ICES Journal of Marine Science

ICES International Council for the Exploration of the Sea
CIEM Conseil International pour l'Exploration de la Mer

ICES Journal of Marine Science (2015), 72(3), 997–1021. doi:10.1093/icesjms/fsu132

Contribution to the Themed Section: 'Risk Assessment'

Original Article

Risk assessment of the environmental impact of Norwegian Atlantic salmon farming

Geir Lasse Taranger[1], Ørjan Karlsen[2]*, Raymond John Bannister[1], Kevin Alan Glover[1], Vivian Husa[1], Egil Karlsbakk[1], Bjørn Olav Kvamme[1], Karin Kroon Boxaspen[1], Pål Arne Bjørn[3], Bengt Finstad[4], Abdullah Sami Madhun[1], H. Craig Morton[1], and Terje Svåsand[1]

[1]*Institute of Marine Research, PB 1870, N-5817 Bergen, Norway*
[2]*Institute of Marine Research, Austevoll Research Station, N-5392 Storebø, Norway*
[3]*Institute of Marine Research, PB 6404, N-9294 Tromsø, Norway*
[4]*Norwegian Institute for Nature Research, PB 5685 Sluppen, N-7485 Trondheim, Norway*

**Corresponding author: tel: +47 4691 2740; fax: +47 5618 2222; e-mail: OrjanK@imr.no*

Taranger, G. L., Karlsen, Ø., Bannister, R. J., Glover, K. A., Husa, V., Karlsbakk, E., Kvamme, B. O., Boxaspen, K. K., Bjørn, P. A., Finstad, B., Madhun, A. S., Morton, H. C., and Svåsand, T. Risk assessment of the environmental impact of Norwegian Atlantic salmon farming. – ICES Journal of Marine Science, 72: 997 – 1021.

Received 23 February 2014; revised 8 July 2014; accepted 10 July 2014; advance access publication 2 September 2014.

Norwegian aquaculture has grown from its pioneering days in the 1970s to be a major industry. It is primarily based on culturing Atlantic salmon and rainbow trout and has the potential to influence the surrounding environment and wild populations. To evaluate these potential hazards, the Institute of Marine Research initiated a risk assessment of Norwegian salmon farming in 2011. This assessment has been repeated annually since. Here, we describe the background, methods and limitations of the risk assessment for the following hazards: genetic introgression of farmed salmon in wild populations, regulatory effects of salmon lice and viral diseases on wild salmonid populations, local and regional impact of nutrients and organic load. The main findings are as follows: (i) 21 of the 34 wild salmon populations investigated indicated moderate-to-high risk for genetic introgression from farmed escaped salmon. (ii) of 109 stations investigated along the Norwegian coast for salmon lice infection, 27 indicated moderate-to-high likelihood of mortality for salmon smolts while 67 stations indicated moderate-to-high mortality of wild sea trout. (iii) Viral disease outbreaks (pancreas disease, infectious pancreatic necrosis, heart and skeletal muscle inflammation, and cardiomyopathy syndrome) in Norwegian salmon farming suggest extensive release of viruses in many areas. However, screening of wild salmonids revealed low to very low prevalence of the causal viruses. (iv) From ~500 yearly investigations of local organic loading under fish farms, only 2% of them displayed unacceptable conditions in 2013. The risk of eutrophication and organic load beyond the production area of the farm is considered low. Despite several limitations, especially limited monitoring data, this work represents one of the world's first risk assessment of aquaculture. This has provided the Norwegian government with the basis upon which to take decisions for further development of the Norwegian aquaculture industry.

Keywords: environmental impact, eutrophication, genetic interaction, organic load, pathogens, risk assessment, salmon lice.

Introduction

Background

The Atlantic salmon (*Salmo salar* L.) farming industry was first started in Norway in the early 1970s and has now grown to become one of the country's largest export industries by economic value. In addition to Atlantic salmon, which is by far the most significant species farmed in Norway, there are also commercial farming of rainbow trout [*Oncorhynchus mykiss* (Walbaum)] and other marine species such as Atlantic cod (*Gadus morhua* L.) and halibut [*Hippoglossus hippoglossus* (L.)]. In 2012, the production of Atlantic salmon and rainbow trout in Norway was 1 232 095 and 74 583 tons, respectively, and a total of 1006 marine farms

was licensed. These farms are distributed along much of Norway's coastline.

Aquaculture of salmonids in Norway, and other countries where these species are farmed in significant numbers, is primarily based around the production of eggs and juveniles in freshwater facilities on land, combined with grow out of fish in open marine cages. During the last decades, technical standards for the production of aquaculture infrastructure has improved dramatically. However, the primary methods for cultivation of finfish have remained similar, with the size of sea cages (up to 160 m in circumference) and the number of stocked smolt (up to 200 000 individuals per cage) increasing.

The rapid expansion of the aquaculture industry, both in Norway and other regions where this form of open-cage production has increased, has not occurred without environmental challenges. However, although a significant body of evidence suggests various environmental impacts of aquaculture, the rapid expansion of this industry means that management guidelines and targets to address potential negative effects have generally not developed in association with the rapid expansion of the industry. Therefore, there is a need for more coordinated efforts to identify hazards related to open sea cage farming and evaluate environmental risks.

Risk analysis

Several approaches have been suggested and discussed for risk analysis of marine ecosystems and marine aquaculture activities (Anon., 2006, 2010; Nash, 2007; GESAMP, 2008; Samuel-Fitwi *et al.*, 2012), and similar approaches of risk analysis and assessment have been adapted to animal welfare including welfare of farmed fish (e.g. EFSA, 2012). According to GESAMP (2008), a risk analysis should first involve hazard identification, then risk assessment of these hazards including the assessment of release, exposure, and consequences, followed by risk estimation/evaluation. The latter preferably related to politically defined thresholds of acceptability or level of protection. Subsequently, this can be followed up by appropriate risk management and appropriate risk communication.

A full risk analysis is based on the ability to quantify both the probability of a certain event and its consequences, but in biological systems it is normally very difficult to quantify these factors precisely. Hence, risk analyses in biological systems are often conducted using broad qualitative categories, by scoring the probability and consequences from low to high (e.g. GESAMP, 2008). This can in turn be based on some semi-quantitative assessment or on expert opinion as suggested by Anon. (2006).

In 2009, the Norwegian government established a set of environmental goals for sustainability in the "Strategy for an Environmentally Sustainable Norwegian Aquaculture Industry" (Anon., 2009b; Table 1). In response to this, the Institute of Marine Research, Norway, initiated a risk assessment of Norwegian salmon farming in 2010, and yearly since (Taranger *et al.*, 2011a, b, 2012a, 2013, 2014). These risk assessments were based on identified hazards and specific endpoints or proxies related to environmental impacts of salmon farming (Table 2). The endpoints/proxies were in turn derived from the governmental goals for environmental sustainability mentioned above. Moreover, evaluation thresholds for some of these endpoints/proxies (acceptance levels of impact or level of protection) were proposed (Taranger *et al.*, 2012b), and subsequently used in the risk assessments in 2013 and 2014. Here, we describe the way in which these assessments have been conducted, the methodological limitations and challenges, as well as future needs to data and analytical tools.

Table 1. The five primary goals for the future development of the Norwegian aquaculture industry as established by the Norwegian government in 2009.

Goals	
Goal 1: Disease	Disease in fish farming will not have a regulating effect on stocks of wild fish, and as many farmed fish as possible will grow to slaughter age with minimal use of medicines.
Goal 2: Genetic interaction	Aquaculture will not contribute to permanent changes in the genetic characteristics of wild fish populations.
Goal 3: Pollution and discharges	All fish farming locations in use will maintain an acceptable environmental state and will not have higher emissions of nutrient salts and organic materials than the receiving waters can tolerate.
Goal 4: Zoning	The aquaculture industry will have a location structure and zoning which reduces impact on the environment and the risk of infection.
Goal 5: Feed and feed resources	The aquaculture industry's needs for raw materials for feed will be met without overexploitation of wild marine resources.

Hazard identification

The first step in a risk assessment is to identify the most important hazards. A range of criteria for hazard identification was proposed by GESAMP (2008). This includes an analysis on how potential hazards relates to undesirable changes in the environment/ecosystem. To this end, potential hazards are evaluated for their possible severity, extent and duration of the change, either based on past experiences, analogue situations, or models. Some of the environmental challenges (i.e. hazards) identified include ecosystem and benthic community effects of organic loading and nutrients (Buschmann *et al.*, 2006; Kutti *et al.*, 2008; Bannister *et al.*, 2014), transfer of parasites to native populations (Krkošek *et al.*, 2005, 2013a, b; Jackson *et al.*, 2013; Skilbrei *et al.*, 2013; Torrissen *et al.*, 2013; Serra-Llinares *et al.*, 2014), disease interactions (Glover *et al.*, 2013b; Madhun *et al.*, 2014a), and ecological (Jonsson and Jonsson, 2006) and genetic interactions with wild populations (Crozier, 1993; Clifford *et al.*, 1998b; Skaala *et al.*, 2006; Glover *et al.*, 2012, 2013a).

Based on the accumulating evidence of the severity, geographical extent and duration and/or reversibility of the various impacts related to open sea cage salmon farming in Norwegian coastal waters, we have based the current risk assessment on the following hazards: (i) genetic introgression of escaped farmed salmon into wild populations, (ii) impact of salmon lice (*Lepeophtheirus salmonis*) on wild salmonid populations, (iii) potential disease transfer from farmed salmon to wild salmonid populations, and (iv) local and regional impacts of organic load and nutrients from marine salmon farms.

Impact of farmed escapees on the genetic integrity of wild Norwegian populations

Risk assessment

In the following chapter, we have considered the following elements of risk assessment; release, exposure, and consequences, in the following manner. Release assessment is defined as the number of farmed salmon escaping into the natural environment, both as reported and unreported numbers of escapees. Exposure assessment is defined as the physical mixing of farmed escaped salmon on the

Table 2. Identified hazards, process of concern, and endpoint of concern for goals 1–3 for the future development of the Norwegian aquaculture industry as established by the Norwegian government in 2009.

Hazard	Process of concern	Endpoint of concern
Genetic interaction (Goal 2)	Farmed escaped salmon successfully interbreed with wild salmon populations	Changes observed in the genetic characteristics of wild salmon populations
Salmon lice (Goal 1)	Salmon lice from fish farming affects wild fish	Salmon lice from fish farming significantly increase the mortality of wild salmonids
Viral diseases (Goal 1)	Disease transmission from fish farming affects wild fish	Viral transmission from fish farming significantly increase the mortality of wild salmonids
Discharges of organic material: (i) local effects (ii) regional effects (Goal 3)	Emissions of organic materials to the surrounding environment	(i) Unacceptable change in sediment chemistry and faunal communities in the production zone (ii) Significant change in bottom communities beyond the production zone—regional impact
Discharges of nutrients: (i) local effects (ii) regional effects (Goal 3)	Emissions of nutrients to the surrounding environment	(i) Nutrients from fish farms results in local eutrophication (ii) Nutrients from fish farms results in regional eutrophication

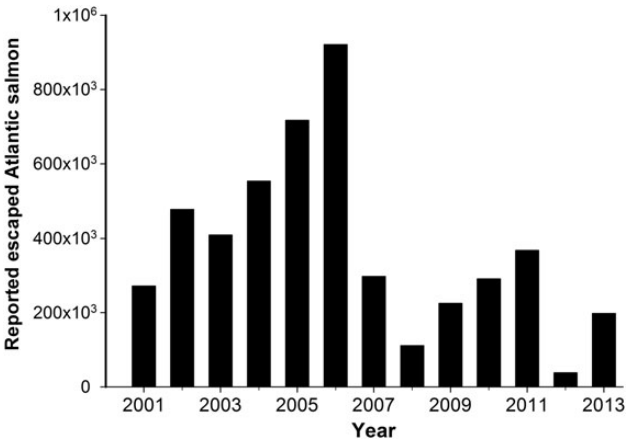

Figure 1. The number of farmed salmon escapes reported to the Norwegian Directorate of Fisheries by fish farmers for the period 2001–2013. Data were taken from the Norwegian Directorate of http://www.fiskeridir.no/.

spawning grounds of wild populations, and the subsequent level of genetic introgression resulting from successful spawning. Finally, we have defined consequence assessment as the consequence of genetic introgression for both the short fitness consequences and the long evolutionary consequences on the native populations.

One of the challenges to the continued development of a sustainable aquaculture industry is containment, and each year, thousands or hundreds of thousands of farmed salmon escape into the natural environment in Norway (Figure 1). Furthermore, the official statistics for numbers of escapees reported to the Norwegian Directorate of Fisheries underestimate the real number of escapees. This has been documented through extensive simulated release experiments and statistical modelling (Skilbrei *et al.*, 2015), and is clearly supported by the fact that the legal authorities in Norway have implemented DNA tracing methods to identify the farm of origin for escapees where they have not been reported (Glover *et al.*, 2008; Glover, 2010; Zhang *et al.*, 2013). While the majority of escapees disappear post-escape (Hansen, 2006; Skilbrei, 2010a, b, 2013), each year, significant numbers of farmed salmon are nevertheless observed in rivers inhabited by wild populations (Fiske *et al.*, 2006, Fiske, 2013). It is therefore considerable potential for genetic interaction between these escapees and native populations.

The Atlantic salmon displays considerable population genetic structure throughout its native range. This variation is partitioned in a hierarchical manner, with the largest genetic differences being observed between populations located in different continents or countries, and the smallest differences being observed among neighbouring populations within regions (Ståhl, 1987; Taggart *et al.*, 1995; Verspoor *et al.*, 2005). This structure reflects various processes, for example recolonization patterns, genetic isolation by distance (Glover *et al.*, 2012), and landscape features which modify population connectivity within regions (Dillane *et al.*, 2008). In addition to differences in allele frequencies of molecular genetic markers, Atlantic salmon populations display different life history characteristics. While much of this phenotypic variation is environmentally caused, some of these differences are influenced by underlying genetic variation, and it is generally accepted that these differences potentially reflect adaptations to their native rivers (Taylor, 1991; Garcia de Leaniz *et al.*, 2007; Fraser *et al.*, 2011).

Norwegian farmed Atlantic salmon dominates global production, originates from over 40 Norwegian rivers, and has been subject to approximately ten or more generations of domestication selection (Gjedrem, 2010). Breeding programmes have successfully selected for fish that outgrow their wild counterparts multiple times under farming conditions (Glover *et al.*, 2009; Solberg *et al.*, 2013a, b). In addition to traits that have been directly selected for in the breeding programmes, genetic changes in non-targeted traits have also been observed, for example in predator awareness (Einum and Fleming, 1997), stress tolerance (Solberg *et al.*, 2013a), and gene transcription (Roberge *et al.*, 2006). In addition, decreased genetic variation, as revealed by molecular genetic markers (Norris *et al.*, 1999; Skaala *et al.*, 2004), and lower estimates of heritability for growth (Solberg *et al.*, 2013a), has been observed in farmed populations. Reduced genetic variation in molecular genetic markers reflects founder effects and genetic drift, driven by limited farmed population sizes, while reduction in heritability for growth is likely to be a result of successful directional selection for this trait over multiple generations.

An early study in Ireland estimated introgression of farmed escaped salmon in a native population based upon escapement

from a nearby farm (Clifford *et al.*, 1998b). However, despite the fact the genetic changes in native wild populations have been observed in molecular genetic markers as a result of farmed salmon introgressing in Canadian (Bourret *et al.*, 2011), Irish (Crozier, 1993, 2000; Clifford *et al.*, 1998a, b), and Norwegian rivers (Skaala *et al.*, 2006; Glover *et al.*, 2012), until a recent breakthrough in a study of 20 Norwegian rivers (Glover *et al.*, 2013a), the cumulative level of introgression of farmed salmon has not been calculated for any native Atlantic salmon population. This is due to the fact that estimation of cumulative introgression of farmed salmon is statistically challenging (Besnier *et al.*, 2011). In the Norwegian study of 20 rivers (Glover *et al.*, 2013a), a combination of Approximate Bayesian Computation, and genetic data for wild-historical, wild-contemporary and a diverse pool of farm samples that were genotyped for a set of collectively informative single-nucleotide polymorphic markers (Karlsson *et al.*, 2011) was used to estimate introgression for a period of 2–4 decades.

Overall, the study by Glover *et al.* (2013a) revealed less introgression of farmed Atlantic salmon in many Norwegian populations (Figure 2) than may be expected based upon the reported numbers of escapees in these populations, and estimations from introgression models (Hindar *et al.*, 2006). The authors concluded that spawning success of farmed escaped salmon has been generally low in many Norwegian rivers, a suggestion consistent with earlier estimates of spawning success in controlled experiments (Fleming *et al.*, 1996, 2000). Nevertheless, results from the study demonstrated high levels of admixture in some native populations, and together with an earlier study using microsatellites, reported decreased genetic differentiation among populations over time (Glover *et al.*, 2012, 2013a). The latter of which is consistent with suggestions that widespread introgression of farmed salmon will lead to erosion of population genetic structure among native populations (Mork, 1991).

Estimating the genetic consequences of farmed salmon introgression on life history traits, population fitness, and long-term evolutionary capacity of wild populations is more challenging than estimating introgression. This is in part due to the fact that wild populations display large natural variation in, for example, marine survival, and at the same time are influenced by a wide range of anthropogenic factors (Parrish *et al.*, 1998), which may potentially mask biological changes caused by introgression of farmed salmon. Nevertheless, comparative studies in Ireland and Norway have demonstrated additive genetic variation for fitness in the wild, with offspring of farmed salmon displaying lower survival than fish of native origin (McGinnity *et al.*, 1997, 2003; Fleming *et al.*, 2000; Skaala *et al.*, 2012). Similar studies conducted on other salmonid species in response to releases of hatchery fish have also arrived at similar conclusions (Araki *et al.*, 2008; Araki and Schmid, 2010).

In summary, the presence of farmed escaped salmon on the spawning grounds of native populations, and the potential for genetic interactions between escapees and wild conspecifics, is of concern. This is because farmed escapees may be genetically different from the recipient wild population for several reasons. (i) Farmed salmon usually do not originate from the same wild population into which they migrate post-escape and will therefore display population genetic differences to the native population. (ii) Farmed salmon have been subject to directional selection and thus differ to all wild salmon for those traits. (iii) Through relaxation of natural selection and inadvertent adaption to the domestic environment, farmed salmon have undergone domestication selection and will also differ to wild salmon.

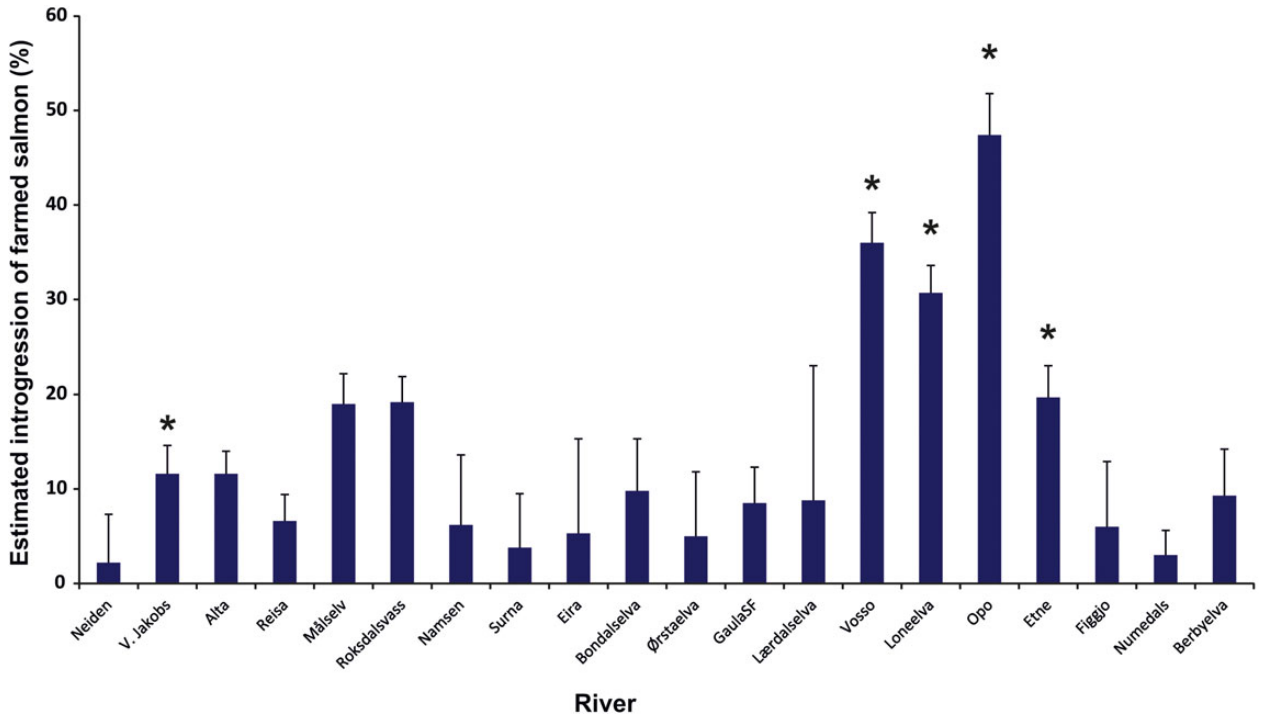

Figure 2. Estimated cumulative introgression of farmed Atlantic salmon in 20 Norwegian Atlantic salmon populations in the period 1970–2008 based upon Approximate Bayesian computation using genetic data. Figure is produced using estimations of admixture from Table 3 in Glover *et al.* (2013a). The computed median level of introgression is 9.1%.

Risk estimation

As part of a national strategy for an environmentally sustainable aquaculture industry (Anon., 2009b), the Norwegian government established the following management goal to prevent genetic interactions of farmed escapees with wild salmon populations: "Aquaculture will not contribute to permanent genetic changes in the genetic characteristics of wild fish stocks" (Table 1). This political target, which forms the basis for the risk estimation, is clearly open for scientific interpretation. However, it was interpreted in a conservative sense for the estimation of risk (Table 2). Thus, any observed genetic change in allele frequencies of molecular genetic markers caused by introgression of farmed salmon would be regarded as permanent genetic change, and therefore in violation of the management goal for sustainability established by the government. The rationale behind this interpretation was first and foremost because molecular genetic markers would be required to directly measure genetic changes in the wild populations. Furthermore, while natural selection will influence the genetic composition of any population, including those where farmed salmon have successfully introgressed, it is unlikely that natural selection will revert the population back to its exact genetic composition before introgression of farmed salmon. This is despite the possibility that natural selection may potentially restore fitness in the natural population.

The documentation of genetic change in a wild population is most directly achieved through the analysis of molecular genetic markers. However, while introgression of farmed Atlantic salmon has been estimated for 20 wild salmon populations in Norway in a 3–4 decade period from 1970 onwards (Glover *et al.*, 2013a), genetic data to estimate introgression of farmed salmon does not exist for the great majority of Norwegian populations. Furthermore, the analysis was being used to address risk of continued and future genetic changes in relation to today's aquaculture industry rather than changes that have already occurred through historical introgression. Therefore, the frequency of farmed escaped salmon observed in wild populations, which is correlated with genetic introgression of farmed escapees over time (Glover *et al.*, 2012, 2013a) was chosen as the indicator to estimate risk of further genetic changes in each wild population for the estimation of risk (Taranger *et al.*, 2012a).

From 2012 onwards, risk was estimated against the below categories for probability of further genetic changes in wild populations caused by introgression of farmed salmon:

No or low risk of genetic change: $<4\%$ incidence of farmed salmon

Moderate risk of genetic change: 4–10% incidence of farmed salmon

High risk of genetic change: $>10\%$ incidence of farmed salmon

The threshold values were set according to knowledge of natural straying (reviewed by Stabell, 1984) and knowledge about the present correlation between frequency of farmed fish and corresponding genetic introgression (Glover *et al.*, 2012, 2013a). Stabell (1984) showed that most fish returned to their natal river, although in two of the experiences referred to, straying rates were as high as 10% and nearly 20%. We have chosen a threshold value for no or low risk for genetic change at a frequency of farmed fish in the river corresponding to the lower part of the natural straying estimates (4%), while 10% as high risk of genetic change from the upper part of the distribution. Especially, the threshold value for the upper limit is uncertain and might be modified according to new knowledge about the corresponding correlation between frequency of farmed fish and actual introgression (Glover *et al.*, 2013a; Taranger *et al.*, 2014).

The frequency of farmed salmon in each river surveyed was based upon autumn data where the frequency has been reported for a series of Norwegian rivers (Fiske, 2013). To estimate risk, the frequency of farmed salmon observed in autumn survey was recomputed into an "incidence of farmed salmon" per population using a formula for normalizing data from summer angling catches and autumn surveys (Diserud *et al.*, 2010). This was done because the percentage of farmed salmon in autumn samples is usually higher than in summer angling catches (Fiske *et al.*, 2006), which is in part because farmed salmon enter rivers later than wild salmon. Thus, without normalization of data, the frequency of farmed salmon in summer and autumn surveys are not directly comparable. While many rivers have both summer and autumn estimates, some only have one or the other estimate and therefore require transforming into what has been defined as the "incidence of farmed salmon". The formula for normalizing data from summer and autumn surveys to create the "incidence of farmed salmon" were obtained from Diserud *et al.* (2010) and are presented below:

$$\begin{aligned}&\arcsin\left(\sqrt{(\text{incidence of farmed salmon})}\right)\\&\quad = 0.116 + 0.888 \times \arcsin\left(\sqrt{(\text{summer frequency})}\right)\\&\arcsin\left(\sqrt{(\text{incidence of farmed salmon})}\right)\\&\quad = 0.044 + 0.699 \times \arcsin\left(\sqrt{(\text{autumn frequency})}\right)\end{aligned}$$

For the risk assessment, the mean incidence of farmed salmon was estimated in 34 rivers distributed along the Norwegian coast using autumn survey data collected in the period 2010–2012. Only rivers having autumn survey data from a minimum of 2 of the 3 years in this survey period were included in the risk assessment. Based on these data, the risk for genetic changes as a result of farmed salmon introgression was low, moderate, or high for 13 (38%), 11 (32%), and 10 (29%) surveyed rivers, respectively (Figure 3).

Limitations

There are a number of challenges and limitations to the conducted risk assessment and estimation, its approach, and calibration against threshold values and potential impacts. One limitation is the fact that the observed frequency of farmed escapees in rivers has been used as the proxy for potential genetic changes in wild populations caused by introgression of the farmed salmon. This is a limitation because the correlation between the frequency of escapees observed in a river, and documented genetic introgression is only modest ($R^2 = 0.47$, $p = 0.0007$) (Glover *et al.*, 2013a). As a result, some rivers display higher and lower levels of genetic introgression from farmed than would be estimated by analysis of the frequency of escapees on the spawning grounds. The consequence of this is that the observed frequency of farmed escapees in each population will not accurately reflect the true risk of genetic changes for all populations, and only by using genetic methods directly will the risk be able to be quantified accurately.

The underlying causes of the lack of a strong relationship between the observed frequency of escapees and genetic introgression are important to identify to help improve the accuracy of the risk assessment in the future. From their genetic study of introgression in 20 Norwegian rivers, Glover *et al.* (2013a) identified both technical and biological elements that are likely to influence the

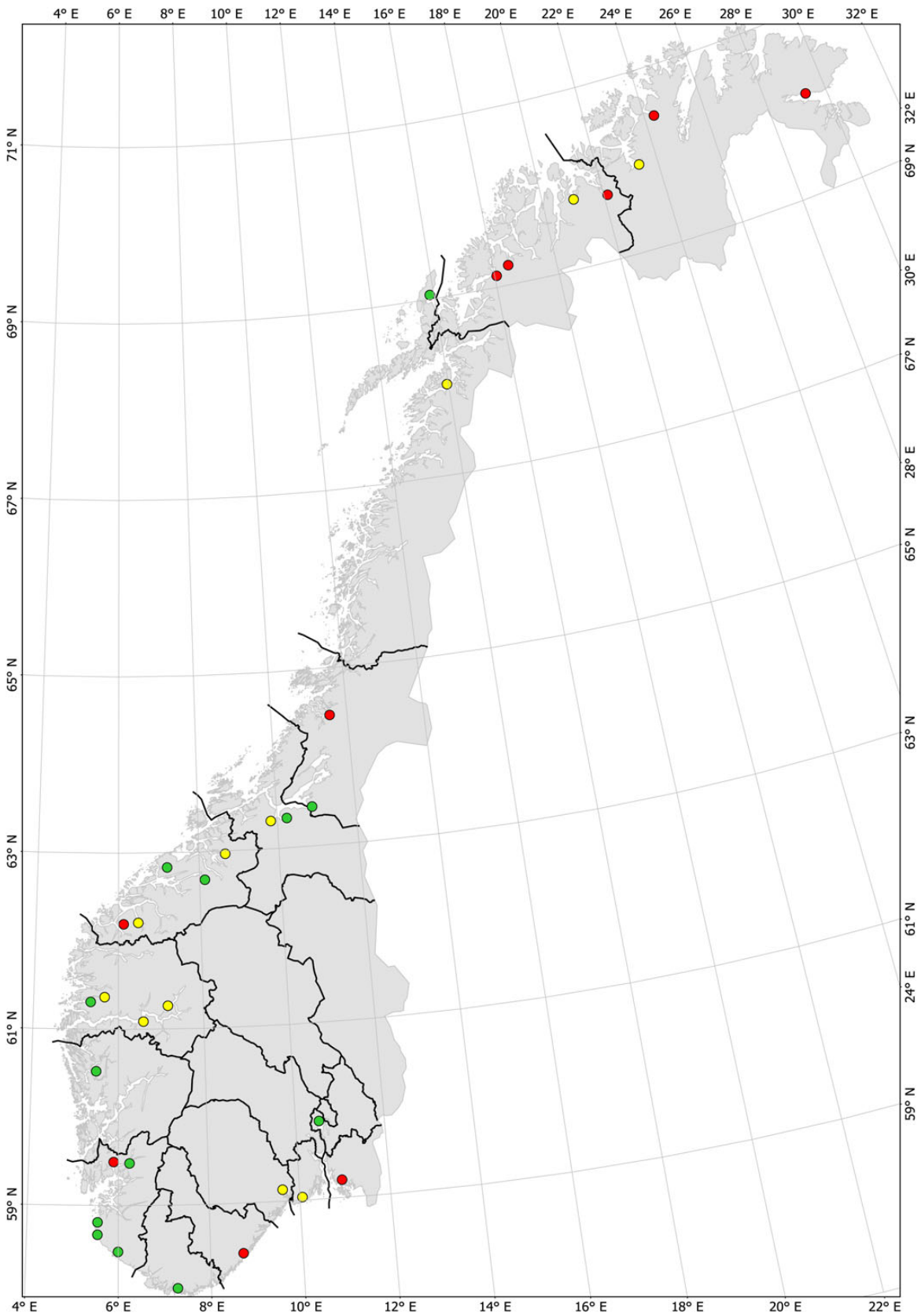

Figure 3. Assessment of risk for genetic changes in 34 wild Atlantic salmon populations in Norway in the period 2010 – 2012. Green point = No or low risk, yellow point = moderate risk, and red point = high risk.

Table 3. Some identified gaps in current knowledge with respect to understanding the potential negative consequences of introgression of farmed salmon in native populations.

Question	Hypothesis to be tested
What is the fitness differential between the offspring of wild, hybrid, and farmed salmon, including multiple generation back-crossed individuals, in different rivers and environments?	To what degree can the results from the few comparative experiments of survival in the wild be generalized for all types of rivers and populations, and is it possible to generalize results?
How strong is natural selection, and will the offspring of farmed salmon readapt to the natural environment?	Lower survival of the offspring of farmed salmon and hybrids in the wild also implies that natural selection will purge poorly adapted individuals from the recipient population, but how strong is natural selection and what is the time-scale of this potential re-adaptation process?
What are the threshold limits of introgression?	What degree of genetic introgression will be tolerated in wild populations before biological, life history and ecological characteristics of the population, and population productivity are compromised?
What is the underlying genomic architecture of domestication and local adaptation in salmonids?	What genetic changes have occurred during domestication, is it possible to identify genetic markers linked with these changes, and fitness in the wild to provide more accurate measurements of functional genetic changes in native populations?

strength of the relationship between the observed frequency of escapees and genetic introgression. Two of the primary components suggested were potential inaccuracies in the frequency of escapees reported for each river (e.g. limited, biased, or non-standardized sampling or reporting), and the fact that the density of the native population, especially on the spawning grounds, may also influence relative success of farmed escapees through spawning competition (Fleming *et al.*, 1996, 2000). In the future, models may be used which include covariables in addition to just the incidence of farmed salmon to predict genetic changes.

A further limitation of the present risk assessment is that it was only conducted for 34 rivers for the 2013 risk assessment. These rivers were chosen as they had autumn survey data published (Fiske, 2013) and thus readily available for assessment of risk for the period 2010–2012. In Norway, there are over 400 salmon rivers and for ∼220 of them the status of the stocks are assessed (Anon., 2013b). Thus, the rivers investigated in the current risk assessment only represent a small proportion of those in Norway. Therefore, it is not possible to make clear regional inferences regarding introgression of farmed salmon, only for a small number of specific rivers. Data for the frequency of farmed salmon exist for a larger number of rivers than are currently included in this risk assessment. However, the quality of some of these data, the reporting and availability of the data are highly variable. It was for this reason that the risk assessment was only conducted for the 34 rivers.

Clearly, there is a significant need to increase efforts to expand and improve the monitoring of escaped salmon in a larger number of Norwegian rivers using data gathered and reported in a standardized manner. This will initially be able to improve estimates of the proportion of escaped salmon and will also provide a better foundation for the collection of representative samples for subsequent use in genetic analysis to validate introgression in rivers. An effort to coordinate data collection of escapees has been initiated within Norway in 2014, and in the future it is predicted that the risk assessment will be conducted in a much larger number of rivers.

In addition to technical and data availability challenges linked with the risk assessment and its implementation, there are gaps in current knowledge which limit the ability to identify threshold tolerance limits for introgression of escapees and the level of potentially detrimental effects on the wild populations. Current knowledge points toward a potential negative effect of introgression of farmed Atlantic salmon on the fitness and future evolutionary capacity of recipient wild populations. This is when taking into consideration data available from experimental comparisons of farmed and wild salmon especially in the natural environment (McGinnity *et al.*, 1997, 2003; Fleming *et al.*, 2000; Skaala *et al.*, 2012), background knowledge of salmon biology, life history and ecology, and extensive information from hatchery-fish supplementation for both Atlantic salmon as well as other salmonid species in both the Atlantic and Pacific (Araki *et al.*, 2008; Araki and Schmid, 2010). Nevertheless, significant gaps in understanding of the biological consequences of introgression of farmed salmon remain. These need to be quantified in the future to make a full assessment of risk of biological consequences following introgression of escapes. The major points are summarized in Table 3.

Salmon lice impact on wild salmonids

Salmon lice (*Lepeophtheirus salmonis*) from salmon farms are recognized as an important hazard to wild anadromous salmonids in Norwegian coastal waters (Serra-Llinares *et al.*, 2014). Salmon lice on farmed salmon produce large amounts of planktonic larvae stages that spread via the water currents and can infect migrating Atlantic salmon post-smolts, as well as sea trout (*Salmo trutta*) and Arctic charr (*Salvelinus alpinus*) that stay in coastal waters (Jones and Beamish, 2011). Hydrodynamic models coupled with biological data show that salmon lice can be transported up to 200 km over a 10-d period, although most dispersed 20–30 km (Asplin *et al.*, 2011; Serra-Llinares *et al.*, 2014). The number of salmon lice allowed on farmed salmon is tightly controlled by Norwegian legislation (www.mattilsynet.no). However, the large number of farmed salmon, with ∼300 million smolts put into sea cages every year along the Norwegian coast, results in worse case releases in the order of more than a billion salmon lice larvae daily from salmon farms in Norway (Taranger *et al.*, 2014).

New analyses reveal strong correlation between salmon farms and lice infections on wild salmonids in Norwegian coastal waters (Helland *et al.*, 2012; Serra-Llinares *et al.*, 2014). The Norwegian salmon lice monitoring programme on wild salmonids demonstrate annual lice epidemics, most likely connected to the density of salmon farms in the surrounding areas as well as the seasonal dynamics of salmon lice infections on farmed salmon (Jansen *et al.*, 2012; Serra-Llinares *et al.*, 2014; Taranger *et al.*, 2014). A series of experiments has shown that salmon lice may affect anadromous salmonids (reviewed in Finstad and Bjørn, 2011; Anon., 2012; Torrissen *et al.*, 2013).

To assess the risk of salmon lice infection on wild populations, we have considered the following elements of risk assessment: release, exposure, and consequences in the following manner. The release

assessment is based on estimating the production and distribution of infectious salmon lice. The exposure assessment is based on estimating the lice infection on wild salmonid populations using different methods for direct measurements of salmon lice infections on salmon and sea trout. The consequence assessment is the effect salmon lice have on salmonid populations in terms of estimated likelihood of increased marine mortality and/or reduced reproduction based on the exposure assessment.

Risk assessment

All salmon farming sites in Norway report the numbers of salmon lice on the fish weekly when the temperature is >4°C. This is reported together with fish biomass and number of individual salmon per cage (reported each month). Based on the number of sexually mature female salmon lice on the fish on each farming site, the number of infectious salmon lice larvae produced from the different salmon farms are calculated (Jansen *et al.*, 2012). However, at present we do not have enough information and validated models to accurately estimate the impact of the salmon lice infections on wild populations based on reported data from the fish farms (Taranger *et al.*, 2013, 2014).

The lice infection on wild salmonid populations is estimated using different methods as part of a national monitoring programme (Serra-Llinares *et al.*, 2014; Taranger *et al.*, 2014). These methods encompass catch of sea trout and Arctic charr in traps or nets (Bjørn *et al.*, 2011a), and salmon post-smolts caught in special surface trawls in fjord systems (Holm *et al.*, 2000; Bjørn *et al.*, 2007b; Holst *et al.*, 2007). In addition, groups of small sentinel cages containing on average 30 farmed salmon post-smolts are placed in the fjords to monitor the salmon lice infection rate. The fish are kept in the cages for 3 weeks before lice are counted on all the fish, and the procedure repeated three times during spring and summer (Bjørn *et al.*, 2011a, 2013). The sampling programme is focused on areas with high salmon farming activity (Figure 4), as well as some fjords that are protected against salmon farming. The assessment in the period 2010–2013 is based on data from 1 to 5 sites per fjord in 13–16 fjord systems annually, and with increasing numbers of fish sampled at each site in the later years (Table 4). More details about the national salmon lice monitoring programme are provided elsewhere (Helland *et al.*, 2012; Bjørn *et al.*, 2013; Serra-Llinares *et al.*, 2014).

To conduct the risk assessment on the potential impact on salmonid smolts that migrate from the rivers in spring and early summer on the one hand, and the risk of sea trout and Arctic charr that stay in fjords and coastal waters during summer, the national monitoring programme covers two different periods. These periods are adjusted for different timing of smolt migration and seawater residence along the Norwegian coast (Anon., 2011), with an earlier sampling window in the southern part of Norway and later further north. This corresponds to two assumed "critical periods", the first during spring when the salmonid smolts leave the rivers and enters the estuaries and fjords (Period 1), and the second period to estimate the accumulated infection rate on sea trout and Arctic charr that remain in fjords and on the coast during summer (Period 2). See also Anon. (2011) for further information on median migration dates and migration speeds for Atlantic salmon post-smolts in Norwegian fjords.

A range of laboratory studies demonstrate the impact of salmon lice on salmon post-smolts (Grimnes and Jakobsen, 1996; Finstad *et al.*, 2000, 2010; Heuch *et al.*, 2005; Wagner *et al.*, 2008). It has been shown that 0.04–0.15 lice per g fish weight can increase stress levels, reduce swimming ability and create disturbances in water and salt balance in Atlantic salmon (Nolan *et al.*, 1999; Wagner *et al.*, 2003, 2004; Tveiten *et al.*, 2010). In sea trout, ~50 mobile lice are likely to give direct mortality (Bjørn and Finstad, 1997), and only 13 mobile lice, or~0.35 lice per g fish weight might cause physiological stress in sea trout (weight range of 19–70 g; Wells *et al.*, 2006, 2007). Moreover, ~0.05–0.15 lice per g fish weight were found to negatively affect sexually maturing Arctic charr (Tveiten *et al.*, 2010).

According to a review by Wagner *et al.* (2008), infections of 0.75 lice per gram fish weight, or ~11 salmon lice per fish, can kill a recently emigrated wild smolt of ~15 g if all the salmon lice develop into preadult and adult stages. Studies of naturally infected wild

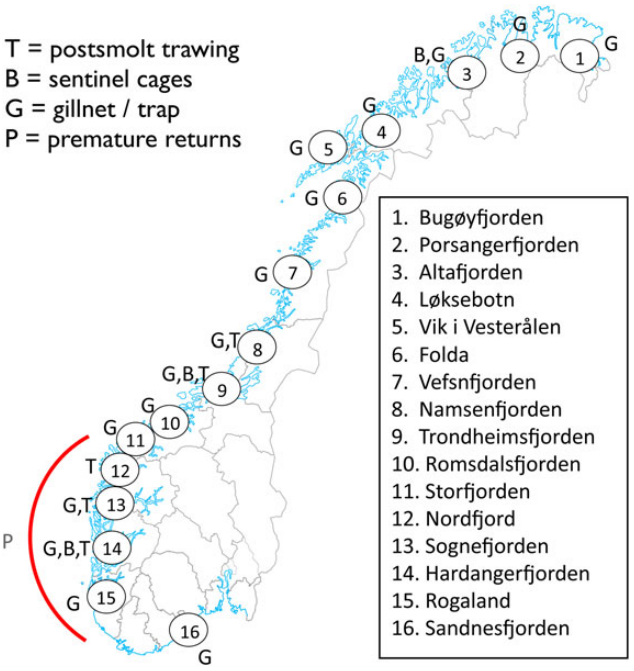

Figure 4. Sampling localities in Norwegian salmon lice monitoring programme applying various techniques such as gillnets and traps, post-smolt trawling, sentinel cages, and recording of premature return to rivers of sea trout during 2010 – 2013. The total number of sampling localities/sites with gillnets and traps has varied between 26 and 32 in the period.

Table 4. The number of sampling locations and number of wild-caught salmonids (mostly sea trout) investigated for salmon lice infestations in the Norwegian salmon lice monitoring programme during 2010 – 2013.

	Sites and number sampled in Period 1 (only fish < 150 g)				Sites and number sampled in Period 2 (all fish sizes)			
Year	2010	2011	2012	2013	2010	2011	2012	2013
Locations	26	31	29	23	26	32	29	23
n	218	422	944	1711	623	806	1144	2368

Period 1 covers smolt migration in spring and early summer, whereas Period 2 covers summer period to assess the accumulated effects on sea trout and Arctic charr. Both periods are adjusted different timing of smolt migration and seawater residence along the Norwegian coast, with an earlier sampling window in the southern part of Norway and later further north.

salmon post-smolts indicate that only those with <10 lice survived the infection (Holst *et al.*, 2007). This is consistent with field studies on salmon lice infections in salmon post-smolts in the Norwegian Sea. Over a decade of surveys, no post-smolts was found with >10 salmon lice, and fish with up to 10 mobile lice were observed to be in poor condition with low blood count and poor growth (Holst *et al.*, 2007). New studies of naturally infected wild salmon post-smolts also show that sea lice are fatal at high infections (Berglund Andreassen, 2013). More work in this field is in progress.

Population-wise effects of salmon lice have been demonstrated on wild salmonids in Ireland and Norway. This was studied by protecting individually tagged Atlantic salmon smolts against salmon lice (using Substance EX or Slice) before they were released into the sea near their respective home rivers (Finstad and Jonsson, 2001; Hazon *et al.*, 2006; Skilbrei and Wennevik, 2006; Hvidsten *et al.*, 2007; Skilbrei *et al.*, 2008, 2013; Jackson *et al.*, 2011, 2013; Anon., 2012; Gargan *et al.*, 2012; Krkošek *et al.*, 2013a, b; Vollset *et al.*, 2014). These studies suggest that salmon lice infections increase the marine mortality in areas with intensive salmon farming activity.

Risk estimation

The risk assessment on salmon lice is based on Goal 1 in the policy document on sustainable aquaculture by the Norwegian government from 2009 (Anon., 2009b, Table 1) stating that "Disease in fish farming will not have regulating effect on populations of wild fish…". This target has in this risk estimation been interpreted as "Salmon lice from fish farming significantly increase the mortality of wild salmonids" (Table 2).

A salmon lice risk index, attempting to estimate the increased mortality due to salmon lice infections, was proposed by Taranger *et al.* (2012a) and are based on the assumption that small salmonid post-smolts (<150 g body weight) will suffer 100% lice-related marine mortality, or return prematurely to freshwater for sea trout, in the wild if they are infected with >0.3 lice g^{-1} fish weight. Furthermore, the lice-related marine mortality is estimated to 50%, if the infection is between 0.2 and 0.3 lice g^{-1} fish weight, 20% if the infection rate is between 0.1 and 0.2 lice g^{-1} fish weight, and finally 0% lice-related mortality if the salmon lice infection is <0.1 lice g^{-1} fish weight.

For larger salmonids (over 150 g), we assume that lice-related mortality or compromised reproduction will be 100% in the group if they have >0.15 lice g^{-1} fish weight, 75% for lice infections between 0.10 and 0.15 lice g^{-1} fish weight, 50% for lice infections between 0.05 and 0.10 lice g^{-1} fish weight, 20% for lice infections between 0.05 and 0.01 lice g^{-1} group, and finally 0% if the salmon lice infection is <0.01 lice g^{-1} fish weight.

For both indices, increased mortality risk or compromised seawater growth or reproduction at population level are calculated as the sum of the increased mortalities/compromised reproduction for the different "infection classes" in the sample, reflecting the distribution of the intensity of salmon lice infections of the different individuals sampled. This assumes that individuals caught with traps, gillnets or trawls are representative for the various salmonid populations in that fjord area. The risk was further scored according to the system proposed by Taranger *et al.* (2012a); as low (<10% estimated increase in mortality; green colour), moderate (yellow) for those with between 10 and 30% increase, and high (red) if the increase is calculated as >30%.

The current assessment is based on these scorings, and we use data from the national monitoring programme on salmon infections in wild salmonids (Bjørn *et al.*, 2010, 2011b, 2012, 2013). Separate result tables are presented for the risk for Atlantic salmon smolts (Period 1; Table 5) and for sea trout and the Arctic charr populations (Period 2; Table 6). The results are sorted by county from south to north and by fjord system.

The results indicate considerable variation in risk between years and sampling locations. Moreover, these data strongly indicate a much higher risk for sea trout (and also Arctic charr in the Northern regions) compared with Atlantic salmon post-smolts and reveal moderate-to-high risk of population-reducing effects on sea trout in most counties with high salmon farming activity. The risk of population-reducing effects for Atlantic salmon varies much more between years and sites, and was low at most sites in 2010 and 2013, but moderate and high at several sites 2011 and 2012.

Limitations

The assessment in the period 2010–2013 is based on data from 13 to 16 fjord systems annually. Despite large field effort (Bjørn *et al.*, 2011b, 2012, 2013), the geographical coverage is insufficient in terms of the distribution of salmon farms and wild salmonid populations along the Norwegian coast. There are also problems considering how well the different sampling methods are representative for the different anadromous populations in that area. We have limited data on salmon lice infections in migrating Atlantic salmon smolts, so the risk assessment for salmon is mainly conducted by the use data on salmon lice infections on sea trout caught in traps and gillnets in Period 1 as proxy for the risk to Atlantic salmon post-smolts. Lice infections on trout may not be directly proportional to lice infections on migrating salmon smolt. It is likely that differences in, for example, migratory behaviour and marine ecology exposes salmon and sea trout smolts for different sea lice infection risk (Anon., 2011), even within the same fjord system (Bjørn *et al.*, 2007a, 2011b, c, 2013; Serra-Llinares *et al.*, 2014). The link between individual lice infections and population effects is also very uncertain. There is therefore uncertainty of the current risk assessment both for Atlantic salmon, and for sea trout and Arctic charr. Moreover, the current data are presented without any estimates of uncertainty, which must be included in future analyses.

Future

The "Strategy for an environmentally sustainable aquaculture industry" (Anon., 2009b) states that no disease, including lice, should have a regulatory effect on wild fish. The monitoring of salmon lice infection of wild salmonids is an important verification of whether this goal is achieved, and whether the measures taken are appropriate and sufficient. An indicator system that allows detection of possible problems needed therefore to be established. Hitherto, this assessment has been based on data from the national monitoring of sea lice. An analysis of the historical data in this monitoring series (2004–2010) shows that both the extent and nature of the data have had some weaknesses that limit the ability to analyse and understand the observed variation in infections on wild fish (Helland *et al.*, 2012; Serra-Llinares *et al.*, 2014). Therefore, monitoring and risk assessment based only on lice counts on wild salmonids is not considered sufficient.

Consequently, a rather radical change in the monitoring, advisory and management system for lice has therefore been proposed (Taranger *et al.*, 2012b, 2013, 2014; Bjørn *et al.*, 2013; Serra-Llinares *et al.*, 2014). This system is based on (i) detection and forecasting of increased production of infectious salmon lice using models, (ii) verification of infection pressure through risk-based

Table 5. Estimated risk for lice-related mortality (%) of Atlantic salmon post-smolts based on lice levels on sea trout caught in traps or gillnets and with weight <150 g in Period 1 at the sites from south to north in Norway in 2010–2013.

County	Fjord	Site	2010	2011	2012	2013
Aust-Agder	Sandnesfjord	Sandnes	0	0	0	
Rogaland	Ryfylke	Hellvik		0	0	0
		Vikedal [a2012]		36	20	
		Nedstrand				3
		Forsand		0	0	
Hordaland	Hardanger	Granvin	0	0		
		Ålvik		54	51	0
		Rosendal	0	69	53	13
		Etne	0	0	16	1
Sogn og Fjordane	Sognefjorden	Balestrand	0	0	2	0
		Vik				0
		Brekke/Dingja	0	35	23	0
Møre og Romsdal	Romsdal	Eresfjord	0	0	0	
		Sandnesbukta				22
		Isfjord	0	0	0	0
		Bolsøy [a2010–2012]	2	10	22	15
		Vatnefjorden				0
		Frænfjorden				7
	Storfjord	Sylte	0	0	37	
		Sykkylven	0	0	0	
		Ørsta	0	5	9	
Sør-Trøndelag	Trondheimsfjorden	Skatval [a2010]	6	0	2	0
		Agdenes	0	90	94	0
		Hitra	0	5	0	0
Nord-Trøndelag	Namsen	Tøtdal	0	0	0	0
		Sitter [a2010–2011]	32	24	71	15
		Vikna			7	98
Nordland	Eidsfjord	Vik [a2010–2012]	0	32	50	
	Folda	Ballkjosen	0	25	13	
		Sagfjord	0	7	7	
	Vefsn	Fagervika	0	3		
		Leirfjord	0	0		
	Velfjord	Indre Velfjord [a]			4	
		Ytre Velfjord [a]			4	
Troms	Salangen	Løksa [a2012]		0	4	
Finnmark	Altafjord	Talvik [a2012]	0	0	1	12
		Skillefjord	5	0	3	26
	Bugøyfjord	Bugøyfjord				
	Porsanger	Handelsbukt	0	0		0
		Kåfjord/Repvåg [a2013]	0	0		0

The colour code refers to the assessment of potential population-reducing effect (red = high, yellow = moderate, and green = low).
[a]Small number of fish caught, all fish were used in the assessment irrespective of body size.

Table 6. Estimated risk for salmon lice-related mortality or compromised reproduction (%) of sea trout based on observations in Period 2 at the various sites from south to north in Norway in 2010 – 2013.

County	Fjord	Site	2010	2011	2012	2013
Aust-Agder	Sandnesfjord	Sandnes	0	2	2	
Rogaland	Ryfylke	Hellvik		0	0	0
		Vikedal		15	0	
		Nedstrand				7
		Forsand		3	0	
Hordaland	Hardanger	Granvin	0	14		
		Ålvik		17	40	32
		Rosendal	55	67	87	38
		Etne	54	3	74	32
Sogn og Fjordane	Sognefjorden	Balestrand	1	0	3	0
		Vik				
		Brekke / Dingja	46	19	72	19
Møre og Romsdal	Romsdal	Eresfjord	0	21	8	
		Sandnesbukta				71
		Isfjord	7	15	25	26
		Bolsøy	14	13	32	73
		Vatnefjorden				98
		Frænfjorden				81
	Storfjord	Sylte	0	0	0	
		Sykkylven	3	10	34	
		Ørsta	25	16	35	
Sør-Trøndelag	Trondheimsfjorden	Skatval	0	13	2	11
		Agdenes	26	40	34	35
		Hitra	8	47	88	41
Nord-Trøndelag	Namsen	Tøtdal	0	9	14	55
		Sitter	65	40	59	62
		Vikna			57	60
Nordland	Eidsfjord	Vik	34	54	59	
	Folda	Ballkjosen	52	45	66	
		Sagfjord	4	52	32	
	Vefsn	Fagervika	19	4		
		Leirfjord	3	0		
	Velfjord	Indre Velfjord			34	
		Ytre Velfjord			28	
Troms	Salangen	Løksa		22	20	
Finnmark	Altafjord	Talvik	3	47	5	18
		Skillefjord	4	55	24	51
	Bugøyfjord	Bugøyfjord		10		
	Porsanger	Handelsbukt	0	0		5
		Kåfjord/Repvåg	0	0		27

Mortality estimates are based on all fish sampled in the period, with different thresholds for small (<150 g) and large (>150 g) fish. The colour code refers to the assessment of potential population-reducing effect (red = high, yellow = moderate, and green = low).

and adapted surveillance on wild salmonids and (c) extended risk assessment based on a considerably larger dataset and fine scale hydrodynamic lice dispersal modelling to assess the effect on wild populations, which then allows adjustment of measures taken by management and industry to reduce this effect to levels within the objective of the strategy. This is now possible due to better knowledge about the relationship between intensive salmon farming activity and infection pressure (Helland *et al.*, 2012; Jansen *et al.*, 2012; Serra-Llinares *et al.*, 2014; Taranger *et al.*, 2014), and better and more accessible farming and environmental data (Jansen *et al.*, 2012; Taranger *et al.*, 2014).

A preliminary analysis indicates that under realistic conditions of lice infections, water currents, temperature and salinity, and relative lice infections may be predicted using a coupled hydrodynamic-biological lice dispersion model. With further calibrations and validation, such a system can probably be developed at least for specific areas along the Norwegian coast. As validation, calibration, and implementation of such a risk-based monitoring system is done, more of the monitoring could be based on the model and less on catch of wild salmonids. The preliminary model results are encouraging in terms of validation and calibration the model predictions against observed infection of wild salmonids. However, considerable research and development remains, where the main challenges are:

- The system for detecting problem areas based on farming data and sea lice infections of notification of problem areas need to be further developed and operationalized, and systems for risk-based and adapted surveillance on wild salmonids must be developed.
- Coupled hydrodynamic-biological lice dispersion models must be validated and calibrated against observed infection levels on wild salmonids in the field.
- Knowledge about the ecological effects of a given infection pressure on stocks of wild salmonids (population-reducing effects) must be increased so that more precise predictions can be developed.

Disease transfer from farmed salmon to wild fish

Background

Infectious diseases represent a major problem in Norwegian fish farming, despite successful development and application of vaccines against a range of pathogens. In addition to lice (considered separately above), viral diseases currently represent the largest disease problems in Norwegian aquaculture (Johansen, 2013). In the period 2005–2012, the four most frequent viral diseases [infectious pancreatic necrosis (IPN), pancreas disease (PD), heart and skeletal muscle inflammation (HSMI), and cardiomyopathy syndrome (CMS)] had 400–500 outbreaks annually (Johansen, 2013). The main reason for the dominance of viral diseases is the lack of effective vaccines. Bacterial diseases, on the other hand, cause only ~20 outbreaks annually, reflecting that the currently used bacterial vaccines provide good protection (Austin and Austin, 2007). Among the parasites, parvicapsulosis due to the myxosporean *Parvicapsula pseudobranchicola* is a problem mainly in northern Norway, whereas heavy gill infections with the microsporidian *Paranucleospora theridion* and the amoeba *Paramoeba perurans* occur mostly in the southern parts of the country. The significance of infections with the former two parasites is unclear, while amoebic gill disease (AGD) has so far been detected only during fall in 3 years (2006, 2012–2013).

For most pathogens, clear evidence for transmission from farmed to wild fish is limited (Raynard *et al.*, 2007). Most of the diseases that currently cause problems in fish farms are likely enzootic, originating from wild fish. This implies that these infections occur or occurred in the past at some "background" level in wild stocks. Such considerations complicate an estimation of the impact of aquaculture, since the "normal" prevalence range of many important disease agents is unknown. However, in two cases exotic pathogens have been introduced in association with farming activities. These have clearly affected wild Atlantic salmon populations.

The ectoparasite *Gyrodactylus salaris* (Monogenea) was first detected in Norway in 1975 (Johnsen *et al.*, 1999). There have been several introductions of *G. salaris* to Norway (Hansen *et al.*, 2003) linked to the import of salmonids from Sweden. Later, the parasite has spread (or has been spread) to many rivers (Johnsen *et al.*, 1999). By 2005, *G. salaris* had been detected in 45 rivers and 39 freshwater farms (Mørk and Hellberg, 2005). Norwegian Atlantic salmon stocks are very susceptible to *G. salaris*, and gyrodactylosis in farmed salmon may lead to 100% mortality if not treated (Bakke, 1991; Bakke *et al.*, 1992; Bakke and MacKenzie, 1993). Mortality in rivers is high, with the density of Atlantic salmon parr being reduced by 50–99% (Johnsen *et al.*, 1999).

Aeromonas salmonicida, the causative agent of furunculosis, was introduced to Norway in 1964, when furunculosis was detected in a single farm that received rainbow trout from Denmark. The disease then spread to other farms and wild fish within a limited area, being detectable there until 1979. A second introduction occurred in 1985, in connection with an import of Atlantic salmon smolts from Scotland. The disease then spread rapidly to farms and wild fish, and in 1992 a total of 550 salmon farms and 74 river systems were affected (Johnsen and Jensen, 1994). This rapid spread of the disease was likely facilitated by frequent escapement events involving infected fish (Johnsen and Jensen, 1994). Mortality due to furunculosis was registered in many rivers among escaped salmon, wild salmon, and trout. Mortality in farmed fish was high, reaching 50%, but the disease was first controlled by antibiotics and subsequently effectively with oil-based vaccines (Sommerset *et al.*, 2005; Johansen, 2013).

These two examples show the devastating effects that introductions of exotic pathogens can have. Even when disregarding agents only known from non-salmonids, there is a large number of potential pathogens (i.e. hazards) infecting salmonids elsewhere that could have significant impact on both salmon farming and wild fish populations in Norway if introduced (Raynard *et al.*, 2007; Brun and Lillehaug, 2010). Import of live fish represents the major threat to both fish farming and wild stocks, since this may lead to the introduction of exotic pathogens. However, *G. salaris* infections have not been detected in Atlantic salmon hatcheries in recent years (Hytterød *et al.*, 2014 and references therein), and the parasite does not survive in seawater. Furunculosis outbreaks in farms are rare, since most farmed salmon is protected through vaccination. Regarding disease transfer from farmed salmon to wild salmonids, these diseases are currently considered to be under control.

The detection of disease in wild fish and estimating disease impact on wild populations is difficult. Clinically affected fish usually disappear quickly in nature (e.g. predated). Epizootics with mass mortality of fish are rare, but have occurred in Norway (Bakke and Harris, 1998; Sterud *et al.*, 2007) and elsewhere (Hyatt *et al.*, 1997; Gaughan *et al.*, 2000). Such episodes are usually caused either by an exotic pathogen introduced to naïve host populations (Bakke and Harris, 1998) or by exceptional environmental

conditions such as high temperature (Sterud *et al.*, 2007). However, infection with native (enzootic) agents under normal environmental conditions can cause disease in individuals and affect an individual's survival or investment in reproduction. Hence, all pathogens may contribute to the regulation of wild populations at some level (May and Anderson, 1979; May, 1983), although the impact may vary and is often the result of a complex interaction between hosts, pathogens, environment, and predators (Dobson and Hudson, 1986; Combes, 2001).

Risk assessment

There is relatively little data available on the infection status of Norwegian wild salmonid stocks with respect to the most important pathogens that affect farmed salmon (e.g. viral agents). The available data mainly concerns returning adult salmon and some local sea trout populations screened with molecular methods (Kileng *et al.*, 2011; Garseth *et al.*, 2012, 2013a, b, c; Biering *et al.*, 2013; Madhun *et al.*, 2014a, b). Studies on the occurrence of viral infections in early life stages of salmonids are only fragmentary (e.g. Plarre *et al.*, 2005).

Due to the limited data available, the disease status (outbreak statistics) in Norwegian fish farming is used as a proxy of the infection pressure from farmed salmon to wild salmonids. Information regarding disease outbreaks on Norwegian fish farms is gathered by the Norwegian Veterinary Institute (NVI) and published annually in their Fish Health Reports (e.g. Johansen, 2013). These data record official diagnoses from NVI, as well as information from the local fish health services. This information is likely to be biased towards the more serious diseases, particularly those that are required by law to be reported to government authorities. Subclinical infections may be common and may also contribute to the spread of pathogens. However, these infections are usually not detected. Despite shortcomings, these data are the best currently available information and give a reasonably good indication of the disease status of the majority of farmed fish in Norway.

Most diseases in Norwegian salmon and rainbow trout farms are represented by only a few outbreaks, often representing geographically separate cases (Johansen, 2013). However, some diseases have a large number of outbreaks/diagnoses, and are those most likely to cause elevated infection pressures that may affect wild populations. At present the most common diseases in Norwegian salmon farming are the viral diseases PD, IPN, CMS and HSMI (Table 7; Johansen, 2013). In addition, AGD due to *Paramoeba perurans* is an emerging problem (Hjeltnes, 2014). The listed viral diseases have caused some 400 or more outbreaks each year since 2005. Outbreaks are often more frequent in certain regions and at certain times of the year, leading to a consideration also of spatial and temporal variation in the potential infection pressure (Table 8).

Table 7. Number of disease outbreaks for the most important diseases in Norwegian salmon farming (Johansen, 2013).

	2005	2006	2007	2008	2009	2010	2011	2012
PD	45	58	98	108	75	88	89	137
HSMB	83	94	162	144	139	131	162	142
IPN	208	207	165	158	223	198	154	119
CMS	71	80	68	66	62	49	74	89

We have considered the following elements of risk assessment regarding the viral agents salmonid alphavirus (SAV), infectious pancreatic necrosis virus (IPNV), piscine myocarditis virus (PMCV), and piscine reovirus (PRV); release, exposure, and consequences as follow. The release assessment is the assumed infection pressure as proxied by the outbreak statistics. The exposure assessment is a consideration of the spatial and temporal concurrence of wild salmonids with release. A consequence assessment should consider two aspects: (i) evidence for virus transmission and (ii) impact of viral infections. However, the impact is in all the considered cases are unknown, and only evidence for virus transmission can be discussed.

Pancreas disease: salmonid alphavirus

PD in Atlantic salmon and rainbow trout is caused by SAV. In Norway, there are currently two regionalized PD epidemics caused by SAV3 (south of Hustadvika, 63° N) and SAV2 (north of 63° N). Experimental studies show transmission of SAV via water, and epidemiological studies provide evidence for horizontal farm to farm spread (Nelson *et al.*, 1995; McLoughlin *et al.*, 1996; Kristoffersen *et al.*, 2009; Stene, 2013; Stene *et al.*, 2014). The virus has been shown to survive for several weeks in the environment (Graham *et al.*, 2007) and thus may be carried long distances with currents (Stene, 2013; Stene *et al.*, 2014). SAV2 may have a different outbreak pattern than SAV3, since outbreaks tend to occur later in the year (Johansen, 2013). For the southern region (SAV3), the period in which smolts migrate and adult salmon return coincides with many SAV3 outbreaks (Table 8; Johansen, 2013). In the northern region (SAV2), most outbreaks occur later in the year. This may signify that most of the smolt migration precedes peak virus spread in the SAV2 region. On the other hand, returning salmon and sea trout are likely more exposed, but screening indicates that very few wild fish are infected (Biering *et al.*, 2013). Infected escaped salmon can enter rivers in fall, possibly exposing wild fish including naïve juveniles to the virus (Madhun *et al.*, 2014a).

Screening of sea trout (Biering *et al.*, 2013; Madhun *et al.*, 2014b) indicates that sea trout in areas with high frequency of PD outbreaks are not infected with SAV. This is in accordance with injection

Table 8. Overview of the main periods where salmon and sea trout reside in coastal areas.

	Coastal area	J	F	M	A	M	J	J	A	S	O	N	D
Smolt migration	South Norway					xx	xx	x					
	Central Norway					x	xx	x					
	Northern Norway						x	xx					
Return	South Norway					x	xx	xx	x				
	Central Norway					x	xx	xx	xx	x			
	Northern Norway					x	xx	xx	xx	x			
Sea Trout (sea)	South Norway				x	xx	xx	xx	x	x	x		
	Central Norway				x	xx	xx	xx	x	x	x		
	Northern Norway				x	x	xx	xx	x	x			

Southern Norway: south Norway up to Sogn and Fjordane, Central Norway: Møre and Romsdal-Trøndelag; Northern Norway: Nordland-Finnmark. x = a few fish in coastal areas, xx = large numbers of fish in coastal areas.

experiments, which suggest that sea trout is more resistant to SAV than salmon (Boucher *et al.*, 1995). SAV infections have been detected in wild salmonids and wild flatfish (Nylund, 2007; Snow *et al.*, 2010; Biering *et al.*, 2013), but PD has not been observed in wild fish.

Altogether, there are yet no data that confirm SAV transmission from farmed salmon to wild fish, but transmission of virus to wild fish is considered likely due to the large number of outbreaks and the documented efficient horizontal transmission of SAV. The probability of transmission of SAV to wild salmon is considered to be moderate for migrating smolts in the southern PD-region due to the temporal overlap between outbreaks and migration, whereas it is considered to be low in the northern PD-region as most of the migration is finished before the major outbreak period. For returning salmon, the probability of infection is considered to be low in both PD-regions based on the available screening results. The probability of SAV transmission to wild salmon is considered to be low in areas with no or few outbreaks. The probability of infection of sea trout during the marine phase is also considered to be low.

Infectious pancreatic necrosis: infectious pancreatic necrosis virus

IPNV is a robust, long-lived birnavirus that infect many different fish species in both fresh water and seawater (e.g. Reno, 1999). The virus is enzootic in Norway. IPN cause significant losses in fish farming in most areas in Norway (Johansen, 2013). However, there are indications of a downwards trend in outbreaks and losses, which might be caused by the increased use of IPN resistant fish (Johansen, 2013). The virus is shed into the water by infected fish, and is spread to other farms by water currents (see, e.g. Mortensen, 1993; Wallace *et al.*, 2005; Raynard *et al.*, 2007; Johansen *et al.*, 2011). A higher prevalence of IPNV has been found in wild fish near salmon farms with clinical outbreaks of IPN, compared with fish at distant sites (Wallace *et al.*, 2005, 2008). Fish surviving an IPNV infection often become persistent carriers of the virus, but viral shedding from carriers has not been demonstrated (Johansen *et al.*, 2011). The prevalence of IPNV in wild fish is low (Brun, 2003; Wallace *et al.*, 2005), and farmed fish are probably a major source of virus in the marine environment. Disease outbreaks in wild salmon have not been described, but mortality in wild marine fish due to IPN has been reported elsewhere (Stephens *et al.*, 1980; Mcallister *et al.*, 1984).

Due to the large number of annual outbreaks, the demonstrated robustness and infectivity of the virus, as well as the wide range of hosts, the probability of IPNV transmission from farmed to wild fish is considered as moderate. The extent and consequences of such transmission are unknown.

Heart and skeletal muscle inflammation: piscine reovirus

HMSI affects farmed salmon along the entire coast of Norway. HSMI outbreaks mainly occur 5–9 months after sea-transfer. The causative agent is an *Orthoreovirus*, PRV (Palacios *et al.*, 2010; Løvoll *et al.*, 2012). The disease can be produced experimentally using infected tissue, infected cell culture, or by cohabitation (Kongtorp *et al.*, 2004; Martinez-Rubio *et al.*, 2012, 2013). PRV is present in high densities in salmon with HSMI, but high infection intensity can be found also in clinically healthy salmon. PRV infections have been detected in wild salmon along the entire coast of Norway and have been detected in sea trout (Biering *et al.*, 2013; Garseth *et al.*, 2013b). Analyses of PRV genotypes in wild Atlantic salmon, farmed salmon, and sea trout have suggested an extensive spread of the virus along the coast, and establishment in wild populations. This spread is probably due to extensive transportation of fish between areas over a long period (Garseth *et al.*, 2013c). However, there are no reports of HSMI in PRV infected wild salmonids (e.g. Garseth *et al.*, 2013b). In Norway sea trout are only rarely (1.4–3%) infected (Garseth *et al.*, 2012; Biering *et al.*, 2013) with PRV. PRV infections have been detected in some marine fish species, but the virus genotype is unknown (Wiik-Nielsen *et al.*, 2012). It is not known how long and in what quantities PRV is shed from infected fish nor viral survival in seawater. However, modelling suggests that the virus can be transported over longer distances than SAV (Aldrin *et al.*, 2010; Kristoffersen *et al.*, 2013). The latter findings suggest that the virus is relatively stable and may spread over large areas.

Since PRV infections are widespread in farmed salmon, may readily be transmitted, and is detectable in >10% of the wild salmon examined, it is considered likely that PRV is transmitted from farmed to wild salmon. HSMI occurrence in wild salmon and other wild fish are unknown (Garseth *et al.*, 2013b, Madhun *et al.*, 2014b).

Cardiomyopathy syndrome: Piscine myocarditis virus

CMS is a serious disease in salmon, and is caused by a Totivirus, PMCV (Løvoll *et al.*, 2010; Haugland *et al.*, 2011). CMS can be transmitted experimentally by injecting heart tissue homogenates from diseased fish, PMCV from cell culture, and by cohabitation (Haugland *et al.*, 2011). Infections are long-lasting, with a gradual development of cardiac pathology.

PMCV has been detected in farmed Atlantic salmon along the entire Norwegian coast, but is not as widespread as PRV. PMCV infection has also been detected in a few wild salmon in Norway (Garseth *et al.*, 2012), and CMS-like lesions have been observed in the hearts of wild salmon before the discovery of the virus (Poppe and Seierstad, 2003). Large-sized wild salmon represent the only known natural reservoir for PMCV. There are no studies on shedding of PMCV from diseased fish or carriers, or on virus survival in water.

Due to the large number of hosts, prolonged infections, and the documented virus spread in cohabitation experiments, we assume that the virus is present in the environment and that the infection pressure around farms harbouring the virus is elevated. Examination of wild returning salmon detected only very few (3/1350) infected with PMCV (Garseth *et al.*, 2012; Biering *et al.*, 2013). Such a low prevalence, which may represent natural rather than fish farming-related infections, suggests that PMCV transmission from farmed to wild salmon is infrequent. Therefore, the probability of infection in wild salmon due to virus released from farms is considered low. Due to a general lack of data, particularly regarding young fish, it is not possible to assess the impact of PMCV infection in wild salmon.

Limitations of the analysis

There is a scarcity of data on infections and lack of evidence for disease in wild salmonids for the four viral agents considered. A large number of outbreaks suggest extensive spread of virus, and consideration of timing also often substantiates an exposure of wild salmonids to the viral agents. A serious limitation in the risk assessment is a lack of information on infections due to these agents in wild fish, particularly in salmon smolts. These may be less susceptible than their farmed peers, but may also suffer mortality due to the infections. Such mortality would likely occur through predation (i.e. virus induced). In either case, the returning 1 or 2 sea-winter salmon could be found to be virus free, as is indeed generally the

case regarding SAV. Juvenile fish is often particularly susceptible to viral infections. Escaped virus infected farmed salmon can enter rivers in fall (e.g. Madhun *et al.*, 2014a), where juvenile salmonids may be exposed to released virus. The impact of SAV, PRV, and PMCV infections on juvenile salmonids is unknown. The abundance of escaped salmon in Norwegian rivers (see above) indeed suggests a high potential for interaction at this stage. Hence, due to lack of data, consequence assessments cannot be done for the four viral agents considered.

Here, we have focused on the four major diseases currently prevalent in Norwegian salmon farming. All are viral diseases, and a large number of outbreaks are reported annually. However, low prevalence does not necessarily mean low impact, and diseases that at present are under control might surge and become a threat to wild populations. Subclinical or apparently benign infections may also have unforeseen ecological effects in nature by affecting survival (i.e. predator avoidance) or recruitment.

We have not considered the possible impact of exotic pathogens on Norwegian salmon farming. There are a large number of potential pathogens (i.e. hazards) infecting salmonids elsewhere that could affect farmed and wild salmonids in Norway if introduced (Raynard *et al.*, 2007; Brun and Lillehaug, 2010). Such introductions may be irreversible, and difficult or impossible to contain, and the main risk factor is the movement of live fish or fertilized eggs.

Future work

To evaluate the effects of salmon and rainbow trout farming on the infection status of wild salmonids, there is a need to increase our knowledge about the complete pathogen repertoire (viruses, bacteria, fungi, and parasites) present in wild fish in areas of high- and low-intensity fish farming. Long-term monitoring of selected wild populations would allow detection of changes in the infection status in the population. Experimental challenge experiments with SAV, PRV, and PMCV must be performed on juvenile Atlantic salmon and brown trout in freshwater. This would better allow a consideration of the threat posed by escaped salmon ascending rivers during fall. A more extensive genotyping of virus from wild and farmed fish would improve our understanding of both virus spread and genetic changes in pathogens that may occur in the high host density of farming areas.

Organic load and nutrients from salmon farms

The salmonid aquaculture industry has continuously restructured since 1999, with reductions in the number of farms, increased farm size and relocation of farms to deeper fjord (50–300 m) and current rich coastal aquaculture sites. During this period, the production has doubled (Gullestad *et al.*, 2011), with typical salmon farm produces between 3000 and 5000 tons in a 18 months period in sheltered coastal waters and as much as 14 000 tons at more dynamic coastal sites. This rapid development has led to increased concerns about the environmental impacts both at present and future predicted finfish production levels.

Increased awareness of elevated discharges of nutrients, excess feed, and faeces to the marine environment has resulted in greater scrutiny of the aquaculture industry (Mente *et al.*, 2006; Taranger *et al.*, 2012a). To this end, the Ministry of Fisheries and Coastal Affairs have stated that "the environmental impact of aquaculture must be kept at an acceptable level and be within the assimilative capacity of the area" (Anon., 2009b).

Assessing the risk of organic enrichment and nutrient overloading of Norwegian finfish aquaculture at local and regional scales focus both on benthic and pelagic systems and is based on a combination of scientific knowledge and industry monitoring data, coupled with best professional judgment, and the precautionary principle.

Risk assessment

Release assessment

Intensive farming of finfish in open sea cages results in the release of organic and inorganic effluents (i.e. carbon, nitrogen, and phosphorus) in the form of waste feed, faeces, and metabolic by-products to the surrounding aquatic environments (Holmer *et al.*, 2005; Strain and Hargrave, 2005). At current production levels in Norway, salmonid farming (1.3 M tons cultivated fish in 2012) releases ~34 000 tons of N, 60 000 tons of C, and 9750 tons of P annually (Taranger *et al.*, 2013).

Expose assessment

Accumulation of these effluents into the marine system can negatively impact the ecosystem by contributing to eutrophication of pelagic systems, fertilization of benthic macrophytes in the euphotic zone, and organic enrichment of benthic systems (Strain and Hargrave, 2005). However, the area of influence (local or regional locations) and degree of enrichment of the environment depends on a number of factors including the size of the farm (i.e. the biomass of fish), the ambient environmental conditions (i.e. hydrodynamics, water depth, wave exposure, topography and substrate type) and the husbandry practices at the individual fish farms (Holmer *et al.*, 2005). In Norway, detailed knowledge about the environmental effects of organic and nutrient enrichment from finfish aquaculture is mainly based on studies around sheltered and coastal fjord aquaculture sites.

At deep aquaculture sites, fish farming effluents can be traced into the wider environment and into benthic foodwebs up to at least 1 km from the farming site (Kutti *et al.*, 2007b; Olsen *et al.*, 2012). At low deposition levels, organic enrichment of benthic sediments (up to 500 m from the farming location) stimulates secondary production in soft bottom communities, resulting in shifts in benthic faunal community structure (Kutti *et al.*, 2007a; Kutti, 2008; Bannister *et al.*, 2014). In addition, excessive loading of organic effluents to sediments often leads to dramatic changes in biogeochemical processes leading to grossly anoxic conditions (Valdemarsen *et al.*, 2012). The emissions of dissolved nutrients from finfish farms are quickly diluted in the water column at dynamic sites and elevated nutrient levels are hardly detected 200 m away from the farm (H. Jansen, IMR, unpublished data; Sanderson *et al.*, 2008).

Consequence assessment

Many studies have investigated benthic impacts of fish farming on soft sediment benthic systems, demonstrating that intensive fish farming modifies biogeochemical processes (Holmer and Kristensen, 1992; Holmer and Frederiksen, 2007; Norði *et al.*, 2011). Remineraliztion of the highly labile organic waste (i.e. fish feed and faeces) results in increased sediment oxygen demand and altered metabolic pathways, and a shift from aerobic (i.e. heterotrophic respiration) to anaerobic (i.e. sulphate reduction and methanogenesis) microbial degradation (Holmer and Kristensen, 1992; Holmer *et al.*, 2003; Valdemarsen *et al.*, 2009). Excessive organic enrichment can thus lead to highly modified sediment conditions (Valdemarsen *et al.*, 2012), impacting the structure and biomass of faunal communities (Kutti *et al.*, 2007b; Hargrave *et al.*, 2008; Valdemarsen *et al.*, 2010; Bannister *et al.*, 2014). Increased release

of dissolved nutrients from fish farming activities may stimulate phytoplankton growth and plankton blooms (Gowen and Ezzi, 1994) and may change the composition of seaweed communities in the littoral zone (Rueness and Fredriksen, 1991; Bokn *et al.*, 1992; Munda, 1996; Pihl *et al.*, 1999; Worm and Sommer, 2000; Krause-Jensen *et al.*, 2007).

To a lesser extent, there are studies that have investigate the effects of intensive fish farming to other habitats and biota including maerl beds (Hall-Spencer *et al.*, 2006; Sanz-Lazaro *et al.*, 2011; Aguado-Giménez and Ruiz-Fernández, 2012), coral reefs (Bongiorni *et al.*, 2003; Villanueva *et al.*, 2006), seaweeds and seagrass beds (Worm and Sommer, 2000; Diaz-Almela *et al.*, 2008; Holmer *et al.*, 2008), megafaunal communities (Wilding *et al.*, 2012), and pelagic and dermersal fish (Tuya *et al.*, 2006; Fernandez-Jover *et al.*, 2007, 2011; Dempster *et al.*, 2011). A consensus of these studies is that if the assimilative capacities of these environments are exceeded, then impacts on individual species, habitats, and ecosystems will be pronounced.

Risk estimation

Organic loading on a local scale

On a local scale the endpoint: *unacceptable change in faunal communities and sediment chemistry in the production zone* is estimated. The criterion of unacceptable change is determined by Norwegian authorities and all salmon farms in Norway are monitored through mandatory investigations (MOM system; Hansen *et al.*, 2001). The MOM-B investigations are performed regularly under and in the closest vicinity of the fish cages and are based on qualitatively determined indicators such as chemical parameters (pH and redox potential), sensory parameters, and presence and/or absence of macro-infauna. The performance of these indicators against predefined thresholds categorizes the farming locations into different environmental conditions (1. low-, 2. medium-, 3. high-organic loading, and 4. organic overloading). The environmental condition 4 represents an unacceptable state when production cannot continue before the farming location has recovered. Data from the monitoring of Norwegian salmon farms are obtained from the Norwegian Directorate of Fisheries, including 2761 electronically reported MOM-B investigations undertaken beneath Norwegian fish farms between 2009 and 2013. The percentage of farms in an unacceptable ecological state (4) has been stable and $<3\%$ the last few years (Figure 5). This is probably a result of better localization of farms. The risk of unacceptable change in sediment chemistry and fauna communities in the production zone is low. However, according to national set thresholds for management of the production zone, low impact on this scale does not reflect pristine conditions, but merely that the farm is managed within acceptable conditions in regard to its local impact.

Several rigorous scientific examinations of benthic impacts have been conducted near salmon farms in western Norway (Hordaland) where benthic carbon loadings, benthic fauna responses, and sediment biogeochemical processes were studied (Kutti *et al.*, 2007a, b; Kutti, 2008; Valdemarsen *et al.*, 2012; Bannister *et al.*, 2014). In addition, there are generic scientific knowledge in respect to the flow of organic waste into benthic foodwebs along the Norwegian coast (Olsen *et al.*, 2012).

Organic loading on a regional scale

The MOM-C system is an extended investigation of several sites (1–5) in the extended influence zone around farms and consists of quantitative measurements of the organic enrichment and the impact on biodiversity in infauna communities. The MOM-C investigations of fauna communities are following the Norwegian Standard (NS 9410) and farming sites are categorized into different environmental states (i.e. very good, good, moderate, poor, and very poor) according to nationally set thresholds (Molvær *et al.*, 1997). Based on the hydrographical conditions around the farm a distant point should be indentified in the most likely accumulation area beyond the production zone. The ecological condition at this distant site could be used as a proxy to estimate the risk of the endpoint; *significant change in bottom communities beyond the production zone (regional impact)*.

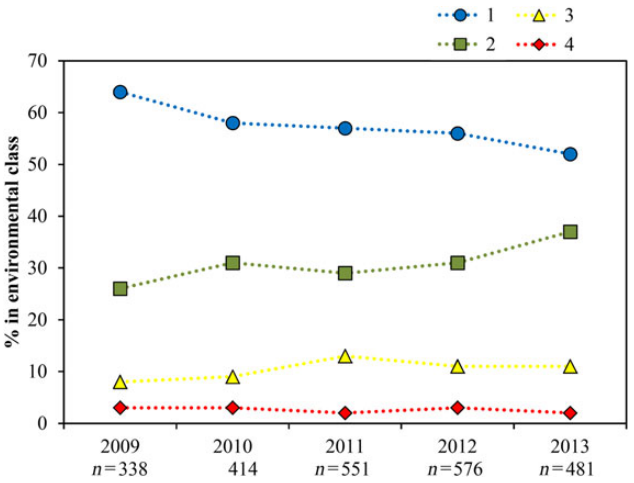

Figure 5. Impact of organic load from Norwegian marine finfish farms monitored by the mandatory MOM-B investigations (NS9410:2007) in the period 2009–2013. Data are given as percentage number of farms with ecological condition: 1 (blue), low organic loading; 2, (green), moderate organic loading; 3 (yellow), high organic loading (maximum allowed loading); 4 (red), overloading of the site, n = number of reported MOM-B investigations (data from Norwegian Directorate of Fisheries).

Data from MOM-C investigations on 122 salmon farms, which represents ~10% of the farms currently operating in Norwegian waters, are compiled from the Norwegian Directorate of Fisheries. The data show that distant sites at 95% of the farms had a high or very high ecological classification according to national set thresholds for Shannon–Wiener diversity index (H′) (Molvær *et al.*, 1997) while 5% was classified in moderate conditions.

To provide an estimate of risk for impact on a regional scale, data have been retained from case studies in the Hardangerfjord (Husa *et al.*, 2014a) and regional monitoring in some other areas in Norwegian coastal waters (Vassdal *et al.*, 2012) according to parameters and thresholds defined in the Norwegian implementation of the European Water Framework Directive (Molvær *et al.*, 1997; WFD, 2000/60/EC; Anon., 2009a). These data show that the ecological conditions in fauna communities and oxygen values in deep regional basins are high to very high in fjords with high salmon farming activity. These findings were also supported by analysis of the relative importance of the extra contribution of organic farm waste to decomposing communities in the deep basins in the Hardangerfjord, estimating that current farming production increased oxygen consumption by 10% and decreased oxygen levels in bottom water with 0.09% (Aure, 2013). However, we do not have sufficient data from the entire Norwegian coastline to make a full risk estimation of the impact of organic loading on a regional scale.

Nutrient emissions on a local scale

Local impact from nutrients and fine particulate material in the euphotic zone are currently not monitored around Norwegian fish farms, and we therefore have no data to estimate the endpoint; *nutrients from fish farms results in local eutrophication*.

Nutrient emissions on a regional scale

To estimate the endpoint, *nutrients from fish farms results in regional eutrophication*, we do not have sufficient data from Norwegian coastal waters to fulfil a complete risk estimation. However, three years monitoring of nutrient values and chlorophyll *a* in the Hardangerfjord area (Husa *et al.*, 2014b) and in Rogaland County, a sensitive area for fish farming due to lower water exchange (Vassdal *et al.*, 2012), show that ecological conditions for these parameters are within national acceptances thresholds (Molvær *et al.*, 1997) suggesting high or very high water quality.

These data coupled with modelling estimations on potential increase in phytoplankton production (Figure 6; Skogen *et al.*, 2009) suggest low risk of regional impacts from aquaculture in Norway. The potential increase in phytoplankton production is based on knowledge about the water transport mechanisms, coupled with typical natural values of nitrogen and phosphorous in the Norwegian Coastal Current and the calculated extra contribution to nutrient concentrations from fish farms in each Norwegian county. Assuming that theoretically all the nitrogen released from fish farms is assimilated in phytoplankton growth, an increase in the natural phytoplankton biomass were calculated and compared with the threshold of a 50% increase in phytoplankton biomass that is defined as eutrophication by OSPAR (Anon., 2010).

Limitations

The risk assessment in the period 2010–2013 is based on a limited dataset, restricted scientific knowledge, and national monitoring methods that require upgrading/revising. On a local scale, the use of the MOM-B monitoring dataset for assessing local impacts of organic enrichment should be used cautiously. The MOM-B system is built on a qualitative assessment limited in its efficacy outside of soft sediment habitats. Using the "MOM system" to monitor the environmental effects to other benthic habitats such as hard, mixed and sandy bottom habitats, seaweed and kelp habitats, or other sensitive habitats including sponge aggregations and cold water corals reefs will lead to uncertain monitoring results. Therefore, reported benthic conditions underneath fish farms should be used cautiously in the risk assessment approach. The number of MOM-C investigations available for this risk assessment was limited to 122 fish farming sites.

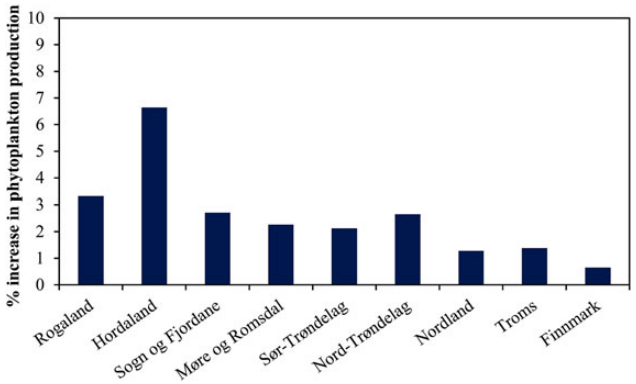

Figure 6. Estimated percentage increase in phytoplankton production due to emissions of dissolved nitrogen from finfish farms in 2012 in each Norwegian county, based on 100% exploitation of the nitrogen to carbon fixation.

Considering there are more or less 1000 fish farming locations along the Norwegian Coastline, the use of these investigations to provide an overview of the impact of organic enrichment from aquaculture within the influence area, should be approached cautiously, given they only represent ~10% of current production sites. Scientific data are restricted to fjord habitats in western Norway, there is a dearth of scientific studies representing the different geographical settings (i.e. southern, mid, and northern Norway) and the different benthic habitat types (coastal sandy sediments, hard bottom habitats, and sensitive and vulnerable habitats).

Furthermore, at regional scales national monitoring programmes have only started to begin (from 2008); therefore, existing knowledge and data of regional impacts from fish farming are mainly restricted to two counties and limited time. These limitations result in a precautionary approach used in the above risk assessment.

Future work

To achieve the goal of the Ministry of Fisheries and Coastal Affairs that the environmental impact of aquaculture must be kept at an acceptable level and be within the assimilative capacity of the area, further scientific research and coastal monitoring efforts are needed. It is crucial that further knowledge is developed on understanding the interaction of organic and nutrient waste release on different habitat types (ca. hard bottom habitats, coastal sandy sediment habitats, and benthic boreal systems). In addition, habitats of ecological significance and sensitive species (ca. coral reefs, sponge aggregations, maerl beds, seagrass meadows, and spawning areas) require detailed investigations to understand their responses to organic and nutrient loadings, thus allowing more informed decisions on ecological impacts to be made. Furthermore, given the heterogeneity of benthic substrates along the Norwegian coastline, and the existing limitations of the MOM-B monitoring standard, improved monitoring tools need to be established to enable monitoring of local impacts from fish farms on non-soft sediment substrates. MOM-C investigations should be performed more often and at a greater number of stations along a gradient from enrichment. Furthermore, sampling for therapeutants and fatty acids should be incorporated in the MOM-C investigations to detect the pressure of drugs on the environment and also to determine if the impacts detected are in fact related to fish farming activities.

Moreover, there is also a need to develop new modelling tools that can predict the dispersal of organic and nutrient wastes from fish farms, which will enable better placement of monitoring stations for both MOM-C and regional monitoring to increase the likelyhood of detecting impacts. Finally, regional monitoring programmes should incorporate greater sampling coverage (i.e. sampling locations) and frequency along the entire Norwegian coastline. Monitoring programmes should also identify possible risk areas for regional impacts and place further emphasis on monitoring in these habitats.

Discussion

The main approach in the risk assessment on the environmental impact of fish farming in Norway was to review the state-of-the-art on various hazards and potential risk factors, review national

monitoring data with relevance for the risk assessment, and score qualitatively the risk of impact in broad categories; low, moderate, and high risk of impact when monitoring data were available (Taranger *et al.*, 2011a, b, 2012b, 2013).

When the first risk assessment of the environmental impact of Norwegian fish farming was initiated in 2010 (Taranger *et al.*, 2011a), only very broad definitions and goals of aquaculture sustainability were presented in a strategy document by the Norwegian government (Anon., 2009b). The goals for sustainability put forward in this document are listed in Table 1, and covers the topics; diseases/parasites, genetic interactions with wild populations, pollution and discharges, marine site structure and zoning, as well as sustainability of feed and feed resources. These goal are very generic and do not specify sustainability indicators and related thresholds for societal/political acceptance of the environmental impact of fish farming in Norway.

The Institute of Marine Research in Norway and the Norwegian Veterinarian Institute were requested by the Norwegian Ministry of Fisheries and Coastal Affairs in 2011 to propose scientifically based sustainability indicators and related thresholds to score the severity of the potential environmental impacts of aquaculture. This included indicators for the risk of genetic introgression of escaped farmed salmon as well impact of salmon lice from fish farming on wild salmonid populations. As a result, indicators and suggested thresholds to score the environmental impact as low, moderate, or high were proposed in 2012 (Taranger *et al.*, 2012a). These recommendations were in part approved and implemented into a governmental report on Norwegian seafood policy in 2013 (Anon., 2013c), and in a newly established Quality Norm for the management and protection of wild Atlantic salmon in Norway approved by the Norwegian government in late 2013 (Anon., 2013a).

As a result, the suggested indicators for environmental impact as the associated suggested thresholds for scoring of the impacts have been adopted in the most recent version of the Risk assessment of Norwegian fish farming (Taranger *et al.*, 2014). They are the basis of the current assessment of risk of genetic introgression and impact on salmon lice on wild salmonid populations. The assessments are based on monitoring programmes on the numbers of escaped salmon in Norwegian rivers over the last 3 years, and the level of salmon lice infections on wild salmonids in Norwegian coastal waters during the last 4 years.

New scientific findings have recently emerged, such as the measured level of introgression of escaped farmed salmon into wild salmon populations in 20 Norwegian rivers (Glover *et al.*, 2013a), and new estimates on the additional marine mortality caused by salmon lice on Atlantic salmon from the rivers Dale and Vosso, in Hordaland, Norway (Skilbrei *et al.*, 2013; Vollset *et al.*, 2014). Such findings assist in developing more quantitative assessments of the impact of number of escaped salmon in rivers, and the impact of salmon lice from farming on wild salmonids, respectively.

In contrast to the situation for genetic introgression and impact of salmon lice, there are very limited monitoring data on potential transfer of other diseases and parasites from salmon farming to wild fish in Norway. Hence, the assessment on the risk of disease transfers to wild fish is mainly based on an analysis of the frequency of disease outbreaks in the ~1000 sites for salmon farming along the Norwegian coast. This is supported with a review on the knowledge of risk of disease transmission for the most relevant pathogens, and some available data on prevalence of pathogens and/or any disease outbreaks in wild Atlantic salmon and sea trout populations in Norway (Taranger *et al.*, 2014).

Regarding the local and regional impact of organic load and release of nutrient from marine salmon farming, such environmental impact indicators and associated monitoring programmes are defined and adapted by the Norwegian fish farming authorities regarding the local zone under and close to the farms, whereas the indicators and monitoring programmes for regional effects are being implemented in some counties in Norway (Vassdal *et al.*, 2012; Husa *et al.*, 2014a). The local zone under and close to the farm is monitored with a risk-based frequency using the relatively simple MOM-B method, while the more sensitive MOM-C method with detailed analysis of the species compositions in soft bottom samples near the farms is only applied occasionally (Hansen *et al.*, 2001). Both these methods have limitations, e.g. they require soft bottom, and are currently under revisions.

Data from regional monitoring has only become available in a few counties in the last years, but new programmes are starting up in several counties. The regional monitoring will to a large degree be based upon environmental indexes and environmental quality elements and related threshold for scoring of quality according to the Norwegian implementation of EUs Water Framework Directive (Anon., 2009a).

The current risk assessment of local of organic matter and nutrient release are based on the mandatory MOM-B monitoring from all farms and MOM-C analyses from a limited number of fish farms (Taranger *et al.*, 2014). The regional impact is evaluated based on available models and some investigations in the counties of Hordaland and Rogaland which both have high salmon farming activity compared with the area of available coastal water. As discussed above under the section on impact of organic matter and nutrients, the analyses have limitations, and a more extensive and improved monitoring programme is needed both on local and regional scale.

Moreover, a range of other potential risk factors, such as use of various pharmaceuticals, transfers of xenobiotics with the feed, use of copper as antifouling agent on sea cages, interactions with fisheries and other ecological impacts of sea cage farming, as well as ecological impacts of catch, transport and use of wrasses as cleaner fish against salmon lice are discussed in the risk assessment of Norwegian fish farming (Taranger *et al.*, 2014), but are not included in the current analysis.

Conclusions and summary of main findings

This represents the first risk assessment of cage-based salmonid aquaculture in Norway, which is world's largest producer of farmed Atlantic salmon. While there are several limitations in the approaches used to estimate the risks, as has been discussed in the sections above, this work has provided the Norwegian authorities with a framework upon which to evaluate the most important identified hazards against environmental goals for sustainability. The primary findings from the present risk assessment can be summarized as follows:

- Based upon the observed frequency of farmed escaped salmon on the spawning grounds of wild populations in the period 2010–2012, 21 of the 34 populations included in the risk assessment were in moderate–to-high risk of experiencing genetic changes due to introgression of farmed salmon. However, a recent study of 20 Norwegian rivers has demonstrated that there is only a moderate correlation between the observed frequency of escapees and introgression of farmed salmon (Glover *et al.*, 2013a); therefore, validation of the level of introgression in a

higher number of native populations will be required in the future.

- During the period 2010–2013, salmon lice infections mainly resulting from salmon farming were estimated at a total of 109 stations covering relevant areas of the Norwegian coastline using wild sea trout as a proxy for local infection pressure on wild salmonids. Twenty-seven of these stations indicated moderate or high likelihood of mortality for wild migrating salmon smolts. For sea trout later in the season, 67 of the stations indicated moderate or high likelihood of mortality on wild sea trout.
- The high frequency of the viral disease outbreaks for PD, IPN, heart and skeletal muscle inflammation, and CMS in Norwegian salmon farming suggests extensive release of the causal pathogens for these diseases in many areas. Migrating wild salmon and local sea trout are likely to be exposed to these pathogens. However, the extent of this exposure and consequences remains largely unknown. Screening of wild salmonids has revealed low to very low prevalence of the viruses SAV, IPNV, PMCV, and low prevalence of PRV in salmon. Furthermore, these viruses have never been documented to cause disease in wild Norwegian salmonids. Thus, a general lack of data prohibits complete risk estimation for these diseases.
- From a total of ~500 yearly investigations of local organic loading under fish farms, 2% of them displayed unacceptable conditions in the benthic sediments and faunal composition in 2013, whereas 11% classified with a high organic loading but still within the threshold. The remaining 87% of the farms had a moderate-to-high ecological conditions. The risk of eutrophication and organic over loading in the benthic communities beyond the production area of the farm is considered low based upon case studies and monitoring data from a limited area of the Norwegian coast.

Given the rapid expansion of open sea cage farming in Norway, and internationally, and the range of ecological impacts that either are demonstrated or suspected, there is an urgent need for better knowledge about such impacts, to implement improved monitoring programmes for the most important hazards, and also to improve procedures for risk assessments including useful environmental risk indicators and to facilitate processes that involves definitions on the societal acceptance levels of the various impacts.

Acknowledgements

We appreciate the excellent work of all those participating in the work with the Norwegian Risk assessment (Risikorapporten). This includes in alphabetical order Jon Albretsen, Jan Aure, Britt Bang-Jensen, Geir Dahle, Arne Ervik, Per-Gunnar Fjelldal, Bengt Finstad, Bjørn Einar Grøsvik, Pia Kupka Hansen, Tom Hansen, Mikko Heino, Peder Andreas Jansen, Daniel Jimenez, Ingrid Askeland Johnsen, Knut Jørstad, Tore Kristiansen, Anja Kristoffersen, Rosa Maria Serra Llinnares, Stein Mortensen, Rune Nilsen, Håkon Otterå, Sonal Patel, Bjørn-Steinar Sæther, Ole B. Samuelsen, Maria Q. Sanchez, Anne Sandvik, Jofrid Skardhamar, Anne Berit Skiftesvik, Ove Skilbrei, Øystein Skaala, Cecilie Skår, Lars H. Stien, Ingebrigt Uglem Terje van der Meeren, and Vidar Wennevik.

References

Aguado-Giménez F., and Ruiz-Fernández J. M. 2012. Influence of an experimental fish farm on the spatio-temporal dynamic of a Mediterranean maërl algae community. Marine Environmental Research, 74: 47–55.

Aldrin M., Storvik B., Frigessi A., Viljugrein H., and Jansen P. A. 2010. A stochastic model for the assessment of the transmission pathways of heart and skeleton muscle inflammation, pancreas disease and infectious salmon anaemia in marine fish farms in Norway. Preventive Veterinary Medicine, 93: 51–61.

Anon. 2006. Report of the Study Group on Risk Assessment and Management Advice (SGRAMA). ICES Resource Management Committee, ICES CM 2006/RMC: 04, Ref LRC ACFM, ACE ACME 71. ICES, Copenhagen.

Anon. 2009a. Classification of environmental condition of water. Directorate group for implementation of the water framework (in Norwegian). Veileder 01:2009. 180 pp.

Anon. 2009b. Strategy for an Environmentally Sustainable Norwegian Aquaculture Industry (in Norwegian). Ministry of Fisheries and Coastal Affairs, Oslo, Norway. 34 pp.

Anon. 2010. Report of the Working Group on Environmental Interactions of Mariculture (WGEIM). ICES CM 2010SSGHIE:08. 58 pp.

Anon. 2011. Status for the Norwegian salmon stocks in 2011 (in Norwegian). Report from Vitenskapelig råd for lakseforvaltning, nr. 3. 285 pp.

Anon. 2012. Salmon lice and effects on wild salmonids- from individual response to effects on stocks (in Norwegian). Thematic Report from Vitenskapelig råd for lakseforvaltning no. 3. 56 pp.

Anon. 2013a. Quality norm from wild populations of Atlantic salmon (*Salmo salar*) (in Norwegian). Ministry of Climate and Environment, Oslo, Norway. 26 pp.

Anon. 2013b. Status for the Norwegian salmon stocks in 2013 (in Norwegian). Report from Vitenskapelig råd for lakseforvaltning no 5. 136 pp.

Anon. 2013c. The worlds leading seafood nation (in Norwegian). White papers no. 22. Ministry of Fisheries and Coastal Affairs, Oslo, Norway. 139 pp.

Araki H., Berejikian B. A., Ford M. J., and Blouin M. S. 2008. Fitness of hatchery-reared salmonids in the wild. Evolutionary Applications, 1: 342–355.

Araki H., and Schmid C. 2010. Is hatchery stocking a help or harm? Evidence, limitations and future directions in ecological and genetic surveys. Aquaculture, 308: S2–S11.

Asplin L., Boxaspen K. K., and Sandvik A. D. 2011. Modeling the distribution and abundance of planktonic larval stages of *Lepeophtheirus salmonis* in Norway. *In* Salmon lice: an integrated approach to understanding parasite abundance and distribution. Ed. by S. Jones , and R. Beamish . Wiley-Blackwell, Oxford, UK.

Aure J. 2013. Farming and oxygen in the Hardangerfjord (in Norwegian). Havforskningsrapporten, Fisken og Havet Særnummer. pp. 42–44.

Austin B., and Austin D. A. 2007. Bacterial fish pathogens. Diseases of farmed and wild fish. Praxis Publishing Ltd., Chichester, UK.

Bakke T. A. 1991. A review of the inter- and intraspecific variability in salmonid hosts to laboratory infections with *Gyrodactylus salaris* Malmberg. Aquaculture, 98: 303–310.

Bakke T. A., and Harris P. D. 1998. Diseases and parasites in wild Atlantic salmon (*Salmo salar*) populations. Canadian Journal of Fisheries and Aquatic Sciences, 55: 247–266.

Bakke T. A., Harris P. D., Jansen P. A., and Hansen L. P. 1992. Host specificity and dispersal strategy in gyrodactylid monogeneans, with particular reference to *Gyrodactylus salaris* (Platyhelminthes, Monogenea). Diseases of Aquatic Organisms, 13: 63–74.

Bakke T. A., and MacKenzie K. 1993. Comparative susceptibility of native Scottish and Norwegian stocks of Atlantic salmon, *Salmo salar* L., to *Gyrodactylus salaris* Malmberg: Laboratory experiments. Fisheries Research, 17: 69–85.

Bannister R. J., Valdemarsen T., Hansen P. K., Holmer M., and Ervik A. 2014. Changes in benthic sediment conditions under an Atlantic salmon farm at a deep, well-flushed coastal site. Aquaculture Environment Interactions, 5: 29–47.

Berglund Andreassen K. 2013. Effects of infections with salmon lice (*Lepeophtheirus salmonis*) on wild smolts of salmon (*Salmo salar* L.) and trout (*Salmo trutta* L.) (in Norwegian). Master thesis, University of Tromsø, Norway. 61 pp.

Besnier F., Glover K. A., and Skaala Ø. 2011. Investigating genetic change in wild populations: modelling gene flow from farm escapees. Aquaculture Environment Interactions, 2: 75–86.

Biering E., Madhun A. S., Isachsen C. H., Omdal L. M., Einen A. C. B., Garseth A. H., Bjørn P. A., *et al.* 2013. Annual Report on health monitoring of wild anadromous salmonids in Norway. Report from the Norwegian Veterinary Institute and the Institute of Marine Research, Bergen, Norway.

Bjørn P. A., and Finstad B. 1997. The physiological effects of salmon lice infection on sea trout postsmolts. Nordic Journal of Freshwater Research, 73: 60–72.

Bjørn P. A., Finstad B., Asplin L., Skilbrei O., Nilsen R., Serra Llinares R. M., and Boxaspen K. K. 2011a. Method development for surveillance and counting of salmon lice on wild salmonids. Rapport fra Havforskningen 8-2011. 58 pp.

Bjørn P. A., Finstad B., Kristoffersen R., McKinley R. S., and Rikardsen A. H. 2007a. Differences in risks and consequences of salmon louse, *Lepeophtheirus salmonis* (Krøyer), infestation on sympatric populations of Atlantic salmon, brown trout, and Arctic charr within northern fjords. ICES Journal of Marine Science, 64: 386–393.

Bjørn P. A., Finstad B., Nilsen R., Asplin L., Uglem I., Skaala Ø., and Boxaspen K. K., *et al.* 2007b. Norwegian national surveillance of salmon lice epidemics on wild Artlantic salmon, sea trout and Arctic charr in connection with Norwegian national salmon rivers and fjords. NINA report 377. 33 pp.

Bjørn P. A., Finstad B., Nilsen R., Uglem I., Asplin L., Skaala Ø., and Hvidsten N. A. 2010. Norwegian national surveillance 2009 of salmon lice epidemics on wild Atlantic salmon, sea trout and Arctic charr in connection with Norwegian national salmon rivers and fjords . NINA Report 547. 50 pp.

Bjørn P. A., Nilsen R., Serra Llinares R. M., Asplin L., Askeland Johnsen I., Karlsen Ø., Finstad B., *et al.* 2013. Salmon lice infections on wild salmonids along the Norwegian coast in 2013. Final report to Norwegian Food Safety Authority. Report from Havforskningsinstituttet, no. 32–2013.

Bjørn P. A., Nilsen R., Serra Llinares R. M., Asplin L., Boxaspen K. K., Finstad B., Uglem I., *et al.* 2012. Salmon lice infections on wild salmonids along the Norwegian coast in 2012. Final report to Norwegian Food Safety Authority. Report from Havforskningen, 31–2012.

Bjørn P. A., Nilsen R., Serra Llinares R. M., Asplin L., Boxaspen K. K., Finstad B., Uglem I., *et al.* 2011b. Final report to the Norwegian Food Safety Authority of the salmon lice infections along the Norwegian coast in 2011. Report From Havforskningen, 19-2011: 34.

Bjørn P. A., Sivertsgård R., Finstad B., Nilsen R., Serra-Llinares R. M., and Kristoffersen R. 2011c. Area protection may reduce salmon louse infection risk to wild salmonids. Aquaculture Environment Interactions, 1: 233–244.

Bokn T., Murray S. N., Moy F. E., and Magnusson J. B. 1992. Changes in fucoid distribution and abundances in the inner Oslofjord, Norway: 1974–80 versus 1988–90. Acta Phytogeographica Suecia, 78: 117–124.

Bongiorni L., Shafir S., and Rinkevich B. 2003. Effects of particulate matter released by a fish farm (Eilat, Red Sea) on survival and growth of *Stylophora pistillata* coral nubbins. Marine Pollution Bulletin, 46: 1120–1124.

Boucher P., Raynard R. S., Houghton G., and Laurencin F. B. 1995. Comparative experimental transmission of pancreas disease in Atlantic salmon, rainbow trout and brown trout. Diseases of Aquatic Organisms, 22: 19–24.

Bourret V., O'Reilly P. T., Carr J. W., Berg P. R., and Bernatchez L. 2011. Temporal change in genetic integrity suggests loss of local adaptation in a wild Atlantic salmon (*Salmo salar*) population following introgression by farmed escapees. Heredity, 106: 500–510.

Brun E. 2003. Chapter 4: Epidemiology. *In* IPN in salmonids – a review, pp. 51–67. Ed. by B. Skjelstad , E. Brun , and I. Jensen . VESO, Oslo, Norway.

Brun E., and Lillehaug A. 2010. Risk profile of the diseases in Norwegian aquaculture. Report 9–2010 from National Veterinary Institute, Oslo, Norway.

Buschmann A. H., Riquelme V. A., Hernández-González M. C., Varela D., Jiménez J. E., Henríquez L. A., Vergara P. A., *et al.* 2006. A review of the impacts of salmonid farming on marine coastal ecosystems in the southeast Pacific. ICES Journal of Marine Science, 63: 1338–1345.

Clifford S. L., McGinnity P., and Ferguson A. 1998a. Genetic changes in an Atlantic salmon population resulting from escaped juvenile farm salmon. Journal of Fish Biology, 52: 118–127.

Clifford S. L., McGinnity P., and Ferguson A. 1998b. Genetic changes in Atlantic salmon (*Salmo salar*) populations of northwest Irish rivers resulting from escapes of adult farm salmon. Canadian Journal of Fisheries and Aquatic Sciences, 55: 358–363.

Combes C. 2001. Parasitism: The ecology and evolution of intimate interactions. University of Chicago Press, Chicago, USA.

Crozier W. W. 1993. Evidence of genetic interaction between escaped farmed salmon and wild Atlantic salmon (*Salmo salar* L.) in a Northern Irish river. Aquaculture, 113: 19–29.

Crozier W. W. 2000. Escaped farmed salmon, *Salmo salar* L., in the Glenarm River, Northern Ireland: genetic status of the wild population 7 years on. Fisheries Management and Ecology, 7: 437–446.

Dempster T., Sanchez-Jerez P., Fernandez-Jover D., Bayle-Sempere J., Nilsen R., Bjørn P. A., and Uglem I. 2011. Proxy measures of fitness suggest coastal fish farms can act as population sources and not ecological traps for wild gadoid fish. PLoS ONE, 6: e15646, 9 pp.

Diaz-Almela E., Marba N., Alvarez E., Santiago R., Holmer M., Grau A., Mirto S., *et al.* 2008. Benthic input rates predict seagrass (*Posidonia oceanica*) fish farm-induced decline. Marine Pollution Bulletin, 56: 1332–1342.

Dillane E., McGinnity P., Coughlan J. P., Cross M. C., de Eyto E., Kenchington E., and Prodohl P., *et al.* 2008. Demographics and landscape features determine intrariver population structure in Atlantic salmon (*Salmo salar* L.): the case of the River Moy in Ireland. Molecular Ecology, 17: 4786–4800.

Diserud O. H., Fiske P., and Hindar K. 2010. Regional impact of escaped farm salmon on wild salmon populations in Norway. NINA Report 622. 40 pp.

Dobson A. P., and Hudson P. J. 1986. Parasites, disease and the structure of ecological communities. Trends in Ecology & Evolution, 1: 11–15.

EFSA. 2012. EFSA Panel on Animal Health and Welfare: guidance on risk assessment for animal welfare. EFSA Journal, 10: 2513, 30 pp. doi:10.2903/j.efsa.2012.2513.

Einum S., and Fleming I. A. 1997. Genetic divergence and interactions in the wild among native, farmed and hybrid Atlantic salmon. Journal of Fish Biology, 50: 634–651.

Fernandez-Jover D., Jimenez J. A. L., Sanchez-Jerez P., Bayle-Sempere J., Casalduero F. G., Lopez F. J. M., and Dempster T. 2007. Changes in body condition and fatty acid composition of wild Mediterranean horse mackerel (*Trachurus mediterraneus*, Steindachner, 1868) associated to sea cage fish farms. Marine Environmental Research, 63: 1–18.

Fernandez-Jover D., Martinez-Rubio L., Sanchez-Jerez P., Bayle-Sempere J. T., Jimenez J. A. L., Lopez F. J. M., Bjørn P. A., *et al.* 2011. Waste feed from coastal fish farms: a trophic subsidy with compositional side-effects for wild gadoids. Estuarine Coastal and Shelf Science, 91: 559–568.

Finstad B., and Bjørn P. A. 2011. Present status and implications of salmon lice on wild salmonids in Norwegian coastal zones. *In* Salmon lice: an integrated approach to understanding parasite

abundance and distribution, pp. 281–305. Ed. by S. Jones , and R. Beamish . Wiley-Blackwell, Oxford, UK.

Finstad B., Bjørn P. A., Grimnes A., and Hvidsten N. A. 2000. Laboratory and field investigations of salmon lice [*Lepeophtheirus salmonis* (Krøyer)] infestation on Atlantic salmon (*Salmo salar* L.) post-smolts. Aquaculture Research, 31: 795–803.

Finstad B., Bjørn P. A., Todd C. D., Whoriskey F., Gargan P. G., Førde G., and Revie C. 2010. The effect of sea lice on Atlantic salmon and other salmonid species. *In* Atlantic salmon ecology, pp. 253–276. Ed. by Ø. Aas , S. Einum , A. Klemetsen , and J. Skurdal . Wiley-Blackwell, Oxford, UK.

Finstad B., and Jonsson N. 2001. Factors influencing the yield of smolt releases in Norway. Nordic Journal of Freshwater Research, 75: 37–55.

Fiske P. 2013. Surveillance of escaped farmed salmon in rivers in the autumn 2010–2012. NINA Report 989. 33 pp.

Fiske P., Lund R. A., and Hansen L. P. 2006. Relationships between the frequency of farmed Atlantic salmon, *Salmo salar* L., in wild salmon populations and fish farming activity in Norway, 1989–2004. ICES Journal of Marine Science, 63: 1182–1189.

Fleming I. A., Hindar K., Mjølnerød I. B., Jonsson B., Balstad T., and Lamberg A. 2000. Lifetime success and interactions of farm salmon invading a native population. Proceedings of the Royal Society of London Series B Biological Sciences, 267: 1517–1523.

Fleming I. A., Jonsson B., Gross M. R., and Lamberg A. 1996. An experimental study of the reproductive behaviour and success of farmed and wild Atlantic salmon (*Salmo salar*). Journal of Applied Ecology, 33: 893–905.

Fraser D. J., Weir L. K., Bernatchez L., Hansen M. M., and Taylor E. B. 2011. Extent and scale of local adaptation in salmonid fishes: review and meta-analysis. Heredity, 106: 404–420.

Garcia de Leaniz C., Fleming I. A., Einum S., Verspoor E., Jordan W. C., Consuegra S., Aubin-Horth N., *et al.* 2007. A critical review of adaptive genetic variation in Atlantic salmon: implications for conservation. Biological Reviews, 82: 173–211.

Gargan P. G., Forde G., Hazon N., Russell D. J. F., and Todd C. D. 2012. Evidence for sea lice-induced marine mortality of Atlantic salmon (*Salmo salar*) in western Ireland from experimental releases of ranched smolts treated with emamectin benzoate. Canadian Journal of Fisheries and Aquatic Sciences, 69: 343–353.

Garseth A. H., Biering E., and Aunsmo A. 2013a. Associations between piscine reovirus infection history traits in wild-caught Atlantic salmon *Salmo salar* L. in and life Norway. Preventive Veterinary Medicine, 112: 138–146.

Garseth A. H., Biering E., and Tengs T. 2012. Piscine myocarditis virus (PMCV) in wild Atlantic salmon *Salmo salar*. Diseases of Aquatic Organisms, 102: 157–161.

Garseth Å. H., Ekrem T., and Biering E. 2013c. Phylogenetic evidence of long distance dispersal and transmission of piscine reovirus (PRV) between farmed and wild Atlantic salmon. PLoS ONE, 8: e82202.

Garseth A. H., Fritsvold C., Opheim M., Skjerve E., and Biering E. 2013b. Piscine reovirus (PRV) in wild Atlantic salmon, *Salmo salar* L., and sea-trout, *Salmo trutta* L., in Norway. Journal of Fish Diseases, 36: 483–493.

Gaughan D. J., Mitchell R. W., and Blight S. J. 2000. Impact of mortality, possibly due to herpesvirus, on pilchard *Sardinops sagax* stocks along the south coast of Western Australia in 1998–99. Marine and Freshwater Research, 51: 601–612.

GESAMP. 2008. Assessment and communication of environmental risks in coastal aquaculture. IMO/FAO/UNESCO-IOC/UNIDO/WMO/IAEA/UN/UNEP Joint Group of Experts on Scientific Aspects of Marine Environmental Protection. Reports and Studies GESAMP No. 76. FAO, Rome. 198 pp.

Gjedrem T. 2010. The first family-based breeding program in aquaculture. Reviews in Aquaculture, 2: 2–15.

Glover K. A. 2010. Forensic identification of fish farm escapees: the Norwegian experience. Aquaculture Environment Interactions, 1: 1–10.

Glover K. A., Ottera H., Olsen R. E., Slinde E., Taranger G. L., and Skaala Ø. 2009. A comparison of farmed, wild and hybrid Atlantic salmon (*Salmo salar* L.) reared under farming conditions. Aquaculture, 286: 203–210.

Glover K. A., Pertoldi C., Besnier F., Wennevik V., Kent M., and Skaala Ø. 2013a. Atlantic salmon populations invaded by farmed escapees: quantifying genetic introgression with a Bayesian approach and SNPs. BMC Genetics, 14: 4.

Glover K. A., Quintela M., Wennevik V., Besnier F., Sørvik A. G. E., and Skaala Ø. 2012. Three decades of farmed escapees in the wild: A spatio-temporal analysis of population genetic structure throughout Norway. PLoS ONE, 7: e43129.

Glover K. A., Skilbrei O., and Skaala Ø. 2008. Genetic assignment identifies farm of origin for Atlantic salmon *Salmo salar* escapees in a Norwegian fjord. ICES Journal of Marine Science, 65: 912–920.

Glover K. A., Sørvik A. G. E., Karlsbakk E., Zhang Z., and Skaala Ø. 2013b. Molecular genetic analysis of stomach contents reveals wild Atlantic cod feeding on piscine reovirus (PRV) infected Atlantic salmon originating from a commercial fish farm. PLoS ONE, 8: e60924.

Gowen R. J., and Ezzi I. A. 1994. Assessment and prediction of the potential for hypernutrification and eutrophication associated with cageculture of salmonids in Scottish waters. Dunstaffnage Marine Laboratory, Oban, Scotland. 137 pp.

Graham D. A., Staples C., Wilson C. J., Jewhurst H., Cherry K., Gordon A., and Rowley H. M. 2007. Biophysical properties of salmonid alphaviruses: influence of temperature and pH on virus survival. Journal of Fish Diseases, 30: 533–543.

Grimnes A., and Jakobsen P. J. 1996. The physiological effects of salmon lice infection on post-smolt of Atlantic salmon (*Salmo salar*). Journal of Fish Biology, 48: 1179–1194.

Gullestad P., Bjørgo S., Eithun I., Ervik A., Gudding R., Hansen H., Røsvik I. O., *et al.* 2011. Effective and sustainable use of areas for aquaculture – land of desire (in Norwegian). Report from an expert panel appointed by Ministry of Fisheries and Coastal Affairs, Oslo, Norway. 198 pp.

Hall-Spencer J., White N., Gillespie E., Gillham K., and Foggo A. 2006. Impact of fish farms on maerl beds in strongly tidal areas. Marine Ecology Progress Series, 326: 1–9.

Hansen H., Bachmann L., and Bakke T. A. 2003. Mitochondrial DNA variation of *Gyrodactylus* spp. (Monogenea, Gyrodactylidae) populations infecting Atlantic salmon, grayling, and rainbow trout in Norway and Sweden. International Journal for Parasitology, 33: 1471–1478.

Hansen L. P. 2006. Migration and survival of farmed Atlantic salmon (*Salmo salar* L.) released from two Norwegian fish farms. ICES Journal of Marine Science, 63: 1211–1217.

Hansen P. K., Ervik A., Schanning M. T., Johannsen P., Aure J., Jahnsen T., and Stigebrandt A. 2001. Regulating the local environmental impact of intensive marine fish farming. II. The monitoring programme of the MOM system (Modelling – Ongrowing fish farms – Monitoring). Aquaculture, 194: 75–92.

Hargrave B. T., Holmer M., and Newcombe C. P. 2008. Towards a classification of organic enrichment in marine sediments based on biogeochemical indicators. Marine Pollution Bulletin, 56: 810–824.

Haugland O., Mikalsen A. B., Nilsen P., Lindmo K., Thu B. J., Eliassen T. M., Roos N., *et al.* 2011. Cardiomyopathy syndrome of Atlantic salmon (*Salmo salar* L.) is caused by a double-stranded RNA virus of the totiviridae family. Journal of Virology, 85: 5275–5286.

Hazon N., Todd C. D., Whelan B., Gargan P. G., Finstad B., Bjørn P. A., and Wendelar Bonga S. E., *et al.* 2006. Sustainable management of interactions between aquaculture and wild salmonid fish. Final report for the SUMBAWS EU project. pp. 1–293.

Helland I. P., Finstad B., Uglem I., Diserud O. H., Foldvik A., Hanssen F., Bjørn P. A., *et al.* 2012. What determines salmon lice infections on wild salmonids? Statistical calculations of data from the national salmon lice surveillance program 2004–2010 (in Norwegian). NINA Rapport, 891. 51 pp.

Heuch P. A., Bjørn P. A., Finstad B., Holst J. C., Asplin L., and Nilsen F. 2005. A review of the Norwegian "National Action Plan Against Salmon Lice on Salmonids": The effect on wild salmonids. Aquaculture, 246: 79–92.

Hindar K., Fleming I. A., McGinnity P., and Diserud A. 2006. Genetic and ecological effects of salmon farming on wild salmon: modelling from experimental results. ICES Journal of Marine Science, 63: 1234–1247.

Hjeltnes B. (Ed.) 2014. Report on fish health—2013 (in Norwegian). Veterinærinstituttet, Oslo, Norway. 44 pp.

Holm M., Holst J. C., and Hansen L. P. 2000. Spatial and temporal distribution of post-smolts of Atlantic salmon (*Salmo salar* L.) in the Norwegian Sea and adjacent areas. ICES Journal of Marine Science, 57: 955–964.

Holmer M., Argyrou M., Dalsgaard T., Danovaro R., Diaz-Almela E., Carlos M. D. E., Frederiksen M., *et al.* 2008. Effects of fish farm waste on *Posidonia oceanica* meadows: Synthesis and provision of monitoring and management tools. Marine Pollution Bulletin, 56: 1618–1629.

Holmer M., Duarte C. M., Heilskov A., Olesen B., and Terrados J. 2003. Biogeochemical conditions in sediments enriched by organic matter from net-pen fish farms in the Bolinao area, Philippines. Marine Pollution Bulletin, 46: 1470–1479.

Holmer M., and Frederiksen M. S. 2007. Stimulation of sulfate reduction rates in Mediterranean fish farm sediments inhabited by the seagrass *Posidonia oceanica*. Biogeochemistry, 85: 169–184.

Holmer M., and Kristensen E. 1992. Impact of marine fish cage farming on metabolism and sulfate reduction of underlying sediments. Marine Ecology Progress Series, 80: 191–201.

Holmer M., Wildish D., and Hargrave B. 2005. Environmental effects of marine finfish aquaculture. *In* Handbook of environmental chemistry, pp. 181–206. Ed. by B. Hargrave. Springer-Verlag, Berlin.

Holst J. C., Jakobsen P. J., Nilsen F., Holm M., Asplin L., and Aure J. 2007. Mortality of seaward-migrating post-smolts of Atlantic salmon due to salmon lice infection in Norwegian salmon stocks. *In* Salmon at the edge, pp. 136–137. Blackwell Science Ltd, Bodmin, Cornwall.

Husa V., Kutti T., Ervik A., Sjøtun K., Hansen P. K., and Aure J. 2014a. Regional impact from fin-fish farming in an intensive production area (Hardangerfjord, Norway). Marine Biology Research, 10: 241–252.

Husa V., Steen H., and Sjøtun K. 2014b. Historical changes in macroalgal communities in Hardangerfjord (Norway). Marine Biology Research, 10: 226–240.

Hvidsten N. A., Finstad B., Kroglund F., Johnsen B. O., Strand R., Arnekleiv J. V., and Bjørn P. A. 2007. Does increased abundance of sea lice influence survival of wild Atlantic salmon post-smolt? Journal of Fish Biology, 71: 1639–1648.

Hyatt A. D., Hine P. M., Jones J. B., Whittington R. J., Kearns C., Wise T. G., and Crane M. S., *et al.* 1997. Epizootic mortality in the pilchard *Sardinops sagax neopilchardu*s in Australia and New Zealand in 1995. II. Identification of a herpesvirus within the gill epithelium. Diseases of Aquatic Organisms, 28: 17–29.

Hytterød S., Lie Linaker M., Hansen H., Mo T. A., and Tavornpanich S. 2014. The surveillance programme for *Gyrodactylus salaris* in Atlantic salmon and rainbow trout in Norway. Surveillance programmes for terrestrial and aquatic animals in Norway. Annual report 2013. Norwegian Veterinary Institute, Oslo. 6 pp.

Jackson D., Cotter D., Newell J., McEvoy S., O'Donohoe P., Kane F., McDermott T., *et al.* 2013. Impact of *Lepeophtheirus salmonis* infestations on migrating Atlantic salmon, *Salmo salar* L., smolts at eight locations in Ireland with an analysis of lice-induced marine mortality. Journal of Fish Diseases, 36: 273–281.

Jackson D., Cotter D., ÓMaoiléidigh N., O'Donohoe P., White J., Kane F., Kelly S., *et al.* 2011. An evaluation of the impact of early infestation with the salmon louse *Lepeophtheirus salmonis* on the subsequent survival of outwardly migrating Atlantic salmon, *Salmo salar* L., smolts. Aquaculture, 320: 159–163.

Jansen P. A., Kristoffersen A. B., Viljugrein H., Jimenez D., Aldrin M., and Stien A. 2012. Sea lice as a density-dependent constraint to salmonid farming. Proceedings of the Royal Society B: Biological Sciences, 279: 2330–2338.

Johansen L. H., Jensen I., Mikkelsen H., Bjørn P. A., Jansen P. A., and Bergh Ø. 2011. Disease interaction and pathogens exchange between wild and farmed fish populations with special reference to Norway. Aquaculture, 315: 167–186.

Johansen R. (Ed.) 2013. Report on fish health—2012 (in Norwegian). Veterinærinstituttet, Oslo, Norway. 44 pp.

Johnsen B. O., and Jensen A. J. 1994. The spread of furunculosis in salmonids in Norwegian rivers. Journal of Fish Biology, 45: 47–55.

Johnsen B. O., Møkkelgjerd P. I., and Jensen A. J. 1999. The parasite *Gyrodactylus salaris* on salmon in Norwegian rivers, status report at start of the year 2000 (in Norwegian). NINA Oppdragsmelding, pp. 1–129.

Jones S., and Beamish R. 2011. Salmon lice: an integrated approach to understanding parasite abundance and distribution. Wiley-Blackwell, Oxford, UK.

Jonsson B., and Jonsson N. 2006. Cultured Atlantic salmon in nature: a review of their ecology and interaction with wild fish. ICES Journal of Marine Science, 63: 1162–1181.

Karlsson S., Moen T., Lien S., Glover K. A., and Hindar K. 2011. Generic genetic differences between farmed and wild Atlantic salmon identified from a 7 K SNP-chip. Molecular Ecology Resources, 11: 247–253.

Kileng Ø., Johansen L-H., and Jensen I. 2011. ILA-virus in wild and farmed fish in Troms (in Norwegian). Nofima report 28/2011, Tromsø, Norway.

Kongtorp R. T., Kjerstad A., Taksdal T., Guttvik A., and Falk K. 2004. Heart and skeletal muscle inflammation in Atlantic salmon, *Salmo salar* L.: a new infectious disease. Journal of Fish Diseases, 27: 351–358.

Krause-Jensen D., Middleboe A. L., Carstensen J., and Dahl K. 2007. Spatial patterns of macroalgal abundance in relation to eutrophication. Marine Biology, 152: 25–36.

Kristoffersen A. B., Bang Jensen B., and Jansen P. A. 2013. Risk mapping of heart and skeletal muscle inflammation in salmon farming. Preventive Veterinary Medicine, 109: 136–143.

Kristoffersen A. B., Viljugrein H., Kongtorp R. T., Brun E., and Jansen P. A. 2009. Risk factors for pancreas disease (PD) outbreaks in farmed Atlantic salmon and rainbow trout in Norway during 2003–2007. Preventive Veterinary Medicine, 90: 127–136.

Krkošek M., Lewis M. A., and Volpe J. P. 2005. Transmission dynamics of parasitic sea lice from farm to wild salmon. Proceedings of the Royal Society B-Biological Sciences, 272: 689–696.

Krkošek M., Revie C., Gargan P. G., Finstad B., and Todd C. D. 2013a. Comment on Jackson et al. "Impact of *Lepeophtheirus salmonis* infestations on migrating Atlantic salmon, *Salmo salar* L., smolts at eight locations in Ireland with an analysis of lice-induced marine mortality". Journal of Fish Diseases, 37: 415–417.

Krkošek M., Revie C. W., Gargan P. G., Skilbrei O. T., Finstad B., and Todd C. D. 2013b. Impact of parasites on salmon recruitment in the Northeast Atlantic Ocean. Proceedings of the Royal Society B: Biological Sciences, 280: 20122359.

Kutti T. 2008. Regional impact of organic loading from a salomonid farm – dispersal, sedimentation rates and benthic fauna response. PhD thesis, University of Bergen.

Kutti T., Ervik A., and Hansen P. K. 2007a. Effects of organic effluents from a salmon farm on a fjord system. I. Vertical export and dispersal processes. Aquaculture, 262: 367–381.

Kutti T., Ervik A., and Høisæther T. 2008. Effects of organic effluents from a salmon farm on a fjord system. III. Linking deposition rates of organic matter and benthic productivity. Aquaculture, 282: 47–53.

Kutti T., Hansen P. K., Ervik A., Høisæther T., and Johannessen P. J. 2007b. Effects of organic effluents from a salmon farm on a fjord system. II. Temporal and spatial patterns in infauna community composition. Aquaculture, 262: 355–366.

Løvoll M., Alarcon M., Jensen B. B., Taksdal T., Kristoffersen A. B., and Tengs T. 2012. Quantification of piscine reovirus (PRV) at different stages of Atlantic salmon *Salmo salar* production. Diseases of Aquatic Organisms, 99: 7–12.

Løvoll M., Wiik-Nielsen J., Grove S., Wiik-Nielsen C. R., Kristoffersen A. B., Faller R., Poppe T. *et al.* 2010. A novel totivirus and piscine reovirus (PRV) in Atlantic salmon (*Salmo salar*) with cardiomyopathy syndrome (CMS). Virology Journal, 7: 309.

Madhun A. S., Biering E., Isachsen C. H., Omdal L. M., Einen A. C. B., Wennevik V., Svåsand T., *et al.* 2014a. Annual report on health monitoring of wild anadromous salmonids in Norway. Report from the Norwegian Veterinary Institute and the Institute of Marine Research, Bergen, Norway.

Madhun A. S., Karlsbakk E., Isachsen C. H., Omdal L. M., Eide Sørvik A. G., Skaala Ø., and Barlaup B. T., *et al.* 2014b. Potential disease interaction reinforced: double-virus-infected escaped farmed Atlantic salmon, *Salmo salar* L., recaptured in a nearby river. Journal of Fish Diseases: in press, DOI: 10.1111/jfd.12228.

Martinez-Rubio L., Morais S., Evensen O., Wadsworth S., Ruohonen K., Vecino J. L. G., and Bell J. G., *et al.* 2012. Functional feeds reduce heart inflammation and pathology in Atlantic salmon (*Salmo salar* L.) following experimental challenge with Atlantic salmon reovirus (ASRV). PLoS ONE, 7: e40266.

Martinez-Rubio L., Morais S., Evensen O., Wadsworth S., Vecino J. G., Ruohonen K., and Bell J. G., *et al.* 2013. Effect of functional feeds on fatty acid and eicosanoid metabolism in liver and head kidney of Atlantic salmon (*Salmo salar* L.) with experimentally induced Heart and Skeletal Muscle Inflammation. Fish & Shellfish Immunology, 34: 1533–1545.

May R. M. 1983. Parasitic infections as regulators of animal interactions. American Scientist, 71: 36–45.

May R. M., and Anderson R. M. 1979. Population biology of infectious diseases: Part II. Nature, 280: 455–461.

Mcallister P. E., Newman M. W., Sauber J. H., and Owens W. J. 1984. Isolation of infectious pancreatic necrosis virus (serotype-Ab) from diverse species of estuarine fish. Helgolander Meeresuntersuchungen, 37: 317–328.

McGinnity P., Prodohl P., Ferguson K., Hynes R., O'Maoileidigh N., Baker N., Cotter D., *et al.* 2003. Fitness reduction and potential extinction of wild populations of Atlantic salmon, *Salmo salar*, as a result of interactions with escaped farm salmon. Proceedings of the Royal Society of London Series B Biological Sciences, 270: 2443–2450.

McGinnity P., Stone C., Taggart J. B., Cooke D., Cotter D., Hynes R., McCamley C., *et al.* 1997. Genetic impact of escaped farmed Atlantic salmon (*Salmo salar* L.) on native populations: use of DNA profiling to assess freshwater performance of wild, farmed, and hybrid progeny in a natural river environment. ICES Journal of Marine Science, 54: 998–1008.

McLoughlin M. F., Nelson R. T., Rowley H. M., Cox D. I., and Grant A. N. 1996. Experimental pancreas disease in Atlantic salmon *Salmo salar* post-smolts induced by salmon pancreas disease virus (SPDV). Diseases of Aquatic Organisms, 26: 117–124.

Mente E., Pierce G. J., Santos M. B., and Neofitou C. 2006. Effect of feed and feeding in the culture of salmonids on the marine aquatic environment: a synthesis for European aquaculture. Aquaculture International, 14: 499–522.

Molvær J., Knutzen J., Magnusson J. B., Rugg B., Skei J., and Sørensen J. 1997. Classification of environmental quality in fjords and coastal waters (in Norwegian). Norwegian Environment Agency, 3. 35 pp.

Mork J. 1991. One-generation effects of farmed fish immigration on the genetic differentiation of wild Atlantic salmon in Norway. Aquaculture, 98: 267–276.

Mørk T., and Hellberg H. 2005. The surveillance and control programme for *Gyrodactylus salaris* in Atlantic salmon and rainbow trout in Norway. *In* Surveillance and control programmes for terrestrial and aquatic animals in Norway, pp. 137–139. Annual report 2004.

Mortensen S. H. 1993. Passage of infectious pancreatic necrosis virus (IPNV) through invertebrates in an aquatic food-chain. Diseases of Aquatic Organisms, 16: 41–45.

Munda I. M. 1996. The northern Adriatic Sea. *In* Marine benthic vegetation, pp. 369–402. Ed. by W. Schramm , and P. Nienhuis . Springer, Berlin, Heidelberg.

Nash C. 2007. Guidelines for ecological risk assessment of marine aquaculture. Aquaculture Europe, 32: 5–11.

Nelson R. T., Mcloughlin M. F., Rowley H. M., Platten M. A., and Mccormick J. I. 1995. Isolation of a toga-like virus from farmed Atlantic salmon *Salmo salar* with pancreas disease. Diseases of Aquatic Organisms, 22: 25–32.

Nolan D. T., Reilly P., and Wendelar Bonga S. E. 1999. Infection with low numbers of the sea louse *Lepeophtheirus salmonis* induces stress-related effects in postsmolt Atlantic salmon (*Salmo salar*). Canadian Journal of Fisheries and Aquatic Sciences, 56: 947–959.

Norði G. A., Glud R. N., Gaard E., and Simonsen K. 2011. Environmental impacts of coastal fish farming: carbon and nitrogen budgets for trout farming in Kaldbaksfjorour (Faroe Islands). Marine Ecology Progress Series, 431: 223–241.

Norris A. T., Bradley D. G., and Cunningham E. P. 1999. Microsatellite genetic variation between and within farmed and wild Atlantic salmon (*Salmo salar*) populations. Aquaculture, 180: 247–264.

Nylund A. 2007. Salmonid Alphavirus (SAV). *In* Review of disease interactions and pathogen exchange between farmed and wild finfish and shellfish in Europe, pp. 41–45. Ed. by R. Raynard , T. Wahli , I. Vatsos , and S. Mortensen . Veterinærmedisinsk Oppdragssenter AS.

Olsen S. A., Ervik A., and Grahl-Nielsen O. 2012. Tracing fish farm waste in the northern shrimp *Pandalus borealis* (Krøyer, 1838) using lipid biomarkers. Aquaculture Environment Interactions, 2: 133–144.

OSPAR. 2010. OSPAR commission 2010. Quality status report 2010. http://qsr2010.ospar.org/en/index.html.

Palacios G., Lovoll M., Tengs T., Hornig M., Hutchison S., Hui J., Kongtorp R. T., *et al.* 2010. Heart and skeletal muscle inflammation of farmed salmon is associated with infection with a novel reovirus. PLoS ONE, 5: e11487.

Parrish D. L., Behnke R. J., Gephard S. R., McCormick S. D., and Reeves G. H. 1998. Why aren't there more Atlantic salmon (*Salmo salar*)? Canadian Journal of Fisheries and Aquatic Sciences, 55: 281–287.

Pihl L., Svenson A., Moksnes P. O., and Wennehage H. 1999. Distribution of green algal mats throughout shallow soft bottoms of the Swedish Skagerrak archipelago in relation to nutrient sources and wave exposure. Journal of Sea Research, 41: 281–295.

Plarre H., Devold M., Snow M., and Nylund A. 2005. Prevalence of infectious salmon anaemia virus (ISAV) in wild salmonids in western Norway. Diseases of Aquatic Organisms, 66: 71–79.

Poppe T. T., and Seierstad S. L. 2003. First description of cardiomyopathy syndrome (CMS)-related lesions in wild Atlantic salmon *Salmo salar* in Norway. Diseases of Aquatic Organisms, 56: 87–88.

Raynard R., Wahli T., Vatsos I., and Mortensen S. 2007. DIPNET – review of disease interactions and pathogen exchange between farmed and wild finfish and shellfish in Europe. p. 452. European Commission/Veterinærmedisinsk Oppdragssenter.

Reno P. W. 1999. Infectious pancreatic necrosis virus and its prevalence. *In* Fish diseases and disorders. Vol 3: viral, bacterial and fungal infections, pp. 1–55. Ed. by P. T. K. Woo , and D. W. Bruno . CABI Publishing, Walingford, UK.

Roberge C., Einum S., Guderley H., and Bernatchez L. 2006. Rapid parallel evolutionary changes of gene transcription profiles in farmed Atlantic salmon. Molecular Ecology, 15: 9–20.

Rueness J., and Fredriksen S. 1991. An assessment of possible pollution effects on the benthic algae of the outer Oslofjord, Norway. Oebalia, 17: 223–235.

Samuel-Fitwi B., Wuertz S., Schroeder J. P., and Schulz C. 2012. Sustainability assessment tools to support aquaculture development. Journal of Cleaner Production, 32: 183–192.

Sanderson J. C., Cromey C. J., Dring M. J., and Kelly M. S. 2008. Distribution of nutrients for seaweed cultivation around salmon cages at farm sites in north–west Scotland. Aquaculture, 278: 60–68.

Sanz-Lazaro C., Belando M. D., Marin-Guirao L., Navarrete-Mier F., and Marin A. 2011. Relationship between sedimentation rates and benthic impact on Maerl beds derived from fish farming in the Mediterranean. Marine Environmental Research, 71: 22–30.

Serra-Llinares R. M., Bjørn P. A., Finstad B., Nilsen R., Harbitz A., Berg M., and Asplin L. 2014. Salmon lice infection on wild salmonids in marine protected areas: an evaluation of the Norwegian "National Salmon Fjords". Aquaculture Environment Interactions, 5: 1–16.

Skaala Ø., Glover K. A., Barlaup B. T., Svåsand T., Besnier F., Hansen M. M., and Borgstrøm R. 2012. Performance of farmed, hybrid, and wild Atlantic salmon (*Salmo salar*) families in a natural river environment. Canadian Journal of Fisheries and Aquatic Sciences, 69: 1994–2006.

Skaala Ø., Høyheim B., Glover K. A., and Dahle G. 2004. Microsatellite analysis in domesticated and wild Atlantic salmon (*Salmo salar* L.): allelic diversity and identification of individuals. Aquaculture, 240: 131–143.

Skaala Ø., Wennevik V., and Glover K. A. 2006. Evidence of temporal genetic change in wild Atlantic salmon, *Salmo salar* L., populations affected by farm escapees. ICES Journal of Marine Science, 63: 1224–1233.

Skilbrei O. T. 2010a. Adult recaptures of farmed Atlantic salmon post-smolts allowed to escape during summer. Aquaculture Environment Interactions, 1: 147–153.

Skilbrei O. T. 2010b. Reduced migratory performance of farmed Atlantic salmon post-smolts from a simulated escape during autumn. Aquaculture Environment Interactions, 1: 117–125.

Skilbrei O. T. 2013. Migratory behaviour and ocean survival of escaped out-of-season smolts of farmed Atlantic salmon *Salmo salar*. Aquaculture Environment Interactions, 3: 213–221.

Skilbrei O. T., Finstad B., Urdal K., Bakke G., Kroglund F., and Strand R. 2013. Impact of early salmon louse, *Lepeophtheirus salmonis*, infestation and differences in survival and marine growth of sea-ranched Atlantic salmon, *Salmo salar* L., smolts 1997–2009. Journal of Fish Diseases, 36: 249–260.

Skilbrei O. T., Glover K. A., Samuelsen O. B., and Lunestad B. T. 2008. A laboratory study to evaluate the use of emamectin benzoate in the control of sea lice in sea-ranched Atlantic salmon (*Salmo salar* L.). Aquaculture, 285: 2–7.

Skilbrei O. T., Heino M., and Svåsand T. 2015. Using simulated escape events to assess the annual numbers and destinies of escaped farmed Atlantic salmon of different life stages from farms sites in Norway. ICES Journal of Marine Science, 72: 670–685.

Skilbrei O. T., and Wennevik V. 2006. Survival and growth of sea-ranched Atlantic salmon, *Salmo salar* L., treated against sea lice before release. ICES Journal of Marine Science: Journal du Conseil, 63: 1317–1325.

Skogen M. D., Eknes M., Asplin L. C., and Sandvik A. D. 2009. Modelling the environmental effects of fish farming in a Norwegian fjord. Aquaculture, 298: 70–75.

Snow M., Black J., Matejusova I., McIntosh R., Baretto E., Wallace I. S., and Bruno D. W. 2010. Detection of salmonid alphavirus RNA in wild marine fish: implications for the origins of salmon pancreas disease in aquaculture. Diseases of Aquatic Organisms, 91: 177–188.

Solberg M. F., Glover K. A., Nilsen F., and Skaala Ø. 2013a. Does domestication cause changes in growth reaction norms? A study of farmed, wild and hybrid Atlantic salmon families exposed to environmental stress. PLoS ONE, 8: e54469.

Solberg M. F., Zhang Z. W., Nilsen F., and Glover K. A. 2013b. Growth reaction norms of domesticated, wild and hybrid Atlantic salmon families in response to differing social and physical environments. BMC Evolutionary Biology, 13: 234.

Sommerset I., Krossøy B., Biering E., and Frost P. 2005. Vaccines for fish in aquaculture. Expert Review of Vaccines, 4: 89–101.

Stabell O. 1984. Homing and olfaction in salmonids: a critical review with special reference to the Atlantic salmon. Biological Reviews of the Cambridge Philosophical Society, 59: 333–388.

Ståhl G. 1987. Genetic population structure of Atlantic salmon. *In* Population genetics and fishery management, pp. 121–140. Ed. by N. Ryman , and F. Utter . University of Washington Press, Seattle.

Stene A. 2013. Transmission of pancreas disease in marine salmon farming in Norway. PhD thesis, Norwegian School of Veterinary Sciences, Oslo, Norway. 117 pp.

Stene A., Viljugrein H., Yndestad H., Tavornpanich S., and Skjerve E. 2014. Transmission dynamics of pancreas disease (PD) in a Norwegian fjord: aspects of water transport, contact networks and infection pressure among salmon farms. Journal of Fish Diseases, 37: 123–134.

Stephens E. B., Newman M. W., Zachary A. L., and Hetrick F. M. 1980. A viral etiology for the annual spring epizootics of Atlantic menhaden *Brevoortia tyrannus* (Latrobe) in Chesapeake Bay. Journal of Fish Diseases, 3: 387–398.

Sterud E., Forseth T., Ugedal O., Poppe T. T., Jørgensen A., Bruheim T., and Fjeldstad H., *et al.* 2007. Severe mortality in wild Atlantic salmon *Salmo salar* due to proliferative kidney disease (PKD) caused by *Tetracapsuloides bryosalmonae* (Myxozoa). Diseases of Aquatic Organisms, 77: 191–198.

Strain P., and Hargrave B. 2005. Salmon aquaculture, nutrient fluxes and ecosystem processes in Southwestern New Brunswick. *In* Handbook of environmental chemistry, pp. 29–57. Ed. by B. Hargrave . Springer.

Taggart J. B., Verspoor E., Galvin P. T., Moran P., and Ferguson A. 1995. A minisatellite DNA marker for discriminating between European and North American Atlantic salmon (*Salmo salar*). Canadian Journal of Fisheries and Aquatic Sciences, 52: 2305–2311.

Taranger G. L., Boxaspen K. K., Madhusn A. S., and Svåsand T. (Eds) 2012b. Suggested first generation method for environmental impact indicators with respect to genetic influences from farmed salmon to wild salmon and the impact of sea lice from farmed fish on wild salmon populations (in Norwegian). Fisken og Havet 13-2012.

Taranger G. L., Svåsand T., Kvamme B. O., Kristiansen T. S., and Boxaspen K. (Eds) 2014. Risk assessment of Norwegian aquaculture 2013 (in Norwegian). Fisken og Havet, Særnummer 2-2014.

Taranger G. L., Svåsand T., Kvamme B. O., Kristiansen T. S., and Boxaspen K. K. 2012a. Risk assessment of Norwegian aquaculture [Risikovurdering norsk fiskeoppdrett] (In Norwegian). Fisken og havet, særnummer 2-2012. 131 pp.

Taranger G. L., Svåsand T., Kvamme B. O., Kristiansen T. S., and Boxaspen K. K. 2013. Risk assessment of Norwegian aquaculture 2012 (In Norwegian). Fisken og Havet, særnummer 2-2013. 164 pp.

Taranger G. L., Svåsand T., Madhun A. S., and Boxaspen K. K. 2011a. Risk assessment of environmental impact of Norwegian aquaculture 2010 [Risikovurdering miljøvirkninger av norsk fiskeoppdrett 2010] (in Norwegian). Fisken og Havet, særnr. 3-2010. 93 pp.

Taranger G. L., Svåsand T., Madhun A. S., and Boxaspen K. K. 2011b. Risk assessment – environmental impact of Norwegian aquaculture. Extracts from Risk assessment of environmental impact of Norwegian aquaculture 2010 Fisken og Havet no. 3-2011. 52 pp.

Taylor E. B. 1991. A review of local adaptation in salmonidae, with particular reference to Pacific and Atlantic salmon. Aquaculture, 98: 185–207.

Torrissen O., Jones S., Asche F., Guttormsen A., Skilbrei O. T., Nilsen F., and Horsberg T. E., *et al.* 2013. Salmon lice – impact on wild salmonids and salmon aquaculture. Journal of Fish Diseases, 36: 171–194.

Tuya F., Sanchez-Jerez P., Dempster T., Boyra A., and Haroun R. J. 2006. Changes in demersal wild fish aggregations beneath a sea-cage fish farm after the cessation of farming. Journal of Fish Biology, 69: 682–697.

Tveiten H., Bjørn P. A., Johnsen H. K., Finstad B., and McKinley R. S. 2010. Effects of the sea louse *Lepeophtheirus salmonis* on temporal changes in cortisol, sex steroids, growth and reproductive investment in Arctic charr *Salvelinus alpinus*. Journal of Fish Biology, 76: 2318–2341.

Valdemarsen T., Bannister R. J., Hansen P. K., Holmer M., and Ervik A. 2012. Biogeochemical malfunctioning in sediments beneath a deep-water fish farm. Environmental Pollution, 170: 15–25.

Valdemarsen T., Kristensen E., and Holmer M. 2009. Metabolic threshold and sulfide-buffering in diffusion controlled marine sediments impacted by continuous organic enrichment. Biogeochemistry, 95: 335–353.

Valdemarsen T., Kristensen E., and Holmer M. 2010. Sulfur, carbon, and nitrogen cycling in faunated marine sediments impacted by repeated organic enrichment. Marine Ecology Progress Series, 400: 37–53.

Vassdal T., Heggøy E., and Johansen P-O. 2012. Marine monitoring Rogaland (in Norwegian). Status report May 2012. UNI Miljø, SAM Marin. E-rapport, 26-2012.

Verspoor E., Beardmore J. A., Consuegra S., De Leaniz C. G., Hindar K., Jordan W. C., Koljonen M. L. *et al.* 2005. Population structure in the Atlantic salmon: insights from 40 years of research into genetic protein variation. Journal of Fish Biology, 67: 3–54.

Villanueva R. D., Yap H. T., and Montano M. N. E. 2006. Intensive fish farming in the Philippines is detrimental to the reef-building coral *Pocillopora damicornis*. Marine Ecology Progress Series, 316: 165–174.

Vollset K. W., Skoglund H., Barlaup B. T., Pulg U., Gabrielsen S-E., Wiers T., and Skår B., *et al.* 2014. Can the river location within a fjord explain the density of Atlantic salmon and sea trout? Marine Biology Research, 10: 268–278.

Wagner G. N., Fast M. D., and Johnson S. C. 2008. Physiology and immunology of *Lepeophtheirus salmonis* infections of salmonids. Trends in Parasitology, 24: 176–183.

Wagner G. N., McKinley R. S., Bjørn P. A., and Finstad B. 2003. Physiological impact of sea lice on swimming performance of Atlantic salmon. Journal of Fish Biology, 62: 1000–1009.

Wagner G. N., McKinley R. S., Bjørn P. A., and Finstad B. 2004. Short-term freshwater exposure benefits sea lice-infected Atlantic salmon. Journal of Fish Biology, 64: 1593–1604.

Wallace I. S., Gregory A., Murray A. G., Munro E. S., and Raynard R. S. 2008. Distribution of infectious pancreatic necrosis virus (IPNV) in wild marine fish from Scottish waters with respect to clinically infected aquaculture sites producing Atlantic salmon, *Salmo salar* L. Journal of Fish Diseases, 31: 177–186.

Wallace I. S., Murray A. G., Gregory A., and Raynard R. S. 2005. The prevalence of infectious pancreatic necrosis virus in wild marine fish from Scotland with reference to clinically infected salmon farms. Abstract in EAFP 12th international Convergence, 12.

Wells A., Grierson C. E., MacKenzie M., Russon I., Reinardy H., Middlemiss C., Bjørn P. A., *et al.* 2006. Physiological effects of simultaneous, abrupt seawater entry and sea lice (*Lepeophtheirus salmonis*) infestation of wild, sea-run brown trout (*Salmo trutta*) smolts. Canadian Journal of Fisheries and Aquatic Sciences, 64: 1360–1369.

Wells A., Grierson C. E., Marshall L., MacKenzie M., Russon I. J., Reinardy H., Sivertsgård R., *et al.* 2007. Physiological consequences of premature freshwater return; for wild sea-run brown trout (Salmo trutta) postsmolts infested with sea lice (*Lepeophtheirus salmonis*). Canadian Journal of Fisheries and Aquatic Sciences, 64: 1360–1369.

Wiik-Nielsen C. R., Lovoll M., Sandlund N., Faller R., Wiik-Nielsen J., and Jensen B. B. 2012. First detection of piscine reovirus (PRV) in marine fish species. Diseases of Aquatic Organisms, 97: 255–258.

Wilding T. A., Cromey C. J., Nickell T. D., and Hughes D. J. 2012. Salmon farm impacts on muddy-sediment megabenthic assemblages on the west coast of Scotland. Aquaculture Environment Interactions, 2: 145–156.

Worm B., and Sommer U. 2000. Rapid direct and indirect effects of a single nutrient pulse in a seaweed-epiphyte-grazer system. Marine Ecology Progress Series, 202: 283–288.

Zhang Z., Glover K. A., Wennevik V., Svåsand T., Sørvik A. G. E., Fiske P., and Karlsson S., *et al.* 2013. Genetic analysis of Atlantic salmon captured in a netting station reveals multiple escapement events from commercial fish farms. Fisheries Management and Ecology, 20: 42–51.

Handling editor: Mark Gibbs