REVIEW

Effects of salmon lice *Lepeophtheirus salmonis* on wild sea trout *Salmo trutta*—a literature review

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ABSTRACT: Salmon farming increases the abundance of salmon lice, which are ectoparasites of salmonids in the sea. Here we review the current knowledge on the effects of salmon lice on wild sea trout. Salmon lice feed on host mucus, skin and muscle, and infestation may induce osmoregulatory dysfunction, physiological stress, anaemia, reduced feeding and growth, increased susceptibility to secondary infections, reduced disease resistance and ultimately mortality of individual sea trout. Wild sea trout in farm-free areas generally show low lice levels. In farm-intensive areas, lice levels on wild sea trout are typically higher, and more variable than in farm-free areas. Lice on wild sea trout are found at elevated levels particularly within 30 km of the nearest farms but can also extend to further ranges. Salmon lice in intensively farmed areas have negatively impacted wild sea trout populations by reducing growth and increasing marine mortality. Quantification of these impacts remains a challenge, although population-level effects have been quantified in Atlantic salmon by comparing the survival of chemically protected fish with control groups, which are relevant also for sea trout. Mortality attributable to salmon lice can lead to an average of 12–29% fewer salmon spawners. Reduced growth and increased mortality will reduce the benefits of marine migration for sea trout, and may also result in selection against anadromy in areas with high lice levels. Salmon lice-induced effects on sea trout populations may also extend to altered genetic composition and reduced diversity, and possibly to the local loss of sea trout, and establishment of exclusively freshwater resident populations.

KEY WORDS: Salmon lice · *Lepeophtheirus salmonis* · Sea trout · *Salmo trutta* · Parasite · Aquaculture · Salmon farming

Introduction

The salmon louse *Lepeophtheirus salmonis* is an external parasite of salmonids in the marine environment, and occurs naturally both in the North Atlantic and North Pacific Oceans. Salmon lice found in the Atlantic and Pacific oceans are regarded as 2 different sub-species (Skern-Mauritzen et al. 2014). From fishery management and conservation perspectives, the effects of salmon lice on wild salmonid populations are potentially problematic in areas with intensive Atlantic salmon *Salmo salar* aquaculture (Finstad et al. 2011). Since farmed salmonids act as hosts, open net cage farms can increase the local production of infective salmon lice larvae in coastal areas. The first outbreaks of salmon lice infestation oc-
curred on Norwegian Atlantic salmon farms during the 1960s, soon after cage culture began (Pike & Wadsworth 1999). Similar outbreaks occurred in Scottish Atlantic salmon farms from the mid-1970s (Pike & Wadsworth 1999). In Ireland, between 1989 and 1991, heavily salmon lice-infested wild sea trout *Salmo trutta* in poor physical condition were recorded for the first time in areas with salmon farming (Whelan 1991, Tully et al. 1993b). Amongst salmonids, sea trout—the anadromous form of brown trout—are perhaps especially vulnerable to salmon lice infestation because most sea trout remain feeding and growing in coastal waters where salmon farms are situated during their marine migration. Since the late 1980s and early 1990s, some sea trout populations in western European countries including Norway, Scotland and Ireland have suffered severe stock declines. Such population declines have been linked to the development of open net cage salmon farming in coastal waters and resultant salmon lice infestation on local wild sea trout stocks (Tully & Whelan 1993, Gargan et al. 2003, 2006a,b, Butler & Walker 2006, Skaala et al. 2014b).

Our aim is to summarize and review existing knowledge on the effects of salmon lice on sea trout. Our review will provide researchers, the aquaculture industry, and fishery managers with a comprehensive and updated overview of documented knowledge on the effects of salmon lice on sea trout. This includes physiological and pathological effects on individual sea trout in laboratory studies, verification of such effects from field studies, quantification of salmon lice levels in wild populations, and specifically, the impacts of salmon lice on sea trout populations. By contrast, previous reviews have largely focused on sea lice effects on salmonids in general, with particular attention given to Atlantic salmon (Pike & Wadsworth 1999, Todd 2007, Costello 2009, Finstad & Bjørn 2011, Finstad et al. 2011, Torrissen et al. 2013). Here, we aimed to provide a complementary and comparative appraisal of the literature pertaining particularly to sea trout, but with qualifying reference to relevant studies of Atlantic salmon. In this paper, we refer to ‘smolt’ and ‘post-smolt’ in relation to brown trout, unless we have specified that the reference concerns another salmonid species.

**The biology of *Lepeophtheirus salmonis***

Salmon lice are marine parasitic copepods of the Family Caligidae. They are planktonic and free-living in the sea during the first, post-hatching, larval life-stages, before they encounter and attach externally to the surface of the host fish. The life cycle of salmon lice comprises 5 phases, namely the nauplius, copepodid, chalimus, preadult and adult phases (Johnson & Albright 1991b, Pike and Wadsworth 1999) (Fig. 1). Each phase comprises 1 or 2 life stages, and the life cycle has a total of 8 life stages. The life cycle was previously divided into 10 stages, but Hamre et al. (2013) found that there are in fact only 2 chalimus stages, and not 4 as previously reported.

The first phase of the life cycle is the free-swimming, and non-feeding, planktonic nauplius phase (2 stages). Nauplius I larvae hatch from the paired egg strings carried by the adult female and are released to the water column. Following the first moult to nauplius II, the larva then mouls to the copepodid phase (comprising a single stage) in which it remains free-swimming and non-feeding. This is the infective stage when the salmon louse must find a host fish to survive. Once the copepodid has attached to a host fish, it mouls to the chalimus phase (2 stages). The sessile chalimus remains attached to the fish by a frontal filament and feeding is restricted to the host skin around the attachment point. This phase is followed by the immature preadult phase (2 stages) and finally the adult phase (one stage). The louse becomes mobile from the first preadult moult onwards and can move over the body surface of the host fish. Preadults and adults can swim in the water column for short periods and perhaps successfully infest other fish. Attached copepodids, chalimus, preadults and adults use rasping mouthparts to feed on host mucus, skin and underlying tissue including blood (Brandal et al. 1976, Costello 2006).

The planktonic stages may last 1–2 mo (Heuch et al. 2005). In areas with strong currents, the free-swimming and infective stages may be widely dispersed from the release source (perhaps up to 100 km or more) (Asplin et al. 2011, 2014). The development rate is temperature-dependent (Wootten et al. 1982, Johnson & Albright 1991a, Stien et al. 2005), and salmon lice can develop into the infectious copepodid stage even during the colder winter months (Box-aspen 2006). Salmon lice are generally absent from sites of low salinity, but various life stages of salmon lice have different salinity tolerances, and this varies with water temperature (Johnson & Albright 1991a, Pike and Wadsworth 1999, Bricknell et al. 2006). In the laboratory at 12°C, copepodids would not develop at salinity lower than 30 (Johnson & Albright 1991a). Copepodids transferred to low salinity water, survived for less than 1 d in waters of salinity 10 or less, and between 2–8 d at salinities of 15–30. Salmon
lice are shed by the host fish within a few days or weeks of fish re-entering freshwater (McLean et al. 1990, Finstad et al. 1995).

Parasitologists conventionally apply 3 distinct terms to define the frequency and abundance of lice on wild salmonids (Bush et al. 1997). ‘Prevalence’ is defined as the proportion, or percentage, of infested hosts in a sample. ‘Abundance’ refers to the mean number of parasites per host sampled, and ‘intensity’ is the mean number of parasites per infested host.

Effects of salmon lice on individual sea trout in laboratory studies

Mechanical damage of fish skin and tissue

In laboratory studies, copepodids tend to show an attachment preference for gills and fins, and especially the dorsal fin. Attachment to the gills may be a laboratory artefact (Wagner et al. 2008). Whilst the attached copepodid typically does not cause visible tissue damage at initial attachment, the damage to host tissues caused by the (sessile) chalimus stages can be visibly obvious but is usually relatively minor, except in dorsal fin areas where damage may be severe for heavily infested fish (Bjørn & Finstad 1998, Dawson 1998, Dawson et al. 1997, 1998, Wells et al. 2006, 2007). The most severe tissue damage arises from the feeding of the mobile preadult and adult stages and may cause mortality for heavily infested fish (Bjørn & Finstad 1998, Dawson 1998, Dawson et al. 1998, Wells et al. 2006, 2007).

Osmoregulatory problems and physiological stress responses

Anadromous fishes such as sea trout experience a physiologically challenging environmental shift when migrating from freshwater to seawater. In seawater, water is lost from the fish by osmosis, whereas salts tend to be gained. The fish would gradually become dehydrated if it did not compensate, which most fishes, including sea trout, achieve by drinking seawater and actively excreting the excess salts through the gills and kidneys (Evans 1979, Marshall & Grosell 2006).

The mechanical damage of the skin, mucus surfaces and dermal tissue caused by salmon lice impairs the barrier between the fish body and seawater, and results in increased leakage of water from the fish and thereby an osmotic and ionic imbalance (Bjørn & Finstad 1997). Reduced haematocrit (volume...
percentage of red blood cells in blood) observed in infested and moribund fish (Bjørn & Finstad 1997, Wells et al. 2006) may be attributable to leakage of blood components (bleeding) due to mechanical damage of skin and tissue, possibly in combination with erythrocyte (red blood cell) shrinkage (dehydration) (Bjørn & Finstad 1997).

Salmon lice have been shown to induce primary, secondary and tertiary stress responses (Pickering 1981, Wendelaar Bonga 1997) in sea trout (Bjørn & Finstad 1998, Dawson et al. 1998, Wells et al. 2006, 2007). Salmon lice-infested sea trout typically show higher levels of plasma cortisol compared to uninfested control fish both in the early days post-exposure, and when the lice are at the attached chalimus developmental stages (Bjørn & Finstad 1997, Wells et al. 2006, 2007). Hence, either of the 2 attached chalimus stages, but particularly the mobile preadult and adult life stages, can cause a stress reaction in the fish as indicated by increased plasma cortisol levels.

Increased plasma chloride levels are indicative of osmoregulatory disturbance and have been observed by the time that the second chalimus stage has developed, with a more severe effect emanating from increasing chalimus densities (Bjørn & Finstad 1997). Hence, the second chalimus stage can cause minor osmoregulatory disturbance in heavily infested sea trout. Severe osmoregulatory problems, as indicated by highly elevated plasma chloride levels and increased plasma osmolality, have been demonstrated when the salmon lice develop to the preadult and adult stages and the lice become mobile (Bjørn & Finstad 1997, Dawson et al. 1998, Wells et al. 2006, 2007). Plasma chloride levels increased with increasing densities of preadult and adult lice, confirming that heavily infested fish were most affected, and moribund fish suffered from a complete osmoregulatory breakdown (Bjørn & Finstad 1997).

The osmoregulatory disturbance indicated by increased plasma chloride levels may be associated both with mechanical damage of the host skin and dermal tissues and with secondary stress responses on osmoregulation. Primary stress responses, such as release of catecholamines and cortisol, may cause structural changes in the gill tissues themselves. Osmoregulatory disturbance may therefore arise as a secondary response from such stress-mediated structural changes (Wendelaar Bonga 1997, Wells et al. 2007).

Given the energy demands related to stress responses, increases in metabolic rate can occur as a secondary stress response to acute and chronic stress. Thus, elevated plasma glucosis (hyperglycaemia), decrease in liver glycogen, and elevated plasma lactate have all been used as stress indicators in fish (reviewed in Wells et al. 2006, 2007). These measures can be further influenced by the metabolic status and feeding history of the host fish. When preadult and adult stages of salmon lice had developed on infested experimental fish, lice-induced elevation of plasma glucosis and plasma lactate (Wells et al. 2006, 2007), as well as depressed liver glycogen (Wells et al. 2007), were recorded.

Growth, behaviour and disease resistance

Salmon lice-infested sea trout have shown a reduced body mass and condition factor compared to control fish (Bjørn & Finstad 1997, Dawson et al. 1998), which may be due to adverse stress responses and dehydration (Pickering 1981, Bjørn & Finstad 1997, Wendelaar Bonga 1997, Wagner et al. 2008). Reduced feeding activity in salmon lice-infested fish has also been recorded, typically after the salmon lice had moulted to the preadult and adult stages (Dawson et al. 1998, Wells et al. 2006, 2007). However, in one study (Wells et al. 2006), this was noted within only 10 d of initial exposure and prior to the development of mobile salmon lice.

Salmon lice may also affect behavioural traits other than feeding. Wells et al. (2006, 2007) and Birkeland & Jakobsen (1997) noted that during the first 2–3 d of the infestation with copepodids, sea trout showed a distinct ‘flashing’ behaviour (lateral turning) or increasing leaping activity in experimental tanks. This behaviour ceased after 7 d, but was subsequently observed again when the salmon lice had reached the mobile stages. Such behaviour has also been described previously as a general response to sea lice infestation (Wootten et al. 1982).

Reduced disease resistance as a consequence of salmon lice infestations in sea trout has not been extensively studied. However, both the mechanical damage to the skin and the primary and secondary stress responses are indicative of a compromised immune system and thereby an increased risk of secondary infection. Bacterial or fungal infections of previously infested fish were recorded when fish were transferred from seawater to freshwater in the laboratory (Wells et al. 2007). Moreover, Bjørn & Finstad (1997) found a reduced lymphocyte-leukocyte ratio, indicative for reduced disease resistance.
Effects related to timing of seawater transfer and fish origin

Physiological effects, reduced feeding and skin damage caused by salmon lice have all been shown to be more severe for fish infested 2 wk after transfer from freshwater to seawater compared to those infested 6 wk after transfer (Dawson et al. 1998). This indicates that salmon lice may be more detrimental for sea trout smolts shortly after entry to seawater than when they have resided there for several weeks, possibly because physiological acclimation is itself a stressful process, and a simultaneous challenge from salmon lice infestation may constitute an additional stressor. Hatchery-reared Atlantic salmon and sea trout smolts may differ from wild fish in many traits and characteristics (Finstad & Jonsson 2001, Wells et al. 2006, 2007), and therefore experimental results from salmon lice exposure of hatchery-reared smolts may not always be representative of wild smolts. However, results from studies of salmon lice effects on wild and hatchery-reared sea trout smolts, and from those of seawater-adapted or newly transferred post-smolts, have been shown to be both comparable and similar (Bjørn & Finstad 1997, 1998, Dawson et al. 1998, Wells et al. 2006, 2007).

Mortality

Salmon lice-induced mortality of hatchery-reared (Bjørn & Finstad 1997, 1998) and wild (Wells et al. 2006, 2007) sea trout post-smolts was observed to commence within 10−20 d of exposure, by which time the salmon lice had reached the mobile preadult and adult life stages. Mortalities in these studies ranged between 25−46% for the infested fish (Bjørn & Finstad 1997, 1998, Wells et al. 2007). Wells et al. (2006) did not record final mortalities in their experiment, because they decided to euthanise the most heavily infested fish for animal welfare reasons. Salmon lice development rates are known to increase with increasing water temperatures (Wootten et al. 1982, Johnson & Albright 1991a, Stien et al. 2005), and fish mortality occurs earlier with increasing temperatures (Bjørn & Finstad 1998, Wells et al. 2006, 2007).

Critical threshold values for detrimental effects

Bjørn & Finstad (1997) showed that for hatchery-reared sea trout with an average mass of 91 g, the most heavily infested fish died as a result of infestation. The relative density of parasites found on moribund fish indicated that >1.0 lice per gram of fish body mass, or 50 preadult and adult lice per fish, may cause mortality in small (60 g) sea trout post-smolts. Given an average lice survival of 63%, a lethal relative density of approximately 1.6 chalimus per gram of fish mass, or >90 larvae for a small sea trout post-smolt (60 g), was suggested as a critical level (Bjørn & Finstad 1997, Finstad & Bjørn 2011). Furthermore, Wells et al. (2006) concluded that 12−13 preadult and adult (i.e. ‘mobile’) salmon lice per fish was a critical intensity which elicited sublethal stress responses in wild post-smolt sea trout (body mass range = 19−70 g). Hence, it has been suggested that a simple, conservative and precautionary approach to manage and protect wild sea trout populations would be to adopt a critical level of 10 mobile lice per fish for sea trout during their first year at sea (Finstad & Bjørn 2011, Finstad et al. 2011).

Recently, a classification system has been suggested for the expected salmon lice-induced mortality of first-time migrant sea trout based on existing knowledge (Taranger et al. 2015). This system predicts no additional mortality risk for sea trout with <0.1 lice per gram of fish body mass, 20% extra mortality for sea trout carrying 0.1−0.2 lice g−1, 50% for sea trout with 0.2−0.3 lice g−1 and 100% mortality for sea trout with >0.3 lice g−1. Studies on the effects of salmon lice on larger, veteran migrants and maturing sea trout are lacking, but a complementary study of Arctic char Salvelinus alpinus L. (Tveiten et al. 2010) suggested that the effects of salmon lice on maturing fish may be more severe than for first-time migrants. Based on that study, Taranger et al. (2015) assumed for veteran migrant and maturing sea trout no additional mortality risk for sea trout with <0.025 lice g−1 body mass, 20% extra mortality for sea trout with 0.025−0.05 lice g−1, 50% for sea trout with 0.05−0.10 lice g−1, 75% for sea trout with 0.10−0.15 lice g−1 and 100% mortality for sea trout with >0.15 lice g−1.

The foregoing threshold level predictions are based on effects in relatively short-term laboratory experiments. Values should therefore perhaps be considered indicative, and not absolute, and require further verification and validation, especially if the objective is to determine critical parasite burdens to guide conservation and management criteria. For example, density dependent mortality of salmon lice developing on a fish may affect estimates of threshold values, and the assumption of a simple linear relationship between lice numbers and lice mortality may not be correct. In addition, fish mortality in the natural environment may be higher than that seen...
in laboratory studies as a consequence of additive effects. The effects of salmon lice have, for example, been shown to be more severe for Atlantic salmon post-smolts impaired also by other influences such as suboptimal water quality (Finstad et al. 2007). Furthermore, compromised fish in the natural environment may experience an elevated mortality risk from predators (Thorstad et al. 2012). A reduced or compromised immune system (Bjørn & Finstad 1997) may incur additional mortality over a longer term, and yet other environmental effects may also exacerbate the effects of salmon lice and the critical threshold levels.

**Effects of salmon lice on individual sea trout in field studies**

**Mechanical damage of fish skin and tissue**

Field studies are important to verify the extent to which laboratory studies are representative of wild fish in natural systems. Similar to results from laboratory studies, fin erosion and haemorrhage at the base of the dorsal fin have been frequently recorded in wild-captured sea trout with heavy burdens of chalimus (McVicar et al. 1993, Dawson 1998, MacKenzie et al. 1998, Skaala et al. 2014a). The patterns reported from laboratory studies, with attachment of chalimi primarily to the dorsal fin and mobile stages present along the dorsal or more anterior body regions, are confirmed from numerous field studies (Tully et al. 1993a,b, Dawson 1998, MacKenzie et al. 1998, Marshall 2003, Urquhart et al. 2008). Cranial lesions and grazing marks on the gill opercula, and along the ventral body surfaces have also been described (McVicar et al. 1993, Tully et al. 1993b).

**Osmoregulatory problems and physiological stress responses**

Primary and secondary physiological stress responses to salmon lice infestation, as exemplified by elevated plasma cortisol, plasma chloride and blood glucose levels, have been documented in wild-captured sea trout, and the elevated cortisol levels were similar to those found in laboratory studies (Poole et al. 2000, Bjørn et al. 2001). Bjørn et al. (2001) concluded that the osmotic imbalance and need for mobilisation of energy stores may have been the result of the integrated stress response attributable to the infestation rather than a result of the mechanical damage caused by the salmon lice. This deduction was based on the observation that chalimus was the predominant life stage, and that only limited skin erosion was observed. Fish body sizes in these studies were <150 g body mass (Bjørn et al. 2001), or an average body length of 18 cm (Poole et al. 2000). For slightly larger fish carrying mobile salmon lice (mean fork length = 23 cm, body mass = 126 g), blood plasma showed a reduction in total protein, serum albumin, and cholesterol compared with sea trout lacking salmon lice or those with copepodids or chalimus stages only (Dawson 1998). Furthermore, plasma glucosis levels increased with lice numbers when all life-stages of salmon lice were pooled (Dawson 1998). The highest estimated cortisol levels in wild-captured sea trout occurred during the period when post-smolts had only recently entered the sea, affirming that post-smolts may be more vulnerable to salmon lice when physiologically adapting to seawater (Poole et al. 2000).

In a controlled experiment, downstream-migrating sea trout smolts were captured in freshwater and held in tanks; 1 group of fish was exposed to seawater (and thereby the natural concentration of lice larvae), whereas an unexposed control group was held in filtered seawater from which salmon lice larvae had been removed (Birkeland & Jakobsen 1997). Salmon lice-induced mortality commenced 11 d after exposure to unfiltered seawater, by which time some lice had developed to the pre-adult stage (water temperature = 17–20°C, mean abundance and intensity of salmon lice per fish = 59). Fish in the exposed group showed severe osmotic problems by this stage, with elevated plasma chloride levels and lower plasma total protein and albumin levels.

The direct observation of mortality is difficult to achieve for free-ranging individual fish in marine waters. Tully & Whelan (1993), Tully et al. (1993a,b) and Birkeland (1996) all reported direct observations of dead and moribund sea trout in estuaries linked to salmon lice infestations. However, fish in the marine environment may die from multiple causes, such as predation, before they may be lost as a direct result of a pathological disease or parasite infestation (Thorstad et al. 2013). Sea louse-infested hatchery-reared sea trout and Atlantic salmon smolts equipped with acoustic transmitters did not show increased mortality during fjord migration compared with uninfected control groups (Sivertsgård et al. 2007). However, the study extended only over a short time period, and during which period the salmon lice could develop only to the chalimus stage of the life cycle.
Growth

Growth patterns of sea trout in freshwater and seawater are generally complex and influenced by a number of environmental factors and characteristics of the fish. Selective salmon lice-induced mortality may mask other potential effects on sea trout growth. It is especially difficult to isolate the effects of salmon lice on fish growth from other possible effects in field studies, because multiple factors may change either independently or in concert over the observational period.

Notwithstanding this caveat, Fjørtoft et al. (2014) compared growth of sea trout from a river in western Norway during 1976–1982, in the absence of local salmon farming, and between 2000–2007 whilst farming was active, based on scale analyses. They demonstrated that fish growth was slower during both their first and second summers at sea during the observational period that salmon farming was active, but there was no difference in growth rate of the same individuals whilst resident in freshwater. The growth reduction after the first summer in the sea corresponded to a body mass reduction of 20–40%.

A gradual decrease in marine growth rates was also detected from scale analyses of sea trout from a Scottish river adjacent to salmon farms (data from 1980 to 1989–1990, 1992–1993, and 1997–2001) (Butler & Walker 2006). Thus, from 1980 to the period 1997–2001, maximum sea age was reduced from 11 to 5 yr. When comparing scale samples from 1926 and 1980, the sea age and marine growth rates did not differ markedly. Butler & Walker (2006) concluded that the decline in growth after 1980 was at least partly caused by salmon lice epizootics emanating from the fish farms established 4 and 7 km from the river mouth in 1987.

For the Burrishoole sea trout stock in Ireland, ratios of sea growth to freshwater growth showed no discernible trend until 1990, after which this ratio showed a marked decrease over the period 1990–1992 (Poole et al. 1996). A significant reduction in marine growth was most likely linked to premature return to freshwater of salmon lice-infested fish (Poole et al. 1996).

Behaviour and migration patterns—premature return to freshwater

Premature return to freshwater of sea trout carrying large numbers of salmon lice has repeatedly been recorded, and has been interpreted as an adaptive behavioural response to salmon lice-induced osmotic dysfunction (Birkeland 1996, Birkeland & Jakobsen 1997, Bjørn et al. 2001, Wells et al. 2007). The return to freshwater may enable the infested sea trout to regain its osmotic balance and survive, because salmon lice have a low tolerance to hyposaline or freshwater conditions (Birkeland 1996). It should be noted that these impacts extended beyond those induced by the chalimus and mobile stages of salmon lice, because high levels of copepodids alone also caused premature freshwater return of sea trout (Birkeland & Jakobsen 1997). Birkeland (1996) concluded that the recorded high salmon lice levels indicated that the post-smolts that returned to freshwater would not have survived had they remained at sea.

The first reports of post-smolt sea trout returning to freshwater prematurely in poor physical condition and with heavy salmon lice infestations, within only a few weeks of their seaward migration, date from the late 1980s and early 1990s in Ireland (Whelan 1991, Tully & Whelan 1993, Tully et al. 1993a,b). Subsequent studies from Ireland, Norway and Scotland have reported similar observations (Birkeland 1996, Birkeland & Jakobsen 1997, Gargan 2000, Bjørn et al. 2001, Butler & Walker 2006, Hatton-Ellis et al. 2006, Pert et al. 2009, Gjelland et al. 2014). It was apparent from relatively early studies that premature return to freshwater may occur as soon as within the first few days, or the first 1–2 wk, at sea (Birkeland & Jakobsen 1997, Bjørn et al. 2001).

The timing of freshwater return was monitored by operating a fish trap in a Norwegian river (Birkeland 1996, Birkeland & Jakobsen 1997). Nearly half (41%) of prematurely returning post-smolts migrated to sea again that same summer, with a median freshwater residency of 38 d following their return to the river (Birkeland 1996). By the time of second descent, most fish had lost the salmon lice, but they also had lost one quarter of their body mass. Whereas the returning post-smolts carried mainly copepodid and chalimus stages of salmon lice, the older migrants showed a larger proportion of mobile preadult and adult salmon louse stages. Several older returning sea trout died. Within 1 wk, 20% of the older migrants were found dead in the river, and they had considerable skin lesions from salmon lice infestations that had become secondarily infected by fungi or bacteria.

Laboratory studies have confirmed that transfer from seawater to freshwater after initial exposure to salmon lice improves the physiological status of the fish and that mortality was reduced compared to fish maintained infested in seawater (Wells et al. 2007). However, secondary bacterial or fungal infection was recorded on a number of the infested fish following
<table>
<thead>
<tr>
<th>Mean (max.) abundance</th>
<th>Mean intensity</th>
<th>Prevalence (%)</th>
<th>Lice life cycle stage</th>
<th>Study period</th>
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<tr>
<td>3.2</td>
<td>4.0</td>
<td>81</td>
<td>Not specified</td>
<td>1972–1973, month not specified, May 1992</td>
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<td>1.0–7.75 (325)</td>
<td>7.0–104.8</td>
<td>14.3–100</td>
<td>Dominated by chalimus stages</td>
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<td>5.0–8.0 (46)</td>
<td>5.0–10.7</td>
<td>75–100</td>
<td>20–26% chalimus</td>
<td>Jun–Aug, 1991 and 1992</td>
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<td>7.0–63.9 (216)</td>
<td>7.0–63.9</td>
<td>75–100</td>
<td>0–79% chalimus, increasing proportion of chalimus with increasing lice abundance</td>
<td>Jun–Aug, 1991 and 1992</td>
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<td>1.4–5.0 (11)</td>
<td>1.4–5.0</td>
<td>25–100</td>
<td>6–33% chalimus</td>
<td>Jun–Aug, 1991 and 1992</td>
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<td>89.8–260.8 (1002)</td>
<td>103.0–272.4</td>
<td>87–96</td>
<td>Mainly chalimus</td>
<td>Jun–Jul 1992</td>
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<td>53.5–623.0 (1179)</td>
<td>53.5–623.0</td>
<td>88–100</td>
<td>Mainly copepodids and chalimus</td>
<td>Jun 1992</td>
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<td>4.66 (41), and 4.42 (55)</td>
<td>5.26 (range = 1–41) and 5.47 (range = 1–55)</td>
<td>96, both years</td>
<td>Mainly preadults and adults</td>
<td>Jun–Nov, 1992 and 1993</td>
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<tr>
<td>3.19 (12), and 1.17 (4)</td>
<td>3.96 (range = 1–12) and 1.75 (range = 1–4)</td>
<td>67 and 81</td>
<td>Not specified</td>
<td>1972–1973, month not specified</td>
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<td>42 (SE = 35)</td>
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<td>82</td>
<td>Mainly chalimus</td>
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<td>71 (SE = 45)</td>
<td>Not given in publication and not calculated</td>
<td>82</td>
<td>Mainly chalimus, but also preadults and adults</td>
<td>May 1996</td>
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<td>0.5–10.9 (84)</td>
<td>2.7–26.7</td>
<td>20–85</td>
<td>Proportion between larvae and mobile stages varied among samples. Proportion of mobile stages always &gt;30%</td>
<td>Aug–Oct 1992, May–Sep 1993</td>
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<td>0.0–7.7 (207)</td>
<td>0–46.4</td>
<td>0–100</td>
<td>Mainly copepodids and chalimus, but increasing proportion of mobile stages from late May and onwards for many locations</td>
<td>Apr–Sep 1994</td>
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<tr>
<td>Not given</td>
<td>Median = &lt;3–8</td>
<td>20–100</td>
<td>Mainly preadults and adults. Dominated by adults also the rest of the summer, but with chalimus appearing in Apr–Oct (never &gt;15% chalimus)</td>
<td>Mar–Dec, 1992–1995</td>
</tr>
<tr>
<td>20.1 (233)</td>
<td>27.9</td>
<td>72</td>
<td>Information not given</td>
<td>May–Jun 1995</td>
</tr>
<tr>
<td>0.0–111</td>
<td>0–156 fish farming</td>
<td>0–100</td>
<td>Information not given</td>
<td>May–Jun, 1993–1997</td>
</tr>
<tr>
<td>49.3–194.9 (471)</td>
<td>53–203</td>
<td>89–96</td>
<td>Chalimus dominated during the entire period</td>
<td>Jun–Sep 1997</td>
</tr>
<tr>
<td>0.6–8.9 (36)</td>
<td>1–13</td>
<td>55–89</td>
<td>Chalimus dominated in Jun, but up to 50% preadults and adults later in summer</td>
<td>Jun–Sep 1997</td>
</tr>
<tr>
<td>0.1–23.6 (134)</td>
<td>0–29.5</td>
<td>0–95</td>
<td>Mainly chalimus</td>
<td>Jun–Aug, 1992–1993</td>
</tr>
<tr>
<td>0.2–13.0 (84)</td>
<td>0–17.3</td>
<td>0–83</td>
<td>Mainly chalimus in Jul and increasing amount of preadults and adults in Aug</td>
<td>Jun–Aug, 1992–1993</td>
</tr>
<tr>
<td>0.75 and 0.33 (11)</td>
<td>Median = 1–2</td>
<td>0–49</td>
<td>Majority preadults and adults</td>
<td>Oct 1998–Apr 1999, Oct 1999–Mar 2000</td>
</tr>
<tr>
<td>0.684 (500)</td>
<td>0–46.4</td>
<td>0–100</td>
<td>Mainly chalimus, but increased proportions of mobile stages in Jul–Sep</td>
<td>Mar–Oct, 1998–2001</td>
</tr>
<tr>
<td>0.6–8.8 (33)</td>
<td>0–8.6</td>
<td>0–81</td>
<td>Preadults and adults dominated in winter, chalimus in Sep–Oct</td>
<td>Mar–Dec 2001</td>
</tr>
<tr>
<td>0.1–3.6 (28)</td>
<td>1.0–4.7</td>
<td>6–80</td>
<td>Preadults and adults dominated in winter, chalimus in Sep–Oct</td>
<td>Mar–Dec 2001</td>
</tr>
<tr>
<td>0.0 (Jun) to 16.7 (Aug) (76)</td>
<td>0 (Jun) to 18.9 (Aug)</td>
<td>0 (Jun) to 80 (Aug)</td>
<td>Chalimus dominated in Jun–Jul, and preadults and adults started to occur in Aug</td>
<td>Jun–Aug 2000</td>
</tr>
<tr>
<td>7.8 (95%, CI = 6.0–10.0) (30 (60))</td>
<td>7.8</td>
<td>100</td>
<td>Preadults and adults</td>
<td>May and Jun 2005</td>
</tr>
<tr>
<td>0.82–7.87</td>
<td>0.24–7.87</td>
<td>29–100</td>
<td>Only copepodid and chalimus stage</td>
<td>May 2007</td>
</tr>
<tr>
<td>0.03–0.37</td>
<td>0.00–0.09</td>
<td>3–23</td>
<td>Information not given</td>
<td>Jul–Dec, 2006–2007</td>
</tr>
<tr>
<td>0.2–20.5 (186)</td>
<td>3.5–30.2</td>
<td>4–77</td>
<td>All stages in May, mainly chalimus in Jun, and increased proportion of adults again thereafter</td>
<td>May–Aug, 2003–2004</td>
</tr>
<tr>
<td>3.3–52.8 (130)</td>
<td>4.6–52.8</td>
<td>73–100</td>
<td>All stages in May, mainly chalimus in Jun and Jul, and subsequently increased proportion of adults</td>
<td>May–Aug, 2003–2004</td>
</tr>
<tr>
<td>0–8.1 (44)</td>
<td>0–12.0</td>
<td>0–83</td>
<td>Information not given</td>
<td>May–Aug, 2008–2012</td>
</tr>
<tr>
<td>0.106 (689)</td>
<td>1.8–114.8</td>
<td>0–100</td>
<td>Information not given</td>
<td>May–Aug, 2008–2012</td>
</tr>
</tbody>
</table>

Table 1. Summary of salmon lice levels found on wild sea trout in the current literature, showing the mean abundance of salmon lice per fish caught per sample (max. number of lice on an individual sea trout is given in parentheses where data available), mean (median where specified) intensity of salmon lice per infested fish in the sample, and percentage prevalence.
of salmon lice infested fish in the sample. Life cycle stage of lice, time of sample collection, capture methods used, sea trout size, study site and extent of fish farming in the area are also summarised. * denotes lice levels given for brown trout and Arctic char combined, because lice levels did not differ among the 2 species.

<table>
<thead>
<tr>
<th>Capture method</th>
<th>Fish size</th>
<th>Study site</th>
<th>Extent of salmon farming in area</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gill nets</td>
<td>Mean = 328 g</td>
<td>North Sea off Yorkshire</td>
<td>No farms</td>
<td>Boxshall (1974)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(England)</td>
<td></td>
<td>Tully et al. (1993a)</td>
</tr>
<tr>
<td>Rod and line</td>
<td>Not given</td>
<td>River Emsaard and Argyll rivers, (1) west coast of Scotland, Rivers Morar, Ewe and Burn (northwest coast of Scotland)</td>
<td>Information not given</td>
<td>Sharp et al. (1994)</td>
</tr>
<tr>
<td>Rod and line</td>
<td>Not given</td>
<td>Rivers Don, Ythan and Hope (north and northeast coast of Scotland)</td>
<td>Information not given</td>
<td>Sharp et al. (1994)</td>
</tr>
<tr>
<td>Fish trap in lower part of river capturing prematurely returned trout</td>
<td>Mean = 19−35 mm</td>
<td>Lenningalselven (Horda-land, Norway)</td>
<td>Intensive farming</td>
<td>Birkeland (1996)</td>
</tr>
<tr>
<td>Gill nets and market</td>
<td>Mean fork length =</td>
<td>East Anglia (England)</td>
<td>No farms</td>
<td>Tingley et al. (1997)</td>
</tr>
<tr>
<td></td>
<td>228 mm, 120 g</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean fork length =</td>
<td>Rivers Bunowen, Bundredroga, Erriff, Culfin, Dawros and Owenglin in Ballinakill District (Ireland)</td>
<td>Information not given</td>
<td>Dawson (1998)</td>
</tr>
<tr>
<td>Electrofishing in river mouth, and Gill nets</td>
<td>Mean = 245 mm</td>
<td>Akeselva and Osloljford (northern Norway)</td>
<td>No farms</td>
<td>Mo &amp; Heuch (1998)</td>
</tr>
<tr>
<td>Seine nets, rod and line, Gill nets</td>
<td>Fork length =</td>
<td>Locations on the west coast (n = 17) east coast (n = 3) and north coast (n = 1) of Scotland</td>
<td>Both from areas with and without intensive fish farming</td>
<td>MacKenzie et al. (1998)</td>
</tr>
<tr>
<td></td>
<td>101−559 mm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beach seine</td>
<td>Mean = 320 mm, 440 g</td>
<td>Skagerakk coast (southern Norway)</td>
<td>No farms</td>
<td>Schram et al. (1998)</td>
</tr>
<tr>
<td>Gill nets, electrofishing, and wolf trap</td>
<td>Not given</td>
<td>North Mayo, South Mayo, Galway and Kerry locations (total n = 10) (Ireland)</td>
<td>Information not given</td>
<td>Byrne et al. (1999)</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 119−209 g</td>
<td>Vesterålen (northern Norway)</td>
<td>Intensive farming</td>
<td>Bjørn et al. (2001)*</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 119−464 g</td>
<td>Ofoten (northern Norway)</td>
<td>Low farming intensity</td>
<td>Bjørn et al. (2001)*</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Not given</td>
<td>Altafjord (northern Norway)</td>
<td>Intensive farming</td>
<td>Bjørn &amp; Finstad (2002)*</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Not given</td>
<td>Lille Porsanger (northern Norway)</td>
<td>Low farming intensity</td>
<td>Bjørn &amp; Finstad (2002)*</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 328 g (SD 63)</td>
<td>Skagerakk coast (southern Norway)</td>
<td>No farms</td>
<td>Heuch et al. (2002)</td>
</tr>
<tr>
<td>Sweepnets</td>
<td>Not given</td>
<td>Laxford Bay (Sutherland, Scotland)</td>
<td>During fallow and production periods at nearby farm</td>
<td>Marshall (2003)</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 668 g (SD 432)</td>
<td>Rankafjord (northern Norway)</td>
<td>No farms</td>
<td>Rikardsen (2004)</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 340 g (SD 314)</td>
<td>Ballafjord (northern Norway)</td>
<td>No farms</td>
<td>Rikardsen (2004)</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 240 g</td>
<td>Lakseljord and Malengkbott (northern Norway)</td>
<td>Low farming intensity</td>
<td>Bjørn et al. (2007)*</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 170 g</td>
<td>Altafjord (northern Norway)</td>
<td>Intensive farming</td>
<td>Bjørn et al. (2007)*</td>
</tr>
<tr>
<td>Bag nets</td>
<td>Mean = 1.16 kg (SD 0.32)</td>
<td>North Esk (east coast of Scotland)</td>
<td>No farms</td>
<td>Urquhart et al. (2008)</td>
</tr>
<tr>
<td>Electrofishing during return to freshwater Gill nets</td>
<td>Mean = 155 mm, 35 g</td>
<td>River Shieldsay (Scotland)</td>
<td>Information not given</td>
<td>Pert et al. (2009)</td>
</tr>
<tr>
<td>Bag nets, sweepnets, Gill nets</td>
<td>Mean = 440−480 mm, 1.06–1.21 kg</td>
<td>Rivers Annan and Carron (west coast of Scotland)</td>
<td>Close to salmon farms</td>
<td>Urquhart et al. (2010)</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 97–383 g, 210–270 mm</td>
<td>Upper Forth Estuary, North Esk, and Stonehaven Bay (east coast of Scotland)</td>
<td>No farming</td>
<td>Urquhart et al. (2010)</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean = 364−490 g, 310–320 mm</td>
<td>Kilsbyfjord in Romsdalsfjord (Norway)</td>
<td>Protection zone with low farm activity</td>
<td>Bjørn et al. (2011)</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean mass = 131–457 g</td>
<td>Five large fjord areas in Norway with restrictions on fish farming (National Salmon Fjords)</td>
<td>&gt;30 km to nearest farm</td>
<td>Serra Linares et al. (2014)</td>
</tr>
<tr>
<td>Gill nets</td>
<td>Mean mass = 85−823 g</td>
<td>Five smaller fjord areas in Norway with some restrictions on fish farming activity (National Salmon Fjords)</td>
<td>&lt;30 km to nearest farm</td>
<td>Serra Linares et al. (2014)</td>
</tr>
<tr>
<td>Bag nets</td>
<td>Mean = 31−35 cm, 263–405 g</td>
<td>Intensive farming</td>
<td></td>
<td>Vollset &amp; Barlaup (2014)</td>
</tr>
</tbody>
</table>

1.06−1.21 kg (west coast of Scotland)
their transfer to freshwater. Although premature return migration can reduce or eliminate the lice infestations on individual fish, it will also involve a fitness cost in terms of reduced growth opportunities (Birkeland 1996), and subsequently reduced resources for egg production, thus reducing female fecundity.

Salmon lice levels in samples of wild sea trout

Salmon lice levels in areas before, or without, salmon farming

Ideally, in order to evaluate whether or not salmon lice levels have become elevated in wild populations, and their possible association with salmon farming, baseline information on lice levels and their year-round population dynamics would be required for time periods preceding the development of fish farming, or from areas lacking fish farming. Historical salmon lice levels on sea trout prior to the industry (Boxshall 1974), and data for areas lacking fish farming (Tingley et al. 1997, Schram et al. 1998, Heuch et al. 2002, Rikardsen 2004, Urquhart et al. 2010), generally show a relatively high prevalence, but low intensity of salmon lice on sea trout (Table 1, Figs. 2 & 3). The natural intensity of salmon lice on sea trout in areas without fish farming may be as low as 0–3 lice per fish, and with a prevalence of 0–20% during late winter and spring (Schram et al. 1998, Heuch et al. 2002, Rikardsen 2004). Intensities increased to a peak of up to 4–8 lice per fish with higher prevalences in the late summer and autumn (Tingley et al. 1997, Schram et al. 1998, Rikardsen 2004, Urquhart et al. 2010). In areas without fish farms, prevalence may range up to 100%, but is most often <70% (Table 1, Figs. 2 & 3). The peak in salmon lice levels on sea trout may occur 1–2 mo later at more northerly locations compared to more southern latitudes, perhaps reflecting seasonal contrasts in temperature and ontogenetic developmental rates for salmon lice. At more northern latitudes in Norway, the peak salmon lice level in sea trout has been noted during the period August–October (Bjørn & Finstad 2002, Rikardsen 2004), whereas at more southerly latitudes this may advance to June–August (Mo & Heuch 1998, Schram et al. 1998, Heuch et al. 2005).

Atlantic salmon, sea trout and Arctic char all are natural hosts of salmon lice and, due to the seasonality of their migrations, there are few of these wild hosts in coastal waters during the winter months. Atlantic salmon feed in the open ocean and traverse coastal areas relatively quickly during the outward migration in the spring (Thorstad et al. 2011). The rate of transit of returning adult salmon through coastal waters also is typically rapid (e.g. Davidsen et al. 2013). These return migrations usually occur during May–September in Norway, but with more extended and variable timing in Scotland and other southerly regions of salmon distribution (Thorstad et al. 2011). By contrast, sea trout and Arctic char often spend weeks or months during the summer in coastal areas and the remainder of the year in freshwater, although a proportion of trout and char populations may reside at sea throughout the year (Jonsson & Jonsson 2011). Because salmon lice cannot survive long in freshwater, the persistence of the parasite population depends upon hosts at sea over the winter months. For wild host populations, these winter components therefore include Atlantic salmon feeding in the open ocean, and the small numbers of sea trout and Arctic char that remain in coastal areas (Klemetson et al. 2003, Jonsson & Jonsson 2011). In areas without salmon farms, the salmon lice populations therefore have few available hosts and appear to encounter a host resource bottleneck in winter (Schram et al. 1998, Heuch et al. 2002, Rikardsen 2004).

The highest levels of salmon lice on sea trout reported for an area without fish farming was a mean abundance of 10.9 lice per fish sampled, and mean
intensity of 11.6 lice per infested fish (Oslofjord, southern Norway) (Mo & Heuch 1998). However, most of the samples in that study showed abundances and median intensities in the range of 0.5–8 and 1.5–10 salmon lice per fish, respectively, with an overall prevalence of 51%. However, it is important to note that 4 heavily-infested individuals (of a total sample of 102 fish) each carried as many as 33–84 lice (of which 43–72% were adults). This shows that even in areas without fish farming a few individual sea trout may carry salmon lice levels that, on the basis of laboratory studies, will probably induce stress in the host fish.

To our knowledge, there are no published records of outbreaks of salmon lice epizootics on sea trout populations that pre-date the commencement of salmon farming. Nevertheless, it is important to emphasize that salmon louse epizootics were reported for Atlantic salmon and brook trout Salvelinus fontinalis (Mitchill, 1814) over the period 1939–1940 at Moser River in Nova Scotia on the Canadian east coast (White 1940, 1942). Notwithstanding a possible publication bias, the lack of known and reported epizootics in areas without salmon farming indicate that this is not a common phenomenon for salmon lice on wild sea trout or other salmonid populations.

Salmon lice levels in areas with salmon farming

Salmon lice levels reported for sea trout in farm intensive areas are generally higher and more variable than in areas without fish farming (Table 1, Figs. 2 & 3). High variation in salmon lice levels can be expected because studies differ in time of the year of the survey, the fish sizes collected, sampling methods, habitats sampled and sample sizes. Moreover, only fish that survived infestation will be caught. Fish captured in gill nets and seine nets may be subject to physical abrasion during capture and removal from the net, thereby resulting in the loss of some salmon lice. When fish are captured in bag nets or other gear where they are retained free-swimming without being killed, lice may move between individual sea trout (K. Vollset and S. Kålås pers. obs.). Furthermore, the place and time of sampling may not be representative of the local sea trout population, and the salmon lice level may be overestimated if only the most heavily infested trout that are returning prematurely to freshwater are caught. Conversely, salmon lice levels on fish captured in estuaries may be underestimated because sampling might be biased towards fish that have only recently arrived and have not been at sea for sufficient time for salmon lice to
Recent Norwegian studies have shown that the risk of mortality was elevated for 12 to 90% of the sampled fish at 1 or more sampling occasions in 5 fjord areas <30 km from the nearest farms (Serra-Llinares et al. 2014). Moreover, Taranger et al. (2015) found that of 109 stations investigated along the Norwegian coast for salmon lice infection, 67 locations indicated moderate-to-high mortality of wild sea trout. Finally, a large-scale study, with nearly 5000 sea trout sampled from 48 sites along the Scottish west coast and Outer Hebrides during 2003–2009 (Middlemas et al. 2013), showed that 13% of the fish carried salmon lice levels above the suggested critical threshold of 13 mobile lice (Wells et al. 2006).

**Interactions between fish farming activity and salmon lice levels of sea trout in coastal areas**

In coastal areas with intensive Atlantic salmon farming, the large disparity in abundance between cultured and wild hosts is such that local larval production of salmon lice most likely originates primarily from farmed salmon and not from wild fish, although all salmon lice hosts potentially cross-infest one another (Tully & Whelan 1993, Heuch & Mo 2001, Butler 2002, Todd et al. 2004, Heuch et al. 2005, Penston & Davies 2009, Jansen et al. 2012, Torrissen et al. 2013). Several studies of wild sea trout have shown increased salmon lice levels with decreasing distance to salmon aquaculture sites (Tully et al. 1999, Gargan 2000, Bjørn et al. 2001, 2011, Bjørn & Finstad 2002, Gargan et al. 2003). Others show increased concentrations of salmon lice larvae in the water column with decreasing distance to salmon farms (Gillibrand et al. 2005, Penston et al. 2008a,b). Moreover, there is additional evidence of a correlation between the abundance of salmon lice larvae in the water column and the number of gravid salmon lice larvae produced by adjacent farms (Penston & Davies 2009). Hence, these studies support a link between salmon farms and salmon lice burdens in sea trout.

A correlation between salmon farming and lice production is even more apparent in farmed areas when farms synchronize their production cycles. During a synchronised 2-yr production cycle, the mean total biomass of fish, and thereby the potential for salmon lice larval production, increased over time (Butler 2002, Revie et al. 2002, Gillibrand et al. 2005). Several studies have shown a relationship between the production cycle in salmon farms and salmon lice levels of sea trout.
levels on wild sea trout, with higher lice levels on trout in the second year of the farm production cycle (Butler 2002, Marshall 2003, Hatton-Ellis et al. 2006, Middlemas et al. 2010, 2013). Biannual cycles of salmon lice epizootics have been observed only in areas with synchronised-year class production, whereas epizootics were observed every spring in areas with a mixed-year class production (Butler 2002).

Gargan et al. (2003), Middlemas et al. (2013), and Serra-Llinares et al. (2014) all included a large number of sampling sites during monitoring of salmon lice levels on wild sea trout over several years. In all 3 studies (respectively from Ireland, Scotland and Norway), the highest levels of salmon lice were found on sea trout sampled in coastal areas within 20–30 km of the farms. In Scotland, the distance to the nearest farm did not influence the probability of infestations above the critical level for physiological impact by salmon lice (based on Wells et al. 2006, Bjørn & Finstad 1997) beyond 31 km, although there was considerable uncertainty around this cut-off distance (95% confidence limits: 13–149 km) (Middlemas et al. 2013). Gargan et al. (2003) found reduced lice levels on wild sea trout recorded at distances >30 km from farms. Chalimus dominated at a distance to farms of <30 km, and preadult and adult lice stages dominated at distances >100 km. Furthermore, Serra-Llinares et al. (2014) found that 41% of the variance of the mean lice abundance on wild sea trout could be explained by the lice production in farms, in areas where active fish farms existed within a distance of <30 km. Elevated salmon lice levels on wild sea trout have, however, also been recorded at greater distances from farms (e.g. >25–30 km) (Bjørn & Finstad 2002, Bjørn et al. 2011).

The distance and directionality of salmon lice larval transport from their release source depend upon multiple variables, including their development rate, water temperature, currents and wind-driven circulation (Gillibrand et al. 2005, Asplin et al. 2011, 2014). Ontogenetic development rates of larvae increase with water temperature (Wootten et al. 1982, Johnson & Albright 1991a, Stien et al. 2005), and larval drift distance may thus increase with decreasing temperatures. Numerical models show that nauplii and copepodids may be transported up to 100 km from their source, although typical dispersal distances are up to ~25 km (Asplin et al. 2011, 2014, reviewed in Costello 2009). In addition, salinity influences salmon lice survival and behaviour (Heuch 1995), which also affects the density of salmon lice in a given area. Hence, although these various studies show potentially considerable variability in the effective dispersal of salmon lice, it is likely that the majority of lice larvae remain relatively close to their source.

Aggregation of salmon lice larvae may occur in certain areas, typically close to land and in embayments (Asplin et al. 2014), and larval distribution is commonly spatially and temporally patchy within a given area (Murray 2002). The movements of wild sea trout themselves will also contribute to variation in their risk of exposure to salmon lice. Fish may move between sites of variable infestation risk, and are not necessarily captured close to the site where they have been infested. Furthermore, premature return to freshwater could reduce the lice infestation rates. Hence, considerable variation in salmon lice levels on wild sea trout, as has been observed in rivers close to farms in Ireland (Gargan et al. 2003), is to be expected. Such complexities may underlie the occasional reports of the lack of a relationship between salmon lice levels and distance to nearby farms, or between lice levels in wild sea trout and those on a nearby fish farm (MacKenzie et al. 1998, Marshall 2003).

Population effects of salmon lice

Population level effects of salmon lice on marine survival and growth of sea trout

Brown trout populations in catchments, tributaries and river stretches accessible from the sea show genetic differentiation, and some of this variability is likely the result of local adaptation (Jonsson & Jonsson 2011). Within populations and rivers, there is little genetic differentiation between sea-migrating and resident individuals (Hindar et al. 1991, Charles et al. 2005, 2006), but, anadromy is a quantitative trait that is controlled by interactions between genetic and environmental factors (Jonsson & Jonsson 1993, 2006, 2011, reviewed by Ferguson 2006). Migrant and resident brown trout within rivers can spawn separately and form discrete populations, or they may spawn together successfully, and thereby constitute freely interbreeding fractions of a single spawning stock (Jonsson & Jonsson 1993, 2006, 2011). The advantages of marine migrations for sea trout include the opportunity of accessing more productive feeding conditions in order to enhance growth, fecundity and thereby evolutionary fitness (Jonsson & Jonsson 1993, 2006, 2011).

Anadromy evolves in response to trade-offs between the costs and benefits of migration compared with
residency, and these are balanced through their effect on fitness (Jonsson & Jonsson 1993, 2006, Bohlin et al. 2001, Ferguson 2006, Solomon 2006). A higher growth rate in freshwater, combined with an increase in the migratory cost, can result in a higher proportion of resident trout (Jonsson & Jonsson 2006). Changes in environmental conditions or genes can, therefore, result in a population shift in life-history strategy (Jonsson & Jonsson 1993, 2006, Ferguson 2006). The likelihood of moderate heritability of anadromy as a trait, in concert with the higher fecundity of larger sea trout, can result in substantial population changes occurring within perhaps only a few generations. Thus, increases in marine mortality and reduced growth of sea trout induced by salmon lice both can shift the selective balance in favour of the freshwater resident life history.

In contrast to the density-dependent freshwater mortality of sea trout that occurs especially during the earliest embryonic and post-emergence life stages, marine mortality seems not to be density-dependent. Mortality in the freshwater phase therefore can have a population regulating effect, whereas mortality in the marine phase (including that attributable to salmon lice) is not regulatory, but has a population reducing effect (see Milner et al. 2003, Einum & Nislow 2011). Elevated mortality during the freshwater phase can, to a varying extent, be compensated by increased growth and survival of the remaining juveniles, whereas there are no compensatory mechanisms for additional mortality in the marine phase. Hence, elevated rates of marine mortality, such as that induced by salmon lice, can result in a proportional reduction in the number of spawning adults. Because sea-run brown trout typically are females (Jonsson & Jonsson 2011), any additional marine mortality has the potential to affect recruitment even more negatively than would be the case for an equal sex ratio.

Reduced marine survival and growth as a result of increased salmon lice levels in farm intensive areas will likely lead to a decreased frequency of sea-run brown trout, as indicated by Gargan et al. (2006b). Catchments offering poor environmental conditions for brown trout during some periods of the year, for example, due to drought or freezing (Borgstrøm & Heggenes 1988, Järvi et al. 1996, Limburg et al. 2001), may be at risk of losing their brown trout populations if the marine mortality is chronically high. Larger catchments with more suitable year-round conditions for brown trout may not be at such risk, but severe reduction or loss of the sea-run migratory form can result in (1) altered genetic composition of populations (which may be regarded as the effective loss of a sea trout population and its replacement by a freshwater resident population with differing population genetic characteristics), (2) reduced genetic diversity, and (3) a greater uniformity in life history characteristics. The loss of access to the improved growth opportunities offered by the marine environment also will lead to a lower abundance of brown trout and reduced recruitment.

Population effects in Ireland

Data for salmon lice intensities on marine salmon farms and wild populations, in addition to observations of the incidence of premature return by sea trout, indicate that salmon lice from marine salmon farms was a significant factor in observed stock collapses in western Ireland in the late 1980s (Tully & Whelan 1993, Tully et al. 1999, Gargan et al. 2003). Data on upstream migration are available since 1970 from the Burrisheoole upstream trap and 1985 for the Tawnyard (Erriff) sea trout kelt trap. Rod catch data and trap records from both fisheries indicate a stable sea trout population structure prior to 1989, dominated by a peak of finnock (sea trout that return to freshwater in the autumn, following a few months at sea), a second peak of maiden sea trout (fish that had spent the previous winter at sea), some older fish and previous return spawners (Poole et al. 1996, Gargan 2000). Subsequent to the 1989 sea trout stock decline in western Ireland there was a marked reduction in the number and proportions of sea age classes, and the stocks were characterised by low returns of finnock and fewer veteran sea trout in the older age classes (Whelan 1993, Poole et al. 1996, Gargan 2000, Poole et al. 2006). The number of ova deposited by sea trout in the Burrisheoole system, estimated to range between 0.49 and 1.61 million before 1987, decreased to <60 000 by 2000 and showed a minimum of 27 500 in 2003 (Poole et al. 2006). O’Farrell et al. (1989) estimated that the percentage contribution to ova deposition of 0-sea age fish was 5.6 %, whereas that of 1-sea age trout was 41 % and 2-sea age fish and older contributed 54 % to ova deposition. Hence, reduced marine survivorship of larger, older spawners that contribute disproportionately to overall egg deposition can exert considerable and rapid impacts at the population level.

Prior to the onset of marine salmon aquaculture in the Burrisheoole system, western Ireland, the percentage of sea trout smolts that survived to return as 0+-sea age finnock in the same year ranged from 11 to
32%, with a historical mean of 21%. Throughout the 1990s (i.e. subsequent to introduction of salmon farming) there was a saw-tooth pattern of finnock return rates, whereby the mean return rate for this period (excluding 1999) was three times lower (6.8%) than the historical average (Poole et al. 2006). Data from 2 other trap facilities in western Ireland (Owengowla and Invermore) indicate a marine survival rate of <2% in the majority of the years during this period (Gargan et al. 2006b). The highest marine survival (19%) for these 2 traps was observed on the Owengowla in 1994, coinciding with whole-bay spring fallowing of salmon aquaculture. Although survival estimates under circumstances of local farm fallowing would require replication in multiple years and locations, these data strongly indicate that salmon lice from marine Atlantic salmon farms made an important contribution to the sea trout stock decline on Ireland’s west coast (Tully & Whelan 1993 Gargan et al. 2003, 2006b, Poole et al. 2006).

Since 1974, the sea trout rod catch has been monitored for 18 west coast fisheries in the Connemara district (Fig. 4). The data show a decline during 1987–1988, from ~10 000 fish caught every year in the 1970s and early 1980s, to only 240 fish caught in 1990 (Whelan & Poole 1996, Gargan et al. 2006a) (Fig. 4). Sampling of sea trout in estuaries was initiated in the Irish mid-west in 1990, and sea trout post-smolts were recorded in all rivers with high levels of predominantly juvenile salmon lice stages (Tully et al. 1993b). This documented decline in sea trout rod catch coincided with the development of salmon aquaculture in western Ireland during the mid-1980s, and has been linked to salmon lice infestation on sea trout (Tully & Whelan 1993, Tully et al. 1999, Gargan et al. 2003). However, in determining whether any reduction in rod catch is reflective of an overall reduction in sea trout stock size, it is important to consider catch per unit effort (CPUE) for the fishery. In this context, the ‘catch and release’ by-law introduced in western Ireland in 1990 may have affected angling effort for some fisheries. Based on analysis of sea trout rod catch and effort data (CPUE), Gargan et al. (2006b) found that the sea trout catch decline recorded between 1988 and 1990 was not related to reduced angling effort, but that a marked reduction in CPUE had indeed occurred.

Following a decline in sea trout stocks in 2 Irish fisheries, Gargan et al. (2006b) recorded that substantial sea trout smolt runs continued for a number of years despite the very small numbers of adult trout returning from the sea. Trend analysis indicated a reduction in sea trout smolt output from both fisheries over the study period, which suggested that although freshwater resident trout contribute significantly to sea trout smolt runs, a reduction in smolt output can be expected after a relatively short period of very poor marine survival. If the individuals that adopted the anadromous strategy had very low marine survival, there would be selection in favour of those with higher genetic propensity for freshwater residence. The declining numbers of smolts produced by the freshwater stock therefore could be explained by such selection against the anadromous life history strategy within a population (Gargan et al. 2006b).

Population effects in Scotland

In Scotland, during the late 1980s, unprecedented declines in sea trout rod fisheries were recorded throughout the west coast region (Walker 1994, Northcott & Walker 1996). Butler & Walker (2006) reported a collapse and a marked shift in population structure of the River Ewe rod-caught sea trout beginning in 1988, linked to salmon lice epizootics following the establishment of salmon farms near the river mouth in the marine embayment of Loch Ewe. Between 1980 and the period 1997–2001, maximum sea age fell from 11 to 5 yr and marine growth rates declined. Butler (2002) further estimated that farmed salmon was probably the primary source of salmon lice (78–97% of parasites) on wild salmon and sea trout populations, and that aqua-
culture facilities comprised the major source of lice to emigrating smolts in springtime on the west coast of Scotland. Taken together, the changes in the River Ewe stock structure could be related to declines in marine growth and survival, which were deduced to have been at least partly attributable to salmon lice epizootics emanating from salmon farms in the adjacent coastal waters (Butler & Walker 2006). This contention was supported by Walker et al. (2006) in comparing contemporaneous catch data for east coast Scotland sea trout stocks. The east coast of Scotland has been essentially free of commercial salmon farming throughout the history of the industry, and sea trout stock structure there remained stable over the same period that the west coast collapses were reported. Further corroborative reports of contemporaneous collapses in other, smaller, sea trout fisheries in the west of Scotland include those for rivers draining into Loch Torridon (McKibben & Hay 2004).

Notwithstanding the clear contrasts in these sea trout stock assessments for east (non-farmed) versus west (farmed) coasts regions, it has to be acknowledged also that the presence or absence of salmon farming is not the only difference between these coastlines. Ideally, comparisons would be drawn between areas or rivers in farmed and non-farmed regions within the Scottish west coast itself, but the development history and extent of the industry is such that suitably large non-farmed, or ‘non-impacted’ areas are not present. Furthermore, even drawing comparisons among specific sea lochs within western Scotland is fraught with difficulty because of the problem of pseudoreplication—no two sea lochs are identical in terms of their size, depth or hydrography. The absence of extensive areas of western Scotland without salmon farming, and which might be designated as ‘controls’ for experimental comparison with salmon farm ‘impacted’ areas, has proven to be a major obstacle to scientists investigating the likely impacts of salmon farming on adjacent wild stocks of sea trout and salmon. The Scottish Salmon Producers Organisation does provide publicly available summary statistics on their website (www.scottishsalmon.co.uk/fish-health-management-report-january-to-march-2015) for the monthly average abundance of adult female parasites on farmed salmon stocks in Scotland. Whilst these summary data can be informative of the overall status of sea lice abundances on farmed stocks, they are summarised by geographic area for 30 regions and lack resolution. Furthermore, the lack of access for scientists to detailed data (e.g. lice levels, and number and size of fish held in particular fish farms) hampers analyses of the likely impacts of salmon farming on local wild sea trout and Atlantic salmon stocks.

Whilst no marine survival data exist for Scottish west coast rivers prior to the sea trout collapse in the late 1980s, low smolt-to-finnock marine survival rates of 0.8–8.1% and 1.0–4.6% were also recorded for the rivers Tournaid and Shieldaig, respectively, over the period 1999–2001 (Butler & Walker 2006) and have been related to salmon lice infestation. Butler & Walker (2006) noted an increase in the abundance of resident (non-anadromous) trout following the sea trout stock collapse in the River Ewe system in western Scotland. Given the reductions in egg deposition resulting from the collapse in adult sea trout abundances, it is possible that lack of competition, and related improvements in freshwater growth rates, might lead to a greater prevalence of freshwater-resident trout in some impacted populations (Butler & Walker 2006).

Population effects in Norway

Sea trout from the majority of sampled sites along the Norwegian coast from Hordaland to Finnmark had salmon lice levels that indicated moderate or high mortality in 2011–2013 (data from the national monitoring programme) (Taranger et al. 2015). The infection levels of salmon lice on anadromous brown trout in the central and outer regions of the intensively farmed Hardangerfjord are among the highest observed in Norway (Skaala et al. 2014b). From 2001 to 2011, all descending smolts and returning sea trout in River Gudalselva, in the central region of Hardangerfjord, were captured in traps, and the smolts were individually tagged (Skaala et al. 2014a). Samples of the emigrant smolt cohorts were treated with Substance EX to prevent early salmon lice infestation. The results show a very low marine survival rate of only 0.6–3.4% for tagged smolts, with the highest survival rates in years with the lowest registrations of farm salmon lice in springtime. The survival rates of Substance EX-treated smolts and controls were respectively 3.41% and 1.76%. Although both these levels of survival are low, they indicate the extent to which spawning abundances of adult sea trout may be reduced in local populations (i.e. in this case by almost one half).

Bjorn et al. (2001) quantified salmon lice levels on sea trout at 2 sites in northern Norway; one ‘exposed’ area subject to extensive salmon farming was compared with an ‘unexposed’ area with little
farming activity. At the exposed location, 47% of
the fish caught in freshwater and 32% of those
captured at sea carried salmon lice at intensities
above the level that has been shown to induce mor-
tality in laboratory experiments (Bjørn & Finstad
1997). Bjørn et al. (2001) concluded that excessive
mortality of the most heavily-infested post-smolts
most likely occurred in that study area and that
high salmon lice levels may therefore have pro-
found negative effects upon wild populations of sea
trout.

Genetic differences among sea trout populations
and effects of salmon lice

The effects of salmon lice on sea trout populations
may vary according to the genetic structure of a
target population. In this regard, Glover et al. (2001)
recorded a clear difference in susceptibility to sal-
mon lice between fish from a freshwater resident
brown trout population and an anadromous popula-
tion when exposed to salmon lice in a laboratory
experiment, as measured by their respective salmon
lice abundances. Subsequently, Glover et al. (2003)
reported significant differences in the abundance,
density, and development rate of salmon lice among
3 sea trout populations in southwest Norway. Their
results suggest that the observed differences in sal-
mon lice level among the 3 sea trout populations
reflect host genetic differences. Also in Atlantic
salmon, differences in infection level are observed
among stocks, which may reflect genetic differences
in their susceptibility to sea lice infestation (Glover
et al. 2004).

Coughlan et al. (2006) sampled DNA from scales
of sea trout in the Burrishoole River, in the west of
Ireland, before and at intervals during aquaculture
activities. Amongst these samples, allelic variation at
a microsatellite marker (Satr-UBA), tightly linked to
a locus critical to immune response, was compared
with variation at 6 neutral microsatellite loci. No sub-
stantial evidence of the variability of a genetic signal
for the immune response genes was observed at neu-
tral microsatellite loci. A significant decline in allelic
richness and gene diversity at the Satr-UBA marker
locus, which preceded a severe sea trout stock col-
lapse, does however appear to be associated with
aquaculture activities. These data therefore suggest
that salmon farming-mediated disease can indirectly
affect the genetic structure of sympatric sea trout
populations by reducing variability at major histo-
compatibility genes.

Population-reducing effects on Atlantic salmon:
relevance to sea trout

Experimental studies have been conducted on the
mortality of salmon lice on Atlantic salmon post-
smolts, comparing fish chemically treated to provide
protection from salmon lice with control groups of
untreated fish. These field studies have been con-
ducted with the presumption that salmon lice origin-
ating from local farm sources might confer in-
creased mortality risk to the untreated control smolts,
and that this effect will extend to the wild Atlantic
salmon smolt population.

All these studies have found greater return rates of
treated salmon smolts, but not in every location or in
each year. The estimated average risk ratio of pro-
tected fish returning to their natal rivers to spawn
compared to unprotected fish ranged from an average
1.14:1 to 1.41:1 (Jackson et al. 2011a,b, 2013,
2014, Gargan et al. 2012, Krkošek et al. 2013, 2014,
Skilbrei et al. 2013, Vollset et al. 2014). Within any
given release group, a risk ratio of 1.14–1.41:1
reflects that 12–29% fewer unprotected than pro-
tected fish ultimately are recaptured as adults. Skil-
br ei et al. (2013) also showed that grilse were 100
grams heavier when treated, suggesting that a pro-
portion of the surviving individuals were infested
with sublethal levels of salmon lice. The most recent
study on releases of treated and untreated salmon
smolts (Vollset et al. 2014) concluded that salmon lice
effects may increase the sea age of returning salmon,
either by influencing their age at maturity or by
disproportionately increasing mortality amongst
those fish that mature early.

These variations in survival estimates may reflect
both the variation in treatment efficacy and the vari-
ation in actual exposure of the released fish to salmon
lice (Skilbrei & Wennevik 2006, Gargan et al. 2012).
Because the effect of such treatments is only tempo-
rary for the first few weeks of the marine migration,
and the acquired dose of the active component will
vary among individuals, it is likely that mortality for
treated fish underestimates the impact of salmon lice.
We should, therefore, be cautious in extrapolating
data from single studies to a population level. None-
theless, comprehensive meta-analyses, long-term
studies, and similar results from an increasing num-
ber of experimental studies, support that mortalities
caused by salmon lice in farm-intensive areas can be
expected to result in 12–29% fewer returning
Atlantic salmon adult spawners.

Atlantic salmon post-smolts migrate through farm-
intensive areas in near-coastal areas only in spring-
time, and perhaps are present there for only a few
days or weeks en route to ocean feeding grounds
(Thorstad et al. 2011, 2012). The salmon louse-
induced mortality impacts from studies of Atlantic
salmon should therefore be regarded as minimum
estimates for sea trout mortality, if protected and un-
protected groups of sea trout were to be compared.
Sea trout normally remain for extended periods
(weeks, months or sometimes even a year or more) in
near-coastal areas. If those coastal areas are charac-
terised by high salmon lice levels, sea trout post-
smolts are likely to be more affected by salmon lice
than are Atlantic salmon. Sea trout typically migrate
downstream and enter the sea for the first time as
smolts in spring or early summer, and may return to
freshwater in the autumn, following a few months
at sea (Fahy 1978, Gargan et al. 2006a, Jonsson &
Jonsson 2009). However, sea trout need not return to
freshwater after their first summer at sea, but can
remain continuously at sea during the summer and
winter until they mature and return to freshwater for
spawning the following year, or even several years
later (Fahy 1978, Jonsson & Jonsson 2009, Skaala et
al. 2014a). Since sea trout remain in coastal areas
later in the spring and summer months than Atlantic
salmon, they are exposed to seasonally higher risks
of salmon lice infestation. Finally, sea trout can re-
main at sea for longer periods than the period of
short-term protection provided by the chemical treat-
ment. Accordingly, results from studies applying these
kinds of experimental methods to sea trout (e.g. Skaala
et al. 2014a) are most likely to be underestimates of
the potential for salmon lice-induced mortality.

Knowledge gaps and research needs

The effect of salmon lice on sea trout is a well-
studied subject, with a large number of published
studies available, as shown in this review. The effects
of salmon lice on individual sea trout are relatively
well documented through both laboratory and field
studies. The most important knowledge gaps are re-
lated to salmon lice impacts at the population level
and in quantifying the reduction in wild sea trout pop-
ulations arising from increased mortality and reduced
growth attributable to salmon lice. The effects of
salmon lice on life history traits, especially of sea trout
population age structure and size at maturation, and
selection against anadromous behaviour in favour of
permanent freshwater residence also are not well un-
derstood. For robust and informed evaluation of the
effects of salmon lice on sea trout populations, field
experiments comparing survival and growth of fish
released to the environment following prophylactic
treatment against salmon lice should be undertaken.
More information also is needed on how salmon lice
planktonic larval stages may spread and be dispersed
in coastal areas, and on the primary environmental
factors that ultimately determine the resultant salmon
lice levels on wild sea trout in a given area.

Wild sea trout populations have generally been
poorly studied, monitored and mapped, although
there is variation in this respect among catchments,
regions and countries. With specific regard to the
marine environment, the behaviour, migration routes
and survival of sea trout are less well understood than
for many other salmonid species. Such information is
essential when interpreting salmon lice monitoring
data on farmed and wild fish, in evaluating the likely
efficacy of any adopted mitigation measures and in
facilitating the formulation of appropriate and relevant
scientific advice on possible mitigation measures.

Overall conclusions

The studies reviewed demonstrate that salmon
farming increases the abundance of salmon lice in
the marine habitat and there is extensive published
evidence that salmon lice in intensively farmed areas
have negatively impacted wild sea trout populations.
The effects of salmon lice on sea trout include in-
creased marine mortality, changes in migratory be-
haviour, reduction of marine growth of individual
fish, and reduced population sizes. These conclusions
are based on:

(1) Studies of salmon lice impacts on individual
sea trout in laboratory and field studies documenting
host tissue damage, osmoregulatory dysfunction and
other physiological stress responses, reduced growth,
and increased susceptibility to secondary microbial
infections and reduced disease resistance;

(2) Documentation of premature return to fresh-
water of sea trout carrying high levels of salmon
lice. Premature return may facilitate individual sur-
vival and recovery from infestation in the short term,
but does compromise growth potential, and thereby
future fecundity, as well as impairing the immune
defence system;

(3) Catch statistics and routine population monitor-
ing utilizing in-river traps that have indicated changes
in population abundance, age structure and altered
life history characteristics in association with the
onset and development of salmon farming in the
adjacent environment;
(4) Monitoring of salmon lice levels on wild fish in relation to spatiotemporal variation in salmon farming intensity and biomass production.

(5) Indications of population-level effects on sea trout derived from monitoring of salmon lice levels on wild fish in relation to experimentally determined threshold levels known to induce physiological compromise and mortality of individual fish.

Because the brown trout is a partially migrating species, reduced marine growth and increased marine mortality will reduce the benefit of marine migrations for individuals in anadromous populations. Potentially, this could result in the loss of anadromous sea trout populations, and the possibility for anadromy is crucial in catchments with environmental conditions unsuitable for brown trout during some periods of the year. Large rivers and catchments with suitable year-round conditions may not be subject to a risk of total loss of brown trout, but a severe reduction in the incidence of the anadromous life-history strategy may result in altered genetic composition of a trout population, the establishment of populations characterised by freshwater residency, and perhaps reduced overall genetic diversity with less variable life-history characteristics. The loss of the enhanced growth opportunities offered by the marine environment may also lead to a lower local abundance of brown trout, altered life-history traits, lowered recruitment and loss of the large veteran migrants popular among fishers. To sustain and enhance sea trout populations, and to ensure a harvestable surplus for fisheries, salmon lice levels need to be reduced in many farm-intensive areas compared to present levels.

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