

Wild Salmon Mortality Caused by Fish-Farm Sea Lice

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It is well established that fish farms raise sea lice levels^{1,2}, and that sea lice can kill juvenile salmon³. However, until now, the impact of sea lice from fish farms on wild salmon has remained unresolved. New research⁴ just published in the *Proceedings of the National Academy of Sciences of the USA* has estimated the total salmon mortality caused by farm-origin sea lice. This bulletin summarizes those research results.

Research Summary

The peer-reviewed paper investigates the impact of sea lice infections on wild salmon caused by fish farms. The study shows that fish farms are changing the way wild salmon get infected with parasites. As a result, up to 95 per cent of wild juvenile pink and chum salmon are dying from sea lice infections in coastal British Columbia.

How do fish farms change sea lice transmission?

Sea lice are natural parasites of salmon. The primary sea lice hosts are adult salmon. Normally, adults are far offshore when juvenile salmon are migrating out to sea and so juveniles rarely encounter lice. However, fish farms put adult salmon in net pens along juvenile salmon migration routes. The result is a cloud of sea lice, more than 80 km long, through which the juveniles must migrate.

How do sea lice kill juvenile salmon?

Sea lice attach to the outside of a fish and feed on mucus, skin, muscle, and blood. Adult salmon can resist infection because they are large and are well armored with scales. However, juvenile salmon are only 1-2 inches long and don't have protective scales. The lice cause open wounds on juvenile fish and can literally eat them alive. It takes only one or two sea lice to kill a juvenile pink or chum salmon.

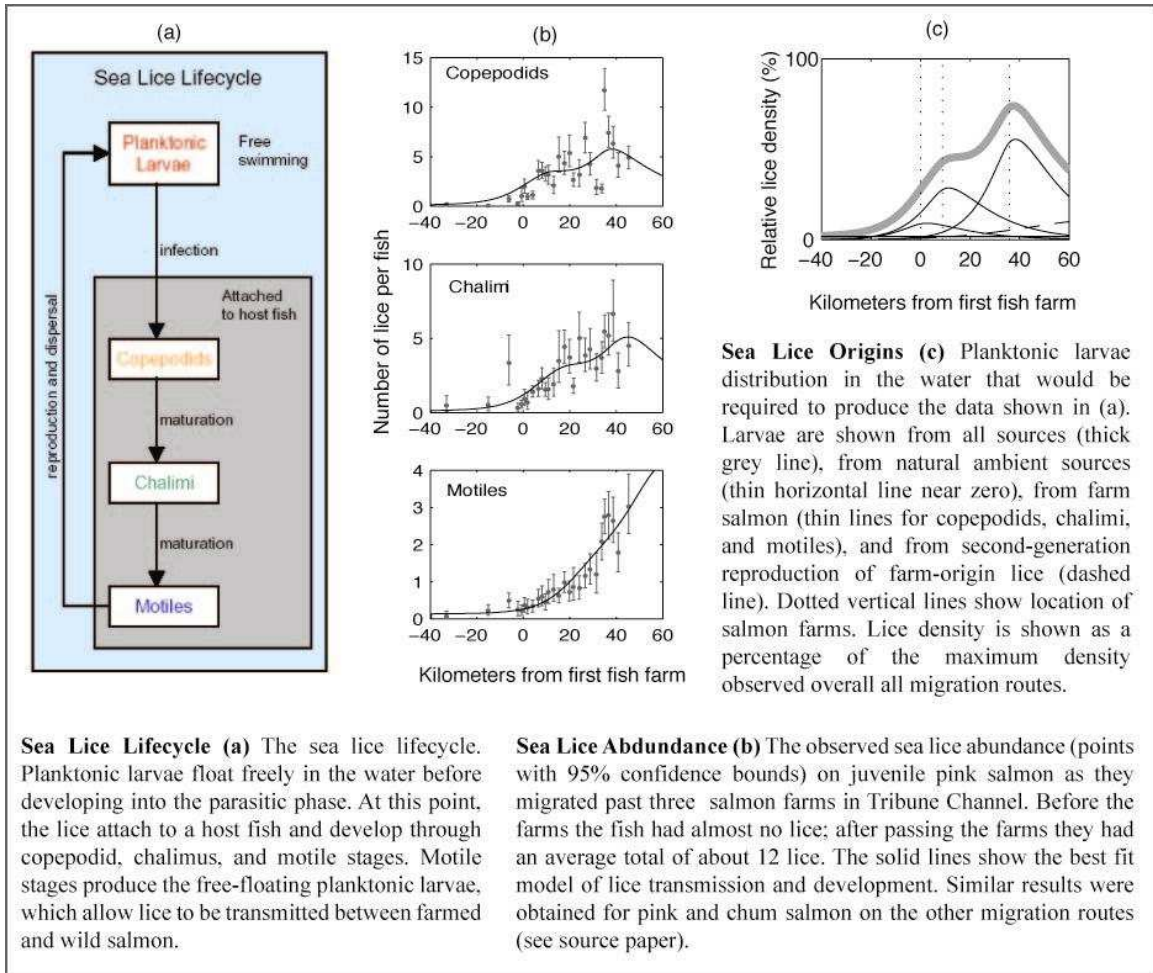
How many juvenile salmon are killed by sea lice?

The study found that an increasing number of juvenile salmon were killed over the migration season. About 9 per cent were killed in early spring when sea lice abundance was low. Later in the season when lice numbers increase, approximately 95 percent were killed. Normally, only a small fraction of juvenile salmon survive to return as adults. Sea lice infections from fish farms are placing a significant additional stress on that small fraction.

Research Approach

The research team consisted of biologists and mathematicians studying sea lice (*Lepeophtheirus salmonis*) on pink (*Oncorhynchus gorbuscha*) and chum (*O. keta*) salmon in coastal British Columbia. The study had three components. First, they surveyed more than 14,000 fish along three migration routes (See Box 1). Using data and

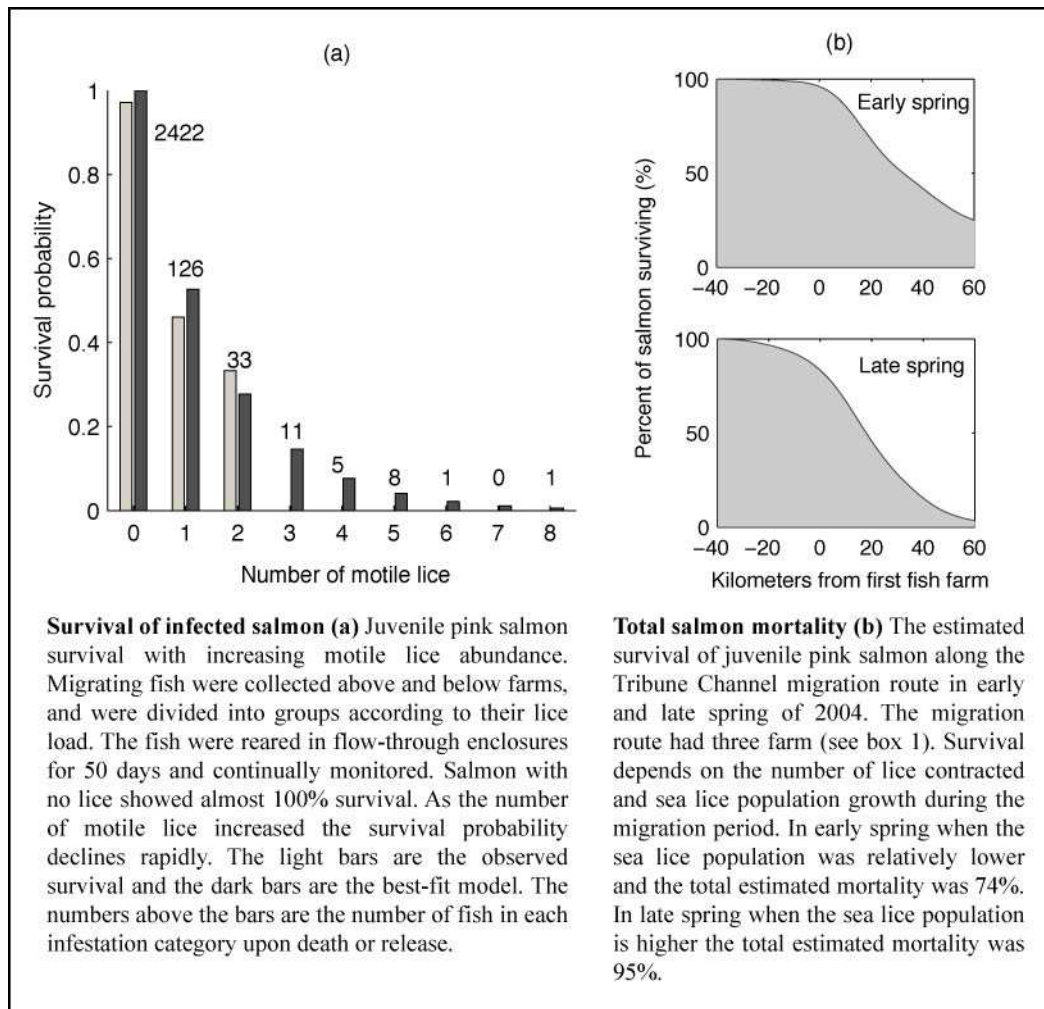
mathematical modeling, they determined how sea lice infections increased as the salmon migrated past fish farms (See Box 2). Second, they conducted experiments with more than 3000 fish to determine the mortality caused by a range of sea lice infections. They found that a single louse can kill a juvenile salmon, and that the odds of dying rapidly reach 100% as infection exceeded two lice (See Box 3). Finally, they stitched the first two components together using mathematical models to estimate the overall mortality of wild salmon caused by sea lice from fish farms (See Box 3).



Box 1. The sea lice lifecycle (a), lice abundances on juvenile salmon migrating past salmon farms (b), and the origins of sea lice (c).

Fish farms increase sea lice infections

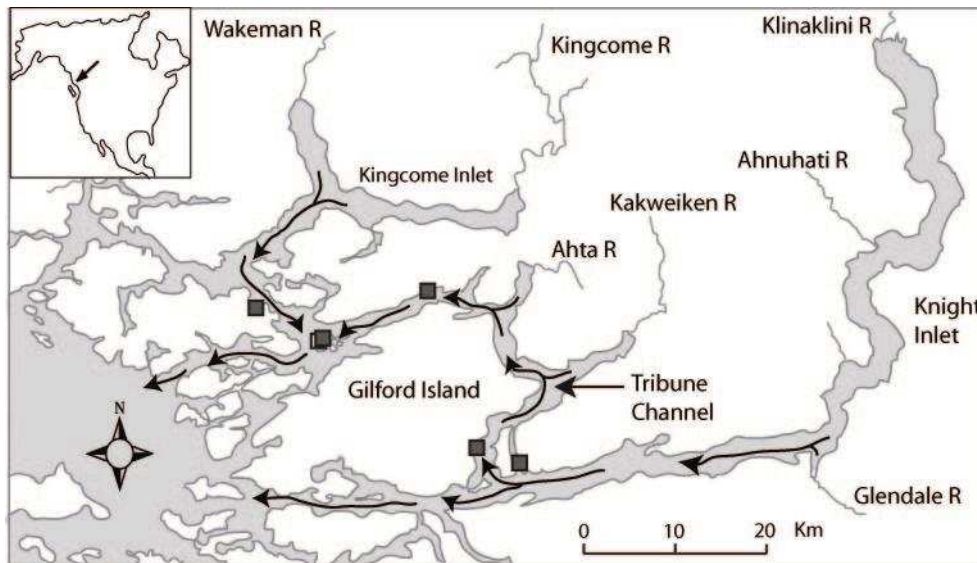
The fish surveys showed that wild juvenile salmon carry a low level of lice before they reach the fish farms, and a much higher level once they pass the farms. (See Box 2.) As the fish continue out to sea, their lice reproduce and create a second generation of parasites that further increases the infection levels. Mathematical modeling shows that farms were the primary source of lice, raising infection for at least 80 km of the salmon's migration route. This means that salmon experience increased infection for the first 2.5 months of their marine life, when they are only 1-2 inches long and lack protective scales. That is, farms infect salmon when they are most vulnerable.



Box 2. Survival of juvenile salmon infected with sea lice (a) and the impact of salmon farms on the survival of juvenile salmon populations (b).

Sea lice infections kill juvenile salmon

The survival experiments showed that almost all the uninfected juvenile salmon survived (See Box 3). As infection increased, salmon survival decreased. A mathematical model was created to predict the survival of fish depending on their infection intensity. To predict the overall salmon mortality caused by fish farms, the survival model was combined with the infection model to estimate juvenile salmon mortality caused by fish farms. The total mortality in migrating juveniles attributed to farm-origin lice ranges from 9-95%. This range encompasses major seasonal variation in sea lice abundance, minor differences between pink and chum salmon, and spatial variation among migration routes.



Implications for Coastal Ecosystems and Economies

As ocean fisheries continue to decline aquaculture has expanded rapidly to meet the global demand for fish⁵. But aquaculture may not help wild fish. This study shows that large scale salmon farming can have a dramatic negative impact on wild salmon. Fish farms can therefore threaten the survival of wild salmon stocks already reduced by other human activities such as fishing and logging. It raises the question of whether aquaculture, as it is currently practiced, can coexist sustainably with natural salmon populations. It also raises the concern that these problems may be repeated in other species and regions as aquaculture continues its rapid growth. Aquaculture challenges the sustainability of coastal ecosystems and economies because coastal ecosystems and economies depend on healthy fish stocks.

Research Published in Peer Reviewed Journal

The paper summarized here, *Epizootics of wild fish induced by farm fish*, was published in the peer-reviewed scientific journal the *Proceedings of the National Academy of Sciences of the United States of America*, Oct 4, 2006. It was authored by Krkosek, M., Lewis, M. A., Morton, A., Frazer, L. N., and Volpe, J. P.

Acknowledgements

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