water buffer zone ("as the fish swims", not "as the crow flies") to ensure true independence of marine finfish farming management areas in New Zealand (whether between year classes on individual farms, or between farms owned by different companies) would be somewhere around 15 km (Figure 2). However, if sealice outbreaks became problematic in New Zealand in the future, the width of an ideal buffer zone may need to be increased to between 18 and 45 km, with the actual minimum distance depending on detailed modelling.

3.3 Differences in biosecurity risk between chinook and Atlantic salmon

When the qualitative risk estimations for the disease agents of salmonids listed in Table 2 are compared between chinook and Atlantic salmon, it becomes apparent that chinook salmon has the lowest disease risk profile (total risk score of 53), due mainly to their resistance to amoebic gill disease (AGD), sealice infections. In contrast, Atlantic salmon are well known to be highly susceptible to many viruses, particularly the OIE listed ISAV, and also are affected by both sealice and AGD. The total risk score for Atlantic salmon (64) was eclipsed only by the risk score obtained by rainbow trout (total risk score 65), due to the latter species high susceptibility to infection by certain viruses such as VHSV, as well as metazoans including AGD and whirling disease (Table 2). Furthermore, it must be considered that whilst chinook salmon, sockeye salmon and rainbow trout are well established in New Zealand, only remnant populations of Atlantic salmon occur, left over from repeated attempts to acclimatise this species to New Zealand waters between the 1860's and 1960's (McDowall 1994). Given the high risk score for diseases of Atlantic salmon, the introduction of new Atlantic salmon genetic stock from overseas is not recommended, due to the inherent risk of importing several exotic diseases, particularly VHS and ISA, but also IPN, salmonid alphavirus, furunculosis and PLB.

3.4 Use of treatments for farmed finfish

Treatments of diseases of seacage farmed fish is a controversial subject, due to issues related to development of microbial resistance to antibiotics (Sitjá-Bobadilla and Oidtmann 2017) and parasitic (sealice) resistance to drugs such as delousing agents (emamectin benzoate, cypermethrin, azamethiphos) (Aaen et al. 2015). Indeed, widespread use of vaccination against major viral and bacterial disease agents has resulted in massive reductions in antibiotic use and massive increases in production in several major salmon farming regions including Norway, Scotland and North America. In Norway, for example, development of effective vaccines led to an approximate 99.8% decrease in antibiotic use (compared to 1987, Sitjá-Bobadilla and Oidtmann 2017) and increased production now exceeding 1 million tonnes of salmon per year, while using <0.17 grams of antibiotics per tonne of

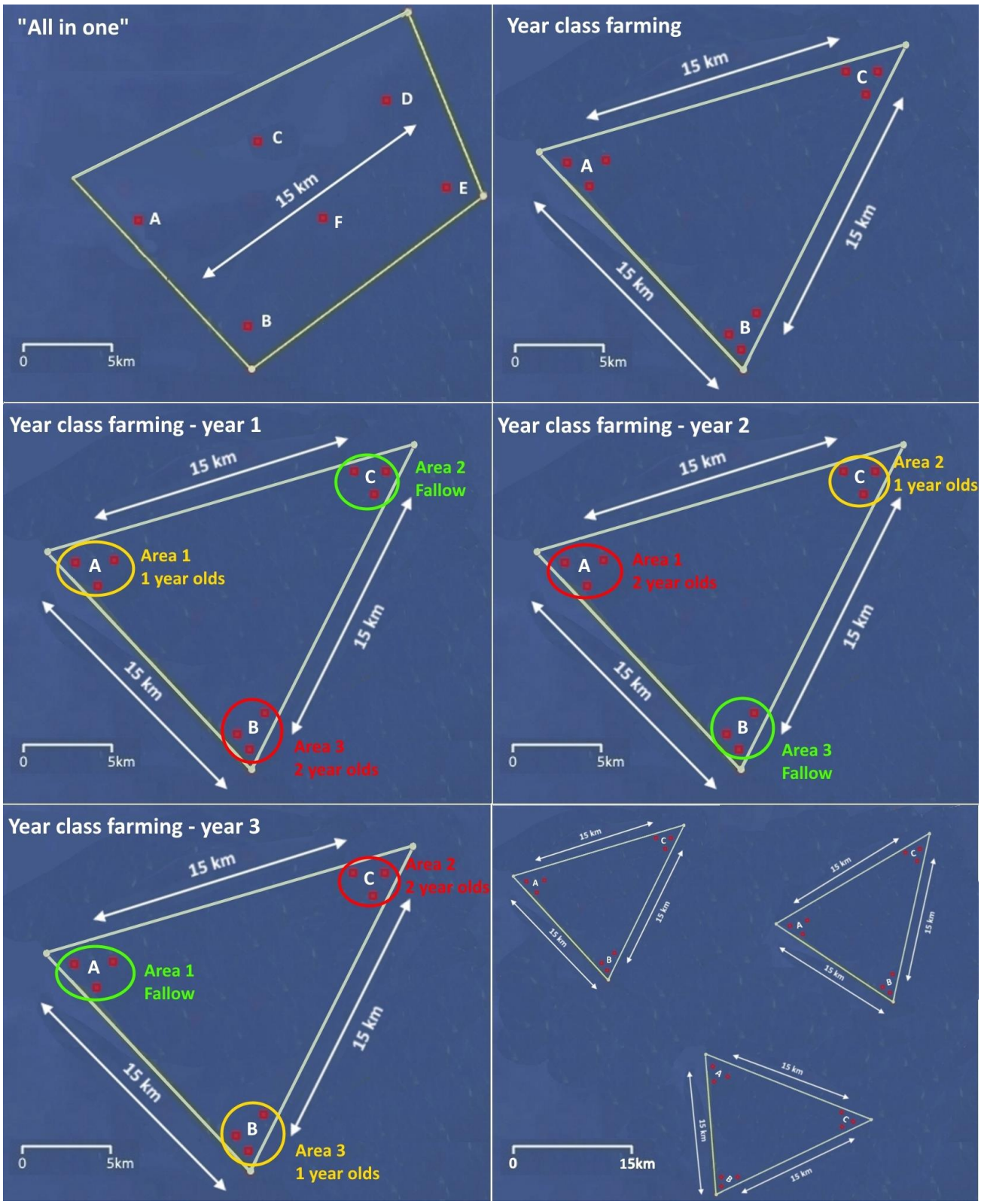

Figure 2. Diagrams showing the difference between the least biosecure "all in one" farming model (upper left), compared to a year class model (upper right) which has the same number of active cages within a similar area, but separates fish into different year classes with ideal (15 km) buffer zones between year classes. This design allows integrated management of the various year classes and regular fallowing (middle 3 diagrams). Different companies can farm in the same geographic region if each farm area is also separated by ideal buffer zones (lower right).

production. In contrast, in Chile antibiotic use has increased in recent years and in 2016 the salmon farming industry used around 0.53 kg of antibiotics per ton of harvested salmon (Miranda et al. 2018). Clearly the appropriate model for New Zealand to copy is the Norwegian model, hence pathogen management strategies for offshore finfish aquaculture should strongly emphasise development of vaccines to combat viral or bacterial disease issues that may arise in New Zealand waters.

In contrast to microbial diseases, control of metazoan parasites has proven to be problematic in the culture of not only salmon (in the case of sealice), but also kingfish due to issues relating to hyperinfestation by monogenean ectoparasites (Chambers and Ernst 2005) and blood flukes. For monogeneans and blood fluke parasites of cultured kingfish and tuna, the drug praziquantel delivered orally or via bath treatment has been shown to be highly effective for control of infections, however praziquantel is not palatable to fish, and therefore is difficult to administer (Williams et al. 2007, Tubbs et al. 2008, Power et al. 2019). Furthermore, unless treatments are 100% effective, there is a long term risk of development of resistance when relying on any drug treatment (Aaen et al. 2015). An alternative bath treatment for both sealice and monogenean ectoparasites is hydrogen peroxide, which is effective and environmentally benign as it breaks down into oxygen, through can be laborious to administer and is stressful to fish (Mansell et al. 2005).

Current integrated pathogen management strategies for control of sea lice in salmon farming in Europe are now relying less on licensed drugs or pesticides such as emamectin benzoate, and more on other husbandry management tools, such as single-cohort stocking, optimized stocking densities, the use of cleaner fish in polyculture, use of cage "skirts" and "snorkel cages" that reduce access of planktonic parasite infective stages to fish, hydrogen peroxide baths or hot water baths for removing sealice when necessary as well as effective fallow periods, while "cutting edge" technologies such as laser removal of sealice may also show promise (Sitjá-Bobadilla and Oidtmann 2017, Brooker et al. 2018). Indeed, "barrier technology" cage design such as innovation of the "snorkel seacage" has been shown to reduce sealice infection burdens in cultured salmonids by up to 75% in commercial sized trials in Europe (Geitung et al. 2019), with the additional bonus of better control of amoebic gill disease, which is usually controlled through freshwater baths (Wright et al. 2017, 2018).

If sealice become problematic in the culture of kingfish and hapuku, use of snorkel type barrier cages may be equally useful for managing infection burdens as they are in the culture of salmon. Indeed, barrier technology or submerged seacages may also be useful for preventing infection by monogenean skin and gill flukes as well. For example, Shirakashi et al. (2013) obtained a reduction of between 80% and 95% in the incidence of skin fluke infection when amberjack (*Seriola dumerili*) cages were submerged at depths of 2 and 4 m, respectively. These results can be explained by positive phototactic behavior of monogenean parasite larvae, which (like those of sealice) become more concentrated in the upper water layers. However, in the case of monogenean infections, regular cleaning of sea cages is a critical part of intregrated pathogen management strategies for these parasites, because monogenean eggs have sticky filaments that effectively attach to most substrates (particularly seacage netting), and the infective larval oncomiracidium stage is most active immediately after hatching, increasing infection pressure immediately around seacages (Chambers and Ernst 2005). This is unlike sealice nauplii which are not immediately infective when they hatch from the egg, but instead may be transported by currents many kilometers away before they moult into the infective copepodid stage.

One final aspect to be considered with respect to treatment of finfish farmed in offshore cages is that their greater isolation from the coast, together with the deeper water depths at which the cages are anchored, will inherently provide greater protection against many diseases of concern. This is because of the better water quality offshore, together with the fact that the larger populations of wild fishes in shallower inshore areas act as reservoirs of infection and vectors for disease introduction into seacages (Dempster et al 2009, Uglem et al. 2009). Because of this, offshore location of seacages is likely to reduce the risk of outbreaks of many viral, bacterial, protozoan and metazoan disease agents. Furthermore, it has been proven that moving seacages to deeper water can provide up to tenfold reduction in risk of infection by some important and problematic parasites such as blood flukes and sealice (Kirchhoff et al. 2011). Reductions in infections of blood flukes are probably because the deeper water interrupts their indirect lifecycle which requires passage through benthic intermediate hosts (polychaetes, bivalves) (Cribb et al. 2011, Warren and Bullard 2019) - a fact which bodes well for reducing risk of infection from other multihost disease agents such as myxozoa which also utilize benthic intermediate hosts; while the reduction in sealice infections reported by Kirchhoff et al. (2011) is probably due to improved dilution of infective copepodid stages.

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Appendix 1 – Definitions for risk estimations(without mitigation)

Risk estimation	Risk score	Definition
Extreme	5	Establishment of disease would cause substantial biological and economic harm at a regional or national level, and/or cause serious and irreversible environmental harm.
High	4	Establishment of disease would have serious biological consequences (high mortality or morbidity or loss of marketability) and would not be amenable to control or eradication. Such diseases would significantly harm economic performance at a regional level and/or cause serious environmental harm which is most likely irreversible.
Moderate	3	Establishment of disease would cause significant biological consequences (significant mortality or morbidity or loss of marketability) and may not be amenable to control or eradication. Such diseases could harm economic performance at a regional level on an ongoing basis and/or may cause significant environmental effects, which may or may not be irreversible.
Low	2	Establishment of disease would have moderate biological consequences and would normally be amenable to control or eradication. Such diseases may harm economic performance at a local level for some period and/or may cause some environmental effects, which would not be serious or irreversible.
Very Low	1	Establishment of disease would have mild biological consequences and would be amenable to control or eradication. Such diseases may harm economic performance at a local level for a short period and/or may cause some minor environmental effects, which would not be serious or irreversible.
Negligible	0	Establishment of disease would have no significant biological consequences and would require no management. The disease would not affect economic performance at any level and would not cause any detectable environmental effects.