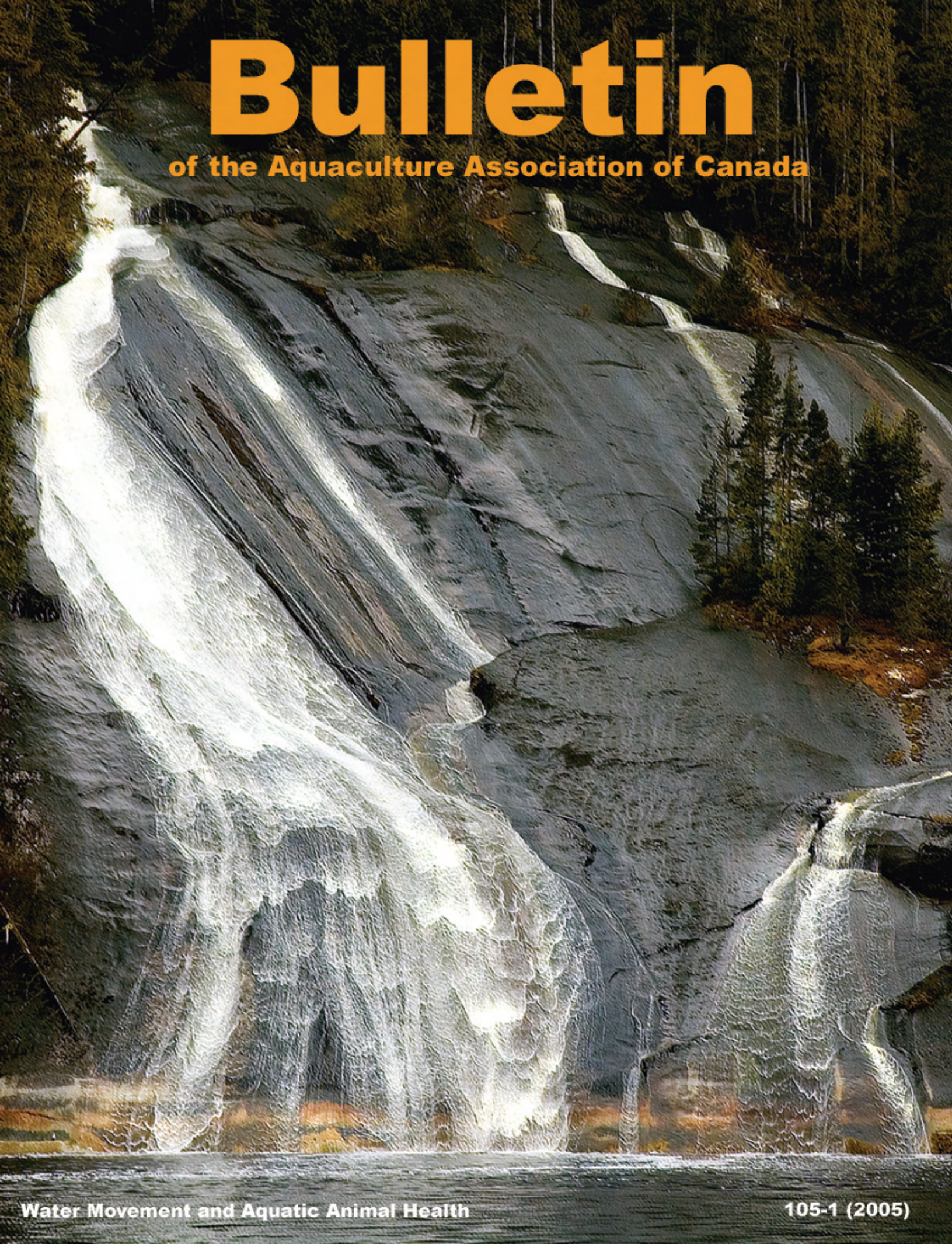
Bulletin

of the Aquaculture Association of Canada



Water Movement and Aquatic Animal Health

105-1 (2005)

St. Andrews Biological Station

Proceedings of the Fourth St. Andrews Aquaculture Workshop

Water Movement and Aquatic Animal Health

21-22 November 2005, St. Andrews, NB

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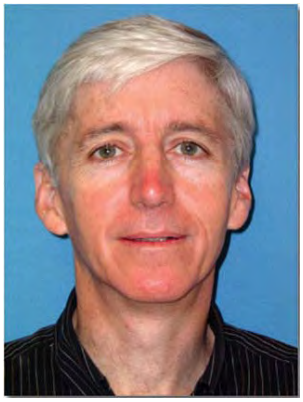
Cover: Waterfall at Glacier Bay, Tribune Channel, BC. A mainland inlet not far from many BC salmon farms. Photo by Bill Pennell, Malaspina University-College, Nanaimo, BC.

Introduction

Water Movement and Aquatic Animal Health Workshop



R. L. Stephenson

It is my pleasure as Director of the St. Andrews Biological Station (SABS) to provide the introductory comments for these proceedings of the Workshop on Water Movement and Aquatic Animal Health which was held 21-22 November 2005 at the historic Fairmont Algonquin Hotel in St. Andrews, New Brunswick.

Management of animal health is one of the most important issues facing the aquaculture industry. The role of water movement in relation to aquatic animal health—while intuitively of great importance—is a complex topic that requires a range of expertise and the collaboration of experts in oceanography, aquaculture, epidemiology, and fish health.

The goal of the workshop was to bring together interested parties from across Canada and other countries to discuss the management of aquatic animal health using oceanographic tools and expertise. It was intended to help regulatory authorities and industry stakeholders better define and manage animal health across the diverse aquatic environments that support Canada's wild fisheries and aquaculture industries.

The concept of the workshop developed from a SABS research project that examined oceanographic influences on the spread of infectious salmon anemia (ISA) among salmon farms in the southwestern part of the Bay of Fundy. The project was led by Dr. Fred Page and funded by the DFO Aquaculture Collaborative Research and Development Program (ACRDP), DFO Science, and the aquaculture industry. Because the outputs from this project were beginning to be used by managers, it was felt that a workshop should be held to compare local experience with that of researchers and managers with similar experience in other geographic areas and on other diseases.

Opening session

Dr. Christine Power, of the Canadian Food Inspection Agency, opened the workshop with a presentation on an investigation into an out-

WMAAH Workshop Committee

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break of avian influenza in British Columbia. The spread of avian flu by wind-borne transmission of the virus in dust shows many similarities to the spread of aquatic animal disease through water.

The remainder of the workshop focused on case studies of managing open-water disease outbreaks in North America and Europe. Experts who had modelled aquatic animal disease transmission using oceanographic tools, along with managers responsible for developing guidelines, policies, and regulations related to infectious disease control, were invited to speak at the workshop. The presentations examined the influences of water movement on transmission, pathogenicity, and success of control measures, in both finfish and shellfish. The material presented encompassed west coast, east coast, and freshwater disease models for North America, as well as trans-boundary disease management experience. In addition, presentations were given by experts familiar with similar problems in the aquaculture industry in Norway and Scotland.

The workshop concluded with discussion sessions on the incorporation of water movement science into aquatic management tools and plans, and on the information gaps that still exist and the research needed to address these issues. Summaries of the discussions are included in these proceedings.

This workshop was the fourth in a series of aquaculture workshops held in St. Andrews since 2002. These St. Andrews Aquaculture Workshops are a collaboration between the St. Andrews Biological Station and the Aquaculture Association of Canada (AAC). For each workshop, SABS develops the scientific program, and obtains financial support, while AAC provides organizational support and publishes the proceedings. The topics of the previous workshops were haddock culture (2002), control of grilsing in salmon (2003), and biotechnology (2004). The Fifth St. Andrews Aquaculture Workshop—Use of Exotic Species in Aquaculture—will be held 11–13 October 2006.

Workshop participants at the lobster dinner held at Anderson House on the campus of the Huntsman Marine Science Centre.

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Financial support for the WMAAH Workshop and this proceedings was provided by:

National Aquatic Animal Health Program (NAAHP)

DFO Science Branch (Ottawa)

New Brunswick Salmon Growers' Association

An Investigation into the Potential Role of Aerosol Dispersion of Dust from Poultry Barns as a Mode of Disease Transmission during an Outbreak of Avian Influenza (H7:N3) in Abbotsford, BC in 2004

Christine A. Power

During the winter and spring of 2004, an avian influenza outbreak occurred in the Lower Fraser Valley of British Columbia. In the early stages of the outbreak, the infected premises were fairly close together (within 2 to 3 km of one another) and downwind from the prevailing NE winds on the Matsqui Prairie at that time of year. While the scientific literature does not define wind movement as a principle source of avian influenza virus transfer, discussions with scientific leaders in the field of avian influenza from Italy, the Netherlands, and the United Kingdom in April 2004 revealed markedly divergent opinions from "highly sceptical" to "a considerable factor to deal with". Clearly revealed was the absence of any testing to support or refute a windborne theory of transmission during recent outbreaks in Italy and the Netherlands. A preliminary assessment of the likelihood of wind playing a role in transmission involved the use of hourly weather data provided by Environment Canada and BC Water Land and Air Protection. This assessment was followed by a more analytic approach provided by the mathematical plume models of the Meteorological Service of Canada's Emergency Response Division. A field study was conducted in early April to evaluate if avian influenza virus was being spread into surrounding areas on dust particles emitted from barns containing infected birds. Air sampling conducted inside an infected barn revealed a high quantitative estimate of viral load per cubic meter of air of 292 $TCID_{50}$. Outdoor samples using low volume air samplers were all negative for the presence of virus. High volume air sampling conducted outdoors confirmed that avian influenza virus was circulating in the air outside barns during the outbreak, yet it remained unclear if the virus was alive and therefore potentially infectious. Current and future plans to further understanding of aerosol dispersion involve collaborations with academia and other government departments. A planned field study will aim to validate models for predicting the movement of generic biological agents from livestock barns into the surrounding environment.

Background of the Outbreak

During the winter and spring of 2004, an avian influenza outbreak occurred in

the Lower Fraser Valley of British Columbia (BC). Over a 3-month period, approximately 13.6 million commercial poultry and 18 thousand backyard birds were destroyed as part of disease control measures implemented by the Canadian Food Inspection Agency (CFIA). Most of the commercial poultry were broilers from uninfected flocks that went directly to slaughter at maturity to be used for human consumption. While 42 commercial operations were found infected, constituting 5% of the operations in the Valley, a wider cull of 410 non-infected poultry flocks took place which affected more than half the producers in the region. The economic impact of this outbreak on the livelihoods of BC poultry producers and the associated support industries was severe and recovery is expected to be protracted. Fortunately, the avian influenza subtype causing disease in the region had minimal effects on persons living in the area or those working with infected poultry. Only two confirmed cases of mild conjunctivitis were reported in disease control workers directly in contact with infected birds over the outbreak timespan.[1,2]

Avian influenza is a contagious viral infection caused by the influenza virus Type "A", which can affect most species of food-producing poultry (chickens, turkeys, quail, guinea fowl, ostriches, emus, ducks, geese, and pheasants), as well as pet birds and wild birds. Avian influenza viruses can be classified as low pathogenicity (LPAI) or high pathogenicity (HPAI) according to the severity of the illness caused in birds. LPAI strains are much more common than HPAI strains in bird populations and typically cause less severe illness and on occasion no clinical signs in infected birds. However, some LPAI strains are capable of mutating into HPAI strains which leads to a severe form of the disease with high mortality. There are many influenza subtypes, two of which include H5 and H7. Historically, only the H5 and H7 subtypes are known to have become highly pathogenic in avian species.[2]

On February 9, 2004 on the northeast corner of the Matsqui Prairie, British Columbia, a broiler breeder producer noticed a mild drop in egg production and feed consumption, and a slight increase in mortality, in a 52-week-old flock of 9200 birds. The farm's veterinarian and the feed company representative investigated the case and samples were submitted to the British Columbia Ministry of Agriculture, Food and Fisheries (BCMAFF) diagnostic laboratory for routine postmortem. Pathogenic findings included unusually firm lungs and inflamed tracheas. The clinical illness appeared to resolve over subsequent days.[1,3]

A diagnosis of avian influenza was made by BCMAFF's poultry pathologist on February 16, 2004. Within a few days, the subtype of the virus was identified as H7N3 by the National Centre for Foreign Animal Disease (NCFAD). This disease event, though mild in impact on the flock, is believed to be the origin or starting point of the HPAI outbreak. This farm is referred to as the "index premises".[1,3]

On February 17, 2004 an adjacent barn on the index premises containing a younger flock of 9030 birds (24 weeks of age) began to show an alarming rise in mortality. On February 19, 1500 birds were found dead. Infection of this second flock with the mutated strain of the avian influenza virus constituted the beginning of the HPAI outbreak event. Three weeks later a second premises 1.6 km away showed signs of infection, then one week later three more premises (2 to 3 km) south and west became infected. Two weeks later 11 additional commercial premises (less than 1 km from one another) were identified as infected and in this way the epidemic began slowly and picked up speed as more flocks became infected.[2]

By the time the outbreak was brought under control, infected farms appeared in three clusters, each of which had a diameter of 5 to 6 km. In a few cases outlying farms were positive on the screening test but flocks did not show clinical signs or appear to contribute to local spread.

Investigation Approach

During the outbreak of high pathogenicity avian influenza (H7N3) in the Lower Fraser Valley, the British Columbia Emergency Operations Centre (BCEOC) established an epidemiology team. A key role of this team was to investigate the sources of the virus on the Matsqui Prairie and the multiple, potential modes of transmission of the virus from farm to farm. This was accomplished through the contributions of many professionals serving on the team and from other federal and provincial departments. We received more than 15 fully referenced reports on key subjects of concern, each carefully written during the outbreak. These reports were used to steer a course through the crisis by providing answers to immediate questions. As well, they provided the knowledge and information required to ensure we were gathering the right data for the final epidemiological analysis of the avian influenza outbreak events.

From early on, the spread of the HPAI virus in the Abbotsford area was investigated from many points of view in order to establish the most probable means of disease spread during the outbreak event. The potential roles of wild birds, ground water, surface water, wind-borne particles, bio-security gaps (inter-farm movement of people and equipment), hatcheries, feed and feed mills, farm service personnel and CFIA staff in their eradication efforts were evaluated by the epidemiology team during the course of the outbreak. While this article focuses on the aerosol aspects of the investigation, a complete report entitled: "The Source and Means of Spread of the Avian Influenza Virus in the Lower Fraser Valley of British Columbia During an Outbreak in the Winter of 2004" is available to the public on the Agency website.[2]

In the early stages of the outbreak, the infected premises were found to be fairly close together (within 2 to 3 km of one another) and down wind from the prevailing NE winds on the Matsqui Prairie at that time of year. While the scientific literature does not define wind movement as a principle source of avian influenza virus transfer, discussions with scientific leaders in the field of avian influenza from Italy, the Netherlands and United Kingdom in April 2004 revealed markedly divergent opinions from "highly sceptical" to "a considerable factor to deal with". Clearly revealed was the absence of any testing to support or refute a windborne theory of transmission during recent outbreaks in Italy and the Netherlands.

Background on Poultry Barn Emissions

Poultry barn emissions in the Lower Fraser Valley have long been a focus of interest by Air Quality Meteorologists in the BC Ministry of Water, Land and Air Protection (BCWLAP) due to concerns over their contribution to summertime air pollution. A consultation with Ministry staff in April proved very informative as they answered many questions concerning the amount of dust emitted from poultry barns and the potential for windborne dispersion. Briefly, an aerosol consists of solid or liquid particles suspended in air.[4] Dust, smoke and fog are examples of aerosols. Dust emissions can be broken down into two basic fractions: visible particles larger than 10 μm in diameter which settle out by gravity, and invisible particles less that 10 μm which are light enough to be suspended in air for long periods.

In the Lower Fraser Valley, many poultry farms use sawdust, a readily available by-product of the lumber industry, as litter for their flocks. Poultry feed, faecal material from birds, along with feathers and dander, also contribute to the dust emissions from barns.

Contained in the BCWLAP report were the results of a study conducted by the

Sustainable Poultry Farming Group in which aerosol emissions from an Aldergrove, BC broiler operation were measured over a full growth cycle. While anyone observing dust emissions from a barn with dimensions of 40 x 400 ft (12 x 122 m) with 20,000 birds bedded on sawdust would agree they are substantial, of particular interest is the invisible portion which can be suspended in air and transported by wind. This fraction constitutes a particle size of less than 10 μm in diameter and is invisible to the naked eye. Emissions of this size range were measured from a 24-in fan over 7 wk revealing output of 25 to 40 g/m^3 per 24-hour period. To illustrate the magnitude, this represents a million-fold elevated concentration of aerosolized dust near a poultry barn fan as compared to outdoor air in a semi-rural area such as Aldergrove. Of all measured total particulate matter (visible and invisible) emitted, 40% was in the invisible size fraction of less that 10 μm in diameter. This indicates that a sizable portion of dust emissions from poultry barns have the potential to remain suspended in the air for up to several days. Given that springtime winds in the Fraser Valley often range between 5 to 10 km/h, poultry barn dust could possibly be found tens of kilometers from their source.[4]

Little is known about the survival of avian influenza viruses in dust particles. Published studies indicate that survival is best in dry air where the relative humidity is less than 50 to 70%. It has been suggested that other factors capable of significantly decreasing the survival of the avian influenza virus in aerosols include ultraviolet radiation, ozone reaction products, air ions, and pollutants while high air salinity found in coastal areas may provide a protective effect on virus survival.[5]

The avian influenza virus survives for many weeks in wet poultry manure at cool spring temperatures (4°C) and for up to 10 days at 25°C. The virus dies within a day or two in dry faeces. The concentration of virus shed in the faeces of infected poultry is very high. A gram of infected faeces can contain as many as ten billion infectious virus particles. Transmission of contaminated manure from an infected premises to a separate susceptible flock can occur through the movement of people, equipment, and vehicles. Barn to barn movement constitutes the highest risk activity for transfer, while deposition of contaminated manure in the vicinity of a susceptible flock is categorized as of somewhat lesser risk. It is thought that a small amount of contaminated dust adhering to boots, clothing or equipment is sufficient to transmit the virus from an infected barn to a susceptible flock.[6] Once infected, the flocks themselves are known to shed enough live virus into their localized environments to be considered as “virus factories”.

Initial Steps in the Investigation

In the early weeks of the outbreak, the CFIA tried several procedures of euthanasia and carcass disposal on the first two infected premises to establish a humane depopulation method and a safe way to dispose of thousands of infected carcasses.

Barns on the index premises underwent bird removal by hand transfer to a conveyor belt that transported the carcasses to an open door at one end of the barn. The carcasses were loaded into a grinder which in turn was emptied into a dump truck for localised transportation for composting. This process of moving carcasses outside the barn followed by grinding for composting was recognized for the potential it held to transmit infected feathers and dust into the wind and surrounding environment. Carcass disposal activities were confined within barns afterwards.

On Premises 2, the CFIA tried out an established euthanasia method provided by a BC poultry industry which, because of the size of the equipment, required that birds be transported outdoors for the process. This led to a significant amount of feather dispersion on the wind and into the surrounding environment. This

method was never repeated and it, too, was held up by government and industry as an unfortunate learning experience.

Did these events contribute to the spread of virus to other premises? This question was investigated through a qualitative risk assessment which evaluated the likelihood of transmission during the known hours of high risk activity on these farms, considering the wind direction and speed at those times, and the timing of infection on Premises 2, 3, 4, and 5. Initially, meteorological data were obtained from the Abbotsford airport hourly observations for this assessment. A consultation with BCWLAP led to additional meteorological data provided from two airports with the daily reports provided in schematics called "wind roses".[4]

The preliminary assessment using wind directions and speed during high risk activities on Premises 1 provided a moderate risk rating for windborne dispersion to Premises 2, a low risk rating for Premises 3, and a negligible risk rating for Premises 4 and 5. The risk of transmission from Premises 2 was estimated as negligible for Premises 3 and low for Premises 4 and 5.[7] This assessment was followed by a more analytic approach provided by the mathematical plume models of the Meteorological Service of Canada's Emergency Response Division. By calculating an estimate of dust (and virus) dispersion parameters from these barns, offering rough "guesstimates" of infectious dose in poultry (by Agency epidemiologists) to the Meteorological Service, the plausibility of airborne spread of virus from these two premises was estimated through modelling of airborne plumes. Similar estimates of risk of transmission were obtained.[8] There exists considerable lack of certainty about the inputs to the model which requires experimental work to clarify.

We concluded that these events may have contributed to the spread of disease. However, in addition to the dust dissemination associated with these events we began to recognize that the volume of exhausted dust from these infected barns may ultimately have been more influential in transmission. The continuous operation of fans required to ventilate birds and disposal crews over many days and nights leads to significantly greater emissions than what would be expected by outdoor activities alone.

As the outbreak progressed, evidence of what appeared to be windborne transmission reinforced the Agency's resolve to minimize any potential airborne transmission. The Agency's on-farm activities were directed by CFIA's HPAI disease control strategy such that once an infected flock was identified, efforts were made to destroy birds as quickly as possible to limit the amount of virus produced. During disposal activities, dead birds were collected indoors and sealed in boxes before being transported off the farm. Barn doors were kept closed as much as possible during the disposal process to prevent air currents from spreading the virus. In-barn composting of birds and litter was introduced by Agency research staff early in the outbreak and conducted thereafter by Agency operational staff. Wherever possible, composting took precedence over removal and incineration of birds.

A Field Study Aimed at Capturing Live Virus Around Infected Barns

In early April 2004, the epidemiological investigators decided to undertake a study to examine airborne spread of virus near infected barns. This study was conducted collaboratively with Defence Research and Development Canada-Suffield (DRDC) of the Department of National Defence (experts in sampling and detection of biological agents in aerosols), Health Canada providing the laboratory testing expertise, and Environment Canada's Meteorological Service which set up a mobile meteorological station locally that provided hourly wind

data to the field crew.

The goal of the study was to evaluate if avian influenza virus was being spread into surrounding areas on dust particles emitted from barns containing infected birds. Agency epidemiologists hypothesized that airborne transmission of the virus might be contributing to the rapid and extended spread of the outbreak.[9]

Air sampling near infected barns

The study assessed air samples collected adjacent to three infected premises using low volume air samplers. On each farm, air samples were collected every fifteen minutes for a 24-hour period both upwind and downwind from the ventilation fans of the barns. Of a total of 240 air samples collected from fixed locations, all were determined to be negative for the avian influenza virus.

Air sampling inside an infected barn

Live virus—virus capable of causing disease—was detected in both of the two samples collected by high volume air sampling inside an infected barn. A quantitative estimate of viral load per cubic meter of air was determined and found to be very high at 292 $TCID_{50}$ (dose lethal to 50% of susceptible tissue culture).

Air sampling in the surrounding area of infected barns

Nine air samples were collected within one kilometer of infected premises using high volume air sampling. Very low levels of virus were detected in one of these samples some 800 meters from an infected barn. Testing was unable to determine if this remote sample of virus was alive or dead.

Although this study confirmed that avian influenza virus was circulating in the air outside barns during the outbreak, it remained unclear if the virus was alive and therefore potentially infectious.

Additional Efforts to Further Study Aerosol Dispersion Using Plume Modelling

The plausibility of airborne spread of virus in the Lower Fraser Valley was the subject of preliminary modelling conducted during the outbreak.[8] In addition, for eight weeks during the outbreak, a mobile weather station dispatched from Vancouver (Environment Canada), was placed on a farm on the Matsqui Prairie to capture local meteorological data for generating retrospective predictions of airborne movement during the outbreak period. In-barn viral concentrations measured in air (by DRDC) and the time sequence of infected premises events in the Lower Fraser Valley collected by the Agency is being used in the analysis. An anticipated outcome of this work is clarification on the role of airborne dispersion in the early infections on the Matsqui Prairie (prior to March 19, 2004) and in the creation of the south and west clusters of infected premises that emerged in early to mid April. This study is being conducted in continued collaboration with Environment Canada's Environmental Emergency Response Division, whose expertise in predicting windborne plumes using complex mathematical models will form the foundation of this project. At the time of writing, this project is mid way towards completion with an expectation for windup by autumn 2006.

Key Questions from a Regulatory Disease Control Perspective

Agency disease control programs would benefit from having information to scientifically delineate a high risk zone around an infected premises for livestock

eradication purposes. With questions such as: What is the radius and concentration of dust emitted from a barn on the ground? What is the radius and concentration of dust emitted from a barn and carriage in the surrounding air? In short, what are the distance bands of probability for high, medium and low contamination and hence corresponding risk?

Agency disease control and zoning programs could also benefit from having environmental contamination predictions for the identification of low risk traffic routes for disposal of livestock. The core question for these purposes is: "What is a safe distance for a barn to be from the road to avoid aerosol dispersion from trucks carrying infected livestock?"

How can these questions be answered through the scientific process?

There are three broad areas of investigation required to make accurate geospatial predictions of risk for the avian influenza virus: range of environmental contamination, viability of the biological agent in the environment, and infectivity of the agent in the target animal species. Each of these dimensions require separate studies.

Environmental contamination. First, through combining expertise from the fields of aerosol physics, meteorology, agricultural engineering, environmental biology and epidemiology the following questions can be probed and clarified: What is the radius and concentration of dust (biological agents) emitted from a barn on the ground? What is the geographical radius and concentration of dust emitted from a barn and carriage in the surrounding air? What are the geographical distance bands of high, medium and low contamination and hence corresponding risk? What level of natural exposure to livestock is expected within these bands?

Agent viability in the environment. Second, little is known about the survival of avian influenza viruses in dust particles due to the limited experimental work conducted to date. The core fields of expertise suggested to address the accompanying questions would be veterinary virology, environmental biology, and aerosol physics. The questions include: What is the viability of the biological agent in dust particles and droplets of varying sizes (weights) under different environmental circumstances (soil, water, air) under varying conditions of temperature, humidity and sunlight.

Infectivity of the agent in the target species. To address the third body of questions requires expertise from the fields of veterinary virology and pathology. These questions revolve around: What is the infective dose and route(s) of transmission of the biological agent in the livestock species of interest? There is a paucity of information in the scientific literature for avian influenza but, nonetheless, it is very much needed for accurate risk predictions.

Overall, the plume modelling approach appears to offer a significant opportunity for advances in science-based decision making in the field of regulatory veterinary medicine.

Plans for a Collaborative Study to Address Localized Dispersion from Barns

A proposed project will address the first body of questions focusing on predicting geospatial dispersion of biological agents from barns and roadsides. Collaborators include the University of Victoria, Defence R and D Canada, Environment Canada – Emergency Response Division, Dycor Inc., and the CFIA.

Existing mathematical models for airborne and ground dispersion of dust from ventilation systems have a solid scientific basis in the field of particle physics and dispersion dynamics. Through the proposed field study, these models will be validated for predicting the movement of generic biological agents from livestock

barns into the surrounding environment. Exhaustion of known quantities of a selected biological agent *(Bti*) from the ventilation systems of barns will be followed by sampling of the nearby environment, including local barns and their livestock. The scientific information to be derived from this project should provide greatly improved estimates of zone size for controlling diseases of barn-housed livestock and assessing the safe distance for vehicle traffic from barns. In addition to validation of existing dispersion models, the development of the field sampling and detection techniques during the course of the field study should lead to the creation of an epidemiological tool capable of detection of any biological agent emitted from a barn ventilation source.

Conclusions

The pattern of disease spread during the avian influenza outbreak in Abbotsford suggested that an airborne mechanism may be involved. Investigative work to date points to the need for applied research involving scientific disciplines beyond the traditional boundaries of veterinary epidemiologists. This offers exciting new challenges and opportunities for an exchange of expertise between scientific disciplines in the fields of epidemiology, environmental biology, virology, particle physics, aerosol dynamics and meteorology. Also, the ability to better understand the mechanism and likelihood of disease transmission of avian influenza by aerosol means will offer important information to a global community wanting to know the real risks associated with avian influenza.

Reference List of Investigation Reports

1. Avian influenza. CAHNet Bulletin Edition 9. Animal Disease Surveillance Unit. Winter 2004. Internet: www.cahnet.org/bulletinsE/CahnetBulletin9english.pdf
2. The source and means of spread of the avian influenza virus in the Lower Fraser Valley of British Columbia during an outbreak in the winter of 2004. An Interim Report. Christine Power, Epidemiologist, CFIA. February 2005. Internet: www.inspection.gc.ca/english/anima/heasan/disemala/avflu/2004sum/repindexe.shtml
3. Comprehensive report on the 2004 outbreak of high pathogenicity avian influenza (H7N3) in the Fraser Valley of British Columbia, Canada. Wayne Lees, Epidemiologist and Lawana Chown, student, Animal Disease Surveillance Unit, June 2004. Internet: www.inspection.gc.ca/english/anima/heasan/disemala/avflu/2004sum/repindexe.shtml
4. Surface wind conditions in the Fraser Valley during the avian influenza (January 11-April 2, 2004). Stephanie Meyn, Air Quality Meteorolgist, BC Ministry of Water Land And Air Protection. April 2004.
5. Literature review of survival of avian influenza virus in aerosol/dust. Pascal Moreau, Animal Health Risk Analysis, Science Branch, May 2004.
6. Literature review of highly pathogenic avian influenza virus survival in manure. Pascal Moreau, Risk Analyst and Scientific Advisor, Animal Health Risk Analysis, Science Branch, March 2004.
7. Case series review: Assessment of the role of wind in disease transmission. Nancy DeWith, Epidemiologist, CFIA contract, April 2004.
8. Evaluation for the potential of dust borne barn to barn transmission of highly pathogenic avian influenza in the Fraser Valley, British Columbia. in March 2004. Carolyn Dubé, Epidemiologist, Animal Health and Production Division and Pascal Moreau, Risk Analyst and Scientific Advisor, Animal Health Risk Analysis, Science Branch, April 2004.
9. Report on the avian influenza aerosol sampling trial in the British Columbia Fraser Valley April 9-19, 2004. Laurie Schofield, Jim Ho, Bill Kournikakis, Research Scientists, Defence R and D Canada-Suffield, Department of National Defence, June 2004.

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National Aquatic Animal Health Program (NAAHP)

Responsibilities, Partnerships…*and* Oceanography

Nancy House

Sharon McGladdery, Nancy House, Jack Taylor, and Brian Jamieson

Canada's National Aquatic Animal Health Program (NAAHP) is aimed at addressing increasingly stringent international trade requirements and improving Canada's capacity to prevent, detect, and respond to diseases that pose a significant threat to our aquatic resources. The NAAHP allows Canada to meet trade obligations laid down by the World Trade Organization Agreement on Sanitary and Phytosanitary Measures and will protect Canada's farmed and wild aquatic resources from serious infectious diseases. The program is led by the Canadian Food Inspection Agency (CFIA) and co-delivered with Fisheries and Oceans Canada (DFO). This federal initiative complements provincial programs aimed at managing endemic diseases that cause significant losses if not actively controlled. Both government disease control responsibilities rely on strong aquaculture industry participation in managing farm-level production (opportunistic) diseases. At all stakeholder levels, however, a common key to success is a clear understanding of the role of the aquatic environment in spreading the infections of concern. This paper covers the federal interest in improving our knowledge of the role of water in order to help design more effective control programs and improve accuracy of disease risk assessments.

What is Canada's NAAHP?

Canada's National Aquatic Animal Health Program (NAAHP) is an initiative to coordinate and build on established federal, provincial, and private industry expertise to optimise handling of outbreaks of infectious diseases that threaten aquatic animal trade (intra- and international) or resource conservation. It is designed to complement regional programs aimed at management of recurrent endemic diseases, as well as aquaculture industry management of production diseases of concern.

The NAAHP addresses increasingly stringent international aquatic animal health standards requiring countries to adopt official aquatic animal health programs that are founded on robust scientific knowledge. These are aimed primarily at reducing the risk of disease spread related to seafood trade but they also form the basis for countries to develop measures to protect their aquatic resources from

exposure to diseases that present a significant threat. These international standards have increased significantly in scope over the last 5 to 10 years as diseases have spread around the world and countries have realised the need to develop disease control programs similar to those well-established for terrestrial food production sectors.

The NAAHP is aimed at proactive health management and early intervention to maximise production efficiency, lower reactive disease control costs, and minimise the need for extreme disease control measures. A pivotal component to ensure these objectives are met effectively is a solid understanding of disease dynamics not only within vulnerable aquatic animal populations, but also of the role of water movement in the spread of infectious disease agents of concern.

"The NAAHP is aimed at proactive health management and early intervention to maximise production efficiency, lower reactive disease control costs, and minimise the need for extreme disease control measures."

The Science Component of the NAAHP

Scientific knowledge encompassing a wide range of expertise is essential for producing the data required for:

i. development of appropriate regulatory frameworks;
ii. accurate risk assessments and related decision-making;
iii. effective surveillance strategies; and
iv. rapid and accurate disease diagnosis.

Classically, this has included pathology, epidemiology, parasitology, microbiology, virology, and molecular pathology, which provides an excellent foundation for understanding the host-pathogen interaction. When this information is combined with related human activities, it often forms the basis for regulatory controls and disease control measures. However, the role of the environment in which the host, pathogen, and humans interact is often underestimated, overlooked, or qualitatively 'guesstimated'. This can pose a significant challenge for development of regulations or disease control policies that are both effective and justified on the basis of 'solid' science.

The recent experience gained with the appearance, impact and efforts at control of infectious salmon anaemia (ISA) in Atlantic salmon (*Salmo salar*) in the Bay of Fundy—covered elsewhere in these proceedings—was one of the major catalysts that highlighted the need to coordinate federal, provincial, and industry aquatic animal health expertise. It also highlighted the need for multidisciplinary scientific expertise input into disease control measures. One key success was the integration of oceanographic expertise and techniques with the epidemiological models developed for ISA. On the basis of this, the new NAAHP recognised the need to include such expertise and information in the development of its scientific program. Similar recognition is reflected in the papers presented at this workshop by participants from the industry, the provinces, and from scientists from other countries and animal disease control backgrounds.

As a backdrop for the workshop, a brief description is given here of the federal program and the rationale for interest in incorporation of water movement techniques within the science component of regulatory aquatic animal disease control and management decision-making.

NAAHP Genesis and Organisation

In spring 2005, federal funding was received to update Canada's federal aquatic animal health program and bring it into line with increasingly stringent interna-

tional aquatic animal health standards. The funding was given to two federal authorities, the Canadian Food Inspection Agency (CFIA) and Fisheries and Oceans Canada (DFO), for co-delivery of the federal regulatory components of NAAHP. The scientific foundation of the NAAHP is managed through DFO and is based on a stringent quality assurance and control program encompassing federal diagnostic and research laboratories, as well as any non-federal laboratories that undertake activities to support the new regulatory laboratory system.

CFIA provides the program direction under the authority of the Health of Animals Act. This brings Canada's aquatic and terrestrial animal health programs under the same legislative umbrella. It also brings aquatic animal disease management under the same Agency responsible for both terrestrial animal and plant diseases. In addition to program leadership, CFIA is responsible for aquaculture health surveillance. DFO is responsible for delivering the science component of the NAAHP. A National Diagnostic Laboratory System (NDLS) is being built on DFO's existing aquatic animal health laboratory infrastructure for delivery of regulatory diagnostics, technology development, and targeted research to support regulatory control of mandatorily reportable diseases. DFO is also responsible for surveillance of wild aquatic resources, as per surveillance programs developed and overseen by the CFIA.

The CFIA programs include risk analysis, import/export control measures and maintenance of the nationally reportable disease list using internationally set criteria for official control measures.[6] They also oversee the NDLS quality assurance and quality control program, and coordinate aquaculture facility surveillance and development/testing of emergency response plans.

National Diagnostic Laboratory System (NDLS)

The National Diagnostic Laboratory System (NDLS) is comprised of four DFO Aquatic Animal Health Centres located at the Pacific Biological Station in Nanaimo, British Columbia; the Freshwater Institute in Winnipeg, Manitoba; the Gulf Fisheries Centre in Moncton, New Brunswick; and the Charlottetown Aquatic Animal Pathogen Biocontainment Laboratory (CAAPBL) in Charlottetown, Prince Edward Island. The CAAPBL is Canada's official aquatic pathogen biocontainment facility for regulatory research.

The National Diagnostic Laboratory System also includes the establishment of a National Centre of Expertise (NCE) for aquatic animal health at the Gulf Fisheries Centre, where both national diagnostic and research activities will be co-housed. The National Laboratory Coordinator (NLC) will be based at the Gulf Fisheries Centre. The NDLS laboratories will provide quality controlled diagnostic and research data to support a new regulatory framework being developed under CFIA's Health of Animals Act.

The NLC will be responsible for ensuring that diagnostic laboratory services are delivered uniformly across the country and meet internationally accepted diagnostic standards. To this end, all federal NDLS laboratories will function under national coordination, direction, and oversight. The CFIA will require all NDLS labs to be ISO 170925 accredited for specified official test procedures, which is consistent with the requirements of CFIA's laboratories in the terrestrial animal health program. Provincial or private diagnostic laboratories interested in joining the NDLS and undertaking federal disease diagnostic activities will also need to be ISO 170925 accredited.

Each federal system laboratory will meet QA/QC standards, prioritize, coordinate and conduct research under the direction of the NLC, contribute to contingency planning and support disease response under CFIA direction. Regional aquatic animal health issues will be managed directly through regional DFO administration and under the Introduction and Transfers Code. In addition, the laboratories will provide confirmation of nationally listed diseases, and provide training to provincial or private laboratories for the screening of listed diseases. The web-based NAAHP database will be used to facilitate national coordination of data from all federal, provincial, and private labs partnered within the NDLS.

The CFIA is developing a proposed national list of reportable and immediately notifiable diseases that are exotic to all or parts of Canada. The management of diseases on this list, where they are exotic, will be a federal responsibility. Reporting of these diseases will be immediate (notifiable) for areas identified as being negative, and at regular intervals (e.g., biannual reportable) where the diseases are endemic. Stakeholders and partners will be consulted prior to finalizing the list, anticipated before the end of 2006. It may take up to two years to make the necessary modifications to the Health of Animals Regulations to fully incorporate all the controls required for the final list of reportable and immediately notifiable diseases.

"In a situation where a reportable or immediately notifiable disease is detected, the NDLS laboratories will report directly to the National Aquatic Animal Health Science Branch, which is the official notification point to CFIA."

In a situation where a reportable or immediately notifiable disease is detected, the NDLS laboratories will report directly to the National Aquatic Animal Health Science Branch, which is the official notification point to CFIA. In the event that a listed disease is detected and reported to the CFIA via this route, the CFIA is responsible for coordinating the emergency response measures required.

During, and after finalisation of, the transfer of regulatory authority from DFO to CFIA, DFO will maintain its responsibility for provision of scientific advice and information via the National Aquatic Animal Health Science Branch (formerly the National Registry for Aquatic Animal Health) for regional/endemic disease issues. This includes scientific advice on disease risks associated with non-NAAHP listed diseases for introductions and transfers committees, as required.

Wild Stock Surveillance

International standards require official aquatic animal health management programs to include surveillance of wild fish, shellfish, and crustaceans, where these are in contact with a stock or population (wild or cultured) that requires certification of proof of freedom from a disease deemed to pose a threat to resources within the country of a trade partner.

The CFIA is responsible for designing the wild surveillance programs necessary for listed disease agents. Several mechanisms can be used for sampling wild population, such as using those collected for research purposes, for stock assessment or habitat investigations, from processing plant inspections, as well as from investigation of wild aquatic animal mortality incidents. Wild population surveillance will focus on the sampling required to reinforce proof of freedom from specified disease agents (certification), or to map the extent of the distribution of a disease agent for the purposes of defining populations that remain free of the infection (zonation). Some sampling of wild stocks may also be required for validation of diagnostic tools using Canadian species or to assess efficacy under Canadian aquatic environmental conditions.

DFO provides the scientific advice on epidemiology, ecology, and oceanographic/hydrographic factors that relate to surveillance or control plans for federally reportable and notifiable disease agents. In this regard, the importance of understanding the role of water movement in the spread of viable infectious agents, or their vectors, cannot be underestimated.

Zoning for Specific Disease and Water Movements

Zones are defined as geographic areas encompassing a population of animals which has a health status that differs from other susceptible populations or species located in a neighbouring or more distant location. An area surrounding a zone positive for a specified disease and which contains species susceptible to that disease is considered negative. The delineation between positive and negative zones is strongly correlated to water movement as well as geography. Effective zonation for an open water population in a freshwater catchment area must include consideration of flow, flood tables, and natural and artificial barriers. Thus, an uninfected population upstream from an infected population can be subject to practical control measures, whereas the reverse would be difficult, if not impossible. The health status of aquatic animals grown in land-based facilities with flow-through or open water discharge is equally important in defining the health status of populations downstream. This concept is well-understood for many freshwater fish producers who choose sites where facilities can be fed by fish-free upstream or well-water sources. In addition, these considerations are incorporated into the OIE Code Chapter on Zonation.[5]

"Experience in both plants and land animal disease control—developed over the last hundred years—can be readily applied to the relatively new aquatic disease control environment."

In open ocean, estuarine, or large river and lake areas, however, the hydrographic component is more difficult to define for disease zonation. For these areas, knowledge about the persistence of the pathogen outside the host, along with knowledge of any vectors important for active (rather than passive) transmission, is essential for overlay over the water movement information for the area. In many cases, such information is lacking and historically have simply been defined based on geographic presence or absence data for samples collected for surveillance. Although useful for mapping purposes, such information is of limited value for disease management and control purposes.

The NAAHP has developed a geographic information system (GIS) web-based database, which uses the mapping component for presence or absence data. However, the zones developed around these data have to be reviewed carefully against the distribution of susceptible species, tidal flow information, drainage basin, and catchment area information. Even where these are definable, the lines drawn may have to be reconsidered in light of human activities that negate use of the hydrographically set boundary. In addition, the dynamic nature of rivers, estuaries, coastlines and human development demands that such zones undergo regular review and revision as necessary.

Of critical importance for control of disease spread to naïve susceptible populations is an accurate definition of the 'buffer zone'. This is the area that borders such populations and which is the focus for targeted surveillance for early detection of spread of an infectious disease towards vulnerable populations. As with water catchment areas, these zones have to be defined taking into account all natural and man-made activities that flow or drain into them. The scale of the buffer zone also has a significant impact on the degree of surveillance activity required for an effective early warning system. That is, the amount of targeted surveillance

necessary to ensure detection of the pathogen if it did spread to the naïve populations in the area. Effective buffer zones have to be based on physical geographic boundaries or water movement characteristics that provide assurance of negligible water exchange between positive and negative host populations (e.g., tidal cycles, estuarine channel flows, embayments). Thus, inclusion of water movements and physical characteristics related to epidemiology of a disease of concern is essential for defining disease control zones that can effectively protect the integrity of negative zones.

As with all zones, however, human activities have to be taken into account to ensure there are no significant activities (fisheries, aquaculture, or completely unrelated activities) that circumvent the natural or geographical boundaries.

Influence and Effect of the Aquatic Environment on Effective Disease Management

There are many challenges in aquatic animal disease management, but it is increasingly recognised that diseases continue to spread internationally despite intense monitoring programs and often stringent control measures. Obviously, much of this spread can be attributed to incomplete knowledge of the epidemiology of the diseases in question, as well as difficult to detect sub-clinical infections. However, the greatest unknown quantity in any disease control program is the role of the aquatic environment in influencing disease spread. This influence applies to the pathogen as well as to biotic and abiotic transmission vectors.

The quality and accuracy of decision making for disease control relies on as complete an understanding of the epidemiology of the disease as possible. In comparing the disease transmission in the aquatic environment with that on land, it is interesting to note that aquatic disease spread has more in common with plant pests than disease in terrestrial farming. Open fields, exposure to air movement and airborne vectors, as well as related wild species counterparts, show some strong parallels to production challenges in the aquatic environment. Experience in both plants and land animal disease control—developed over the last hundred years—can be readily applied to the relatively new aquatic disease control environment. The new NAAHP co-delivery with a CFIA lead will ensure this experience is brought into the multidisciplinary approach required for effective aquatic animal disease control.

Canadian Experience

There are two examples where water movement has played an important, but different, role in the introduction or distribution of aquatic disease. In 2002, the introduction of *Haplosporidium nelsoni* or multinucleate sphere X (MSX) to oysters in the Bras d'Or Lake system of Cape Breton Island, Nova Scotia showed no hydrographic link to infected oyster stocks located along the eastern seaboard of the United States. Also, the well known preference of this pathogen for proliferation in lower salinity, suggests that the spread north was unlikely via ocean drift—albeit possible—with its unknown putative intermediate host. Although water currents did not play an obvious role in introducing MSX to Canada, they have been important in influencing the distribution of the disease within the Bras d'Or Lake system. Detailed information on this work is provided elsewhere in these proceedings.[4] After 3 years, spread of infections appears primarily related to hu-

man transfers of oysters between leases within the Bras d'Or Lake system.

The second example is also well-described elsewhere in these proceedings. Infectious salmon anaemia (ISA) infects Atlantic salmon in the Bay of Fundy, New Brunswick and Maine, USA. Initial attempts to prevent spread of the disease between Canadian and American salmon farms, located meters away from each other across the Maine–New Brunswick border, were doomed to failure based on the proximity of the farms and the strong tidal influences mixing the waters shared by both countries at the mouth of the Bay of Fundy and Gulf of Maine. The subsequent development of bay management areas (BMAs) defined by water currents is described by Ellis et al.,[1] along with similar experiences and strategies developed in Scotland[2] and Norway.[3]

Summary

The creation of an internationally credible science-based National Aquatic Animal Health Program provides Canada with the tools to meet international trade obligations to protect Canada's wild and farmed aquatic resources. The regulatory disease control responsibilities of the NAAHP share the same information needs as control programs aimed at the management of endemic and opportunistic diseases—notably as comprehensive an understanding as possible of the dynamics of infectious agents in the aquatic environment being used to grow seafood.

For the evolving federal aquatic animal health program, diseases falling under regulatory control will be managed by control measures that incorporate oceanographic factors into the decision-making process. In recognition of the dynamic environment and evolving aquaculture and aquatic epidemiological knowledge, these measures will also be designed to be flexible and subject to regular review.

References

1. Ellis S, Gustafson L, Giray C, Robinson T, Marenghi F, Merrill P. 2005. Hydrographics and the epidemiology of ISA: Findings from Maine and New Brunswick. *Bull. Aquacul. Assoc. Canada* 105-1:44-51.
2. Murray A, Amundrud TL, Gillibrand PA. 2005. Models of hydrodynamic pathogen dispersal affecting Scottish salmon production: Modelling shows how Scotland eradicated ISA, but not IPN. *Bull. Aquacul. Assoc. Canada* 105-1:79-86.
3. Rolland J. 2005. Infectious salmon anaemia (ISA) management in Norway. *Bull. Aquacul. Assoc. Canada* 105-1:87-91.
4. Stephenson M, Petrie B. 2005. Oceanographic influences on the management of MSX disease in American oysters *(Crassostrea virginica)* in Atlantic Canada. *Bull. Aquacul. Assoc. Canada* 105-1:67-78.
5. OIE. 2005. *Aquatic Animal Health Code*. 8th edition. World Organisation of Animal Health, Paris. 178 p.

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Blythe Chang

Application of a Tidal Circulation Model for Fish Health Management of Salmon Farms in the Grand Manan Island Area, Bay of Fundy

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One mechanism that may facilitate the spread of viral diseases among salmon farms is waterborne transport of the virus. In Norway and Scotland, disease spread is managed by using hydrographically-defined control and surveillance zones, based on one tidal excursion around farms. We investigated the application of similar zones for the management of infectious salmon anemia (ISA) among salmon farms in the southern Grand Manan Island area in the Bay of Fundy. We estimated tidal excursions around farms using two approaches: a simple method based on 5-km radius circles around farms and a more complex method using a 3-dimensional tidal water circulation and particle transport model. Using both methods, we determined the overlaps of the predicted control zones with farm sites and with control zones of farms in the area. We also examined the potential for waterborne disease spread between farms in the southern and eastern areas of Grand Manan Island.

Introduction

The viral disease infectious salmon anemia (ISA) first appeared among marine salmon farms in the southwestern New Brunswick (SWNB) area of the Bay of Fundy in the summer of 1996.[1] Epidemiological studies in Norway indicated that the ISA virus was likely transmitted via infected salmon, fish wastes, and/or processing effluents, and that seawater was a significant route for disease transmission.[2,3] These studies found that proximity of a farm to ISA-infected farms, slaughterhouses, and processing plants was among the greatest risk factors, especially where the separation distance was less than 5 km. In Scotland, the spread of ISA over large distances was related to transfers of live fish and harvest collection, while at the local scale, ISA may have been transported among neighboring farms by water currents.[4-6]

The pattern of occurrence of ISA in SWNB[1,7] suggests that both large-scale vectors and smaller-scale passive transport via seawater have occurred. Murray[6] suggested that in areas with high tidal currents and farms located close together, such as SWNB, local transmission mechanisms such as passive spread through water may be more important than in areas with lower currents and greater separation between farms.

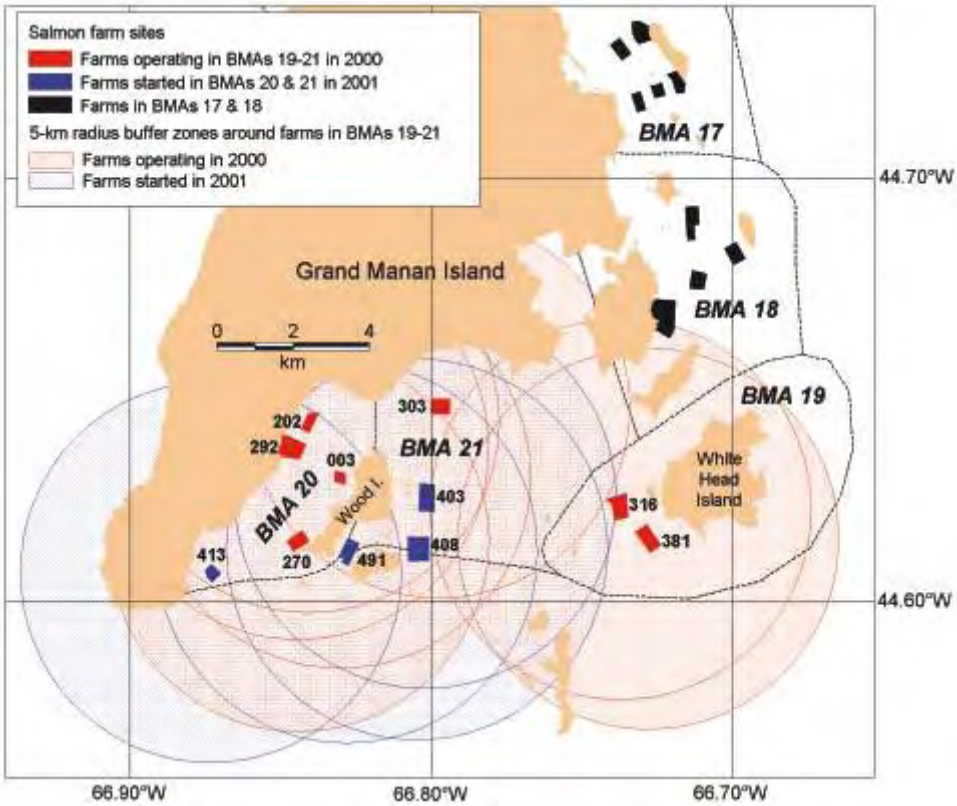
Norway and Scotland have implemented hydrographically-based control zones to reduce the potential for waterborne transport of the ISA virus. Within control

zones around infected farms there are requirements for fallowing and disinfection of farms, restrictions on fish transfers, and increased fish health surveillance. In Scotland, the control zone is defined as a circle, with a radius equal to one tidal excursion from the centre of a farm.[4,8] The maximum tidal amplitudes in Scotland (estimated from maps of tidal currents) translate into tidal excursions of 7.1 km in mainland Scotland and 3.6 km in Shetland.[8] In Norway, the control zone is defined as a circular area with a radius of at least one tidal excursion (but not less than 5 km) from the centre of a farm, or an equivalent area determined from hydrological or epidemiological data.[9] Because most farms in Norway are in areas of weak tides, the actual distance of one tidal excursion will often be less than 5 km.[4] Both Scotland and Norway also use larger zones where increased fish health surveillance is required. In Norway, the surveillance zone includes all farms whose control zones overlap with the control zone of an infected farm,[9] while in Scotland, the surveillance zone (or management area) includes all farms having overlapping control zones where at least one farm is infected.[4]

One of the responses to the ISA outbreak in SWNB was to organize the salmon farms into Bay Management Areas (BMAs), within which all farms should synchronize smolt placement, harvesting, and fish health management strategies.[10] Our project was initiated to provide oceanographic data that could be used to confirm or revise these BMA boundaries, and to assist in fish health management and farm site selection. We estimated control zones based on one tidal excursion using two methods. A simple approach, in which tidal excursions around farms were estimated as a 5-km radius circular zone, was compared to a more complex approach in which tidal excursions were estimated using a tidal circulation and particle tracking model. The southern Grand Manan Island area was the initial geographic focus. We also examined potential water linkages between farms in the southern and eastern Grand Manan Island areas.

The mean tidal amplitude in the Grand Manan area is about 5 m. In the year 2000, there were 7 salmon farms operating in this area, within 3 BMAs (Fig. 1). Since 2001, 4 additional farms have operated in this area. The minimum distance between each farm and its nearest neighbor (the shortest distance via water between

Figure 1
Salmon farm sites and 5-km radius buffer zones in the southern Grand Manan Island area (BMAs 19–21). Also shown are salmon farm sites in the eastern Grand Manan Island area (BMAs 17 and 18).



site boundaries) ranged from 0.3 to 2.8 km (average 1.0 km) in 2000 and from 0.3 to 1.9 km (average 0.9 km) in 2001. There are currently 9 salmon farms operating in the area. The shortest distance between a farm in the eastern area and a farm in the southern area is 2.4 km (between farms 172 and 316).

We previously reported preliminary results from this project[11] and additional details can be found in two technical reports.[12,13] Other reports[14-16] describe the application of this approach to other areas of SWNB. The purpose of this paper is to describe the approach used in those reports, using the Grand Manan Island area as an example, and to include some new analyses.

Methods

A simple method of estimating the zone equivalent to one tidal excursion around each farm involved drawing a circle with a 5-km radius around the centre of the site or, if known, around the centre of the cage cluster. We excluded land areas which fell within the circle, as well as any water areas separated from the farm site by land. These circle-based zones were created using the MapInfo Profes-

Table 1. Surface areas of 5-km radius buffer zones (excluding land and areas of water separated from the farm by land) and model-derived tidal excursion areas, and maximum straight line displacement of model particles during one tidal excursion (12.4 h), for salmon farms in the southern Grand Manan Island area. Farms shown in bold italics started operating in 2001.

Farm	Surface area of 5-km radius buffer zone (km^2)	Surface area of model-derived tidal excursion area (km^2)	Maximum straight line displacement from starting point during one tidal excursion (km)
BMA 20			
202	32.0	2.2	3.2
292	35.6	3.3	3.4
003	48.4	2.9	2.8
270	53.4	9.3	4.1
413	***59.0***	***4.0***	***3.6***
Mean ± SD	45.7 ± 10.3	4.3 ± 2.6	3.4 ± 0.5
BMA 21			
303	46.8	5.3	3.1
403	***63.5***	***21.9***	***5.0***
408	***71.6***	***31.0***	***6.5***
491	***63.2***	***8.6***	***4.4***
Mean ± SD	61.3 ± 9.0	16.7 ± 10.3	4.8 ± 1.4
BMA 19			
316	68.2	49.0	13.6
381	70.3	81.6	13.2
Mean ± SD	69.2 ± 1.0	65.3 ± 16.3	13.4 ± 0.3
All southern Grand Manan Island area farms			
Mean ± SD	55.6 ± 13.0	19.9 ± 24.0	5.7 ± 4.0

sional® 7.0 buffer tool and are henceforth referred to as buffer zones. The maximum possible area of a 5-km radius buffer zone (i.e. if there were no land areas within the zone) was 78.5 km^2. We determined the overlaps of each 5-km radius buffer zone with farm sites and with the buffer zones of farms in the study area.

More precise estimates of the tidal excursions around farms were made using a 3-dimensional particle tracking model (version August 2004)[17] customized for our study area. The model estimates the tidal currents by dividing the geographic area into triangles (finite elements) and by numerically solving the equations of motion at each x,y,z,t grid point. A depth profile of the current is calculated at

Table 2. Tables of overlaps of tidal excursion areas and farms sites, using two methods of estimating tidal excursion areas. ● indicates an overlap of the originating farm's 5-km radius buffer zone with farm sites (top table) and with farm buffer zones (bottom table); ■ indicates an overlap of the originating farm's model-derived tidal excursion area with farm sites (top table) and with model-derived tidal excursion areas (bottom table). Shaded areas indicate farms which started operating in 2001.

Overlaps of tidal excursion areas with farm sites			Originating farm										
			BMA 20					BMA 21				BMA 19	
			202	292	003	270	413	303	403	408	491	316	381
Receiving farm site	BMA 20	202	●■	●■	●	●	●	●	●	●	●		
		292	●■	●■	●	●	●	●	●	●	●		
		003	●	●	●■	●■	●	●	●	●	●		
		270	●	●	●■	●■	●	●	●	●	●		
		413	●	●	●	●	●■				●		
	BMA 21	303	●	●	●	●		●■	●	●	●		
		403	●	●	●	●		●	●■	●■	●■	●	
		408	●	●	●	●■		●	●■	●■	●■		
		491	●	●	●	●■	●	●	●	●	●■		
	BMA 19	316							●			●■	●■
		381										●■	●■

Overlaps among tidal excursion areas			Originating farm										
			BMA 20					BMA 21				BMA 19	
			202	292	003	270	413	303	403	408	491	316	381
Receiving farm tidal excursion area	BMA 20	202	●■	●■	●	●	●	●	●	●	●	●	●
		292	●■	●■	●	●	●■	●	●	●	●	●	●
		003	●	●	●■	●■	●	●	●■	●■	●	●	●
		270	●	●	●■	●■	●	●	●■	●■	●■	●	●
		413	●	●■	●	●	●■	●	●	●	●		
	BMA 21	303	●	●	●	●	●	●■	●■	●■	●■	●	●
		403	●	●	●■	●■	●	●■	●■	●■	●■	●■	●■
		408	●	●	●■	●■	●	●■	●■	●■	●■	●■	●■
		491	●	●	●	●■	●	●■	●■	●■	●■	●	●
	BMA 19	316	●	●	●	●		●	●■	●■	●	●■	●■
		381	●	●	●	●		●	●■	●■	●	●■	●■

each corner of every triangle every 2.07 seconds. The model is fully non-linear, has up to 21 depth levels, and has variable horizontal resolution (minimum ~50 m). The model also simulates wetting and drying of intertidal areas. Although the generic model code has the capability of including boundary forcing using multiple tidal constituents, internal water density and surface winds, we only used boundary forcing by the principal lunar semi-diurnal (M_2) tide for the simulations presented here.

Thirty-six model particles were released every hour over a 12-h period (to represent conditions over a tidal cycle) from a 200 × 200 m grid located at the approximate centre of each farm or, if known, at the cage cluster (i.e. a total of 12 releases and 432 particles from each farm). The particles were released and maintained at 1 m below the sea surface. The position of each particle was recorded every 20 min during one tidal cycle (12.42 h). Some particle tracks were shorter than one tidal cycle, because the tracks terminated if they hit the shore. To delineate the geographic area covered by one tidal excursion, the marine surface area in the vicinity of each farm was divided into 100 × 100 m square cells. A farm's tidal excursion area was then estimated by combining all cells that were intersected by at least one of the 432 particle tracks released from that farm.

We tabulated the overlap of each model-derived tidal excursion area with farm sites and with the tidal excursion areas of farms. The model-derived tidal excursions also allowed us to measure the intensity of the overlaps: by determining the number of releases (out of 12) that had at least one particle overlapping farm sites and the number of particles (out of 432) that overlapped farm sites.

Results

Estimated control and surveillance zones of farms in the southern Grand Manan Island area

Control zones for farms operating in the southern Grand Manan Island area, as estimated by 5-km radius buffer zones, ranged in size from 32.0 to 71.6 km^2 (Table 1, Fig. 1). Buffer zones of farms operating in 2000 overlapped an average of 4.1 farm sites and all 7 buffer zones (including the originating site and buffer zone), while buffer zones of farms operating since 2001 overlapped an average of 7.4 farm sites and 10.6 buffer zones (Table 2). In all instances, the overlaps of buffer zones with farm sites were reciprocal; where one farm's buffer zone overlapped a second farm's site, the second farm's buffer zone overlapped the first farm's site.

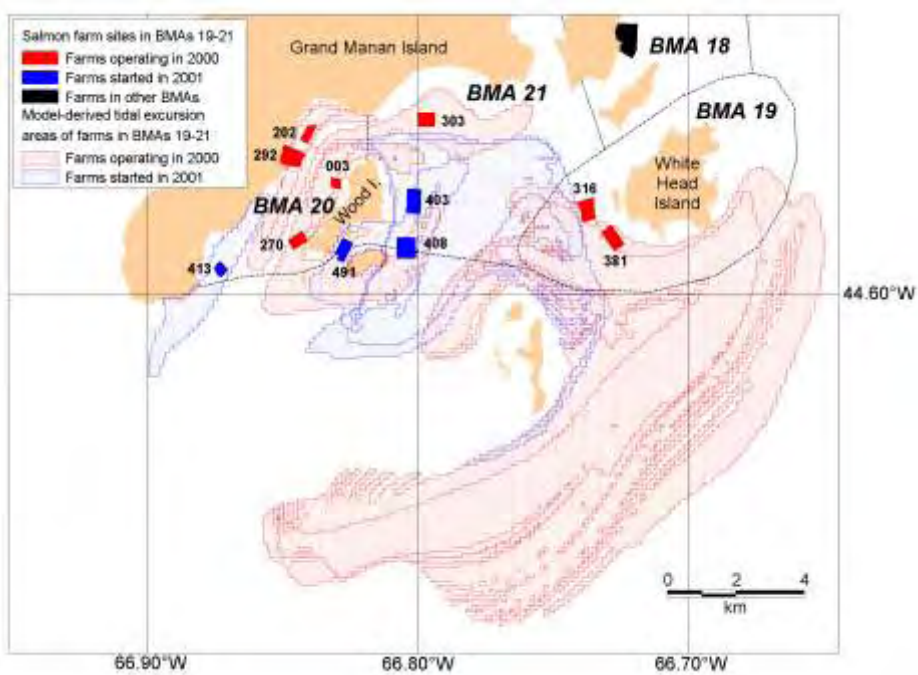


Figure 2
Salmon farm sites and model derived tidal excursion areas in the southern Grand Manan Island area (BMAs 19- 21).

The overlaps of buffer zones with farm sites in 2000 indicated two completely separate groups of farms: farms in BMAs 20 and 21 in one group and farms in BMA 19 in the other group. With the addition of 4 new farms in 2001, the overlaps of buffer zones with farm sites still suggested two groups, but they were not completely isolated: there were some overlaps between farms in BMAs 21 and 19. The overlaps among buffer zones, both in 2000 and since 2001 suggested that all southern Grand Manan Island farms should be in one surveillance zone.

Control zones, as estimated using model-derived tidal excursions, were generally elongated, rather than circular and were smaller than the corresponding 5-km radius buffer zones, except in the case of farm 381 in BMA 19 (Table 1 and Fig. 2). The model tidal excursion areas resulted in fewer overlaps than the 5-km buffer zones (Table 2). For farms operating in 2000, the model tidal excursion areas overlapped an average of 1.9 farm sites and the same number of tidal excursion areas (including the originating farm site and tidal excursion area), while for farms operating since 2001, there was an average of 2.1 over-

Figure 3
Model particle tracks for each of 12 releases (0 to 11) started at hourly intervals and tracked for one tidal excursion (12.4 h) from farm 403. Finfish farms are shown as small white polygons. The larger blue polygon is the farm's total tidal excursion area. Release 3 started near the time of high water and release 9 started near the time of low water.

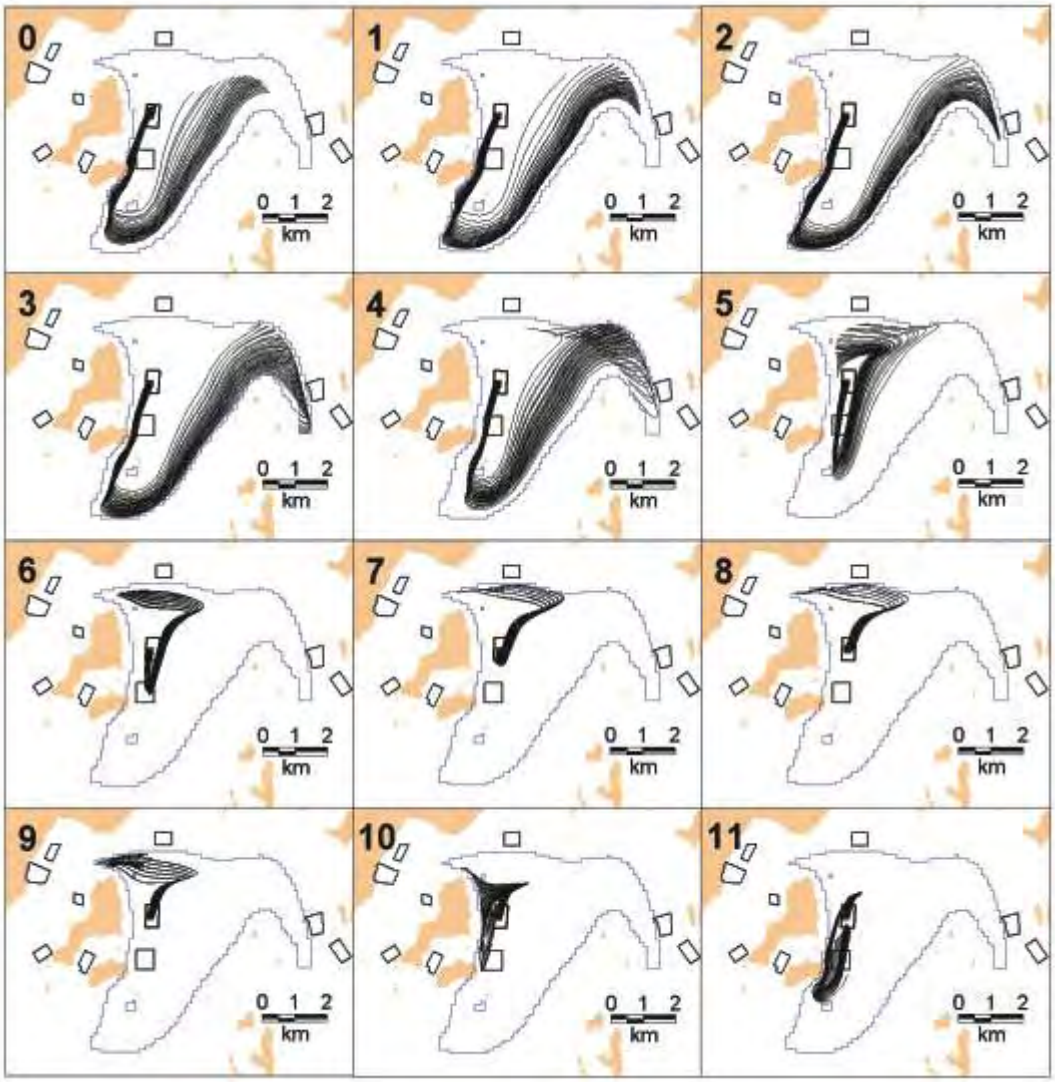


Table 3. Numbers of model-derived particle tracks from each farm which overlapped farm sites. Also shown, in brackets, are the numbers of particle releases which had overlapping particle tracks. There were 12 particle releases, at hourly intervals, from each farm site. Each release consisted of 36 particles, for a total of 432 particles from each farm. Particles were tracked for one tidal excursion (12.4 h). For farm 202, the number of particle tracks overlapping the originating farm site was slightly less than 432, because one point in the particle release grid lay just outside this farm's site boundaries. Shaded areas indicate farms which started in 2001.

No. of overlapping particle tracks			Originating farm of model particles										
			BMA 20					BMA 21				BMA 19	
			202	292	003	270	413	303	403	408	491	316	381
Receiving farm site	BMA 20	202	427(12)	289(9)	0	0	0	0	0	0	0	0	0
		292	216(6)	432(12)	0	0	0	0	0	0	0	0	0
		003	0	0	432(12)	33(4)	0	0	0	0	0	0	0
		270	0	0	2(1)	432(12)	0	0	0	0	0	0	0
		413	0	0	0	0	432(12)	0	0	0	0	0	0
	BMA 21	303	0	0	0	0	0	432(12)	0	0	0	0	0
		403	0	0	0	0	0	0	432(12)	166(5)	41(3)	0	0
		408	0	0	0	60(3)	0	0	221(9)	432(12)	82(6)	0	0
		491	0	0	0	16(3)	0	0	0	0	432(12)	0	0
	BMA 19	316	0	0	0	0	0	0	0	0	0	432(12)	0
		381	0	0	0	0	0	0	0	0	0	0	432(12)

laps with farm sites and 4.5 overlaps with tidal excursion areas. For farms operating in 2000, all overlaps between tidal excursion areas and farm sites were reciprocal; however in 2001, there were four instances where overlaps were not reciprocal (Table 2).

The overlaps of tidal excursion areas with farm sites operating in 2000 indicated that the three BMAs in the southern Grand Manan Island area were completely separate. However, for farms operating since 2001, there was some water exchange between farms in BMAs 20 and 21, while BMA 19 remained separate. The overlaps among tidal excursion areas in 2000 indicated that surveillance zones for all farms would only include farms within one BMA, while the overlaps since 2001 indicated that the surveillance zones for 7 of the farms would include farms in more than one BMA.

Figure 3 shows the individual hourly releases from farm 403 (similar maps for other farms in this area can be found in Page et al.[12]). The model particle tracks show that the displacement of a particle from its starting point does not increase constantly over time; at times, due to the change in the tide, the particle may move closer to its starting point.

The highest number of releases overlapping a farm site was 9 (apart from the 12 releases overlapping each originating farm site; see Table 3). This occurred in two instances: releases from farm 292 overlapping its nearest neighbor, site 202; and releases from farm 403 overlapping its nearest neighbor, site 408. In all other instances, there were 6 or fewer releases overlapping farm sites.

A map of the spatial distribution of the density of all particle tracks from farm 403 is shown in Fig. 4 (for other farms see Page et al.[12]). All 432 particle tracks over-

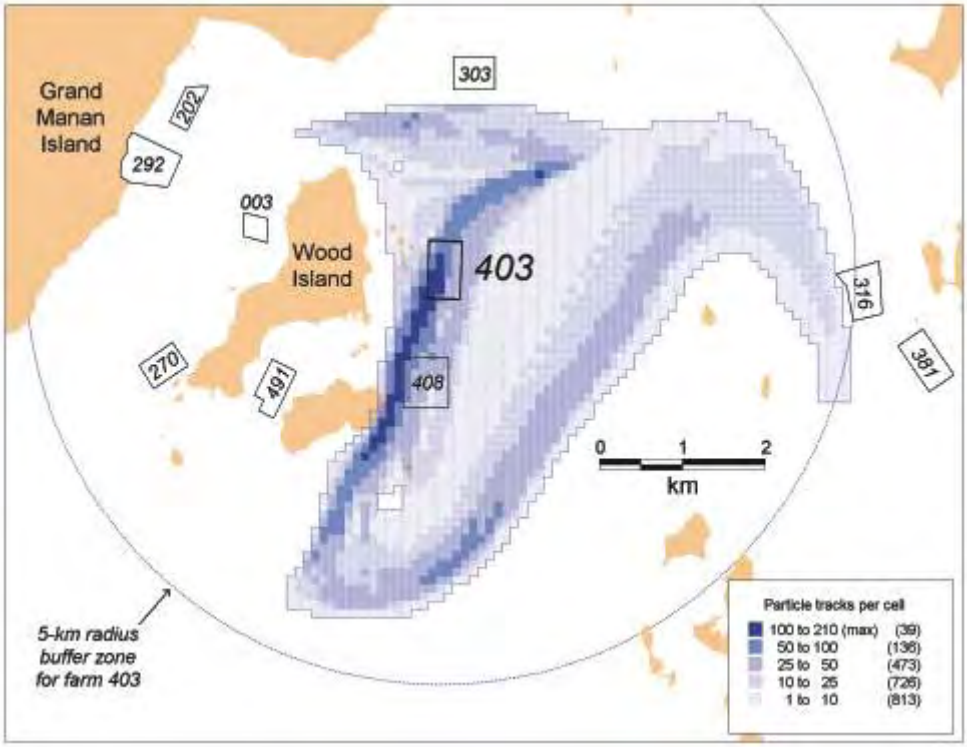
lapped with the originating farm's site, with the exception of farm 202, where only 427 particle tracks overlapped the same farm's site, because one of the points in the starting grid for that farm was located just outside of the farm's site boundaries (Table 3). There were just three other instances where greater than 50% of the particle tracks overlapped a farm site: 289 particle tracks (67%) from farm 292 overlapped farm site 202; 221 particle tracks (51%) from farm 403 overlapped farm site 408; and 216 particle tracks (50%) from farm 202 overlapped farm site 292.

Estimated water exchange between farms in the southern and eastern Grand Manan Island areas

There was just one overlap of a 5-km radius buffer zone from the southern area with a farm site in the eastern area and vice versa (Fig. 5): the buffer zone of farm 172 (BMA 18) overlapped farm site 316 (BMA 19) and the buffer zone of farm 316 overlapped farm site 172. None of the buffer zones of farms in BMA 17 overlapped with buffer zones of farms in the southern area (BMAs 19 to 21), but buffer zones of farms in BMA 18 overlapped with the buffer zones of farms in BMAs 19 to 21 in the southern area.

There were four overlaps of model-derived tidal excursion areas of farms in the eastern area overlapping farm sites in the southern area (Fig. 6): the tidal excursion area of farm 172 (BMA 18) overlapped farm sites 316 and 381 (BMA 19) and the tidal excursion area of farm 300 (BMA 18) overlapped the same two farm sites in BMA 19. The intensity of these overlaps was relatively small: 1 to 3 of the hourly releases and 29 to 33 of the particles released in each case. There were no overlaps of tidal excursion areas of farms in the southern area with farm sites in the eastern area. The tidal excursion areas of two eastern Grand Manan Island farms (farms 172 and 300 in BMA 18) each overlapped with the tidal excursion areas of two farms

Figure 4
Model-derived tidal excursion area of farm 403 in the southern Grand Manan Island area. The shading represents the number of model-derived particle tracks intersecting each 100 × 100 m square cell within the farm's total tidal excursion area. Thirty-six particles were released from the farm site at hourly intervals over a 12-h period (total of 12 releases and 432 particles) and tracked for one tidal excursion (12.4 h). Farm sites are shown as small white polygons. Numbers in parentheses in the legend are the numbers of 100 × 100 m cells within each range.



in BMA 19 and two farms in BMA 21 in the southern Grand Manan Island area.

Discussion

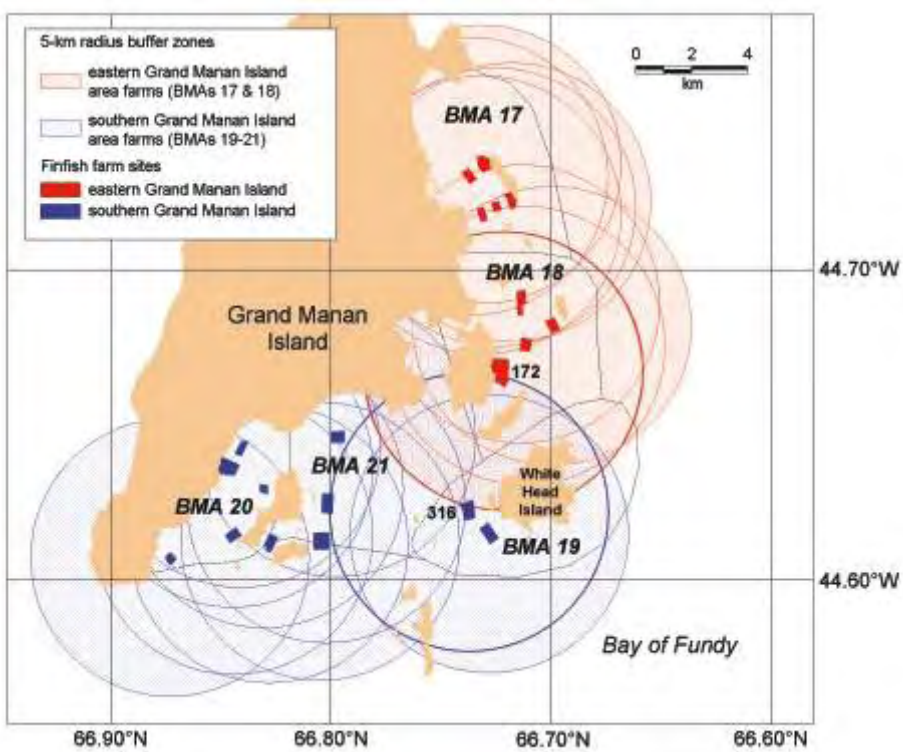
In this study we compared simple and more complex estimates of the tidal excursions around salmon farms in the southern Grand Manan Island area, as predictors of the potential for waterborne spread of disease among farms. The simple approach, using 5-km radius circular zones, can be easily done using commercially-available software. The more complex approach required the development of a tidal circulation model customized to our study area. This latter approach is more expensive and time-consuming but provides more precise estimates of the tidal excursion areas around farms.

Our choice of a 5-km radius for the simple approach was based on preliminary information on the current speeds in the area, as well as epidemiological studies and management practices in Norway.[2,3,9] The particle trajectories estimated by our model suggest that this was a reasonable choice for our study area, although it underestimated the maximum distance travelled by most particles released from farms in BMA 19 and overestimated the maximum distance travelled by most particles released from farms in BMA 20.

If we use 5-km radius buffer zones to delineate control zones (as in Norway and Scotland) for farms operating in the southern Grand Manan Island area in 2000, we would find that all farms in BMAs 20 and 21 would share the same control zone, while the two farms in BMA 19 would be in a separate zone. The overlaps among buffer zones suggest that all southern Grand Manan Island area farms should be in one surveillance zone.

If we use the model-derived tidal excursions to delineate control zones for southern Grand Manan Island area farms in 2000, we find that most of the control zones would include just one other farm (except the control zone of farm 303 would contain no other farms) and none would include any farms outside the originating farm's BMA. The new farms added in 2001 did not substantially increase the average number of farms per control zone, but did create a link from farms in BMA 20 to BMA 21, although not in the reverse direction. The overlaps

Figure 5
Map showing 5-km radius buffer zones around farms in the eastern and southern areas of Grand Manan Island. Thicker outlines indicate the buffer zone of one farm in the eastern area (farm 172) which overlapped a farm site in the southern area (farm 316) and the buffer zone of one farm in the southern area (farm 316) which overlapped a farm site in the eastern area (farm 172).



among model tidal excursion areas suggest that the surveillance zones in 2000 would only include farms in one BMA, but in 2001 most surveillance zones would include farms in more than one BMA.

Both methods of estimating water exchange areas indicated linkages between the southern and eastern Grand Manan areas. However, the model-derived tidal excursion areas indicated that the linkages were primarily in one direction, from two farms in BMA 18 toward farms in the southern area, and were relatively low in intensity.

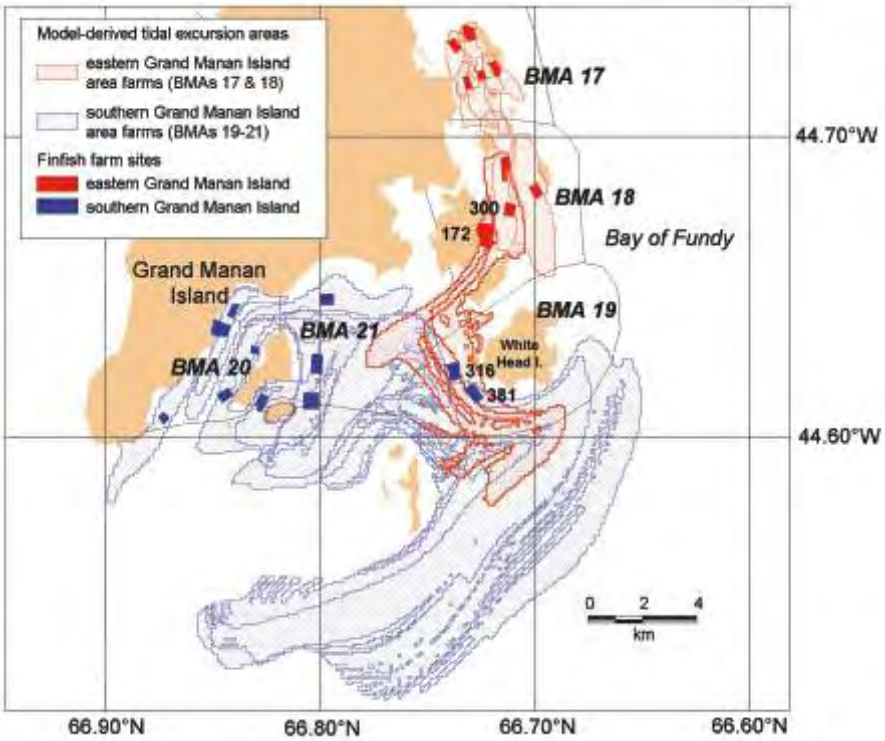
The modeling approach allows us to quantify the overlaps among farms, both temporally and spatially. Because the hourly releases were spread over the tidal cycle, the number of releases that have overlaps with farm sites or tidal excursion areas (Table 4, Fig. 3) provides an indication of the temporal nature of the overlaps. For example, if the overlaps of particles released from one farm overlapped with another farm in 3 of the 12 hourly releases, this represents about 25% of the tidal cycle. Such information could be used by farmers to time activities, such as harvesting, to minimize potential impacts on neighboring farms.

The number of model particle tracks from a farm which overlap other farm sites (or other tidal excursion areas) provides an estimate of the overall intensity of the overlap. This information could be used to the predict the risk of waterborne spread of disease between farms located too close together to achieve 100% separation or where there are socioeconomic pressures to place a management area boundary in a certain location. This methodology could also be used to estimate the cumulative risk of a particular farm to virus originating from more than one farm.

It must be mentioned that we do not know enough about the ISA virus to confidently translate the relative exposure index into a risk of infection. Information is lacking on rates of viral shedding from infected fish, survival of the virus in seawater, and amount of virus required to infect a fish, especially in field conditions. In a related study of ISA outbreaks among salmon farms in Cobscook Bay, Maine and adjacent areas of SWNB, it was found that tidal dispersion, as predicted by the model methodology used in our study, did play

Figure 6

Map showing model-derived tidal excursion areas of farms in the eastern and southern areas of Grand Manan Island. Thicker red outlines indicate the tidal excursion areas of two farms in the eastern area (farms 172 and 300) which overlapped farm sites in the southern area (farms 316 and 381 in BMA 19). None of the tidal excursion areas of farms in the southern area overlapped any farm sites in the eastern area.



a significant role in the spread of ISA in that study area, although the predictive power of model-estimated water movement alone was relatively small and many other factors were also important.[18] Nevertheless, the authors suggested that the use of model-predicted tidal excursions would provide benefits for the control of disease transmission among salmon farms.

Our model-predicted tidal excursion areas were determined completely by the M_2 tide. Although the M_2 tide is the major component of the tide in this area, other factors do play a role, and when they are included in the model the particle trajectories and exposure maps will be modified to some degree. The model approach can also be customized with different time scales and different release and particle trajectory parameters, such as the depth of particle release, vertical movement behavior, the duration of particle infectiousness, the density of the release grid, and the temporal sequence of release.

Fish health specialists, members of the salmon aquaculture industry, and regulators have indicated to us that the more precise estimates of water exchange areas obtained using the model justify the increased expense and effort required, as compared to the results obtained using simple circle-based estimates.[19] Ideally we would like to see such an approach implemented before aquaculture sites are approved, so that unwanted water exchange scenarios could be avoided.

Acknowledgments

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References

1. McGeachy SM, Moore MJ. 2003. Infectious salmon anemia in New Brunswick: an historical perspective and update on control and management practices (1997-2002). In: International Response to Infectious Salmon Anemia: Prevention, Control, and Eradication: Proceedings of a Symposium; 3-4 September 2002; New Orleans, LA. (O Miller, RC Cipriano, tech. coords.). *US Dept. Agriculture, Animal and Plant Health Inspection Service; US Dept. Interior, US Geological Survey; US Dept. Commerce, National Marine Fisheries Service (Washington, DC). Tech. Bull.* 1902:145-153. Available from: hht://www.aphis.usda.gov/vs /aquaculture/isa-proceedings.pdf. Accessed 24 October 2005.
2. Vågsholm I, Djupvik HO, Willumsen FV, Tveit AM, Tangen K. 1994. Infectious salmon anaemia (ISA) epidemiology in Norway. *Prev. Vet. Med.* 19:277-290.
3. Jarp J, Karlsen E. 1997. Infectious salmon anaemia (ISA) risk factors in sea-cultured Atlantic salmon *Salmo salar*. *Dis. Aquat. Org.* 28:79-86.
4. Joint Government/Industry Working Group on Infectious Salmon Anaemia. 2000. *Final Report of the Joint Government/Industry Working Group on Infectious Salmon Anaemia (ISA) in Scotland.* Scottish Executive, Fisheries Research Services, Marine Laboratory, Aberdeen. Available from: http://www.marlab.ac.uk/FRS.Web/uplaods/Documents/ JGIWGReport.pdf. Accessed 24 October 2005.
5. Murray AG, Smith RJ, Stagg RM. 2002. Shipping and the spread of infectious salmon anemia in Scottish aquaculture. *Emerg. Infect. Dis.* 8:1-5.
6. Murray AG. 2003. The epidemiology of infectious salmon anemia in Scotland. In: International Response to Infectious Salmon Anemia: Prevention, Control, and Eradication: Proceedings of a Symposium; 3-4 September 2002; New Orleans, LA. (O Miller, RC

Cipriano, tech. coords.). *US Dept. Agriculture, Animal and Plant Health Inspection Service; US Dept. Interior, US Geological Survey; US Dept. Commerce, National Marine Fisheries Service (Washington, DC). Tech. Bull.* 1902:55-62. Available from: http://www.aphis.usda.gov/vs/aquaculture/isa-proceedings.pdf. Accessed 24 October 2005.

7. McClure CA, Hammell KL, Dohoo IR, Nerette P, Hawkins LJ. 2004. Assessment of infectious salmon anaemia virus prevalence for different groups of farmed Atlantic salmon, *Salmo salar* L., in New Brunswick. *J. Fish Dis.* 27:375-383.
8. Stagg RM. 2003. The eradication of an outbreak of clinical infectious salmon anemia from Scotland. In: International Response to Infectious Salmon Anemia: Prevention, Control, and Eradication: Proceedings of a Symposium; 3-4 September 2002; New Orleans, LA. (O Miller, RC Cipriano, tech. coords.). *US Dept. Agriculture, Animal and Plant Health Inspection Service; US Dept. Interior, US Geological Survey; US Dept. Commerce, National Marine Fisheries Service (Washington, DC). Tech. Bull.* 1902:111-124. Available from: http://www.aphis.usda.gov/vs/aquaculture/isa-proceedings.pdf. Accessed 24 October 2005.
9. Norwegian Animal Health Authority (Statens Dyrehelsetilsyn). 2002. *Contingency Plan for Control of Infectious Salmon Anaemia (ISA) in Norway*. Norwegian Animal Health Authority, Oslo. Available from: http:/dyrehelsetilsynet.mattilsynet.no/dyrehelse/bekjempelsesplaner/bekjempelsesplan_ila/isa_contingency_plan/. Accessed 24 October 2005.
10. New Brunswick Department of Agriculture, Fisheries and Aquaculture. 2000. *Bay of Fundy Marine Aquaculture Site Allocation Policy*. New Brunswick Department of Agriculture, Fisheries and Aquaculture, Fredericton. Available from: http://www.gnb.ca/0177/e-fundy.html. Accessed 24 October 2005.
11. Page FH, Chang B, Losier R, Greenberg D, McCurdy P. 2004. Water circulation and management of ISA in the southwest New Brunswick salmon culture industry. *Aquacul. Assoc. Canada Spec. Publ*. 8:64-68.
12. Page FH, Chang BD, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2005. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry of southern Grand Manan Island, Bay of Fundy. *Can. Tech. Rep. Fish. Aquat. Sci.* 2595.
13. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2006. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry of eastern Grand Manan Island, Bay of Fundy. *Can. Tech. Rep. Fish. Aquat. Sci.* 2621.
14. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2005. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry of Cobscook Bay, Maine and adjacent southwestern New Brunswick. *Can. Tech. Rep. Fish. Aquat. Sci.* 2598.
15. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2005. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry of Letete Passage, Back Bay, Bliss Harbour, and Lime Kiln Bay in southwestern New Brunswick. *Can. Tech. Rep. Fish. Aquat. Sci.* 2599.
16. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2006. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry in Passamaquoddy Bay, southwestern New Brunswick. *Can. Tech. Rep. Fish. Aquat. Sci.* 2622.
17. Greenberg DA, Shore JA, Page FH, Dowd M. 2005. A finite element circulation model for embayments with drying intertidal areas and its application to the Quoddy Region of the Bay of Fundy. *Ocean Modell.* 10:211-231.
18. Gustafson LL, Ellis SK, Beattie M, Chang BD, Dickey D, Robinson TL, Marenghi FP, Moffet P, Page FH. (submitted for publication). Hydrographics and the timing of infectious salmon anemia outbreaks among Atlantic salmon (*Salmo salar* L.) farms in the Quoddy region of Maine, U.S.A. and New Brunswick, Canada. *Prev. Vet. Med*.
19. Page FH, Chang BD, Greenberg DA. 2004. Fish Health and Oceanography Project of the Aquaculture Collaborative Research and Development Program: final project report. *Can. Tech. Rep. Fish. Aquat. Sci.* 2543.

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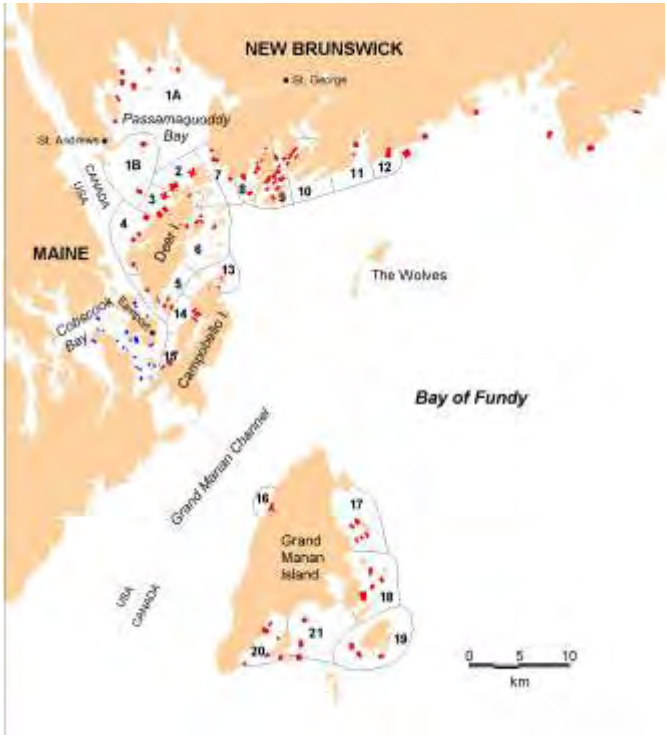
Mike Beattie

Water Movement and Fish Health in the Salmon Farming Industry of Southwestern New Brunswick

M. J. Beattie, S. M. McGeachy, B. D. Chang, and F. H. Page

Salmon farming in southwestern New Brunswick is the largest single agri-food business in Atlantic Canada. The industry has suffered significant losses from infectious salmon anemia (ISA) over the past decade and in response the New Brunswick Department of Agriculture, Fisheries and Aquaculture has developed and implemented a series of disease management and control strategies, and these have been coordinated with similar efforts in the adjacent United States. Local knowledge of water movement has contributed significantly to the developments on both sides of the Canada–United States border and it is anticipated that water movement will continue to be an important consideration in the ongoing evolution of disease management and control strategies. Additional applications of existing knowledge concerning water movements are identified, as are some priority research areas that will contribute to anticipated management needs.

Figure 1
Map of southwestern New Brunswick and adjacent areas of Maine, showing locations of salmon farms and Bay Management Areas (BMAs) in 2005. Salmon farms in New Brunswick are shown in red; farms in Maine are blue.



Introduction

The finfish aquaculture industry within southwestern New Brunswick (SWNB), Canada is dominated by Atlantic salmon (*Salmo salar*) farming. The salmon industry is the largest grossing single product agri-food industry in Atlantic Canada. In 2004 the industry consisted of 96 licensed salmon farms that produced 35,000 tonnes of salmon with a value of Can$175 million.[1] The industry is located throughout SWNB (Fig. 1) and in the adjacent eastern Maine area of the United States. The industry is also experimenting with the production of haddock, cod, and halibut. Shellfish, mainly blue mussels and scallops, and multi-trophic (salmon-mussels-seaweed) farming are also being explored.

There are several diseases that can, or have the potential to, affect farmed salmon in this area. The agents for several finfish diseases are known to attach to substrates that can be transported by water (Table 1).[2-6] The most economically-significant disease affecting farmed salmon in SWNB over the past decade is infectious salmon anemia (ISA). ISA first affected

Table 1. Some finfish diseases for which there is evidence of waterborne transport, including potential waterborne substrates.

Disease	Potential waterborne substrates
Bacterial diseases	
Furunculosis *(Aeromonas salmonicida)*	Lipids, sediments, sea lice, plankton
Bacterial kidney disease (BKD) *(Renibacterium salmoninarum)*	Feces, sediments
Yersiniosis/Enteric redmouth disease (ERM) *(Yersinia ruckeri)*	Feces
Viral diseases	
Infectious pancreatic necrosis (IPN)	Fish silage, sea lice
Infectious salmon anemia (ISA)	Mucous, feces, blood, sea lice
Pancreas disease (PD)	Blood, processing wastes
Infectious hematopoietic necrosis (IHN)	Mucous, feces

Information sources: Holm,[2] Evelyn et al.,[3] Traxler et al.,[4] Austin and Rayment,[5] and Rolland and Nylund[6]

farmed salmon in this area in 1996[7] and has been present every year since. Initially, four farms with 1995 year-class fish were infected. This rose to 25 farms in the 1997 year-class (Fig. 2). For the 1998 through 2004 year-classes, the number of infected farms varied between 24 and 3 farms with a trend toward fewer infected farms over time.

Infectious Salmon Anemia: Risk Factors

Research from various salmon farming areas of the world has identified several risk factors associated with the spread of ISA (Table 2).[8-11] Although most of the studies have not explicitly included water movement as a potential factor in their analyses, they have included the distance from an infected farm and this has consistently been a statistically significant factor in the presence of the disease.

In SWNB, farms are located relatively close together, particularly in comparison to areas such as Norway and Scotland. For example, within the Lime Kiln Bay, Bliss Harbour and Back Bay area there are 17 farms within a circular area of 5-km diameter (Fig. 3). Tidal excursion analyses[12-18] indicate that water is exchanged between many farms in the SWNB area and that some of the exchange is between farms located on opposite sides of the international border between Canada

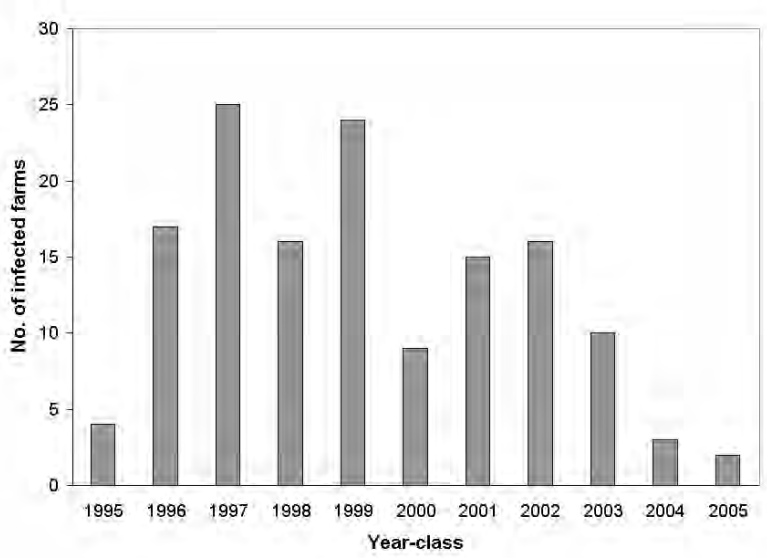


Figure 2
Number of salmon farms in southwestern New Brunswick infected with infectious salmon anemia (ISA), by year-class (up to December 2005).

and the United States (Fig. 4). Hence, water transport of pathogens is likely to be a significant mechanism for disease spread within and between salmon farms in the SWNB and eastern Maine areas.

The implication of disease exchange is supported by statistical analyses. A risk analysis study conducted by McClure et al.[11] on data from salmon farms within SWNB in 2002 indicated that the risk of developing ISA decreased with increasing distance between infected and uninfected farms. Specifically, when the distance was < 0.5 km the risk was 5.5 times greater than when the distance was ≥ 5 km.

Table 2. Risk factors associated with the spread of ISA in Norway and the southwestern New Brunswick area of Canada

Risk Factor	References
Norway	
• proximity to an ISA-infected farm	8,9
• proximity to a fish processing plant discharging improperly disinfected effluent	8,9
• delay in removal of infected stocks	8,9
• transfer of live fish between seawater sites	8
• co-existing multiple generations on a farm (multi-year-class site)	8
• increased distance from hatchery to farm site	9
• purchase of smolts from multiple hatcheries	8,9
• no separation of smolts from older fish for the first three months in seawater	8
Southwestern New Brunswick, Canada	
• large initial populations of fish in cage	10
• moderate fish density (number/volume)	10
• higher smolt weight (≥ 99 g)	11
• higher cumulative mortality during first year in seawater (i.e. reduction in the general health of smolts after transfer to seawater)	10,11
• higher frequency of weight sampling (additional stress)	10
• co-existing multiple generations on a farm (multi-year-class site)	10
• higher number of mortality dives per week (perhaps related to increased stress on fish or transfer of disease between sites if divers visited multiple sites with same gear)	10,11
• sites belonging to companies with more than one site (sharing of equipment or personnel between sites)	10
• delivery of dry feed to the site by the feed company	10,11
• a reduction in the health or productivity of fish in the first cold-water season	10
• high abundance of pollock in the cage	11
• less frequent sea lice treatments	10,11
• fewer months feeding moist feed after smolt transfer	10
• shorter distance between an uninfected site and an infected site	10,11
• processing vessels passing within 1 km of the site	11
• depth of nets < 10 m	11
• depth of water under cages > 3 m (possibly higher stress due to higher currents)	11

When the distance was 0.5 to 2 km, the risk was 2.0 times greater and when the distance was 2 to 5 km the risk was 1.2 times greater than when the distance was ≥ 5 km. The analysis also indicated that sites having harvest boats passing ≤ 1 km of the site had 7.5 times the risk of contracting ISA than sites where the vessels passed at a distance > 1 km.

Another study[19] explicitly utilized estimates of the tidal excursion pathways as proxies for disease transport in an analysis of risk factors associated with ISA outbreaks in SWNB and eastern Maine. That study found that about 11% of the variation in the occurrence of ISA infected farms was associated with a farm being downstream of an infected farm.

In addition to the water transport, the magnitude of the water current within a farm may influence the health status of caged fish and their susceptibility to disease. For example, McClure et al.[11] have suggested that strong water currents may cause fish, particularly smolts, to get pushed against the net, resulting in skin and fin damage, and this may reduce the healthiness of the fish and provide open sores for the ISA virus to enter.

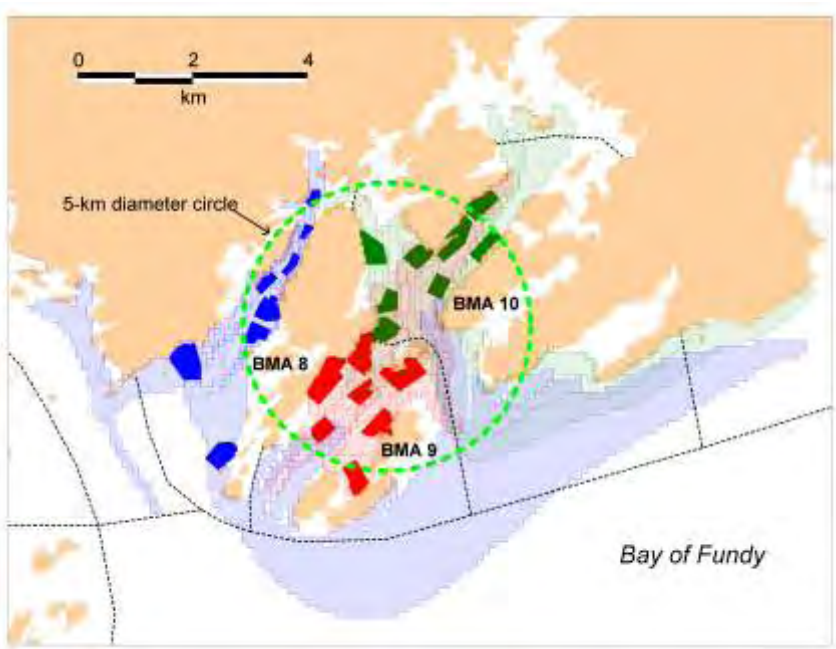
Infectious Salmon Anemia: Management Strategies

In SWNB the New Brunswick Department of Agriculture, Fisheries and Aquaculture (NBDAFA) is responsible for the development of fish health management regulations and control strategies for the aquaculture industry and has implemented several management and control strategies (Table 3). NBDAFA's fish health management program has focused on reducing the exposure of the caged salmon to the ISA virus. Biosecurity protocols (vessel traffic, harvesting procedures, etc.), a fish health monitoring program, early removal of fish from positive cages, vaccination, and a Bay Management Area strategy have all been implemented.[20,21]

Single year-class farming

One of the management practices implemented by NBDAFA is single year-class farming with fallowing between salmon generations. This approach was first used in Norway to control the spread of ISA and was introduced in SWNB in 1998. Prior to implementation of this strategy in SWNB, most farms were multi-year-class sites. However, when single year-class farming was first introduced, farms were allowed to have some "holdovers", i.e. a 3- to 4-month overlap of generations in the third year of production (no fallowing between generations) to allow for a constant supply of market fish during the late spring and early summer periods. As the industry has adjusted to single

Figure 3
Salmon farms in Bay Management Areas (BMAs) 8 (Back Bay), 9 (Bliss Harbour) and 10 (Lime Kiln Bay), with model-predicted tidal excursion areas. The small solid polygons are farm sites; larger shaded areas are tidal excursion areas. Farms and tidal excursion areas for BMA 8 are shown in blue, for BMA 9 in red, and for BMA 10 in green.



year-class farming, the number of holdover sites has declined considerably. For example, in 2002 there were holdovers at 20 farms in SWNB; in 2003, the number of holdover sites was reduced to 3; and in 2004 there was just one holdover site. New strategies have been introduced to allow maintaining of market fish for a longer time, while having a complete fallow, including having a few dedicated holdover sites in low risk areas and later smolt entry.

Canada–United States coordination

Another major aspect of the management strategy has been for NBDAFA to work closely with the United States Department of Agriculture to coordinate ISA management strategies between Maine and New Brunswick. Farms in SWNB are very close to farms in adjacent Cobscook Bay, Maine and tidal excursion analyses indicate that water is exchanged between farms that are on opposite sides of the Canada–United States border (Fig. 4).[14,19]

Because of this potential for pathogen exchange, considerable coordination of activities between the two jurisdictions has been developed. This includes coordination of: 1) farm service vessel movements (common harvest and feed vessels); 2) diagnostic testing and fish health surveillance programs (same farm companies and same private veterinarians, but different diagnostic laboratories); 3) site designation and depopulation criteria; and 4) smolt placement.

Bay Management Areas

Another major aspect of the ISA management strategy has been to divide the SWNB area into geographically distinct Bay Management Areas (BMAs) (Fig. 1). Although water movement was a relatively minor consideration when these BMAs were initially defined in 1998, efforts now underway to redefine the BMA boundaries and reduce the number of BMAs from 21 to about 6 have been largely

Table 3. Infectious salmon anemia (ISA) management and control strategies that have been implemented by the New Brunswick Department of Agriculture, Fisheries and Aquaculture (NBDAFA).

Management and Control Strategy	Year Implemented
ISA Surveillance Program including monthly private vet visits; testing of moribund fish by IFAT, PCR and virology	1997-1998
Cage by cage depopulation and fallowing	1998
Control and containment of processing wastes	1998
Disinfection of equipment, divers, etc.	1998
Smolt entry restrictions	1998
Single year-class restructure	1998-2002
Bay Management Area (BMA) fallowing	2003
Audit of processing plants	2003
Vessel traffic restrictions and harvest boat certification	2003
Wharf usage restrictions	2003

based on water movement information. One of the main goals of the refinement of the BMA structure is to have "buffer zones" (zones without farms) between adjacent BMAs. The rationale is that this will help to eliminate "stepping stones" for disease transfer between BMAs, as well as prevent cycling of disease between BMAs. When creating these "buffer zones" water movement will be a major criterion—ideally, there should be no overlap between tidal excursion areas of farms in adjacent BMAs, but in the event that cannot be attained, water movement information can be used to minimize such interactions. As an example, Figure 3 shows the model-predicted tidal excursion areas for farms in 3 adjacent BMAs in SWNB: BMA 8 (Back Bay), 9 (Bliss Harbour) and 10 (Lime Kiln Bay). The considerable overlap among the tidal excursion areas in these 3 BMAs suggests that they should be combined into one larger BMA. In practice, NBDAFA and the salmon industry have been treating these areas as one BMA for several years. The new BMAs will also incorporate knowledge of water transport between salmon farming areas in eastern Maine and SWNB.

Impacts of management measures

The implementation of these ISA management strategies and control measures has coincided with a reduction in the prevalence of ISA in SWNB (Fig. 2). For example, the number of farms with multiple year-classes on site (holdover of fish) declined from 20 sites in 2002 to 1 site in 2004. This corresponds with a reduction in the number of farms with ISA from 16 in the 2002 year-class to 3 in the 2004 year-class. Table 4 compares the numbers of depopulated cages or fish and the time until the first appearance of ISA on multi-year-class farms and single year-class farms in SWNB. It is clear that at single year-class farms there were fewer infected cages or fish, as well as a longer time before the onset of ISA infection.

Details on the numbers of infected farms and numbers of fish removed from four odd year-class Bay Management Areas (BMAs) since the 1997 year-class are given in Table 5. In all of these BMAs there has been a general decline in the numbers of fish removed in subsequent year-classes, and in the case of Seal Cove (one of the initial ISA outbreak locations in SWNB) there has been no ISA re-infection.

Future Role of Water Movement in Disease Management

As mentioned above, water movement considerations have played a significant role in the development and implementation of ISA management and control strategies in SWNB and eastern Maine. It is anticipated that water movement considerations will continue to play an important role in disease management in the

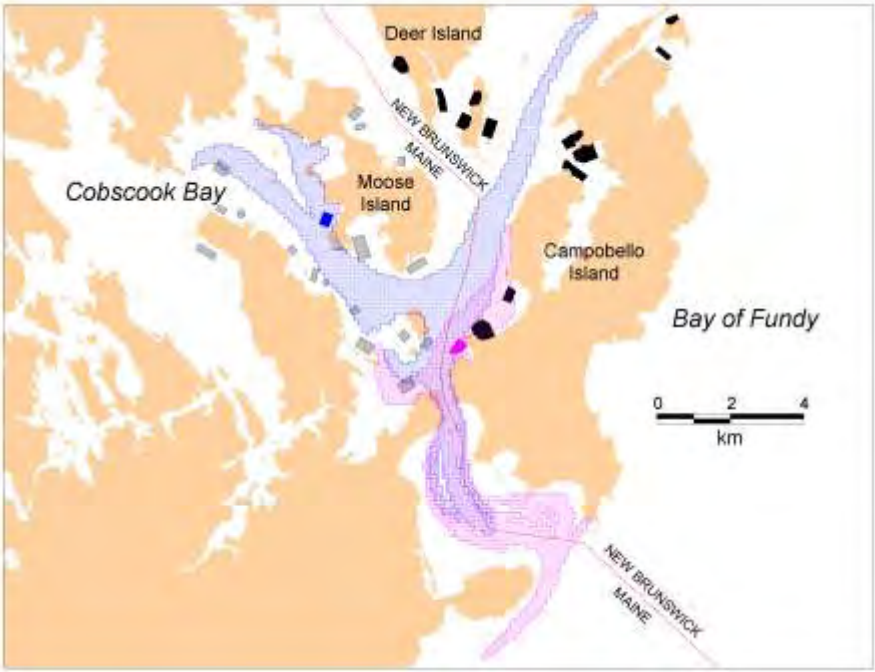


Figure 4
Two examples of model-predicted tidal excursion areas (one for a farm in New Brunswick and one in Maine), which overlap farm sites on both sides of the border. The two farm sites are shown as small polygons (pink for the site in New Brunswick and blue for the site in Maine). The larger pink and blue shaded areas are the tidal excursion areas for these two farms. Other farm sites are small black (New Brunswick) and grey (Maine) polygons.

future. There are several areas in which the existing water movement information can be incorporated into industry and fish health management practices to help reduce water- borne exchange of pathogens between farms. There are also some areas needing additional research.

Application areas

Existing knowledge of water movement can be used to:

- Help determine the zones of influence for each farm site: "downstream" effects for specific diseases. This would be useful for defining containment or control areas for new disease outbreaks, potential new farm sites, and Bay Management Area boundaries. It is recognized that the utility of these definitions will depend in large part on the accuracy of water and pathogen transport information.
- Establish if there is a best time within a tidal cycle to harvest diseased fish from an infected farm so that the spread of waterborne pathogens to other fish farms is minimized. This would be useful to companies when they plan and implement their harvesting strategies.
- Identify the strength of water currents and water exchange between farms for consideration in the placement of smolts. Some smolts may be more susceptible to disease than others and hence farmers may wish to put these smolts into farms that are least likely to experience exposure to pathogens and/or stressful environmental conditions.
- Help redesign local farm grids so cage orientation at each farm optimizes water exchange through cages and minimize exchange between cages.
- Help define the best routes for harvest vessels, in order to minimize the potential for disease spread to fish farms.

Research areas

Areas of research that should include consideration of water movement include:

- The development of sea lice population dynamics models that explicitly include water movement as an integral part of the models. Sea lice are often identified as a vector for the transfer of diseases between cages and farms and it would be useful to have the capability to predict the geographic location and timing of the spring "pulse" in sea lice populations so the industry could take this into consideration when planning their farm stocking schedules.
- The establishment of the probability of exposure to plankton blooms. Areas with historically high probabilities of plankton blooms could be avoided or abandoned, smolt entry times could be scheduled to avoid pending blooms, and

Table 4. Time from smolt entry until the first appearance of infectious salmon anemia (ISA) and the numbers of cages or fish depopulated at farms infected by ISA in southwestern New Brunswick, comparing multi-year-class and single year-class farms.

Year-class	1999		2002	
Farm type	Multi-year-class	Single year-class	Multi-year-class	Single year-class
Mean time from smolt entry until ISA infection	6 months	12 months	6.75 months	12 months
Mean number of cages or fish depopulated at infected farms	6 cages	3 cages	207,000 fish	103,000 fish

harvesting schedules could be adjusted to allow removal of market fish before a pending bloom occurs.

- The development of a water exchange based planning tool for multi-user group interactions.
- The prediction of the transport pathways of diseases from untreated effluent of wild fish processors and of invasive species or disease agents from untreated ballast water of ships. Such predictions could be incorporated into an analysis and planning tool that would identify interactions with potentially susceptible populations within the transport and dispersal domain.
- The estimation of the holding capacity or sustainable biomass within BMAs from both environmental and fish health perspectives. For the latter, water movement considerations may help reduce environmentally-induced stress to caged salmon through direct water movement linkages or indirectly through poor flushing and water quality processes. When combined with more refined water movement information concerning transport pathways between farms, the onset of disease within cages and its subsequent spread to other cages and farms may be reduced. The information may also contribute to minimizing the risk of transferring diseases from wild fish to cultured fish and vice versa.
- The use of water movement knowledge to help identify the location of possible disease reservoirs. This knowledge could be used to help place farms outside these areas or in areas that would not contribute pathogens to the reservoirs.
- Examination of water movement and disease spread patterns in an attempt to aid in the identification and quantification of disease risk factors. In general, data on the water transport pathways could be incorporated into statistical explorations identifying disease risk factors. More specifically, the knowledge of water transport pathways could be combined with the pathogenicity of ISA genotypes in a refinement of the statistical identification of risk factors. So far 12

Table 5. Numbers of farms infected by infectious salmon anemia (ISA) and numbers of fish removed due to ISA in four Bay Management Areas (BMAs) in southwestern New Brunswick.

BMA	No. of farms in BMA	1997 year-class		1999 year-class		2001 year-class		2003 year-class	
		No. of ISA-infected farms	No. of fish removed	No. of ISA-infected farms	No. of fish removed	No. of ISA-infected farms	No. of fish removed	No. of ISA-infected farms	No. of fish removed
10 Lime Kiln Bay	10	9	583,880	3	25,577	2	95,000	1	15,000
9 Bliss Harbour	7	6	583,990	4	215,707	2	418,700	1	15,000
8 Back Bay	8	3	114,760	3	33,000	1	62,030	1	15,000
20 Seal Cove	4	3	206,820	0	0	0	0	0	0
Totals	29	21	1,489,450	10	274,284	5	575,730	3	45,000

Table 6. Genotypes of infectious salmon anemia virus (ISAv) found in salmon farms in southwestern New Brunswick, based on comparisons of segments 6 and 8 of the virus in samples collected from October 2004 to September 2005. Initial testing of samples was by polymerase chain reaction (PCR), with follow-up testing of PCR-positive samples using the indirect fluorescent antibody technique (IFAT).

Segment 6	Segment 8	Number of Farms	Number of Bay Management Areas	Comments
H0	European	12	8	October 2004–May 2005
H2	North American	3	3	
H2	European	1	1	July 2005
H2a	North American	1	1	April–May 2005
H2b	North American	1	1	May–July 2005
H4	North American	5	4	strong IFAT; all 2003 year-class
H4a	North American	5	3	strong IFAT; all 2003 year-class
H4b	North American	2	2	weak IFAT; 2004 year-class
H8	European	1	1	July 2005; same site H2 European
HRPC!	North American	1	1	2004 year-class
HRPC2	North American	1	1	strong IFAT; 2003 year-class; October 2004–March 2005
HRPC3	North American	1	1	March 2005

genotypes have been identified in salmon farms in SWNB (Table 6), not all of which are virulent. Some of these genotypes are widespread, while others have only been detected at one farm. Some have been found in only one year-class, and most are only present during part of the year.

Conclusions

Water movement considerations have played a significant role in the development and implementation of ISA management and control strategies in SWNB and eastern Maine. It is anticipated that water movement considerations will continue to play an important role in disease management in the future.

References

1. Statistics Canada. 2005. *Aquaculture Statistics: 2004.* Catalogue no. 23-222-XIE. Statistics Canada (Ottawa, ON). Available online at www.statcan.ca/english/freepub/23-222-XIE/23-222-XIE2004000.pdf. Accessed 4 January 2006.
2. Holm JA. 1999. Disease prevention and control. In: *Manual of Salmonid Farming* (S Willoughby), p. 195-259. Fishing News Books, Oxford.
3. Evelyn TPT, Kent ML, Poppe TT, Bustos P. 1998. Bacterial diseases. In: *Diseases of Seawater Netpen-Reared Salmonid Fishes* (ML Kent and TT Poppe, eds.), p. 17-35. Fisheries and Oceans Canada, Pacific Biological Station, Nanaimo.
4. Traxler GS, Kent ML, Poppe TT. 1998. Viral diseases. In: *Diseases of Seawater Netpen-Reared Salmonid Fishes* (ML Kent and TT Poppe, eds.), p. 36-45. Fisheries and Oceans Canada, Pacific Biological Station, Nanaimo.
5. Austin B, Rayment JN. 1985. Epizootiology of *Renibacterium salmoninarum*, the causal agent of bacterial kidney disease in salmonid fish. *J. Fish Dis.* 8:505-509.

6. Rolland JB, Nylund A. 1998. Infectiousness of organic materials originating in ISA-infected fish and transmission of the disease via salmon lice (*Lepeophtheirus salmonis*). *Bull. Eur. Assoc. Fish Pathol.* 18:173-180.

7. McGeachy SM, Moore MJ. 2003. Infectious salmon anemia in New Brunswick: an historical perspective and update on control and management practices (1997-2002). In: International response to infectious salmon anemia: prevention, control, and eradication: proceedings of a symposium; 3-4 September 2002; New Orleans, LA. (O Miller, RC Cipriano, tech. coords.). *US Dept. of Agriculture, Animal and Plant Health Inspection Service; US Dept. Interior, US Geological Survey; US Dept. Commerce, National Marine Fisheries Service (Washington, DC). Tech. Bull.* 1902:145-153. Available online at www.aphis.usda.gov/ vs/aquaculture/ isa-proceedings.pdf. Accessed 24 October 2005.

8. Vågsholm I, Djupvik HO, Willumsen FV, Tveit AM, Tangen K. 1994. Infectious salmon anaemia (ISA) epidemiology in Norway. *Prev. Vet. Med.* 19:277-290.

9. Jarp J, Karlsen E. 1997. Infectious salmon anaemia (ISA) risk factors in sea-cultured Atlantic salmon *Salmo salar*. *Dis. Aquat. Org*. 28:79-86.

10. Hammell KL, Dohoo IR. 2005. Risk factors associated with mortalities attributed to infectious salmon anaemia virus in New Brunswick, Canada. *J. Fish Dis.* 28:651-661.

11. McClure CA, Hammell KL, Dohoo IR. 2005. Risk factors for outbreaks of infectious salmon anaemia in farmed Atlantic salmon, *Salmo salar*. *Prev. Vet. Med.* 72:263-280.

12. Page FH, Chang B, Losier R, Greenberg D, McCurdy P. 2004. Water circulation and management of ISA in the southwestern New Brunswick salmon culture industry. *Aquacul. Assoc. Canada Spec. Publ*. 8:64-68.

13. Page FH, Chang BD, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2005. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry of southern Grand Manan Island, Bay of Fundy. *Can. Tech. Rep. Fish. Aquat. Sci.* 2595.

14. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2005. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry of Cobscook Bay, Maine and adjacent southwestern New Brunswick. *Can. Tech. Rep. Fish. Aquat. Sci.* 2598.

15. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2005. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry of Letete Passage, Back Bay, Bliss Harbour, and Lime Kiln Bay in southwestern New Brunswick. *Can. Tech. Rep. Fish. Aquat. Sci.* 2599.

16. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2006. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry of eastern Grand Manan Island, Bay of Fundy. *Can. Tech. Rep. Fish. Aquat. Sci.* 2621.

17. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2005. Water circulation and management of infectious salmon anemia in the salmon aquaculture industry in Passamaquoddy Bay, southwestern New Brunswick. *Can. Tech. Rep. Fish. Aquat. Sci.* 2622.

18. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2006. Application of a tidal circulation model for fish health management of salmon farms in the Grand Manan Island area, Bay of Fundy. *Bull. Aquacul. Assoc. Canada* 105-1:22-33.

19. Gustafson LL, Ellis SK, Beattie MJ, Chang BD, Dickey DA, Robinson TL, Marenghi FP, Moffet PJ, Page FH. (submitted for publication). Hydrographics and the timing of infectious salmon anemia outbreaks among Atlantic salmon (*Salmo salar* L.) farms in the Quoddy region of Maine, USA and New Brunswick, Canada. *Prev. Vet. Med.*

20. NB DAFA.2000. *Bay of Fundy Marine Aquaculture Site Allocation Policy*. New Brunswick Department of Agriculture, Fisheries and Aquaculture, Fredericton, NB. Available online at www.gnb.ca/0177/e-fundy. html. Accessed 24 October 2005.

21. NBSGA. 2004. *New Brunswick Salmon Growers Environmental Policy and Codes of Practice.* New Brunswick Salmon Growers Association, Letang, NB. Available online at www.nbsga.com/articles/2004-Codes%20and%Policy%20June%202004%20Complete %20Document%20(1).pdf. Accessed 4 January 2006.

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Hydrographics and the Epidemiology of ISA: Findings from a High-Risk Region in Maine and New Brunswick

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The history and patterns of infectious salmon anemia (ISA) incidence in the Quoddy region of Maine and New Brunswick have raised questions about the role of waterborne transmission in the spread of disease. We summarize our observations and epidemiologic efforts to understand the importance of hydrographics in the dispersal of the ISA virus (ISAv). Included are descriptions of year-class carryover and infection summaries, virus survival and seawater detection studies, and a retrospective analysis of the relationship between ISA outbreaks on Atlantic salmon sites connected by a single tidal exchange. The resulting findings help to solidify concerns that waterborne transmission of ISAv is a viable mechanism of disease transfer in the Quoddy region, though only one component of a complicated hierarchy of risk factors. We conclude by describing a plan to re-configure bay management zones to achieve better hydrographic separation between zones and farmed salmon year-classes in Maine and New Brunswick.

Introduction

Oceanography is central to the study of marine organisms and ecosystems. The integration of oceanographic concepts into the study of aquatic animal pathogen movements, however, is not commonplace. The importance of water circulation to regulatory boundaries, biosecurity protocols, disease containment plans, and ultimately the dissemination of waterborne pathogens, is recognized,[1] but hydrographic information pertinent to pathogen dispersal is not uniformly applied. Contributing to this problem is the incomplete availability of information on localized tidal exchange and pathogen persistence characteristics. Also contributing is the scarcity of scientific efforts to gather and test evidence of waterborne dispersal of disease in the field. Oceanographic and epidemiologic resources centered on the Quoddy region of Maine and New Brunswick offered a unique opportunity to clarify the importance of hydrographics to the local spread of ISA. We provide an overview of our efforts to chronicle disease outbreaks, assess pathogen survival and dispersal in the water column, and empirically test modeled movements of tidally-dispersed particles against observed outbreaks of ISA in the field.

History and Local Conditions

ISA was first identified in Norway in 1984 and subsequently detected in farmed Atlantic salmon *(Salmo salar)* in southwestern New Brunswick, Canada in 1996.[2]

By 1997, the viral disease had spread to several farms in a three-bay management area in New Brunswick; in 1999, 24 Canadian sites were affected by ISA, including 9 of 12 sites on the west side of Deer Island.[3] Some of these sites were as close as 2 km to US farmed salmon operations in neighboring Cobscook Bay.

The proximity of ISAv-infected sites raised concerns for the US farmed salmon industry for several reasons. First, since the early 1990s, some companies have operated salmon farms on both sides of the international border (Fig. 1). Boats, fish, equipment, and personnel historically have moved across the marine border freely. Secondly, water circulation models[4,5] suggested that dilute concentrations of virus particles released from southern Deer Island could reach US salmon farms via the mouth of Maine's Cobscook Bay. The likelihood of waterborne transmission appeared substantial. Third, sea lice (*Lepeophtheirus salmonis* and possibly *Caligus* spp.), which can move on tidal currents, are documented vectors of ISAv.[6,7]

Figure 1
The bi-national Atlantic salmon farming region of interest in Maine and New Brunswick. Basemap data provided by ESRI.

In 2001, ISA viral infection was detected in Maine[8] at salmon sites in Cobscook Bay (Fig. 2). In December 2001, the Secretary of Agriculture declared an ISA disease emergency, which permitted funds to be allocated to USDA's Animal and Plant Health Inspection Service (APHIS) to assist Maine's salmon industry over a 2-year period. Between the beginning of the outbreak and the emergency declaration, a group of fish health veterinarians and biologists developed ISA disease control standards based on existing New Brunswick ISA policies and practices implemented by the Norwegian salmon industry. The final standards were published in early 2002 as the USDA APHIS Infectious Salmon Anemia Disease Standards.[9]

The APHIS ISA Program was implemented in early January 2002 in partnership with the Maine Department of Marine Resources (DMR), which regulates site leasing and has the legal authority to issue quarantines and depopulation orders. Maine DMR also oversees enforcement resources for Maine's nearshore marine waters. Because fish in 16 of 17 active cage sites in Cobscook Bay had already been reported to be infected with ISAv by the end of 2001, a strategy of com-

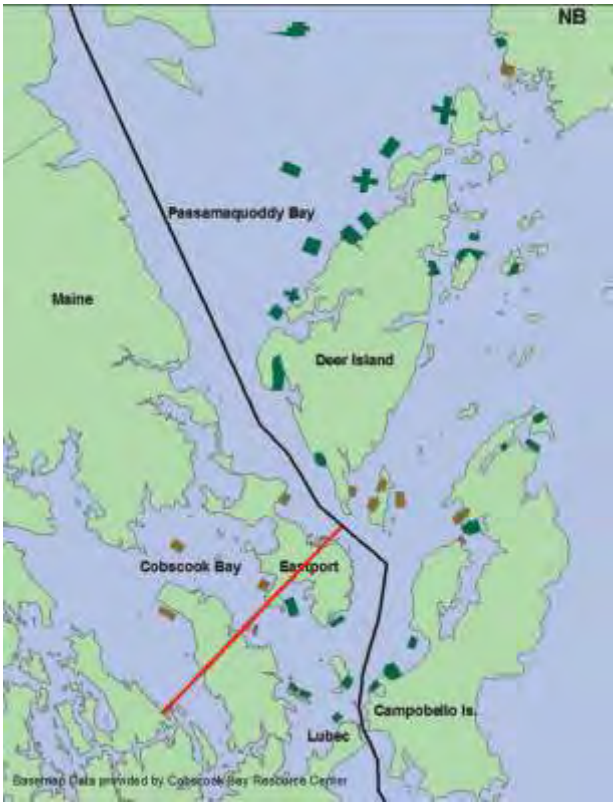


Figure 2
Atlantic salmon farms active during 2002-2004 in the Cobscook and Passamaquoddy region. The international boundary is represented by a black line. The red line depicts a bay management boundary arbitrarily drawn to separate year-classes in the United States. Sites with 2002 entry year-class fish are shown in green. Sites holding alternate year-class fish during the same time period are shown in brown. Basemap data were provided by the Cobscook Bay Resource Center.

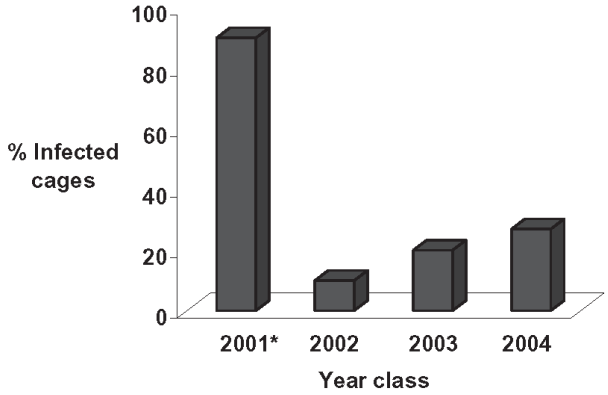
plete, bay-wide depopulation was undertaken immediately. Over 1 million salmon had either died or been depopulated prior to the program's initiation and an additional 1.6 million first-year fish were culled in the program's first 2 weeks. Compensation, covering 60% of the industry's average cost of production, was paid on the 1.6 million fish destroyed under the program. The bay-wide eradication was widely presumed to have helped prevent the virus from traveling farther west along the Maine coast to approximately 20 other salmon farming sites situated in marine waters up to 100 miles west of Cobscook Bay. After the depopulation, all cages, nets, feed systems, other farming equipment, and vessels were cleaned and disinfected during a planned 3.5-month fallow of the entire bay. Six farms in Cobscook Bay's southern half, delineated by an arbitrary line designed to split the region into two management zones (Fig. 2), were repopulated in May 2002 at reduced density and with a single-year class of salmon.

Both the industry and USDA committed to strict biosecurity guidelines, since many risk factors identified in the transmission of ISA are related to biosecurity issues.[9] These include handling and disposal of processing wastes, blood, and stun water; mortality removal and disposal; controlling movements of vessels, equipment, and human traffic; maintenance and use of disinfection stations; and fallowing and sea-lice management. Mandatory monthly surveillance and integrated pest management plans were imposed by regulators and/or voluntarily adopted by industry. Vaccination was voluntary, but universally employed, in those first few years.

Despite a succession of new cases in neighboring New Brunswick, sites in Maine remained ISA-free for 13 months after the spring 2002 re-stocking. However, in June 2003, surveillance testing of moribund fish and/or fresh mortalities at all US marine sites indicated low-level ISAv infection in salmon from one cage at each of two sites in Cobscook Bay. Fish in the infected cages were harvested and/or culled within 2 weeks of detection. Fish in three more cages on those two sites were declared infection-positive by the end of 2003. However, both farms brought the majority of their fish to market successfully, without any indication of further spread of infection to neighboring farms that year.

The next year-class (2003) to enter the bay was sited in the northern zone of Cobscook Bay (Fig. 2), and fared similarly to the class before it. ISAv was detected 13, 14 and 15 months post-entry at the three 2003 year-class sites. In a situation referred to as 'carryover', the 2003 year-class had been stocked 8 months prior to full removal of the 2002 year-class in the region (though the classes were separated by the arbitrary zonation system shown in Figure 2). However, because the line separating the management zones into north and south sectors was based on practical rather than hydrographic considerations, actual separation between the two year-classes was not achieved. Consequently, the overlap between classes resulted in an effective carryover of previous year-class fish, and a holdover period that ended 6 months prior to the initial ISAv detection in the 2003 year-class. Similar conditions (again with an overlap of year-classes across the north-south zone boundary in Cobscook Bay) led to even earlier detection (during the effective holdover) and more costly removals

Figure 3
Proportion of ISAv infected cages in Maine's high-risk Cobscook and Passamaquoddy region, by year-class. 2001* (pre-program) figures were estimated from clinical observations in combination with results from voluntary laboratory submissions. All other year-class figures are based on laboratory results from the ISA active surveillance program. Total stocked cages varied by year class: 140 cages in 2001, 88 in 2002, 80 in 2003, and 52 in 2004.



for the following 2004 year-class.

Figures 3 and 4 are graphical depictions of the losses suffered over the 2001 to 2004 year-classes in Maine's high-risk Cobscook and Passamaquoddy region. Fish were not stocked in the region in 2005, in anticipation of the implementation of a more effective bay management strategy in 2006. Figure 3, which shows the proportions of infected *cages*, demonstrates strong improvement since the implementation of the control program in 2002. However, a review of the proportion of infected *sites* over the same time-span (Fig. 4) shows a conflicting message of minimal effect. These contrasting patterns, together with the knowledge that many other documented risks had been successfully controlled, initiated further investigation into the role of waterborne transmission in the region. Evolving discussions with oceanographers[4,10,11] confirmed suspicions that existing bay management zones were not hydrographically distinct. The emerging patterns also provided indirect demonstration of the potential role of holdovers, in regions unified by tidal exchange, in the spread of virus from older, carrier, or pathogen-tolerant fish to more recently introduced, naïve year-classes.

Environmental Sampling and Pathogen Survival

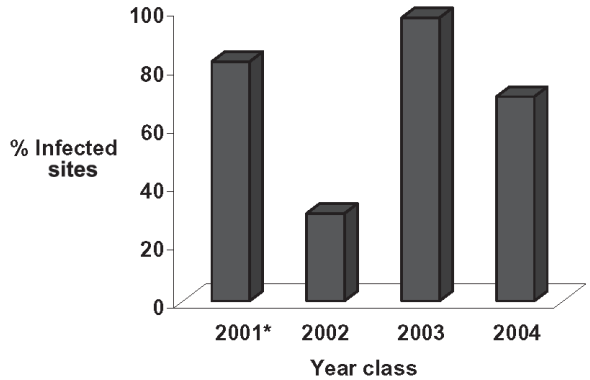
During the initial culling activities associated with the ISA Program, efforts were undertaken to attempt to detect ISAv from a wide variety of environmental samples including a number of alternate fish species,[12] boat hulls, water, nets, cage floats, and pontoons. Evidence of ISAv was isolated through cell culture or detected by molecular techniques (RT-PCR) in each of these broad categories, except for the nets.

Later, in an effort to learn more about ISAv, the disease it produces, and more effective means of detection and control, USDA APHIS Veterinary Services, Micro Technologies, Inc., the NB Department of Agriculture, Fisheries and Aquaculture (NB DAFA), and a cooperative Deer Island salmon producer partnered to observe, sample, test, and research an ongoing outbreak at a site on the west shore of Deer Island. We visited the site 9 times between January and June of 2003 and sampled water, fish, boat hulls, cage surfaces, mussels, and sediments during each visit. Genetic material was repeatedly detected by RT-PCR in all sample types except mussels and sediments (Table 1).

In a complementary study, we learned that viral survival in non-sterile seawater varied greatly with temperature, ranging from 1 to 2 days at 16°C to up to 14 days at 4°C.[13] A simultaneous analysis of water sampled from within and around the perimeter of a clinically-diseased site recovered RT-PCR evidence of ISAv genetic material dispersed through the water column up to 1.5 km from the site.[13]

All of these findings, coupled with the need for some thorough epidemiological and statistical support for a more realistic management strategy based on effective zonation, led us to pursue the empirical study we address next.

Figure 4
Proportion of ISAv infected sites by year-class in Maine's high-risk region of Cobscook and Passamaquoddy bays. 2001* sites preceded the ISA Program. Sites were designated infected if one or more cages were found positive by two laboratory tests (culture, RT-PCR, and/or IFAT) in two fish. Total sites varied by year class: 8 sites in 2001, 6 in 2002, 3 in 2003, and 4 in 2004.



Empirical Field Study

Anecdotal evidence supports the inference of an active role for tidal dispersal of virus, and there is labora-

tory rationale for waterborne transmission. Risk factor studies from both sides of the Atlantic also strongly imply 'proximity to infected farms' as a risk factor for ISA.[14-17] A goal of risk factor studies is to explore a wide array of general risks. In the case of ISA transmission factors, water movement as a general category (proximity) would easily qualify in this respect. Proximity risks are typically approximated using straight-line (or elliptical) distances from a source of infection, and a site's disease status is often evaluated at a single-point-in-time or as a cumulative measure. However, proximity risks may be mediated by hydrographic exchange of waterborne virus, or by 'external' issues that could fall under the broader category of proximity (e.g., mixing of crew off-site, sharing of equipment and vessels, predators or parasites, overlapping traffic patterns, and shared environmental stressors).

To more closely define the specific role of hydrographics in the field transmission of ISA, we conducted a retrospective analysis of disease incidence in the Quoddy region. In this analysis we explored the apparent impact of modeled water circulation patterns specific to the region, against month-to-month changes in the spatial incidence of disease over time.[18] The hydrographics were still approximated (e.g. focusing on one rather than two or three tidal excursions), but based on the best available oceanographic knowledge of tidal influence in the region.[4,5,11] The details of this study will be reported elsewhere,[18] but a summary of the highlights follows.

Our goal was to test for spatiotemporal evidence that disease distribution might track water circulation patterns. We looked at the location and timing of ISA outbreaks (cage-by-cage and month-by-month) across 32 sites in the Quoddy region (Fig. 2). We focused on the spring-entry 2002 year-class, primarily because of the uniform availability of records, and defined an outbreak case as any cage testing positive for ISAv in 2 fish by 2 tests under the ISA surveillance programs. The Quoddy region is a historically active salmon farming region that has been heavily impacted by ISA for a number of years. Seventy-eight percent of our 32 study sites dealt with ISAv in one or more cages at some point during the targeted production cycle.

Hydrographic relationships between sites were estimated from tidal excursion maps generated from Fisheries and Oceans Canada's tidal circulation and particle transport models.[4,5,11] These models predict water direction and strength from the principal lunar tide and local bathymetry, and have been substantiated by years of field data from current meters and drifters. From these maps we generated a list of farms considered 'upstream' (or within one tidal excursion) of each of our 32 study sites. In the example shown in Figure 5, models suggest that Site A (shown in black) receives tidal flow from one upstream site (in green), and distributes tidal flow to six downstream sites (in red) within a single 12.4-hour tidal cycle.

We compared disease incidence data to proximity risk as defined by the tidal exchange models using time-series cross-sectional (TSCS) regres-

Table 1. Proportions of positive results from environmental samples submitted for ISAv RT-PCR testing.

Sample type	Number RT-PCR +	Total submitted	Proportion
Boat hulls	5	15	0.33
Sediments, mussels	0	38	0
Sea lice	43	44	0.98
Cage pontoons	24	45	0.53
Seawater, on-site	23	45	0.51
Seawater, off-site	30	85	0.35
Salmon	207	285	0.73

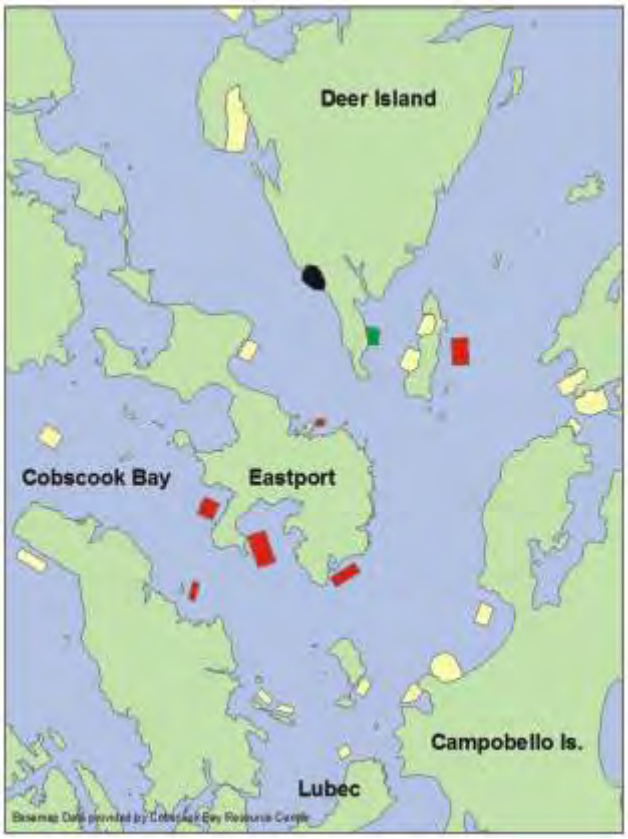
sion. If we had only one observation per site (e.g., ISA status at the end of the cycle), a standard regression format would be appropriate. Similarly, if we monitored one site repeatedly over time (e.g., recording the number of new ISA cages each month), a time series analysis could be a useful analytic tool. But because data for multiple months existed for each of multiple sites, TSCS regression was a more constructive approach. TCSC was originally designed as an econometrics method—to look at factors driving economic growth, or political uprisings, or recessions, across nations or regions and over time. It has also been used in the human medical field (e.g., to identify socioeconomic factors relevant to health).[19]

Our findings indicated that the number of new ISA outbreak cages detected on a given site in a given month was associated with several predictive factors.[18] Statistically significant predictors of new outbreaks each month included 1) the number of outbreaks on-site in previous months, 2) the number of cages susceptible on-site at the beginning of the month, as well as 3) whether holdovers from a previous year-class were on-site during the stocking of the 2002 year-class. However, the variable with the strongest influence was the number of new outbreaks *upstream* in the same month. We reviewed this relationship on a week-by-week basis to find that the greatest predictive association for downstream outbreaks fell on upstream events 2 to 3 weeks prior. This lag period corresponds well with viral incubation periods described in ISAv laboratory trials.[20,21]

We also broke the dataset down, site by site, to identify hot-spots or specific sites where the timing of outbreaks was best predicted by hydrographics. Using this approach, we found two clusters: along northwestern Deer Island and the eastern tip of Estes Head. These were locations where upstream events predicted downstream outbreaks, over time, more often than chance would allow. Note that a number of simultaneous events had to come together to be included in this list: an infected and shedding population upstream, a susceptible population downstream, and ultimately a downstream outbreak—all occurring within the 'correct' frame of time. Such a sequence would need to happen with some regularity over time to show up as a cluster, or hot-spot, in this analysis.

It is important to recognize that the predictive power (R^2) of our model was low. Statistical studies can indicate two things about relationships. They can relate the regularity (or presumed repeatability) of the effect across the dataset (typically disclosed in the *p* value). They can also indicate the strength of the relationship (reflected in odds ratios, or the R^2 value, or other similar measure). For example, in humans, one's chosen profession is thought to predict longevity to some degree. This relationship may occur regularly across datasets, but many other factors (e.g., lifestyle and genetics) will control a portion of the total risk. Our situation with ISA transmission and water movement is similar. Hydrographic proximity reveals some, likely reliable, information about ISA incidence in this region, but

Figure 5
An example of a site (Site A, in black) with different modeled upstream and downstream influences. The model identifies one upstream site (in green) within a single tidal exchange, and 6 downstream sites (in red) within the same tidal period. Basemap data were provided by the Cobscook Bay Resource Center.



other factors (many unaccounted for in our analysis) play a role as well.

Factors of possible or probable relevance that were not tested in our study include disease severity or chronicity, biosecurity, parasites, wild fish presence, storm events, fishing/dragging seasons, genetics and vaccination histories of the salmon, and husbandry or site characteristics that could influence immune response. The latter category might include high energy vs. low energy sites, stocking densities, transfers or splits, predation, periods of extreme or stunted growth, or concurrent disease. Additionally, over the last 16 months, surveillance, diagnosis and control efforts have been significantly complicated by the emergence of several new genotypes of the ISA virus, including an apparently non-pathogenic genotype closely resembling a similar genotype in Europe. We're also concerned that we have yet to identify an environmental reservoir for the virus. Wild salmonids—as speculated by researchers across the Atlantic[22,23]—or sea lice, or other species of animals, may play that role.

Conclusion

From this series of studies and observations, we concluded that hydrographics likely do play a role in the transmission of ISA in the Quoddy region, though the influence appears to be limited in degree. Because even a few outbreaks are too many, the information gained through this analysis has been used to further the re-configuration of regional management strategies. These epidemiological findings, supported by tidal modeling and GIS-assisted evaluation of environmental risk factors have led to a new bi-national bay management strategy to be implemented starting in spring 2006. The aim of this strategy is to include, within a single management zone, all of Cobscook and Passamaquoddy bays, and sites off Deer and Campobello islands, as well as the St. Andrews area. In defining the boundaries of bay management zones hydrographically, we hope to reduce the chances of hydrographic spread of ISAv from one bay management zone to the next. A goal is to delay the onset of the index case of ISA in a given production cycle, and subsequently improve chances of a full production cycle for each new year class. Additionally, given the predisposition of ISAv (as an orthomyxovirus) for genetic shift and drift, it is sensible to limit opportunities for future mixing of virus genotypes and environments as much as possible.

References

1. Murray AG, Peeler EJ. 2005. A framework for understanding the potential for emerging diseases in aquaculture. *Prev. Vet. Med*. 67: 223-235.
2. Mullins JE, Groman D, Wadowska D, 1998. Infectious salmon anemia in salt water Atlantic salmon (*Salmo salar* L.) in New Brunswick, Canada. *Bull. Eur. Assoc. Fish Pathol.* 18:110- 114.
3. McGeachy SM, Moore M J. 2003. Infectious salmon anemia in New Brunswick: an historical perspective and update on control and management practices (1997–2002). In: International response to infectious salmon anemia: Prevention, Control, and Eradication: Proceedings of a Symposium; 3–4 September 2002; New Orleans, LA (O Miller and R Cipriano, tech. coords.). *US Dept. Agriculture, Animal and Plant Health Inspection Service; US Dept. Interior, US Geological Survey; US Dept. Commerce, National Marine Fisheries Service (Washington, DC). Tech. Bull.* 1902:145–153.
4. Page FH, Chang BD, Greenberg DA. 2004. Fish health and oceanography project of the Aquaculture Collaborative Research and Development Program: final project report. *Can. Tech. Rep. Fish. Aquat. Sci.* 2543: vi+47 p.
5. Greenberg DA, Shore JA, Page FH, Dowd M. 2005. A finite element circulation model for embayments with drying intertidal areas and its application to the Quoddy Region of the Bay of Fundy. *Ocean Model.* 10:211-231.
6. Nylund A, Hovland T, Hodneland K, Nilsen F, Lovik P. 1994. Mechanisms for transmission of infectious salmon anaemia (ISA). *Dis. Aquat. Org*. 19:95-100.

7. Rollaand JB, Nylund A. 1998. Infectiousness of organic materials originating in ISA-infected fish and transmission of the disease via salmon lice (*Lepeopththeirus salmonis*). *Bull. Eur. Assoc. Fish Pathol.* 18:173-180.

8. Bouchard DA, Brockway K, Giray C, Keleher W, Merrill PL. 2001. First report of infectious salmon anemia (ISA) in the United States. *Bull. Eur. Assoc. Fish Pathol.* 21:86-88.

9. USDA APHIS (United States Department of Agriculture Animal and Plant Health Inspection Service) Veterinary Services, Maine Department of Marine Resources and Maine Aquaculture Association. 2002. *Infectious Salmon Anemia Program Standards*. April 30, 2002.

10. Brooks DA. 2004. Modeling Tidal Circulation and Exchange in Cobscook Bay, Maine. Available online at cobscook.tamu.edu/review/Cobscook.NEN_final.pdf.

11. Chang BD, Page FH, Losier RJ, Greenberg DA, Chaffey JD, McCurdy EP. 2005. Water circulation and management of infectious salmon anemia in the salmon culture industry of Cobscook Bay, Maine and adjacent southwestern New Brunswick. *Can. Tech. Rep. Fish. Aquat. Sci*. 2598: iii+54 p.

12. MacLean SA, Bouchard DA, Ellis SK. 2002. Survey of nonsalmonid marine fishes for detection of Infectious Salmon Anemia virus and other salmonid pathogens. In: International Response to Infectious Salmon Anemia: Prevention, Control, and Eradication: Proceedings of a Symposium; 3–4 September 2002; New Orleans, LA (O Miller and R Cipriano, tech. coords.). *Tech. Bull.* 1902:135–143. Available online at www.aphis.usda.gov/vs/aquaculture/isa-proceedings.pdf.

13. Giray C, Bouchard DA, Brockway KA, Merrill PL. 2004. Environmental persistence of infectious salmon anemia virus. In: *Abstracts for the Proceedings of the Second Maine Atlantic Salmon Technical Advisory Committee Research Forum.* University of Maine, Orono, Maine, USA, 7 January 2004, p. 12.

14. Jarp J, Karlsen E. 1997. Infectious salmon anaemia (ISA) risk factors in sea-cultured Atlantic salmon *Salmo salar*. *Dis. Aquat. Org*. 28:79-86.

15. Vagsholm I, Djupvik HO, Willumsen FV, Tveit AM, Tangen K. 1994. Infectious salmon anaemia (ISA) epidemiology in Norway. *Prev. Vet. Res*. 19:277-290.

16. Gustafson LL, Ellis SK, Bartlett CA. 2005. Using expert opinion to identify risk factors important to infectious salmon anemia (ISA) outbreaks on salmon farms in Maine, USA and New Brunswick, Canada. *Prev. Vet. Med*., 70(1-2):17-28.

17. McClure CA, Hammell KL, Dohoo IR. 2006. Risk factors associated with outbreaks of infectious salmon anemia in farmed Atlantic salmon, *Salmo salar*, in New Brunswick, Canada. *Prev. Vet. Med.* (in press).

18. Gustafson LL, Ellis SK, Beattie MJ, Chang BD, Dickey DA, Robinson TL, Marenghi FP, Moffett PJ, Page FH. In submission. Hydrographics and the timing of infectious salmon anemia outbreaks among Atlantic salmon (*Salmo salar L.*) farms in the Quoddy region of Maine, USA and New Brunswick, Canada. Submitted to *Prev. Vet. Med.* July, 2005.

19. Shi L, Macinko J, Starfield B, Xu J, Regan J, Politzer R, Wulu J. 2004. Primary care, infant mortality, and low birth weight in the states of the USA. *J. Epidemiol. Community Health* 58:374-380.

20. Totland GK, Hjeltnes BK, Flood PR. 1996. Transmission of infectious salmon anaemia (ISA) through natural secretions and excretions from infected smolts of Atlantic salmon *Salmo salar* during their presymptomatic phase. *Dis. Aquat. Org.* 26:25-31.

21. Mikalsen AB, Teig A, Helleman AL, Mjaaland S, Rimstad E. 2001. Detection of infectious salmon anaemia virus (ISAV) by RT-PCR after cohabitant exposure in Atlantic salmon *Salmo salar. Dis. Aquat. Org.* 47:175- 181.

22. Raynard RS, Murray AG, Gregory A. 2001a. Infectious salmon anaemia virus in wild fish from Scotland. *Dis. Aquat. Org*. 46:93-100.

23. Devold M, Falk K, Dale OB, Krossoy B, Biering E, Aspehaug V, Nilsen F, Nylund A. 2001. Strain variation, based on the hmagglutinin gene, in Norwegian ISA virus isolates collected from 1987 to 2001: indications of recombination. *Dis. Aquat. Org*. 47:119-128.

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Dario Stucchi

Modelling the Transport and Dispersion of IHN Pathogens in the Broughton Archipelago, British Columbia

D. J. Stucchi, R. F. Henry, and M. G. G. Foreman

Numerical circulation models of the tides and the mean flow field in the Broughton Archipelago, BC are presented and described. Tidal currents are energetic in the outer regions of the model domain and at constrictions and sills inside the archipelago. The mean seaward surface flow driven by freshwater discharge is a prominent feature of the sub-tidal circulation. Model simulations of the transport and dispersion of IHN viral particles released from several finfish farm sites showed that the surface estuarine flow transports the viral particles many kilometres seaward, but the details of the distances travelled and pathways are site specific. Distances travelled during the two-day lifetime given for viral particles ranged from 3 to 33 km depending upon location of the release site. The simulations showed that within the two-day lifetime or time window used for the IHN viral particles some finfish farm sites were directly downstream of, or connected to, the site that was the source of the pathogen.

Background

On the British Columbia mainland coast and to the east of Queen Charlotte Strait lies a group of islands commonly referred to as the Broughton Archipelago (Fig. 1). The archipelago is composed of several large islands, notably its namesake and Gilford Island, and many smaller islands, drying rocks and reefs. Two major fjords, Kingcome and Knight Inlets, lie to the east of the archipelago and penetrate deeply into the coastal mountain range. The circulation in the archipelago and adjacent fjords and straits is primarily tidal in nature. Strong tidal currents and mixing are conspicuous features of Johnstone and Queen Charlotte Straits [(1)] and in the narrow passages through the archipelago. Local runoff drives a prominent estuarine surface flow especially in Knight Inlet.[(2)] The Klinaklini River at the head of Knight Inlet is the largest river in the region and reaches peak discharge in the summer. The surface circulation is also forced by local winds.[(3)]

The waters of the Broughton Archipelago and adjoining fjords and watersheds of the region support the production of most species of Pacific salmon (*Oncorhynchus*). The Broughton Archipelago is also noted as a major salmon farming area. There are a total of 26 finfish farm tenures distributed through the passages and channels of the archipelago (Fig. 1). In 2002, the farms of the Broughton produced 20,000 tonnes of Atlantic salmon (*Salmo salar*) [(4)] accounting for about 25% of the province's total farmed salmon production.

Infectious hematopoietic necrosis (IHN) virus is a rhabdovirus pathogen that is endemic in the Pacific Northwest and infects all five species of Pacific salmon (*Oncorhynchus*), Atlantic salmon *(S. salar)* and rainbow trout *(O. mykiss)*.[(5)] In British Columbia the IHN virus is most often associated with sockeye salmon (*O.*

nerka) in the freshwater environment either as fry or spawning adults.[6,7] Atlantic salmon are very susceptible to the IHN virus, and in 1992 the first reported epizootic of IHN in farmed Atlantic salmon was reported in BC.[8] Over the course of the next four years the infection spread to 13 farm sites located within 20 km of the index site.[7] The second epizootic of IHN in farmed Atlantic salmon occurred from 2001 to 2003 and affected 36 Atlantic salmon farms in five separate areas, one of which was in the Broughton Archipelago.[7] Both epizootics resulted in significant mortalities and economic loss to the aquaculture industry in BC.

In the 2001 epizootic, the coincidence in time of the initial cluster of IHN disease outbreaks and the returning adult salmon migration in the local waterways suggested that wild salmon may have been the source of the infection of the Atlantic salmon. The studies of Traxler et al.[9] demonstrated that Atlantic salmon become infected when placed close to infected fish and the rapid spread of the disease to other adjacent pens in the farm imply that the transmission is likely waterborne. Once an IHN disease outbreak is established in a farm it then becomes a reservoir or source of IHN viral particles. Ocean currents will disperse and transport the viral particles into the surrounding waterways and possibly transmit the disease to other finfish farms in the area. The pathways followed, distances travelled and temporal extent of the waterborne transport of the pathogen will depend on the local ocean currents. The spread of the disease to adjacent farm sites may also depend on factors such as the virulence of the particular variant of the pathogen, the health of the host farm population, the inactivation time of the viral particles in seawater, and the concentration of and duration of exposure to the pathogen at finfish farm sites.

Our goals in this contribution are to a) describe the numerical circulation models of the Broughton Archipelago that we have been developing, b) simulate using particle tracking programs, the transport and dispersion of IHN pathogen based on

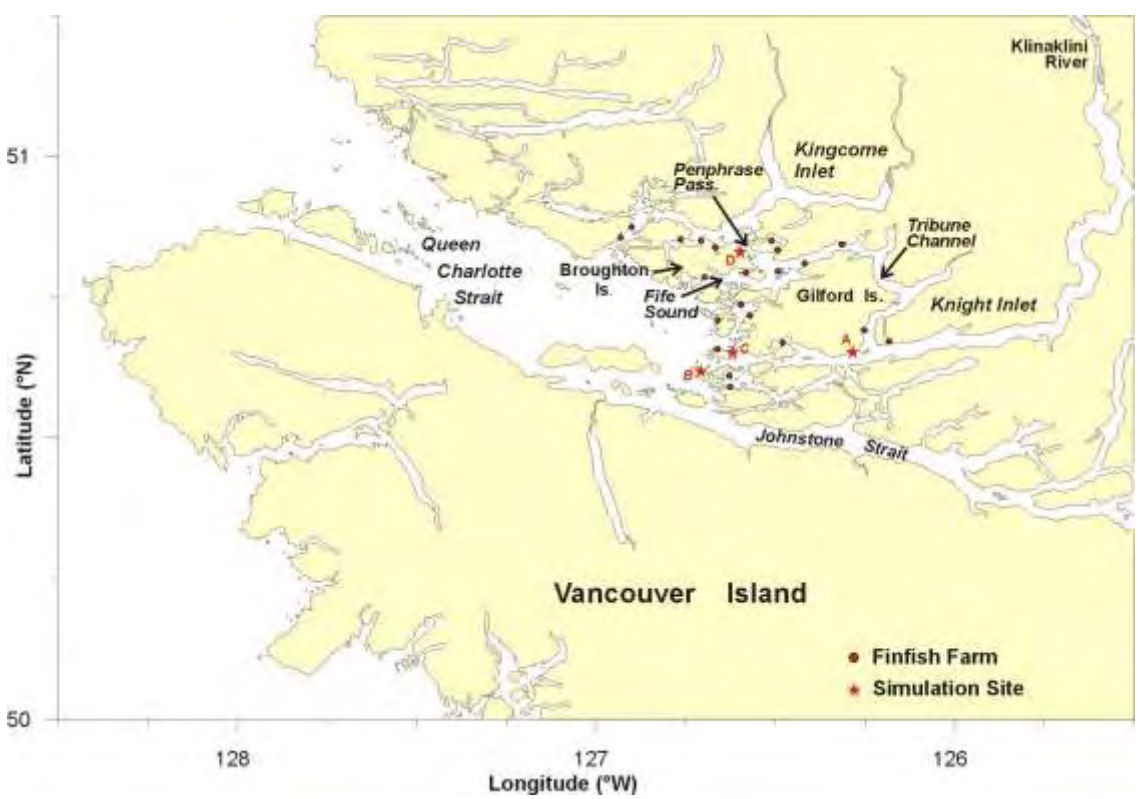


Figure 1
Site map of region showing principal rivers, waterways, islands, locations of finfish farms, and the particle release sites A, B, C and D.

the currents computed in our circulation models, and c) discuss the significance that these simulations may have for the waterborne transmission of the disease between farm sites in the archipelago.

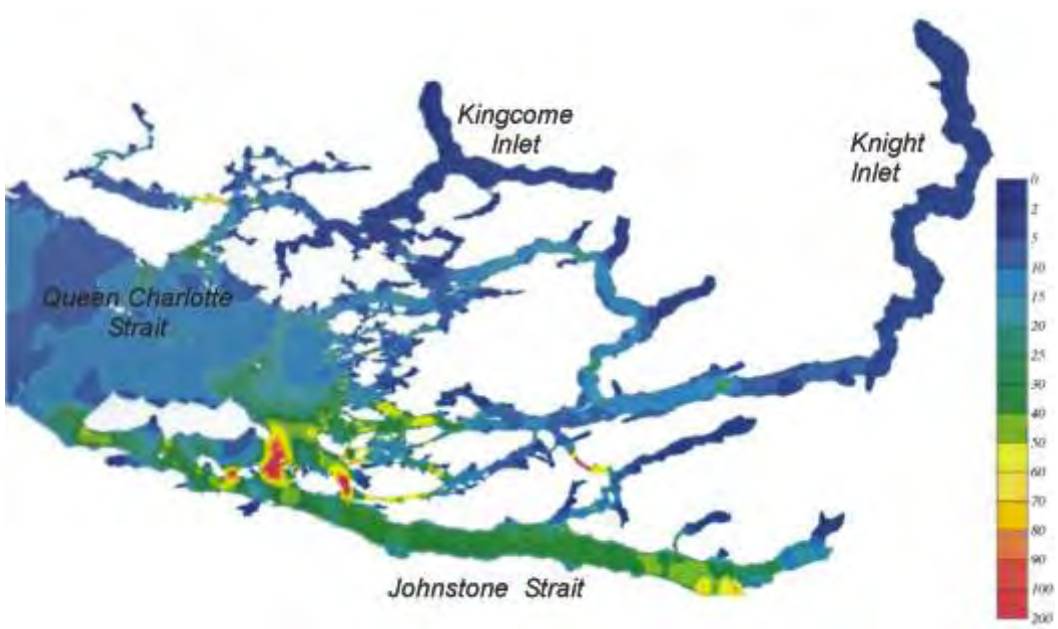
Numerical Circulation Model

The model domain stretches 150 km from the southern end of Johnstone Strait to the northern end of Queen Charlotte Strait and inland about 100 km to the head of Knight and Kingcome Inlets, and includes the Broughton Archipelago (Fig. 1). This is a large and topographically complex region that presents many modeling challenges. The model uses variable size triangular grid elements that vary in length from approximately 6 km in Queen Charlotte Strait to about 50 m in some of the narrow passages. The variable grid element size provides a better representation of the complicated coastline and bathymetry in the Broughton Archipelago than the uniform rectangles that are often required by other numerical model methods. Model bathymetry and coastline were obtained from the Canadian Hydrographic Service nautical charts. A more complete description of the models and comparisons of model results with observations are detailed in Foreman et al.[10]

The two-dimensional (2D), frequency-domain finite-element model TIDE2D [11] was used to compute the tidal currents. TIDE2D computed amplitudes and phase lags of tidal elevation and depth-averaged velocity components at all nodes of the model grid. Eight constituents were used to approximate the tidal currents in TIDE2D (M_2, S_2, N_2, K_2, K_1, P_1, O_1, and Q_1), and in this region they account for most (80% to 90%) of the energy in the tidal elevation and currents. Tidal elevation forcing was specified along the open sea boundaries using historical observations from tide gauges near those locations.

The main features of tidal currents in the model domain are the conspicuously strong currents at the southern end of Queen Charlotte Strait, the western end of Knight Inlet and in Johnstone Strait. In contrast, the tidal currents are generally weak in the fjords and embayments (Fig. 2). Localized areas of strong tidal currents are evident over sills and at horizontal constrictions in the waterways of the region.

Figure 2
Map of root mean square (rms) vertically-averaged tidal currents (cm/s) in the model domain.



TIDE3D, a harmonic finite element method developed by Walters,[11,12] was used to compute three-dimensional (3D) diagnostic buoyancy currents or mean flow field (Z0) arising from a specified density field for the region. The 3D density field used to force the diagnostic TIDE3D calculation of the mean flow field was derived from the summer climatology (July, August and September) of all temperature and salinity profiles obtained from 1955 to the present in the model domain.

The computed mean surface flow field exhibits a generally seaward flow in the fjords, passages and straits in the model domain (Fig. 3). This seaward surface flow is generally consistent with observations and our understanding of the oceanography of these regions.[1-3] Comparisons of the model and observed mean surface flows at the locations where surface flow measurements were taken indicate that there was agreement in the seaward direction of the flow, but the magnitudes were substantially different.[10]

Particle tracking simulations were carried out with a modified version of the DROG3D program [13] distributed by the Quoddy modelling group. DROG3D calculates the positions and tracks of the particles using the TIDE2D depth-averaged tidal velocities and mean surface flows computed by a diagnostic TIDE3D. The DROG3D program was modified to permit the release of particles at different stages of the tide. Another modification was made to permit specification of the length of time each particle was tracked.

The 'markers' or particles, which represent IHN viral particles, are passive and neutrally buoyant and are transported through the model grid by the computed currents (tides + mean surface flow). In the simulations, particles were released hourly from farm sites into the surface layer over a 15-day period in order to encompass one complete fortnightly cycle (neap and spring tides) of the tidal currents. Each particle was tracked for two days (2-day lifetime) as this appears to be an appropriate time scale for the inactivation time of IHN viral particles.

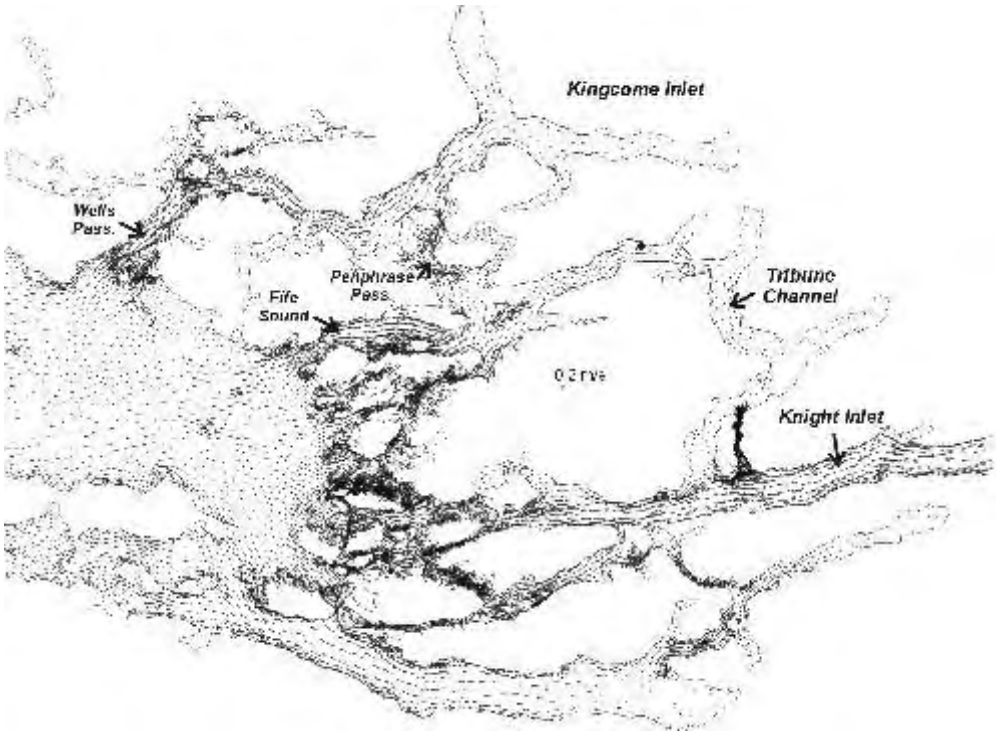


Figure 3
Mean surface flow or Z0 flow field produced by the diagnostic TIDE3D model and the average summer density field.

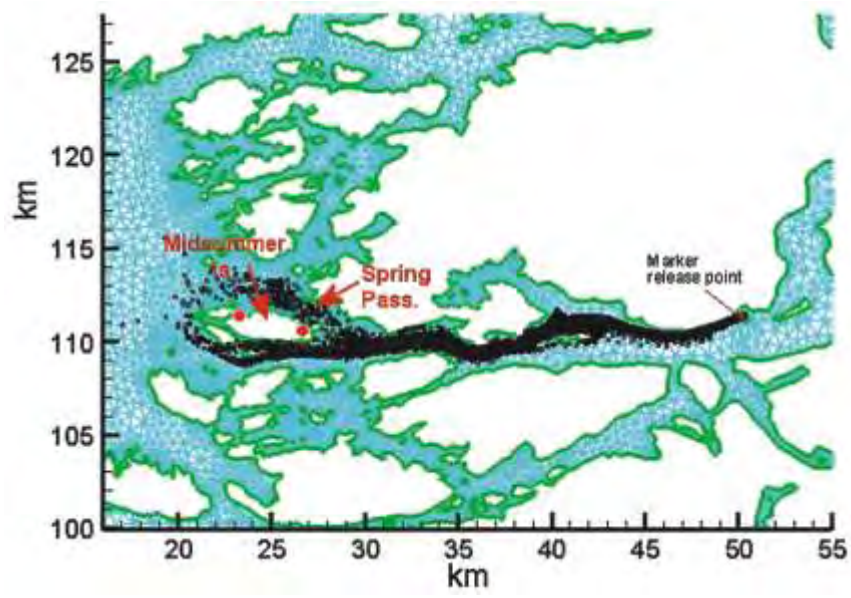


Figure 4
Results of particle tracking simulation for site A.

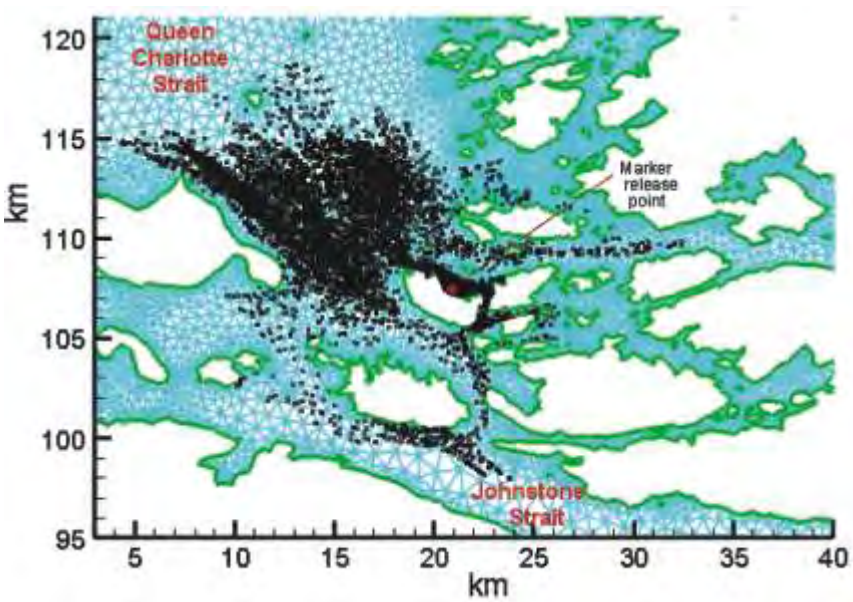
Results

Particle tracking simulations were conducted for many farm sites and locations of interest in the model domain but herein results from four farm sites (A, B, C and D in Fig. 1) are presented in Figures 4, 5, 6 and 7. The diagrams that follow show the positions of each particle plotted hourly for two days (48-hour lifetime) after its release.

Site A

Particles released at site A experience the relatively strong seaward (westward) surface currents of Knight Inlet (Fig. 4). Up-inlet or eastward movement of the particles on the flooding tide was only slight and no particles reached the junction of Tribune Channel and Knight Inlet which is located 1.5 km to the east of site A. Most of the particles travelled westward through the lower reaches of Knight Inlet to Queen Charlotte Strait; however, some particles travelled through Spring Passage. Those particles moving through Spring Passage passed close to finfish farms located on Midsummer Island, possibly exposing the cultured fish to the IHN pathogen. The maximum distance travelled by particles released for this location was 33 km—the largest travel distance of all the sites simulated.

Figure 5
Results of particle tracking simulation for site B.



Site B

Unlike the particles released from site A, particles released from site B were dispersed over a large area. Most particles were transported into Queen Charlotte Strait where they were widely dispersed. The strong tidal currents transported a small number of particles southward into Johnstone Strait and a very small number eastward into Knight Inlet. Maximum travel distances from the release point were about 19 km in the two-day lifetime of the particles.

Site C

The simulation for particles released at this site, which is located at the eastern end of Midsummer Island, were transported into Queen Charlotte Strait by the mean seaward surface flow and dispersed over a large area. A small number of particles were carried eastward into Knight Inlet by the strong flooding currents in Spring Passage. A noteworthy feature of this simulation is the concentration and passage of particles past the finfish farm at the north-western end of Midsummer Island only 4 km away from the release point. Maximum travel distances in two days were about 18 km.

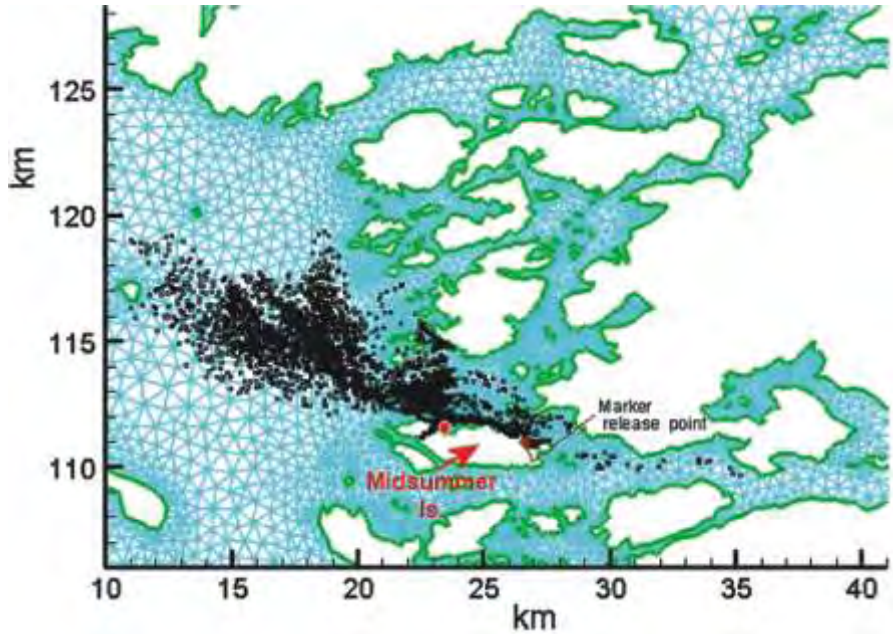
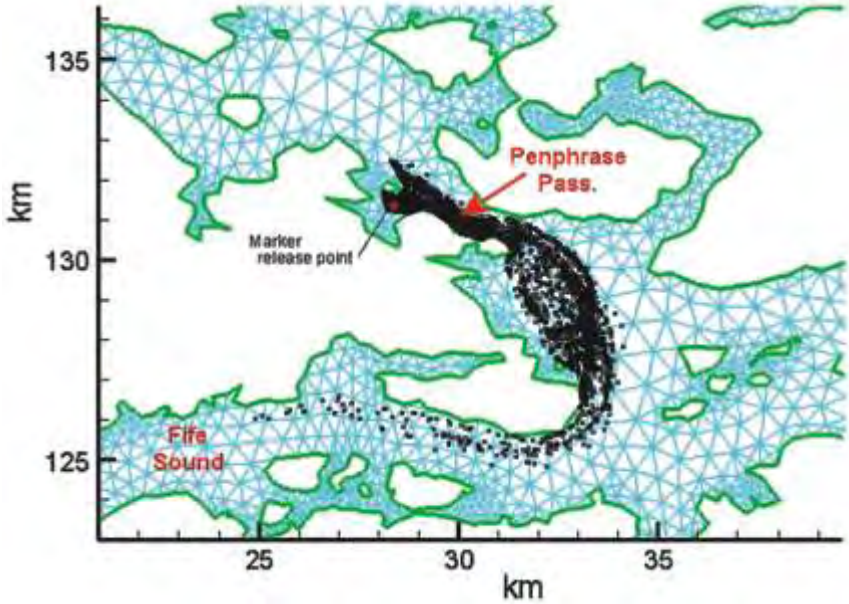


Figure 6
Results of particle tracking simulation for site C.

Site D

The release point for this simulation was located in a protected bay, and movement of the particles out of the bay was slow. However, once the particles entered Penphrase Passage the strong tidal current and mean surface flow quickly transported the particles southeast through the passage and then westward into Fife Sound (Fig. 7). Maximum travel distance in two days was about 16 km from the release point. It is of note that the particles that enter Fife Sound did not travel far enough in two days to reach an active finfish farm site on the north shore of Fife Sound.

Figure 7
Results of particle tracking simulation for site D.



Discussion and Conclusions

The particle tracking simulations based on numerical circulation models are useful tools for investigating the role of water movement in transporting and dispersing pathogens. The simulations provided an estimate of the distances particles were transported from their sources, the routes the particles travelled and a time history of particle positions. The modelled surface currents in the Broughton Archipelago and Knight Inlet transported passive, neutrally buoyant viral particles seaward.

The distances the particles travelled from the release sites, the routes taken and the dispersion were site specific. Maximum travel distances raged from 33 km in the case of releases from site A to only 3 km for releases undertaken in very sheltered bays. The extent of the dispersion and distance varied with the strength of the tidal currents and the mean surface flows. The simulations showed that within the two-day lifetime or time window used for the IHN viral particles some finfish farm sites were directly downstream of, or connected to, the farm site that was the source of the pathogen. For sources located close to Queen Charlotte Strait the energetic tidal current combined with the seaward surface flow resulted in a widespread dispersion and transport of pathogens into the strait.

As with other numerical circulation models, our Broughton Archipelago models are based on simplifying assumptions and approximations. Furthermore, in some regions of the model domain observational data used to force the model were limited. Consequently, interpretation of the particle tracking simulations should take into consideration the limitations and uncertainties of the model currents. A discussion of several of the important limitations follows, and a more detailed comparison of the model with observations is provided by Foreman et al.[10]

"The spread and severity of an epizootic result from the interaction among the pathogen, its host and the environment. The role of water circulation in the spread of the disease organism is but one part of the environmental component of the problem."

Winds are known to influence the surface circulation in fjords, and Baker and Pond[3] have shown that in Knight Inlet the winds are an important factor driving surface layer flow. However, because of the scarcity of long-term local wind measurements and the absence of a high resolution, atmospheric circulation model for this topographically complex region, wind forcing was not included in the circulation model.

In the absence of adequate measurements of freshwater discharge into the model domain, except for the Klinaklini River, the estuarine circulation in the model was driven indirectly by the average summer density field, derived from the climatology of the temperature and salinity observations in the region. Observations were most prevalent in Knight Inlet and Queen Charlotte Strait, sparse in some of the major channels (e.g. Tribune Channel and Fife Sound) through the archipelago and nonexistent in most of the minor passages and embayments. Consequently, in those areas that had little or no data coverage the uncertainty in the modelled currents was higher than in those regions with better data coverage.

The tidal model TIDE2D computes a depth-averaged current which is a simplification of the more complicated velocity profiles that have been observed in fjords. In shallow waters where the flows are strong the vertical-averaged velocity provides a better estimate of the tidal currents than in the very deep areas of the model domain. Overall the depth-averaged tidal currents will underestimate the surface tidal currents.

To the extent that the models accurately represent the surface tidal currents and the mean surface (estuarine) flow, the implications that the simulations have for the waterborne transmission of the IHN virus from finfish farms are site specific. The simulations based on the two-day lifetime or inactivation time of the viral particles showed that some finfish farms were connected by water movements within this two-day time window. Using longer lifetimes for the pathogen would enlarge the area for potential waterborne disease transmission.

The spread and severity of an epizootic result from the interaction among the pathogen, its host and the environment. The role of water circulation in the spread of the disease organism is but one part of the environmental component of the problem. The inactivation time of IHN virus in sea water and the factors governing the inactivation of the virus are also part of the environmental component. We have identified several limitations of our numerical circulation models and we are

continuing to improve the realism of modelled currents within the Broughton Archipelago. Improvements in our circulation models and better understanding of the factors affecting the inactivation of the viral particles in sea water will enable us to assess and understand the role of water movements in the spread and development of this serious disease in Atlantic salmon farms. The results of improvements in our models and additional circulation measurements will be described in future manuscripts.

Acknowledgments

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References

1. Thomson RE. 1981. Oceanography of the British Columbia coast. *Can. Spec. Publ. Fish. Aquat. Sci.* 56, 291 p.
2. Farmer DM, Freeland HJ. 1983. The physical oceanography of fjords. *Prog. Oceanogr.* 12(2):147-220.
3. Baker P, Pond S. 1995. The low-frequency residual circulation in Knight Inlet, British Columbia. *J. Phys. Oceanogr.* 25:747-763.
4. British Columbia Ministry of Agriculture and Lands. 2005. *Fish Production and Fallowing in the Broughton Archipelago Area.* Available at www.agf.gov.bc.ca/fisheres/health/Sealice/Prod_ Fallowing_BA pdf.
5. Kurath G, Garver KA, Troyer RM, Emmenegger EJ, Einer-Jensen K, Anderson ED. 2003. Phylogeography of infectious haematopoietic necrosis virus in North America *J. Gen. Virol.* 84:803–814.
6. Traxler GS, Rankin JB. 1989. An infectious hematopoietic necrosis epizootic in sockeye salmon *Oncorhyncus nerka* in Weaver Creek spawning channel, Fraser River system, B.C., Canada. *Dis. Aquat. Org.* 6:221-226.
7. Saksida S. 2002. *Investigation of the 2001-2002 IHNV Epizootic in Farmed Atlantic Salmon in British Columbia.* Government of British Columbia. Ministry of Agriculture, Food and Fisheries. Available at www.agf.gov.bc.ca/fisheries/health/ihnv_report_2003.pdf
8. St-Hilaire S, Ribble C S, LaPatra SE, Chartrand S, Kent ML. 2001. Infectious hematopoietic necrosis virus antibody profiles in naturally and experimentally infected Atlantic salmon *Salmo salar. Dis. Aquat. Org.* 46:7–14
9. Traxler GS, Roome JR, Kent ML. 1993. Transmission of infectious hematopoietic necrosis virus in seawater. *Dis. Aquat. Org.* 16:111-114.
10. Foreman MGG, Stucchi DJ, Zhang Y, Baptista AM. 2006. Estuarine and tidal currents in the Broughton Archipelago. *Atmos.-Ocean* (in press).
11. Walters RA. 1988. A finite element model for tides and currents with field applications. *Commun. Appl. Numer. Meth.* 4:401-411.
12. Walters RA. 1992. A three-dimensional, finite element model for coastal and estuarine circulation. *Cont. Shelf Res.* 12:83-102.
13. Blanton BO. 1995. *DROG3D: User's Manual for 3-Dimensional Drogue Tracking on a Finite Element Grid with Linear Finite Elements.* University of North Carolina, Chapel Hill. Manual available at www.opnml.unc.edu/Particel_Tracking/part_track/html.

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Sonja Saksida

Water Movement and Fish Health Management of Infectious Diseases on Salmon Farms in British Columbia

Sonja Saksida, Joanne Constantine, and Jim Brackett

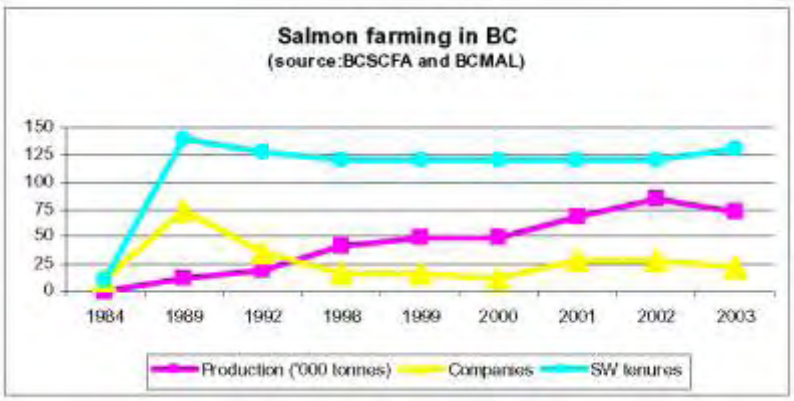
In British Columbia (BC), understanding the local water movement patterns is essential not only in obtaining and maintaining farm tenures in the marine environment but also in management of infectious diseases in farmed salmon. The impact water patterns have on disease management decisions depends on the pathogen, the host, and the pathogen/host relationship. The level of importance of the role of water movement in the management of diseases known to occur in BC farmed salmon is examined using the examples of salmon rickettsial syndrome (SRS), furunculosis, and infectious hematopoietic necrosis (IHN).

Aquaculture in British Columbia

Salmon farming in British Columbia (BC) began in the early 1970s using the Pacific salmon species *Oncorhynchus tshawytscha* (chinook salmon), *O. kisutch* (coho salmon), and *O. mykiss* (rainbow trout). The Atlantic salmon (*Salmo salar*) was introduced to BC as a farmed species in the mid 1980s.

As of 2002, annual production of farmed salmon had increased to 83,000 metric tons despite a substantial decrease in the number of operating companies from the peak of the late 1980s (Fig. 1). Presently, at least 90% of the total production comes from only six companies. Much of the growth in production between the 1980s and the present is not due to increased saltwater tenures, which have only slightly increased (range 121 to 140) since the 1990s. Currently, only approximately 60% of the tenures are in use. More significantly, the increase is the result of the switch to Atlantic salmon, which have better survival and growth rates, allowing for production expansion. Atlantic salmon now make up approximately 82% of farmed salmon production in BC.[1,2] In addition, other factors such as improved husbandry and fish health practices (i.e. increased availability of efficacious vaccines, less handling and grading, prevention of disease through screening, etc.) have contributed to increased production despite the reduced numbers of sites available for expansion.

Figure 1
Number of companies, salt water (SW) tenures, and farmed salmon production from the mid 1980s to 2003 in British Columbia.

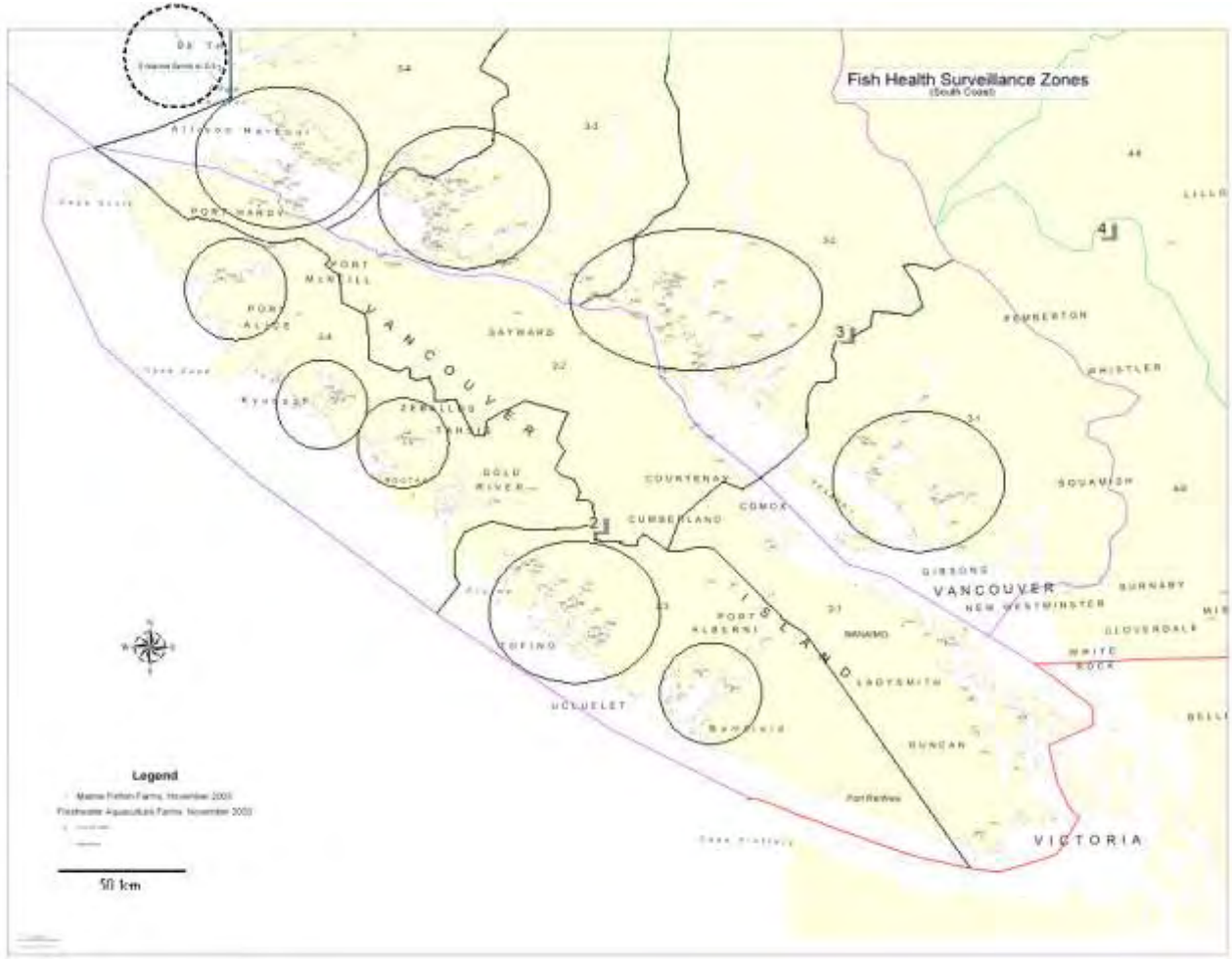


Water Movement Around BC Farms

Salmon farms are located predominantly around Vancouver Island, with the majority of the farms on the eastern side or "inside passage" between Vancouver Island and the BC mainland (Fig. 2). The mainland coastline is made up of a large number of inlets or fjords. As a result of the large river systems, the high rainfall, and the glaciated mountains, a significant portion of this coastal area experiences estuarine circulation: net seaward movement of surface water.[3] In general the movement of surface water toward the sea is strongest during the freshet. The surface water layer can extend to great depths; for example in farms located in zone 3-3 (Fig. 2) the depth of the surface water layers range from 10 to 40 m.[4] Average surface currents of up to 15 cm/sec are usually found on typical farms in BC with rip tides sometimes reaching 50 cm/sec on some farm sites.

Ample water movement around a farm is considered essential for the health of the farmed fish and the environment, and is therefore important in maintaining the rights to operate a farm on the lease. As part of the lease/license requirements for operation of a farm in BC, the BC Ministries of Agriculture and Lands (BCMAL) and Environment (BCMOE) require routine benthic sampling directly under the pen systems and within a defined

Figure 2
Location of farming areas located around Vancouver Island, British Columbia. The black lines outline the Fish Health Surveillance zones and the circles outline the farming regions that share water.
(source: http://www.agf.gov.bc.ca/fisheries/health/fish_health_zone_map.pdf)



distance from the pen system (the footprint) to ensure minimal impact.

Fish Health Management Considerations

Farmed salmon health issues are managed at a population level. There are many factors to be considered when making decisions on how to manage a health problem involving an infectious agent.

The level of effect that the pathogen has on the susceptible salmon populations should be considered, including:

- infectivity: what proportion of the exposed population will become infected with the pathogen;
- pathogenicity: what proportion of the infected population will develop disease;
- virulence: what proportion of the diseased population will become very sick or die; and
- incubation period: the period of time from infection to disease.[5]

When determining a management strategy, it is also necessary to have an understanding of how the pathogen moves between susceptible hosts both within a population (transmission) and between populations (spread). Transmission and spread can occur either by direct or indirect methods.[5] Table 1 outlines some of the various ways that pathogens can be transmitted or spread by direct and indirect methods.

Direct methods involve the movement of pathogen as a result of direct contact with an infected individual or their immediate by-products such as feces, blood, or gametes (in the case of vertical transmission). Direct methods are thus very important in the movement or transmission of infection or disease within a population.

Alternatively, indirect transmission or spread of the pathogen involves the movement of the pathogen itself. This can be done through another host (such as another species of fish or shellfish, etc.), attached to fomites (i.e. non-living material), or directly in the water (waterborne). As a consequence, indirect methods play a crucial role in the spread of infections/disease between susceptible populations.

Waterborne spread of pathogens is of particular interest when considering a management plan, especially since salmon farming involves net-pen systems in the ocean. In BC, fish health professionals normally assume that the pathogens

Table 1. Modes of transmission or spread of pathogens. The ticks (✔) indicate modes of transmission or spread for the causitive agents of SRS, furunculosis, and IHN.[6,7]

	SRS	Furunculosis	IHN
Direct Transmission or Spread			
Horizontal—fish to fish (direct contact, feces, cannibalism)	✔	✔	✔
Vertical—parent to progeny	✔	✕	✔
Indirect Transmission or Spread			
Vector—other species of fish, sea lice, etc.	✔		✔
Vehicle—through fomites (people, equipment)	✕	✔	✔
Waterborne—current, carried by boats	✔	✔	✔

causing diseases in farmed salmon are capable of surviving in water for a variable length of time. Thus water flow patterns are considered during the development of a treatment plan or control program. The importance that water movement has in a management decision, however, is usually dependent on the seriousness of the disease (i.e. how pathogenic or virulent, is it a listed disease, etc.), the rate of spread, and the availability of efficacious treatments.

The following is an outline of three diseases found in BC farmed salmon requiring different approaches to management: net-pen level, farm level, or area management. The influence water movement plays in developing management decisions is discussed.

Pen level management

Salmon rickettsial syndrome (SRS) is a disease that occurs in Atlantic salmon in a few salmon farming regions in British Columbia. The causative pathogen is *Piscirickettsia salmonis*.[6,7]. In BC, SRS is considered a "chronic" condition with relatively low infectivity and pathogenicity. Only a small proportion of the exposed population on a farm develops disease. Table 1 summarizes the methods of transmission/spread of SRS; waterborne transmission/spread is known to occur.

The overall mortality rates associated with SRS in farmed Atlantic salmon are considered low and known cases often go untreated for extended periods of time. When therapeutic intervention is determined to be appropriate, farm-wide treatment may be recommended, but more often treatment is provided only to the cages of concern because of the factors described above. The orientation of the farm may be assessed to determine an appropriate treatment regime. Figure 3 displays two farm orientations with respect to local water patterns. If the disease was located in farm A then the veterinarian may consider treating only the affected pens; however if the disease was on farm B then treatment may be given to the affected pens plus the pens immediately adjacent to the affected pens or possibly the entire farm.

Chronic diseases such as SRS are managed largely by targeting individual or small groups of pens within the farm. Water movement is not a major consideration in the control programs.

Farm level management

Aeromonas salmonicida is the causative agent of furunculosis, a bacterial disease diagnosed in Atlantic salmon in BC.[6,7] This pathogen is considered moderate to highly pathogenic and virulent in Atlantic salmon. In BC, furunculosis can be considered an acute disease because of its apparently rapid spread within a population and its short incubation period (days). Table 1 summarizes the methods of transmission/spread of furunculosis. Waterborne transmission/spread is known to occur and considered an important contributing factor in the movement of the pathogen between pens within a farm.

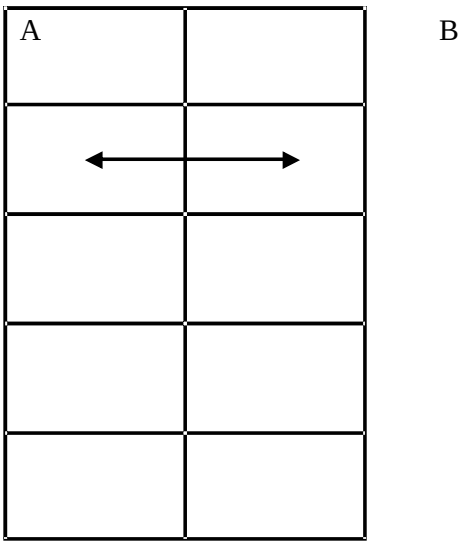


Figure 3
Two possible directions of water flow within a pen system. Pen system A has water flowing perpendicular to the system. Pen system B has water flowing parallel to the system.

Furunculosis has caused severe disease and mortality on saltwater farms containing unprotected Atlantic salmon populations. However, it has not been found to spread between seawater farms in BC. When furunculosis is diagnosed, the normal course of action is to treat the entire farm with one of the available in-feed antimicrobial treatments. Fortunately, in recent years, the prevalence of furunculosis has been declining due to an efficacious vaccination program. Presently, all farmed Atlantic salmon are vaccinated.

In summary, when dealing with acute diseases such as furunculosis, the management strategy is to manage at a farm level (i.e. treat the entire farm). In these situations waterborne movement of the pathogen is considered an important method of transmission within the farm.

Area management

Infectious hematopoietic necrosis (IHN) is a viral disease caused by a rhabdovirus (IHN virus). It has caused serious disease in farmed Atlantic salmon in BC. Atlantic salmon farmers in BC have faced two epidemics of infectious hematopoietic necrosis (IHN): 1992-1996 and 2001-2003. IHN has not been diagnosed in farmed salmon outside these two outbreaks.[8,9] The disease is considered endemic in sockeye salmon. Atlantic salmon, which have only relatively recently been introduced as a farmed species, are highly susceptible to the infection. In farmed Atlantic salmon in BC, IHN is a peracute disease. The IHN virus is highly virulent and the incubation period is short. The infection spreads quickly through a farm population and potentially to other farms resulting in significant mortalities.

Table 1 outlines the modes of transmission/spread of IHN. An in-depth discussion of the most recent IHN epidemic is provided below to illustrate control of this type of disease in which waterborne transmission plays a significant role.

IHN epidemic 2001-03

During the 2001-03 epidemic, 36 farms became infected with IHNv.[9] The epidemic was devastating: the disease spread to five farming regions and an estimated 12 million fish died or were destroyed during the epizootic. The costs of the epidemic after the first 23 outbreaks was estimated to be over $40 million in direct inventory losses and over $200 million in lost sales, with the final costs most likely significantly higher. The cumulative mortality on smolt farms (< 700 g) was reported to be as high as 77% and for harvest-sized populations cumulative mortality rates were reported to be as high as 53%.[9]

There is no treatment and at the time of the epidemics no efficacious vaccine for IHNv was available; therefore management of the disease is much different than for the other disease situations discussed. The virus is capable of surviving in surface water and is infective at low titres, making waterborne spread a significant factor. It was recognized that the infection/disease spread quickly through individual farms and then to other farms located in the vicinity. Therefore, a large component of controlling the epidemic was conditional on understanding local water movement patterns and how they affect the spread of the disease.

In addition, there was another type of water movement that may have been responsible for the spread of IHN between farms within an area. That is the movement of IHNv infected water by the larger supply vessels servicing the farming industry (i.e. feed delivery and mortality removal). These vessels are believed to have inadvertently moved infected water (as ballast or engine coolant water) between farms within a region.

Waterborne exposure of the pathogen played a significant role in spreading the disease into other farming areas. Atlantic salmon smolts being transported in well boats are believed to have been exposed to effluent discharged from a plant processing Atlantic salmon from an infected farm. Although the effluent was treated using a protocol recommended by provincial and federal government authorities, the method may have been ineffective in killing all the virus.

The control and eradication of this disease from the farmed population required a coordinated effort of fish health professionals (both private and government), farming companies, processors, third party suppliers, and scientists. The control measures contained two components: measures to reduce the number of IHN virus and measures to reduce the movement of the virus.

Management and control measures used to reduce the pathogen numbers

It was apparent during the epidemic that the largest risk for infection came from the presence of farms in the area with infected or diseased Atlantic salmon that were shedding virus. Furthermore, it was recognized that the virus was most virulent in the Atlantic salmon smolt populations and these infected populations were likely shedding large amounts of virus into the water. To reduce this risk, several farms with smolts culled their entire population; the owners were never compensated for either the cost of the fish or the cost of culling. Other measures implemented to reduce the number of pathogens included increased frequency of removal of dead and moribund fish from affected sites and earlier harvesting. Processing plants also established effective water treatment procedures to reduce the risk of discharging infected water back into the sea.

"The most important action that contributed to the eradication of IHN within an area during the last epidemic was the establishment of high risk areas or zones."

Management and control measures used to reduce the spread of pathogen

The most important action that contributed to the eradication of IHN within an area during the last epidemic was the establishment of high risk areas or zones. These zones were defined by the presence of infected sites, and presumed local surface water flow patterns. Activities were restricted to within the zones (equipment movement, transport of staff, harvesting, etc.). Biosecurity was increased at a farm level. There was an increase in monitoring of non-infected farms and the high risk activity of transporting fish using transport pens was stopped.

Increased biosecurity was placed on third-party activity which included introducing dedicated vessels that travelled exclusively to infected sites or non-infected sites. Furthermore, water pick-up and discharge protocols were established to reduce the risk of inadvertent movement of infected water. To reduce the risk of spread to other areas, protocols were established for smolt transport that stipulated the regions where pumping of water into the well boats should be stopped. This was based on presumed water movement around the processing plants and within the infected zones.

Finally, farms were only re-populated once all the infected/exposed populations had been removed from the area. Near the end of the epizootic, infected fish were moved to a contained region to allow for clean up to occur. Some companies elected to repopulate the site with chinook salmon, which are not susceptible to IHNv.

The implementation of these management measures reduced the length of time to control the epidemic by almost 50%: 23 months for 2001-03 compared to 44 months for the 1992-06 outbreak.[8,9] Many of these measures are now part of companies' Fish Health Management Plans implemented by BCMAL government regulation in anticipation of the next outbreak.[10]

As discussed, diseases such as IHN in farmed Atlantic salmon must be managed on an area basis, in addition to critical on-farm control measures. Understanding water movement as an essential component in the spread of the pathogen between farms is an important consideration in developing control and management procedures.

Summary

Salmon farms in British Columbia are separated by relatively large distances. Water movement in farming areas is largely estuarine in influence. Knowledge of larger water flow patterns, along with specific knowledge of water movement locally and within farms assists in the development of appropriate health management programs. These programs are also based on characteristics of the pathogen, including infectivity, pathogenicity, virulence, and modes of transmission. Consideration of all of these factors will determine the level of control: by pen, by farm, or by area management.

In BC, the possibility of waterborne transmission for most infectious pathogens is accounted for in the design of health management programs. The relative importance of water movement in each disease is dependent on the factors discussed.

Increased understanding of water movement within farming regions and within farms will assist in better design and implementation of health management programs.

References

1. British Columbia Salmon Farmers Association. 2005. *2002 Production Values.* Available online at www.agf.gov.bc.ca/fish_stats/pdf/Seafood_Industry_YIR_2002.pdf. Accessed November 2005.
2. British Columbia Ministry of Agriculture and Lands. 2005. Aquaculture Statistics. Available online at www.agf.gov.bc.ca/fish_stats/statistics-aqua.htm. Accessed November 2005.
3. Thomson RE.1981. Oceanography of the British Columbia coast. *Can. Spec. Publ. Fish. Aquat. Sci.* 56, 291 p
4. Stucchi D, Fisheries and Oceans Canada, Institute of Ocean Sciences, personal communication.
5. Baldock C. 2000. *Epidemiology Notes*. University of Tasmania. July 23-27, 2000.
6. Kent ML, Poppe TT. 1998. *Diseases of Seawater Netpen-Reared Salmonid Fishes.* Pacific Biological Station, Fisheries and Oceans Canada, Nanaimo.
7. Bruno DW, Alderman DJ, Schlotfeldt HJ. 1997. *What Should I Do? A Practical Guide for the Marine Fish Farmer.* European Association of Fish Pathologists.
8. St-Hilaire S, Ribble CS, Stephen C, Anderson E, Kurath G, Kent ML. 2002. Epidemiological investigation of infectious hematopoietic necrosis virus in salt water net-pen reared Atlantic salmon in British Columbia Canada. *Aquaculture* 212:49-67
9. Saksida SM. 2004. I*nvestigation of the 2001-2003 IHN Epizootic in Farmed Atlantic Salmon in British Columbia.* Available online at www.agf.gov.bc.ca/fisheries/health/IHNV_report_2003.pdf. Accessed November 2005.
10. BCMAL. *Fish Health Management Plans implemented by BCMAL government regulation in anticipation of the next outbreak.* Available online at www.agf.bc.ca/fisheries/health/IHNV_Isolation_Control_Procedures.pdf. Accessed November 2005.

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Oceanographic Influences on the Management of MSX Disease of American Oysters *(Crassostrea virginica)* in Atlantic Canada

Mary Stephenson

Mary Stephenson and Brian Petrie

Multinucleate sphere "X" (MSX disease), caused by *Haplosporidium nelsoni*, was detected in American oysters (*Crassostrea virginica*) in the Bras d'Or Lakes, Cape Breton, Nova Scotia in October 2002. Disease monitoring of oysters and other bivalves that had been in place since 1988 had not detected the presence of MSX; therefore this detection was considered to be a new introduction of an exotic pathogen. Because MSX was a World Organisation for Animal Health (OIE = Office Internationale des Epizootics) listed disease, Fisheries and Oceans Canada reported the detection to the Chief Veterinary Officer for Canada and, in consultation with affected government and industry stakeholders, implemented controls aimed at disease containment. In surveys conducted throughout Atlantic Canada to establish the geographic distribution of the disease, MSX was detected solely in multiple areas of the Bras d'Or Lakes. Since the infective life-stage of *Haplosporidium nelsoni* is believed to be waterborne, it was essential to consider oceanographic influences on disease dispersion. The hydrographic connectivity within the Bras d'Or Lakes and subsequent links to the rest of Atlantic Canada were reviewed. Historic oceanographic data were re-analyzed and flushing times were used to predict particle transit times within the lakes. Based on the results of the surveillance, analysis of oceanographic profiles, and consideration of industry activities, it was advised that a zonation approach be taken to manage the disease. The Bras d'Or Lakes of Cape Breton were defined as MSX-positive. Sub-zoning within the lakes enabled industry activities to continue while protecting areas not yet impacted by MSX. It was also advised that increased disease monitoring be done in a buffer zone that encompassed the Atlantic coast of Cape Breton. This approach was accepted and licences issued with conditions reflecting this zonation strategy. Review of the MSX management is ongoing.

Introduction

In October 2002, *Haplosporidium nelsoni*, the causative agent of MSX (multinucleate sphere 'unknown') disease of American oysters, *Crassostrea virginica*, was detected for the first time in oysters in Canadian waters.[1,2] The affected stocks were in a unique population located in the Bras d'Or Lakes of Nova Scotia (Fig. 1). These oysters were oceanographically and physically isolated from previously affected oyster populations, the closest of which was in the Piscataqua

River on the Maine–New Hampshire border, USA.[3] MSX is an OIE-listed (Office Internationale des Epizootics, World Animal Health Organization) disease of American oysters, due to the severity of its impact on naïve populations. Mortality levels on impacted oyster leases in Cape Breton were over 80 to 90%, reinforcing the severity of the disease. Initial management decisions to prevent the spread of MSX were based on controlling industry activities.

The importance of oceanography was immediately noted due to the unique oceanographic and geographic features of the Bras d'Or Lakes. In this paper, the Bras d'Or Lakes are defined as Whycocomagh Basin west, Whycocomagh Basin east, St. Patrick's Channel, North Basin, St. Andrew's Channel, Great Bras d'Or Channel, Bras d'or Lake, East Bay, and West Bay (Fig. 1 and 3). Oceanography was included in the review of historic disease information, disease survey results, and human activities, to provide the scientific advice used for implementing mitigative measures to prevent spread of MSX to unaffected populations in the Gulf of St. Lawrence and Atlantic shore of Nova Scotia.

Historic Disease Information

Molluscs in Atlantic Canada were considered free of all OIE-listed diseases based on active surveillance performed by Fisheries and Oceans Canada (DFO) since 1990, as well as on historic disease investigations dating back to the 1930s.[4-6] Since 1990, over 8000 American oysters have been screened for disease to meet the requirements for inter-provincial transfers through DFO's National Code on Introduction and Transfer of Aquatic Animals, and Fishery (General) Regulations, aimed at protecting fish habitat from negative disease, genetic, and ecological impacts.[7] In addition, significant sampling of oysters had been undertaken for disease research projects. Based on this information, a map of endemic parasites and disease profiles was developed that enabled the industry to make 'like-to-like' disease profile transfers within Atlantic Canada. The information gathered reinforced the historic data on Malpeque disease (caused by an unidentified infectious agent) and demonstrated that oysters within the southern Gulf of St. Lawrence are homogenous and differ from those in the Bras d'Or Lakes.[8] Oysters from the Bras d'Or Lakes remain susceptible to Malpeque disease when exposed to sub-clinical carriers from the southern Gulf of St. Lawrence.

MSX was listed by the OIE as a reportable disease until the end of 2005. Its listing was based on the devastating effects of the disease on *C. virginica* stocks on the Atlantic Coast of the United States.[9] It first appeared in 1957 in Delaware Bay and by 1959 had spread to other major oyster beds in Chesapeake Bay.[10,11] Mortalities

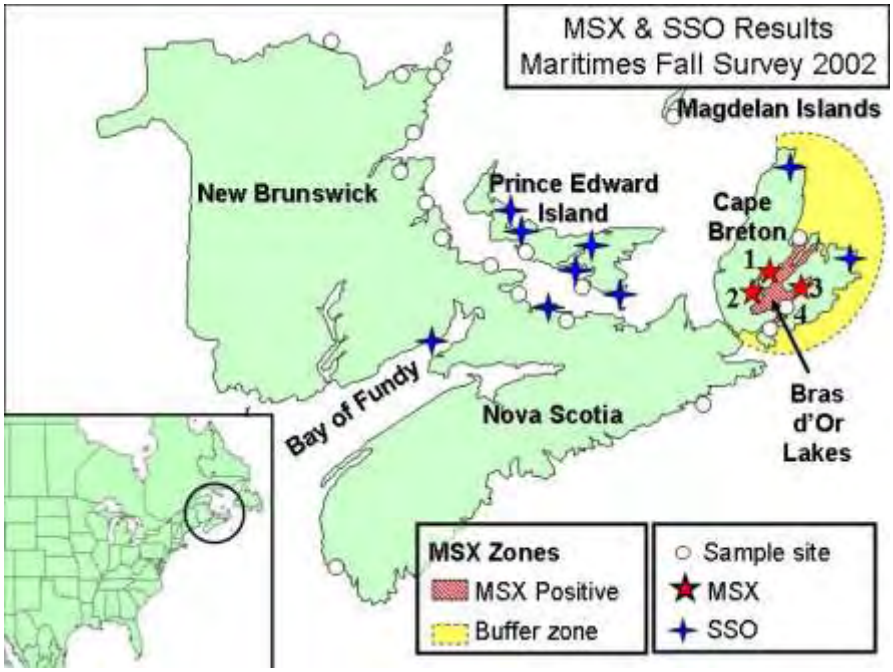


Figure 1
Map of Atlantic Canada showing the results of the disease survey and MSX zonation. Within the Bras d'Or Lakes the MSX-positive areas were: 1. St. Patrick's Channel (multiple sample sites), 2. Gillis Cove, and 3. Eskasoni. MSX was not detected at site 4, Chapel Island.

were approximately 90 to 95% and the current oyster industry in Chesapeake Bay is estimated to be 10% of pre-MSX values.[12] The disease spread north and south over the years, attributed often to human activities related to transfers of sub-clinical infected oysters.[13] The most northerly report of an MSX epizootic occurred in 1996 in the Piscataqua River on the Maine–New Hampshire border.[3] Low levels of MSX have also been detected in Pacific oysters *(Crassostrea gigas)* in Korea, Europe, and the west coast of the United States, but infections are limited and have had a negligible impact on the health of this species.[9,14,15]

In affected populations of *C. virginica* in the eastern US, new infections are detected in early summer (June) and continue through to late fall. The heaviest mortalities occur in late summer and fall.[16-18] A second wave of mortalities is observed the following spring as metabolic activity increases and infected oysters appear unable to recover. Temperature and salinity are the primary environmental influences on MSX proliferation in *C. virginica*. MSX is found in salinity ranges of 10 to 25 ppt and seawater temperatures of 3º to 25ºC, with epizootics occurring when conditions approach 20 ppt and 20ºC. However, it is unclear if these parameters are important to the oyster, the parasite, or its putative intermediate host.

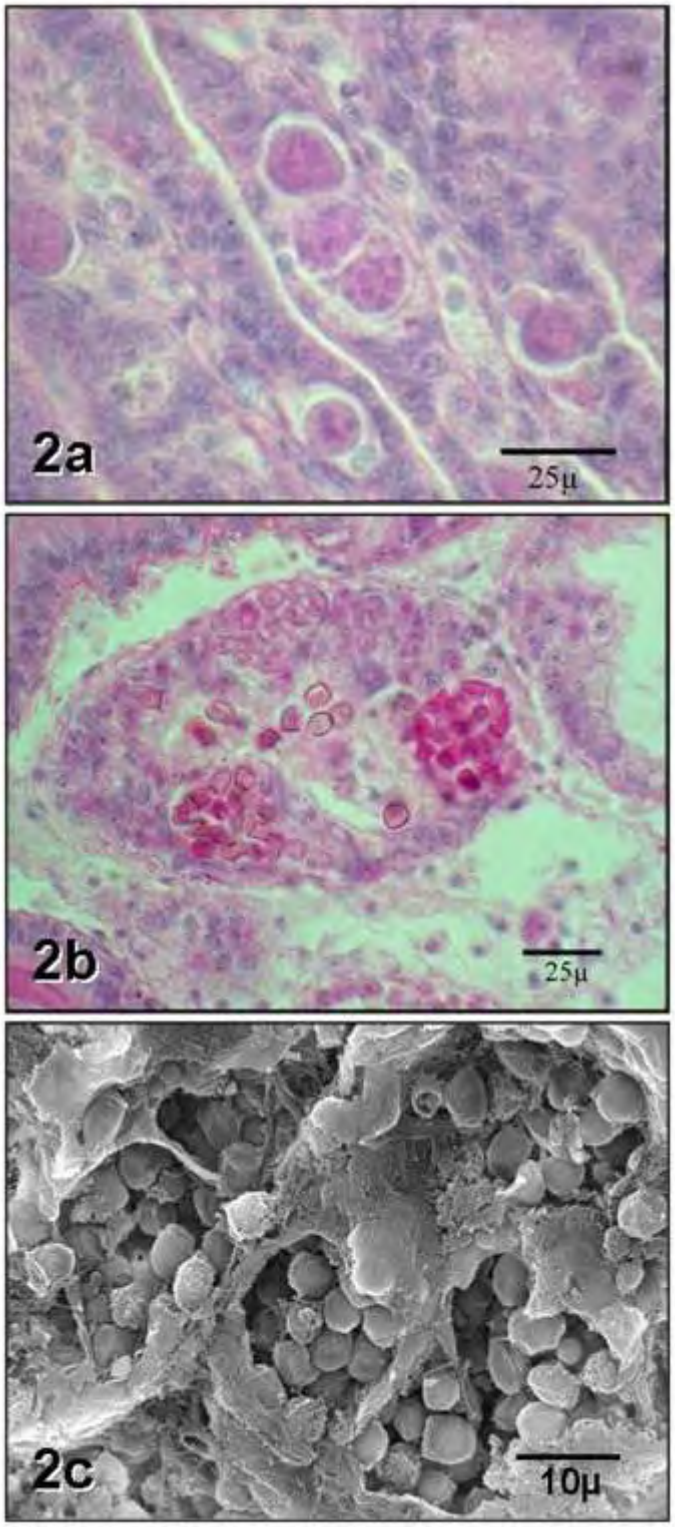
The natural barrier to disease created by the cold waters of the Bay of Fundy and the Atlantic coast of Nova Scotia, as well as an absence of oyster stocks along much of the Atlantic shore of Nova Scotia, were thought to provide protection from diseases such as *Perkinsus marinus* (Dermo disease), *H. nelsoni* and *H. costale* (SSO disease) that plague oysters in the eastern United States.[19] It was recognised that if these diseases were to emerge in the warm waters of the Gulf of St. Lawrence and Bras d'Or Lakes the impact on oyster stocks would likely be as devastating as those experienced by stocks in the United States. The value of the Atlantic Canadian oyster industry has been estimated at Can$10 million, with approximately 90% of production coming from the southern Gulf of St. Lawrence and the remainder from Cape Breton.[20]

Temperature and salinity profiles within Atlantic Canada, and specifically within the Bras d'Or Lakes, are within the ranges documented as suited for MSX proliferation.[21] However, winter ice and duration of freezing conditions in Atlantic Canada have not been a factor in American oyster investigations and, therefore, were an unknown influence on and hope for control of MSX in Canadian oyster stocks.

Although the distribution and seasonal dynamics of MSX in *Crassostrea virginica* in the United States have been established, little is known about its life cycle outside the oyster. Moreover, the lack of knowledge concerning the potential intermediate host of *H. nelsoni* complicates attempts at disease containment and mitigation. As with other haplosporidian oyster infections, the spore stage, found almost exclusively in the oyster's digestive gland epithelium, is believed to be the infective stage (Fig. 2).[12] However, experimental infections of naïve oysters have consistently failed to demonstrate this as a direct mechanism of transmission of MSX. In addition, MSX disease does not appear to depend on oyster density. This also infers that an intermediate stage may persist in an unknown host or state for extended periods. Thus, a waterborne host or carrier is suspected.[22, 23] The distribution of *H. nelsoni* in both the Pacific and Atlantic Oceans suggests the intermediate host may be a non-specific ubiquitous vector in the ocean environment and in the Bras d'Or Lakes. The environmental species profile within parts of the Bras d'Or Lakes, notably St. Partrick's Channel where MSX was first detected, has been described as 'Virginian' (i.e. similar to the

Figure 2

Heavy infections of *Haplosporidium nelsoni* observed in *Crassostrea virginica* in Atlantic Canada. 2a) plasmodial infection within the connective tissue of the gill (H & E stain, light microscopy 400x), 2b) spores of *H. nelsoni* within the digestive tubules (H & E stain, light microscopy 1000x), and 2c) spores of *H. nelsoni* within the digestive tubules (scanning electron microsopy) (Photos by A. Veniot).



mid-Atlantic coast of the United States, where *H. nelsoni* has had the greatest impact).[24] This is not clearly ecological and may reflect shipping and other human links to the eastern USA.

Human Activities

Human activities may have played a part in the introduction of MSX into Canadian waters. Although deliberate importation of oysters to the Bras d'Or Lakes is prohibited, recreational boating and commercial shipping traffic—that have direct links to Delaware and Chesapeake bays—have been documented.[25] MSX may have been introduced from hull fouling, ballast water discharge, or simply by disposal of shells by visiting consumers. Although it is doubtful that a direct link between these activities and the initial introduction can be verified, it is important that they are considered in the analysis of its potential spread both within the lakes and to other parts of Atlantic Canada.

Following the detection of MSX, industry activities were reviewed and protocols for the harvest of oysters were developed with the stakeholders. These included preventing re-soaking oysters from Cape Breton in open waters and ensuring that oysters were processed in plants that were not discharging effluent into open water.

Results of MSX Disease Survey

Following detection of MSX in oysters from St. Patrick's Channel in the Bras d'Or Lakes of Cape Breton, a meeting with key stakeholders was held to review both current and historic industry activities. It was determined that oysters had been transferred from the Bras d'Or Lakes to other parts of Atlantic Canada, putting oyster populations in the southern Gulf of St. Lawrence at risk.

Sites chosen for surveillance represented

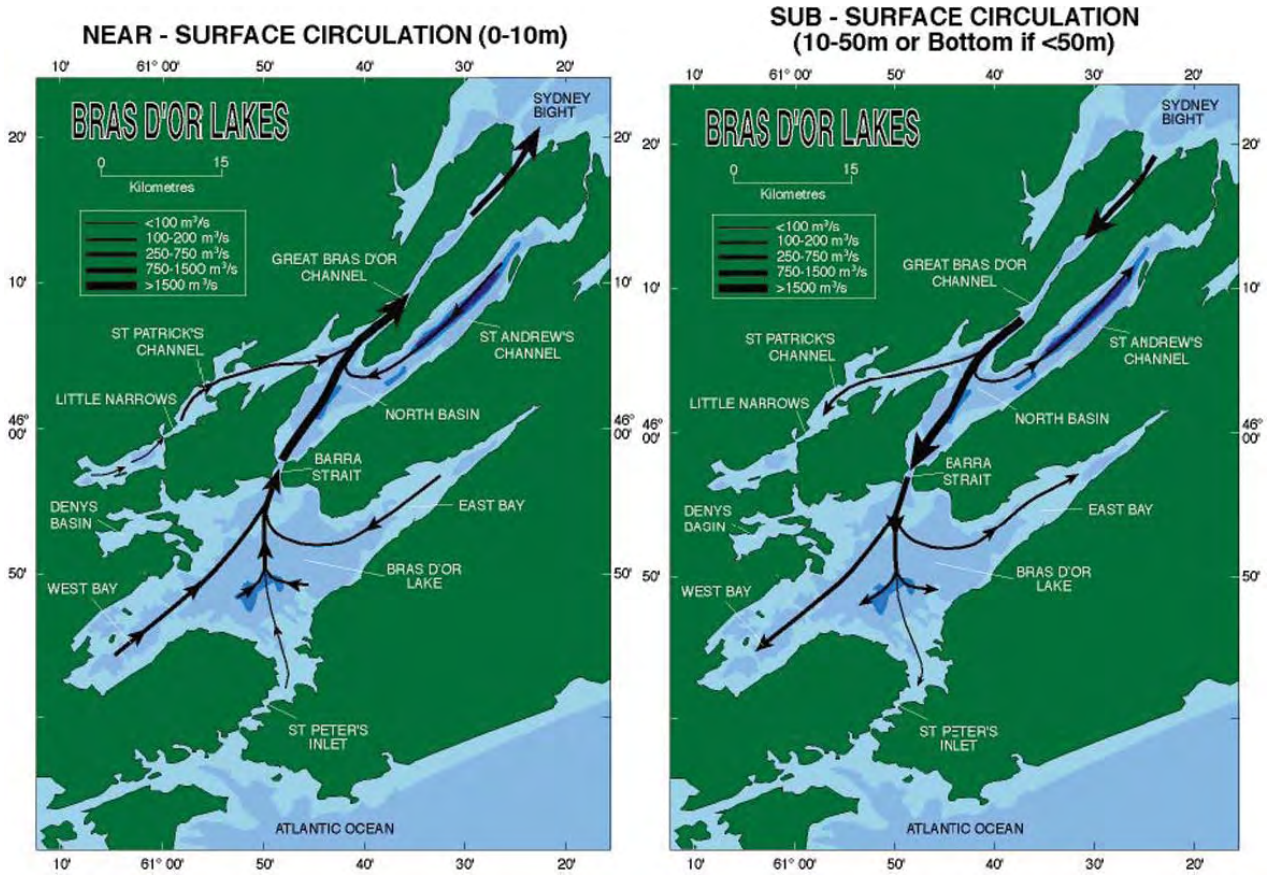
commercially-important bays linked directly through transfers from the Bras d'Or Lakes, those linked indirectly (oysters from directly linked sites), or sites with no oyster transfer links.[26]

A sample size of 60 was chosen to detect a presumed pathogen prevalence occurring in 5% of the population. Approximately 3000 oysters were sampled from 39 sites.[9,27] Diagnostic testing followed OIE guidelines. Histopathology was used for disease screening, and polymerase chain reaction (PCR) and in-situ hybridization for infection confirmation. The molecular confirmatory tests were required for differentiation between plasmodial stages of *H. nelsoni* and *H. costale.* When both haplosporidians were detected by PCR, an in-situ hybridization test reaction was used to identify the dominant infection within individual oysters.[28]

Results from the 2002 fall/winter survey are shown in Figure 1. MSX was confirmed in three areas of the Bras d'Or Lakes: 1) St. Patrick's Channel (multiple sample sites), 2) Gillis Cove, and 3) Eskasoni. MSX was not detected in the southern basin at Chapel Island. Dual infections of *H. nelsoni* and *H. costale* were detected by PCR, but *H. nelsoni* was confirmed as the dominant infection. SSO was detected by histology and confirmed by PCR at low prevalences and intensities in areas of higher salinity outside the Bras d'Or Lakes. OIE was notified of this new occurrence of *H. costale* in Canada.[29]

With MSX and SSO disease distributions documented, two main questions were considered:

Figure 3
Near-surface and sub-surface circulation in the Bras d'Or Lakes.[21] Figure reproduced with the permission of the Proceedings of the Nova Scotia Institute of Science.



- Should oysters be moved freely within the Bras d'Or Lakes to areas where these diseases had not yet been detected?
- Should oysters from areas of the Bras d'Or Lakes, where MSX had not been detected, be exported to waters believed to be free of MSX?

To address these questions, consideration of the oceanographic influences within the Bras d'Or Lakes was required.

Bras d'Or Lakes—Oceanography

The mean circulation in the Bras d'Or Lakes is estuarine; near-surface waters flow predominantly out of the lakes into Sydney Bight driven by freshwater inflow, and deeper waters feature a return flow from the Bight into the lakes (Fig. 3).[21,30] The Bras d'Or Lakes are semi-enclosed with only three points of exchange with oceanic waters: i) the Great Bras d'Or Channel and ii) the Little Bras d'Or Channel connect with the sea in the north; and iii) St. Peter's Canal, a lock system that opens occasionally to allow boats in and out of the lakes, to the south. The exchange between the lakes and the ocean is predominantly through the

Figure 4
Box model depth ranges and flushing times (concentration decreases to 1/e of original value, e-folding) by region: Whycocomagh Basin east (WBE), Whycocomagh Basin west (WBW), West Bay (WB), East Bay (EB), St. Andrew's Channel (St.A), St. Patrick's Channel (St.P), Bras d'Or Lake (BdOrL), North Basin (NB) and Great Bras d'Or Channel (GbdOr).

Region	WBE	WBW	WB	EB	St.A	St.P	BdOrL	NB	GBdOr
Layer					**Layer Depth Ranges (m)**				
1	0-10	0-10	0-10	0-10	0-10	0-10	0-10	0-15	0-15
2	10-Btm	10-Btm	10-Btm	10-Btm	10-50	10-Btm	10-Btm	15-30	15-Btm
3					50-Btm			30-Btm	
					Flushing Time Estimates (d)				
1	3.2	2.1	2.5	67	57	12	25	5	1.7
2	670	701	45	81	54	8	92	2.6	1.4
3					259				
					Particle Transport Times (d)				
1			10.5	11.4	20.5	38.2, 24.3	3.5 70.4 9.1 13.4 5.7	7.5 30	20.1 2.5
2			23.9	20.4	306	42.1, 12.8	18.7 421 30.1 72.1 9.4	42.4 20.1	25.6 3.5
3									

Great Bras d'Or Channel, which is about 320 m wide and a maximum 16 m deep at its mouth, the narrowest point.[21]

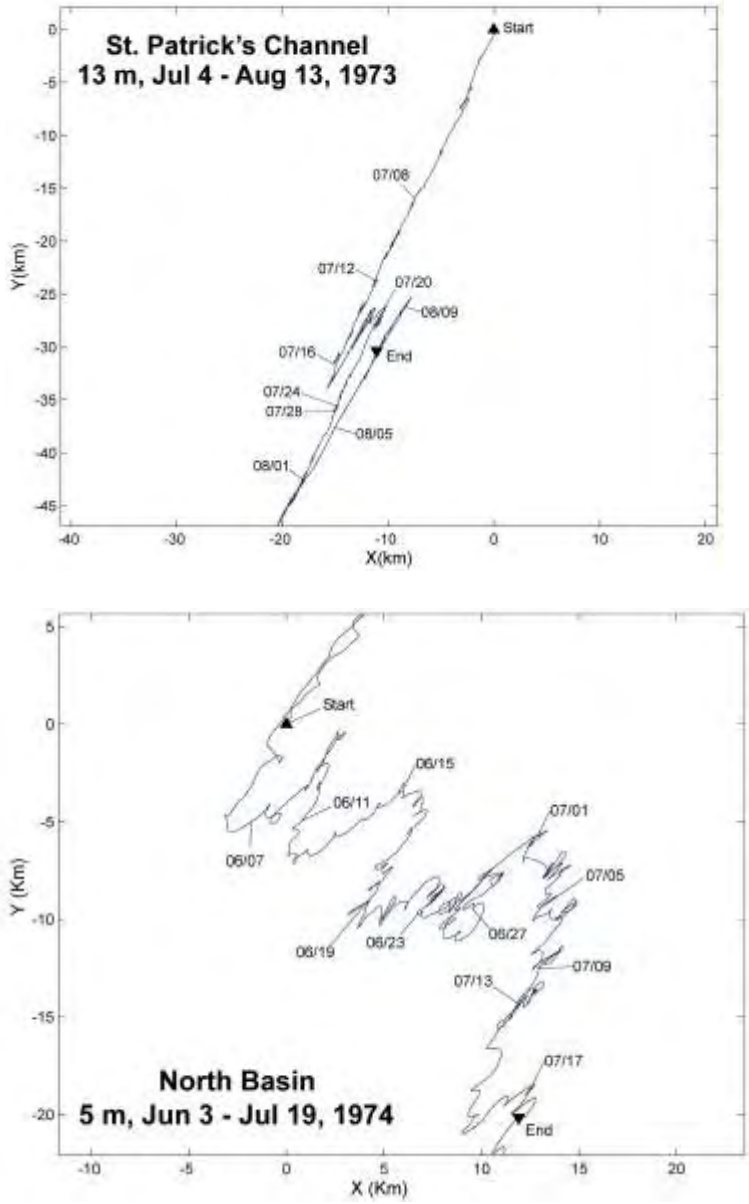
Flushing times from a box model

Estimates of the flushing times in the Bras d'Or Lakes were made from a box model based on mass, heat, and salt conservation.[31] The lakes were divided into 9 geographic areas: Whycocomagh Basin west (WBW), Whycocomagh Basin east (WBE), St. Patrick's Channel (ST. P), North Basin (NB), St. Andrew's Channel (ST.A), Great Bras d'Or Channel (GBDOR), Bras d'Or Lake (BDORL), East Bay (EB), and West Bay (WB). Within each geographic area, 2 or 3 vertical compartments were delineated depending on the area (Fig. 4). The model gave a best fit to the distribution of water properties throughout the lakes and allowed currents and vertical mixing rates to be calculated. A dye was introduced into the different compartments of the model and its concentrations were followed over time.[31] The times for the concentrations to be reduced to about 37% (i.e., 1/e, where e is the base of natural logarithms, a commonly used decay scale) of their initial values were compiled. In the upper layers, flushing times ranged from days (WBE, WBW, WB, NB and GBdOr), to weeks (St.P, BdOrL), to months (EB, St.A).

Another approach to tracing the potential spread of waterborne particles in the lakes was use of existing current meter data to create paths that the object could take if it experienced the flows measured by the instrument.[32] This assumed that the observations are representative of the flow over the area the particle is projected to move. With the complicated geometry of the Bras d'Or Lakes, this is not a reliable assumption—particularly because of changes in the coastline, particles could be projected to end up inland. Conversely, the instrument provides a more likely indication of the strength of the circulation in the area, such that the overall distance the particle is projected to travel may be reasonably accurate.

Examples of the paths that particles would take if they experienced the flows recorded by the current meters are shown in Figure 5. Records from St. Patrick's Channel show a nearly bi-directional flow, with currents directed along the local bathymetry with occasional reversing. The observations from North Basin feature considerably more

Figure 5
Progressive vector diagrams for St. Patrick's Channel and North Basin, Bras d'Or Lakes, NS.



variability imposed on a gradual drift to the southeast.

For some basins, several current meter time series were available. We used the vector-averaged current speeds from the lakes and the lengths of the major areas to give rough estimates of the time it would take a particle to move over the length of a particular basin (Fig. 4).

Both approaches give similar results. Time scales associated with flushing or particle movement within the lakes vary from days to a few months. The shortest flushing times, ~2 days, are in the tidally-energetic Great Bras d'Or Channel where the mean currents are among the strongest in the lakes. The longest times—several hundreds of days—are in the isolated, deep portion of Whycocomagh Basin (which features anoxic (western basin) or low oxygen saturations), and in the deep part of St. Andrew's Channel (where reduced oxygen saturations have also been observed). In the remaining areas of the lakes, flushing times vary from 2 to 90 days and particle transit times vary from 2 to 400 days, with most estimates in the range of 10 to 70 days.

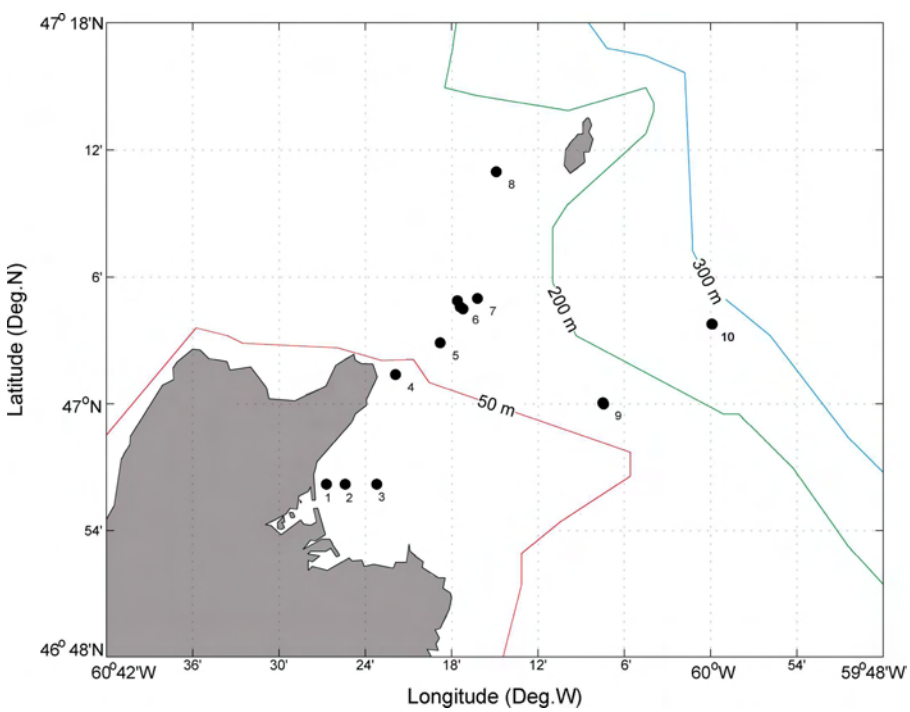
These results indicate that waterborne materials could be carried through regions of the lakes within seasonal time scales.

Currents in Sydney Bight–Northern Cape Breton

The mean circulation in Sydney Bight is dominated by the outflow from the Gulf of St. Lawrence estuary.[33] This outflow becomes the Nova Scotia Current which has been tracked into the Gulf of Maine.[34] The general conclusion is that flow from the Bras d'Or Lakes is most likely to move south through the Sydney Bight and onto the Scotian Shelf. Notably, this makes the transmission of MSX from Maine to Cape Breton by ocean currents unlikely. The modelling of the circulation in Sydney Bight has concentrated mainly on seasonal time scales and has not dealt with current variability. However, there are some in situ measurements from Sydney Bight that allow us to address the variability of the mean flows.

A considerable amount of current meter data has been collected at Cabot Strait off the northern tip of Cape Breton from Cape North (about 1 to 2 km off the coast) towards St. Paul Island. The observations cover the months of June to October and provide 86 monthly means at depths ranging from 13 to 290 m (Fig. 6). Though the dominant mode of the circulation was out of the Gulf, 13 of the 86 monthly averages featured flow into the Gulf. Of these 13, seven

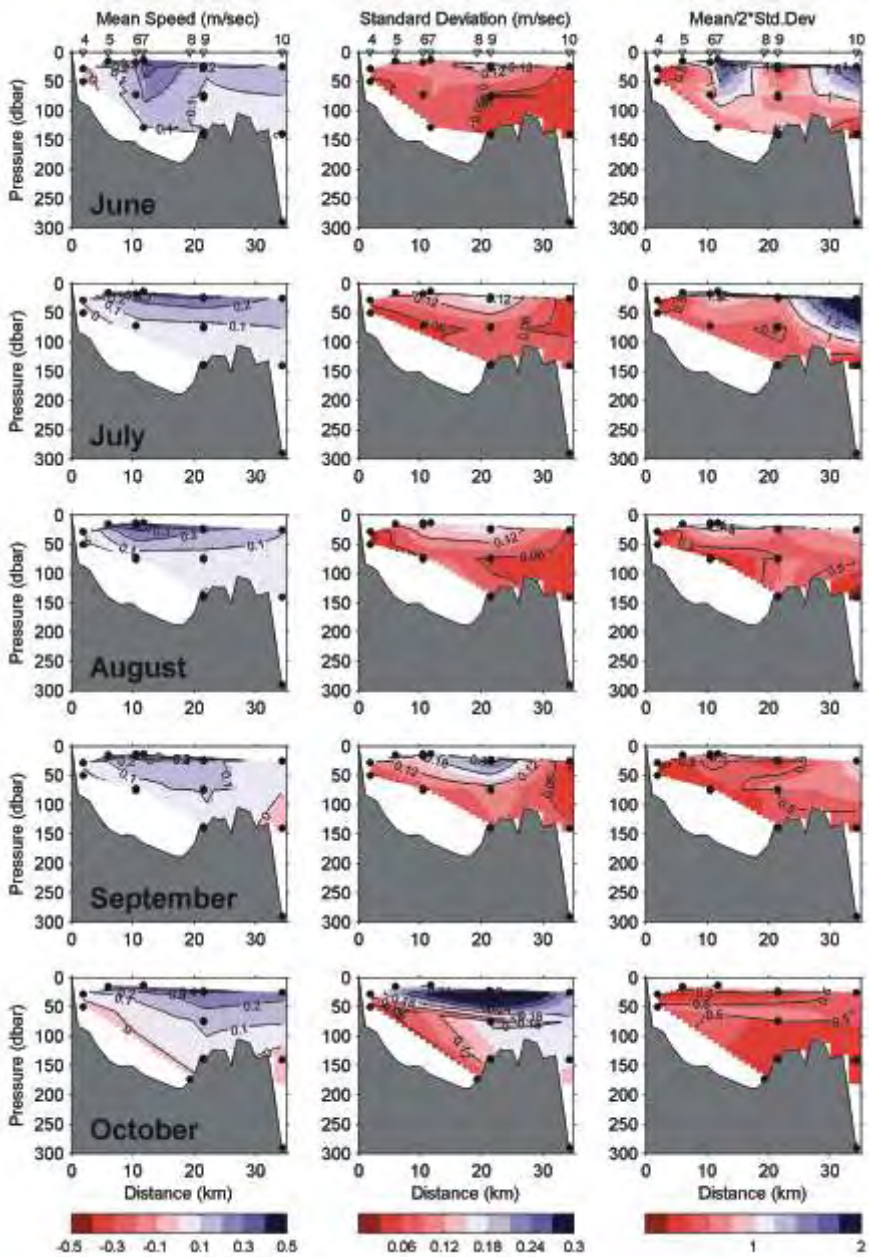
Figure 6
Data moorings 1 to 10 in the Cabot Strait used to create section plots of currents, current variability (standard deviation), and the ratio of monthly mean flow to 2 times the standard deviation.



were from the mooring closest to the coast at 28 and 50 m depth. We also compared the variability of the low frequency current (tides and inertial period motions were filtered out) and the mean flow component into and out of the Gulf. The mean flow component was divided by two times the standard deviation in the same direction (Fig. 7). A value of >1 indicates that the mean flow is larger than the low frequency component for 95% of the time. In 71 of the 86 cases, the ratio was < 1; in 32 of the 86 cases, the ratio was < 0.5. These statistics indicate that despite the strength of the outflow from the Gulf, the low frequency variability is strong enough to reverse the currents for a significant amount of time in the months of August to October when MSX transmission is possible.

Figure 7

Section plots of currents, current variability (standard deviation), and the ratio of monthly mean flow to 2 times the standard deviation from June to October, at depths ranging from 13 to 290 m.



Discussion

To understand the dynamics of MSX in Atlantic Canada and predict the potential for spread, the results of the disease survey were reviewed along with the oceanographic information described above. As well, human activities involved with the oyster industry, recreational boating, and commercial shipping were considered.

The Bras d'Or Lakes are described as a single body of water and are nearly completely surrounded by land. The oyster populations at risk within the lakes occur in the shallow, near-shore bays encircling the coastline, separated by the deep waters of the lakes.[24] The physical separation between the oyster beds may provide limited protection from heavily infected populations; however, it may not protect them from the natural dispersal of the infective stage or putative intermediate host. The connectivity of

the various bays within the lakes is demonstrated by the flushing times and estimates of particle distribution. This analysis suggests a waterborne particle, such as the infective stage of *H. nelsoni*, could be dispersed throughout the lakes within one season. This information supports the concern that, although MSX was not detected at the southern basin of the lakes, oysters may have been exposed to the disease and are not yet expressing clinical signs at levels the sampling was not designed to detect (i.e. < 5% prevelance).

Although hydrographic connectivity within the lakes was established, active transfer of clinically-infected oysters to areas within the lakes where MSX had not yet been detected was not supported. Distance between oyster beds, coupled with the varying environmental profiles within the lakes, suggests that although the infective stage of MSX could be distributed to various areas within a single season, environmental conditions could decrease the impact at local levels.

In the Cabot Strait, the nearshore mean flow back into the Gulf of St. Lawrence observed during August–October indicates that a waterborne particle like the spore stage of *H. nelsoni* could be introduced into the Gulf. However, extensive penetration into the Gulf beyond the Cabot Strait area cannot be established with these current meter data alone. Therefore, there would be a risk of enhancing the spread of MSX if infected animals from within the semi-contained ecosystem of the Bras d'Or Lakes continued to be physically transferred and re-soaked in northern Cape Breton. This activity would be a deliberate breach of the natural oceanographic barrier created by the Great Bras d'Or Channel to an area with hydrographic links to the oyster populations in the Gulf.

Conclusions

To determine the delineation of MSX zones for disease management, oceanography, disease distribution, and human activities were taken into consideration. Subsequently, geographic boundaries were used to define bodies of water affected by MSX.[26]

Thus the Bras d'Or Lakes were described as an MSX-positive zone (Fig. 1). Within this zone, sub-zones were established to protect areas of the lakes linked hydrographically and by human activities, but showing no signs of MSX infection.

A buffer zone requiring increased disease monitoring was delineated around Cape Breton, predominantly on the Atlantic shore, but with sentinel populations on the Gulf of St. Lawrence shores.

For SSO, another disease previously listed by the OIE and detected in Atlantic Canada during the enhanced surveillance for MSX disease, no control measures were implemented. This was due to the broad distribution of SSO and its detection at low prevalences and intensities. In addition, no mortalities or reduced condition of oysters were attributed to this infection in Canadian oyster populations.

The scientific advice was accepted and implemented through licensing mechanisms to industry. This multi-disciplinary approach to MSX management decisions enabled industry activities to proceed while scientific investigations continued. The MSX monitoring continues and the containment measures are reviewed in light of all new information. In this context, *H. nelsoni* has survived prolonged Canadian winter temperatures. In addition, MSX is infamous for 'good' and 'bad' years, depending on environmental conditions. Canadian information suggests that MSX will persist in this northernmost extension of its geographic distribution; but stakeholders remain hopeful that temperature, salinity and, most importantly, oceanographic barriers, will provide the best foundations for reducing the long-term impact of MSX on oyster production in Atlantic Canada.

Acknowledgments

Detection, response, and management of on OIE listed disease requires the cooperation and collaboration of many people. Special thanks go to Michelle Maillet, Anne Veniot, Nellie Gagné, and Jeannette Arsenault of the the Shellfish Health Unit, Gulf Fisheries Centre and to Gary Meyer of the Pacific Biological Station. Provincial colleagues provided valuable advice and knowledge and coordinated the collection of field samples—notably Andrew Bagnall, Lou Clancy, Roland Cusack, Carl Huntington, Neil MacNair, Matt Smith, Richard Gallant, Robert Rioux, Sylvio Doiron, Jacques Mallet, Marie-Josée Maillet and Marcel Légere. Support and leadership was also provided by Unama'ki Institute for Natural Resources and Eskasoni Fish and Wildlife Commission; specifically Charlie Dennis, Allison MacIsaac and Robin Stuart. The DFO multidisciplinary team who responded to the MSX outbreak included Dan Bedell, Pierre Gautreau, Maureen Butler, Joan Reid, Rick Young, Gerard Blanchard, Janet Langille, Christiane Parcigneau, Colin MacIsaac and Darrell Harris. Collaboration and advice from Gene Burresson and Nancy Stokes at the OIE reference lab for MSX, Virginia Institute of Marine Sciences, Virginia, USA. Special thanks go to Maurice Mallet, Sharon McGladdery and René Lavoie for their guidance and insight during this most difficult of situations. The cooperation and collaboration of members of the aquaculture associations and industry associations are also acknowledged. The individuals who suffered tremendous personal losses and who stick to working on the water continue to inspire and teach us to strive for a better understanding of MSX.

References

1. Stephenson MF, McGladdery SE, Maillet M, Veniot A, Meyer G. 2003. First reported occurrence of MSX in Atlantic Canada. *J. Shellfish Res*. 22:355.
2. OIE. 2005. *Aquatic Animal Health Code.* World Animal Health Organization. Available online at www.oie.int/eng/normes/fcode/en_summaire.htm.
3. Barber BJ, Langan R, Howell TH. 1997. *Haplosporidium nelsoni* (MSX) epizootic in the Piscataqua River Estuary (Maine New Hampshire, USA). *J. Parasitol.* 83:148-150.
4. McGladdery SE, Drinnan RE, Stephenson MF. 1993. A manual of the parasites, pests and diseases of Canadian Atlantic bivalves. *Can. Tech. Rep. Fish. Aquat. Sci*. 1931, 121 p.
5. McGladdery SE, Stephenson MF. 1991. Parasites and diseases of suspension and bottom-grown shellfish from eastern Canada. *Bull. Aquacul. Assoc. Canada*. 91-3:64-66.
6. McGladdery SE, Stephenson MF. 1994. A viral infection of the gonads of eastern oyster (*Crassostrea virginica*) from Atlantic Canada. *Bull. Aquacul. Assoc. Canada.* 94:84-86.
7. DFO. 2003. *National Code on Introduction and Transfers of Aquatic Organisms.* Fisheries and Oceans Canada. Available online at www.dfo-mpo.gc.ca/science/aquaculture/ code/ prelim_e.htm.
8. Bower SM, McGladdery SE, Price I. 1994. Synopsis of infectious diseases and parasites of commercially exploited shellfish. *Ann. Rev. Fish. Dis.* 4:1-199.
9. OIE. 2003. *Aquatic Animal Health Manual.* World Animal Health Organization. Available online at www.oie.int/eng/normes/fmanual/A_summary.htm.
10. Haskin HH, Stauber LA, Mackin JA. 1966. *Minchinia nelsoni* n. sp. (Haplosporidia, Haplosporididiidae) causative agent of the Delaware Bay oyster epizootic. *Science* 153:1414-1416.
11. Ford SE, Haskin HH. 1982. History and epizootiology of *Haplosporidium nelsoni* (MSX), an oyster pathogen in Delaware Bay 1957-1980. *J. Invert. Pathol*. 40:118-141.
12. Burreson EM, Ford SE. 2004. A recent review of information on the Haplosporidia, with special reference to *Haplosporidium nelsoni* (MSX disease). *Aquat. Living Resour.* 17:99-517.
13. Ford SE. 1992. Avoiding the transmission of disease in commercial culture of molluscs, with special reference to *Perkinsus marinus* (Dermo) and *Haplosporidium nelsoni* (MSX). *J.*

Shellfish Res. 11:539-546.

14. Friedman CS. 1996. Haplosporidian infection of the Pacific oyster, *Crassostrea giga*s (Thunberg), in California and Japan. *J. Shellfish Res.*15:597-600.
15. Burreson EM, Stokes NA, Friedman CS. 2000. Increased virulence in an introduced pathogen: *Haplosporidium nelsoni* (MSX) in the eastern oyster *Crassostrea virginica*. *J. Aquat. Anim. Health* 12:1-8.
16. Ford S, Powell E, Klinck J, Hofmann E. 1999. Modeling the MSX parasite in eastern oyster *(Crassostrea virginica)* populations. I. Model development, implementation, and verification. *J. Shellfish Res*. 18:475-500.
17. Paraso MC, Ford SE, Powell EN, Hofmann EE, Klinck JM. 1999. Modeling the MSX parasite in eastern oyster *(Crassostrea virginica)* populations. II. Salinity effects. *J. Shellfish Res*. 18:501-516.
18. Powell EN, Klinck JM, Ford SE , Hofmann EE, Jordan SJ . 1999. Modeling the MSX parasite in eastern oyster *(Crassostrea virginica)* populations. III. Regional application and the problem of transmission. *J. Shellfish Res*. 18:517-537.
19. Andrews JD. 1984. Epizootiology of diseases of oysters *(Crassostrea virginica)*, and parasites of associated organisms in eastern North America. *Dis. Mar. Org.* 37(1/4):149-166.
20. Fisheries and Oceans Canada. Statistical services commercial landings. Updated Dec 2005 online at www.dfo-mpo.gc.ca/communic/statistics/commercial/landings/seafisheries/index_e.htm.
21. Petrie B, Bugden G. 2002. The physical oceanography of the Bras d'Or Lake. *Proc. N. S. Inst. Sci*. 42:9-36.
22. Sunila I, Karolus J, Lang EP, Mroczka ME, Volk J. 2000. Transmission of the haplosporidian parasite MSX *Haplosporidium nelsoni* to the eastern oyster *Crassostrea virginicain* an upweller system. *Dis. Aquat. Org*. 2000:42(2):153-155.
23. Ford SE, Xu Z, Debrosse G. 2001. Use of particle filtration and UV irradiation to prevent infection by *Haplosporidium nelsoni* (MSX) and *Perkinsus marinus* (Dermo) in hatchery-reared larval and juvenile oysters. *Aquaculture* 94:37-49.
24. Tremblay MJ. 2002. Large epibenthic invertebrates in the Bras d'Or Lake. *Proc. N. S. Inst. Sci*. 42:101-126.
25. Carver CE, Mallet AL. 2002. *An Assessment of the Risk of Ballast Water-Mediated Introduction of Non-indigenous Phytoplankton and Zooplankton into Atlantic Canadian Waters.* Mallet Research Services Ltd., Dartmouth, Nova Scotia. Vi + 91p. Available online at http://as01.ucis.dal.ca/ramweb/papers-total/DavidLees/BW%20Report%202002.PDF
26. Subasinghe RP, McGladdery SE, Hill BJ. 2004. Surveillance and zoning for aquatic animal diseases. *FAO Fish. Tech. Pap.* 451, 73 p.
27. Fisheries and Oceans Canada. 1984 (revised 2004). Fish Health Protection Regulations: Manual of compliance. *Fish. Mar. Serv. Misc. Spec. Publ. 31 (revised)* iv+50 p.
28. Stokes NA BurresonEM. 2001. Differential diagnosis of mixed Haplosporidium costale and *Haplosporidium nelsoni* infections in the eastern oyster, *Crassostrea virginica*, using DNA probes. *J. Shellfish Res.* 20:207-213.
29. Stephenson MF, Maillet M, Veniot A, Gagné N, Arsenault J. 2005. Dynamics of MSX and SSO in *Crassostrea virginica*, American oysters in Atlantic Canada. *J. Shellfish Res.* 24: 677.
30. Gurbutt PA, Petrie B. 1995. Circulation in the Bras d'Or Lake. *Estuar. Coast. Shelf Sci.* 41:611-630.
31. Gurbutt PA, Petrie B, Jordan F. 1993. The physical oceanography of the Bras d'Or Lake: data analysis and modelling. *Can. Tech. Rep. Hydrogr. Ocean Sci*. 147, 61 p.
32. Krauel DP. 1975. The physical oceanography of the Bras d'Or Lake 1972-1974. *Fish. Mar. Sci. Serv. Tech. Rep.* 570, 357 p.
33. Han G, Loder JW, Smith PC. 1999. Seasonal mean hydrography and baroclinic circulatation in the Gulf of St. Lawrence and on the eastern Scotian and southern Newfoundland Shelves. *J. Phys. Oceanogr*. 29:1279-1301.
34. Sutcliffe WH Jr, Loucks RH, Drinkwater KF. 1975. Coastal circulation and physical oceanography of the Scotian Shelf and the Gulf of Maine. *J. Fish. Res. Bd. Can.* 33:98-115.

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Models of Hydrodynamic Pathogen Dispersal Affecting Scottish Salmon Production: Modelling Shows how Scotland Eradicated ISA, but not IPN

Alexander Murray

Alexander G. Murray, Trisha L. Amundrud, and Philip A. Gillibrand

Coupled hydrodynamic and particle tracking models that are commonly used to simulate water quality can be adapted to investigate the advection of other particles, including pathogens, and are therefore a useful tool in disease control. The Fisheries Research Services Marine Laboratory of Scotland has developed numerical models to investigate the spread of diseases affecting the salmon aquaculture industry in order to advise on appropriate disease control measures. Three such models are described here. First, a simple tidal excursion model was successfully used as part of an eradication program of the viral disease infectious salmon anaemia (ISA). Second, a model combining simple hydrodynamics and particle tracking of particles with different biological properties was developed. This model confirms the suitability of the tidal excursion model for understanding control of ISA, but suggests other pathogens such as infectious pancreatic necrosis virus or larval sea lice may be transported well beyond tidal excursion limits. Third, a more complex hydrodynamic model of Loch Torridon (north-west Scotland) was combined with a sea lice particle model to generate concentrations of sea lice larvae. Predicted locations of high lice levels are highly dependent on wind conditions, indicating that the local environment can greatly influence transport. The three models demonstrate that different models with different levels of hydrodynamic and biological sophistication are required to understand and control various pathogens.

Introduction

The waters of the oceans are in constant movement at scales ranging from molecular diffusion to global circulation; in many coastal waters the most obvious movement is the semi-diurnal tidal cycle. This constant motion carries not only water but particles that are suspended in that water such as pollutants or larval organisms.

Particles suspended in the water include many pathogens of aquatic animals that must go through a phase where they are suspended freely in the water in their search for new hosts. Micro pathogens such as viruses and bacteria are colloidal and completely passive in their movement with respect to water. Larger parasites may have some swimming ability, although few larval parasites can swim with

speeds and endurance comparable to tidal currents. However, even when pathogens are passive, their biological parameterization in the model, such as their survival time, is important for modelling where the water may carry infectious pathogens.

Due to this interaction between hydrodynamics and biology, a useful approach to estimate areas at risk from a release of pathogens at a point source is to use a combination of hydrodynamic and particle tracking models. These models may be of varying complexity and in this paper we discuss three models that have been used by the Fisheries Research Service (FRS) Marine Laboratory, an agency of the Scottish Executive, to assess potential pathogen dispersal around salmon farms.

A Tidal Excursion Model

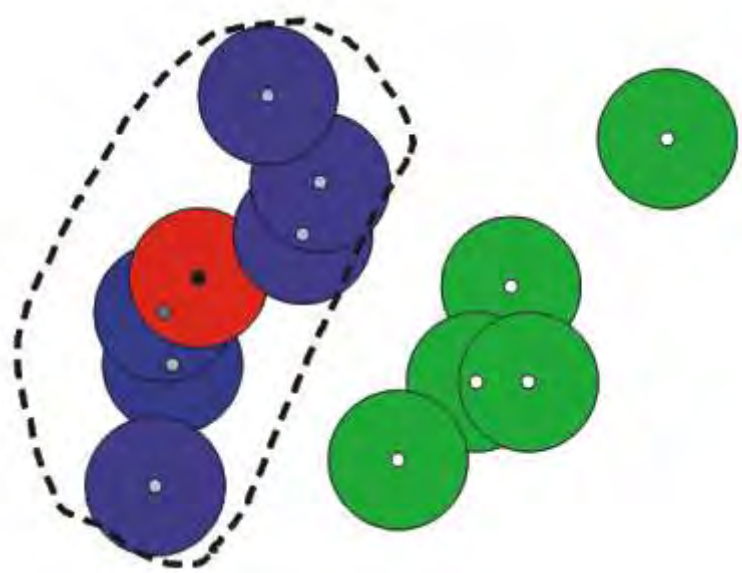
FRS began to develop formal models of pathogen dispersal as a result of an outbreak of infectious salmon anaemia virus (ISAV). This occurred in 1998 to 1999 and was confirmed on 11 sites and officially suspected on many more[8] with costs to the industry estimated at over £25M (Can$51M). Most of the site-to-site spread was shown to be due to movements of fish or shipping; however local transmission of infection between neighbouring sites through the advection of water was thought possible. Recent analysis of ISAV outbreaks in Norway has also shown local association of outbreaks,[2] again probably reflecting transmission through the water.

Because of the potential for transmission through water, FRS devised zones for surveillance and fallowing of farms should a case of ISAV be confirmed in a particular area. The model devised[8] was based on tidal excursion distance X_t around a farm:

$$X_t = UT/\pi$$

where U is the tidal current amplitude (maximum spring tide current) and T was the tidal period (12.42 hours). This generated approximate tidal excursion distances of 7.3 km for mainland Scotland and 3.6 km for the Shetland Isles[8] using average tidal currents. The area within 1 tidal excursion distance of an infected farm was described as the infected zone, while all sites whose tidal excursion distances overlapped the infected zone or other sites within the surveillance zone, were labelled as in the surveillance zone (Fig. 1).

Figure 1
The tidal excursion model with infected (red) and surveillance (blue) zones shown. Tidal excursion around arms outside the surveillance zone (green) are also shown. The site in the middle of the red zone is infected and must be culled, while other sites within the surveillance zone are placed on increased surveillance, which is only lifted after the last site is fallowed.



The tidal excursion model can be considered successful for the case of ISAV control as it formed part of a successful eradication program. However, it was based on the assumption that infectious viruses were not transported outside the tidal excursion zone. In practice some increased surveillance did occur over a

40-km radius zone around infected sites, allowing for the possibility of transport outside the tidal excursion zone.

A Simple Coupled Hydrodynamic and Particle-Tracking Model

After the ISA epidemic was stamped out, FRS developed more sophisticated pathogen dispersal models to test assumptions behind the tidal excursion model and its potential for further application. With this aim, we developed and coupled a simple hydrodynamic model and a simple particle-tracking model using experimental data to parameterize the biological processes.

We apply the model with biological parameters appropriate to two viruses: ISAV and infectious pancreatic necrosis virus (IPNV), which is widespread in Scottish salmon farms.[6] The model is also fitted with parameters to describe dispersal of larval sea lice, a major parasite of farmed and wild salmonids.

Physical processes

The hydrodynamic model incorporates three types of water motion: tidal advection currents, residual advection currents and turbulent diffusive currents (Fig. 2). Motion due to each type of current is calculated for a model time step Δt. The tidal advection current, M_t, is sinusoidal with velocity depending on the time of the tidal cycle τ, returning particles to their original location over a tidal cycle:

$$M_t = U \sin (2\pi\tau/T)$$

The residual advection current, M_a, has a simple constant rate, c, that moves particles in a constant direction and represents such features as net outflow in an estuary, large-scale coastal currents, or prolonged wind in a given direction.

$$M_a = c$$

Turbulent diffusive currents, M_d, move particles in random directions at a strength dependent on a diffusion coefficient D, (in the following, $D = 10^4$ cm^2s^{-1}). This current disperses particles released from a point source with time

$$M_d = \zeta\sqrt{D\Delta t}$$

Here ζ is a random value between -1 and 1.

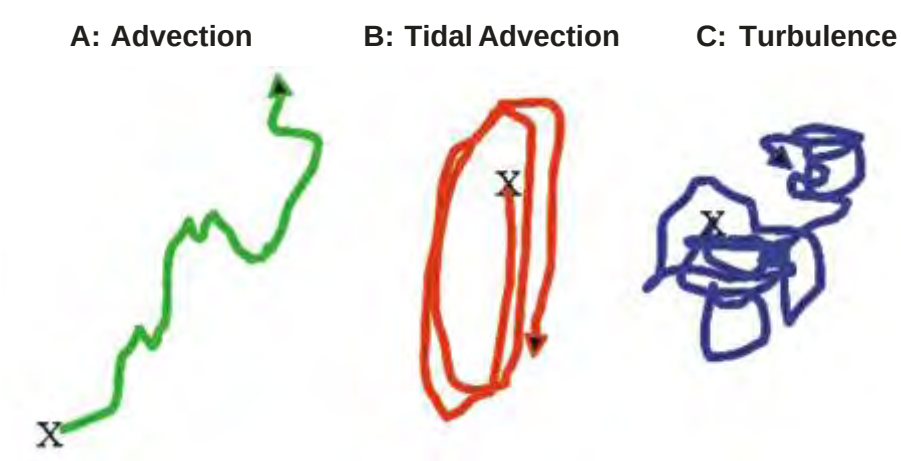


Figure 2
Patterns of movement of particles due to: residual advection (a), tidal advection (b), or turbulent diffusion (c). Two particles will follow the same track under (a) or (b), but under (c) no two tracks are identical.

These movements are combined to generate the net movement of a particle. Tidal and residual currents are restricted to the x-axis only while diffusive movements are allowed to occur throughout the horizontal plane.

$$\Delta X/\Delta t = M_t + M_a + M_{dx}$$
$$\Delta Y/\Delta t = M_{dy}$$

Particles move with the currents over a period of 10 days. Because of turbulent diffusion, particles released simultaneously will disperse with time.

Biological processes

For the parameterization of biological processes, viral shedding is assumed to occur over a period of 5 days (based on experimental observation of both IPNV and ISAV shedding). Shedding of sea lice is different, as elevated shedding occurs shortly after events such as bath treatment of infected farms, and so lice are assumed to be shed over only 1 tidal cycle. The number of pathogens represented by each model particle is found by multiplying the number of pathogens shed per unit weight of fish per hour by the biomass of fish on the farm and dividing by the number of simulated particles shed per hour (120 for viruses or 240 for lice).

For the examples shown here we use shedding rates of 17×10^9 $TCID_{50}$ mL^{-1} kg^{-1} h^{-1} for ISA and 1.7×10^9 $TCID_{50}$ mL^{-1} kg^{-1} h^{-1} for IPNV based on experimental work in preparation. For sea lice, we estimate shedding of 10 larvae that survive to the copepodid stage kg^{-1} h^{-1}. These figures are intended illustratively and will be subject to refinement as analysis of experimental data is completed.

During their time of transport, pathogens could die or lose infectivity. Thus particles representing viruses decay with time; decay rates of 10% h^{-1} are used to simulate ISAV (a rough estimate based on work in preparation showing a decay rate in sterile water of 5 to 10 times that of IPNV) and 1.6% h^{-1} for the more robust IPNV.[9] Thus after an hour a particle initially representing 1 unit of ISAV represents 0.9 units or, for IPNV, 0.984 units. Sea lice are simulated as having a fixed life-span with sealice becoming infectious copepodids after 4 days and dying after a further 7 days.[3]

Results

Modelled concentrations of particles are found by summing the number of particles present in spatial boxes defined by the gridded model domain, separately for each time step of the simulation, and dividing by the volume of these boxes (100 × 100 × 4 m). This gives the relative infectivity of a grid cell throughout the simulation, rather than a snapshot of infectivity at the conclusion of the simulation. These local concentrations of particles are multiplied by the number of pathogens each particle represents to give concentrations of pathogens at each time step.

For the viruses, the highest concentration present in each box, at any time during the simulation, is found. This is then divided by a minimum infectious dose (we use 10 for ISAV $TCID_{50}$ mL^{-1} and 0.1 $TCID_{50}$ mL^{-1} for IPNV; these estimates are subject to the same reservations as shedding rate estimates) to give a relative concentration. If the relative concentration exceeds 1 then a risk of infection being induced locally exists on at least one occasion in the simulation.

For sea lice, concentrations are summed over all time steps in the simulation run, multiplied by time-step length, to give an output with units of infectious sea-lice larvae-hours m^{-3}. This is a measure of the number of times a fish would be exposed to risk of infection if it were present in a particular area for the period of the model

run. Sealice have no minimum infectious dose and infection is a cumulative affair.

Results from the ISAV dispersal simulations are all fairly similar, regardless of advection velocity (Fig 3a). This suggests that ISAV is unlikely to be found far beyond the distances due to tidal excursion and hence that the tidal excursion model is an adequate representation of ISAV dispersal.

Results from the IPNV dispersal simulations are more sensitive to the velocity of any advection current (Fig. 3b), with even a small velocity resulting in the potential for IPNV to be transported kilometers out of the tidal excursion zone at concentrations exceeding minimum infectious doses. Note that it is not certain, or merely possible, that sites at the distances shown will be infected. Variable currents, or increased turbulence, could increase the area at risk of infection at a given distance from the source, but the maximum distance of risk would be reduced. The existence of risks far outside the tidal excursion distance suggests that model is an inadequate description of IPNV dispersal.

Results from the sealice dispersal simulation are quite at odds with the tidal excursion model. Not only is it quite possible for lice to be transported out of the tidal excursion zone, but the

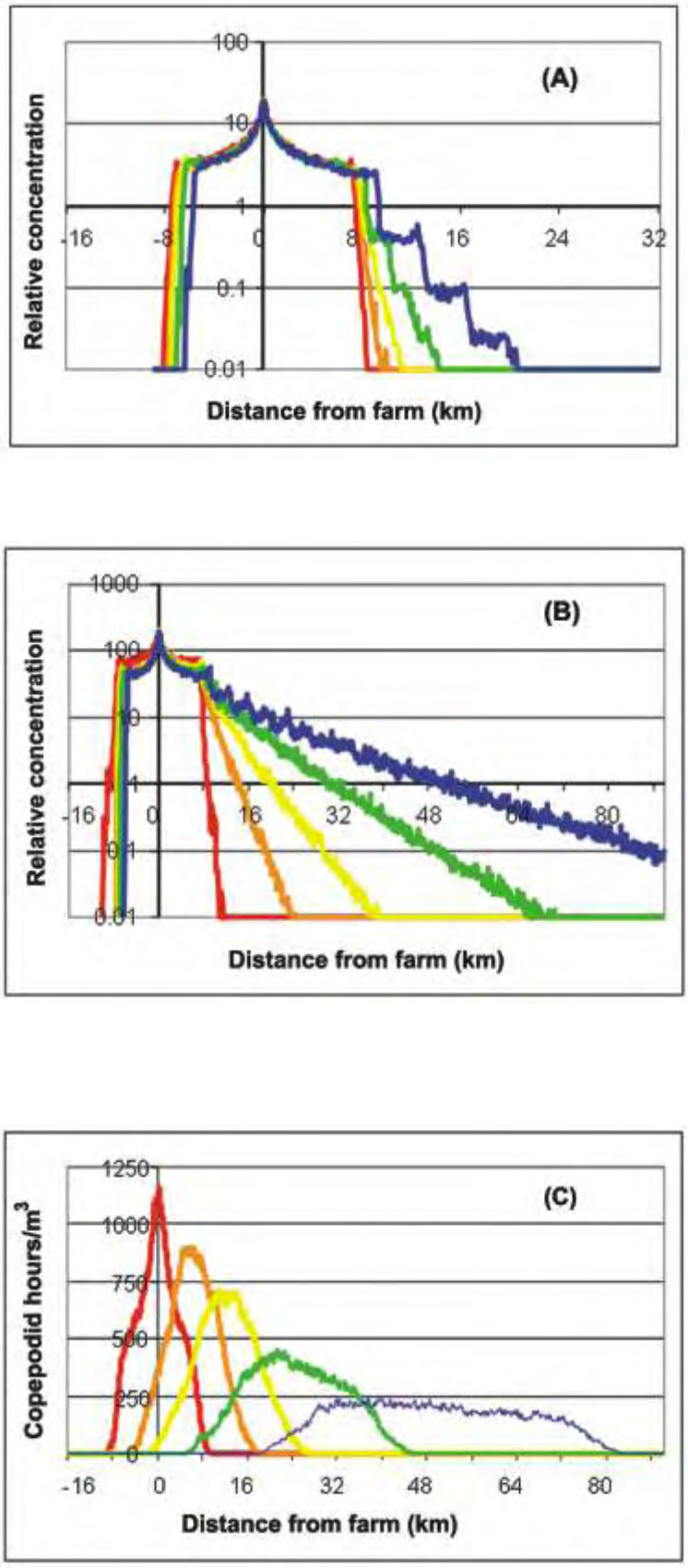


Figure 3
Log concentrations of virus relative to minimum critical dose for (A) ISAV and (B) IPNV, and concentration of (C) sea lice against the distance along X-axis from a source farm containing 1000 tonnes of salmon. Distributions are shown for residual current speeds of zero (red), 1 (orange), 2 (yellow), 4 (green) and 8 (blue) cm s^{-1}. The currents under zero residual current are tidal excursion model with turbulence, and hence similar to the assumptions of the tidal excursion model.

location of highest infection risk is also transported outside the zone with even quite weak currents, and indeed risk at the origin becomes small under moderate advection. Clearly the tidal excursion model is inadequate for sealice, but more complex models may provide more information on the formation of concentrations of larval lice.

The patterns of risk appear to be relatively robust to assumptions about shedding rate or minimum infectious dose, but do appear sensitive to pathogen decay rate. Low turbulence had little effect on sea lice or ISAV dispersal, but extended distance for IPNV risk by a few kilometers. More work will be done as data become available.

A More Sophisticated Coupled Model of Sea Lice in Loch Torridon

FRS has developed a more complex model of the dispersal of larval sea lice as part of a study of the circulation in Loch Torridon in north-west Scotland. This fjordic system consists of 3 basins: outer and inner Loch Torridons, with Loch Sheildaig lying between the two. A number of salmon farms are located in the system and particular interest has been placed in the interaction between sea lice larvae near the mouth of the Sheildaig River, which supports a wild sea trout population, and a farm in Loch Shieldaig nearly 5 km away (Fig. 4).

To study such interactions a 3-dimensional hydrodynamic model of the loch has been developed. The model is based on the GF8 model of the Saint Lawrence estuary and fitted for Torridon bathymetry.[4] The model has been forced using tidal elevation at the loch mouth, plus wind and freshwater inputs. Model output includes surface currents in the X and Y dimensions (for computational reasons the model is not orientated north-south). Vertical currents are not used as sea lice larvae are assumed to maintain themselves within the first 4 m of the surface as suggested by observational studies.[4]

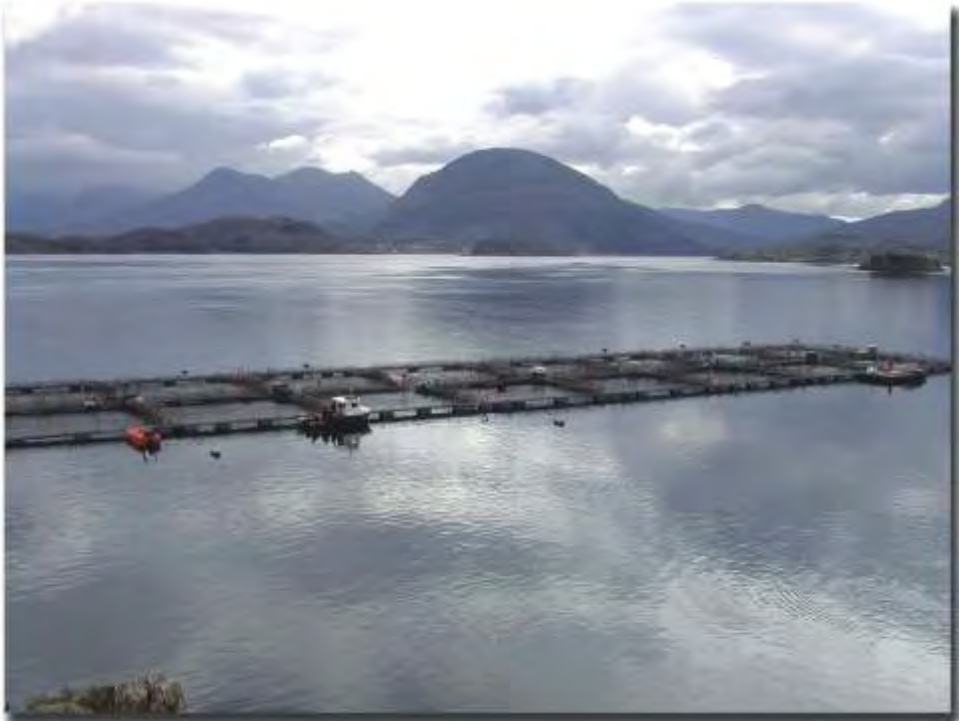


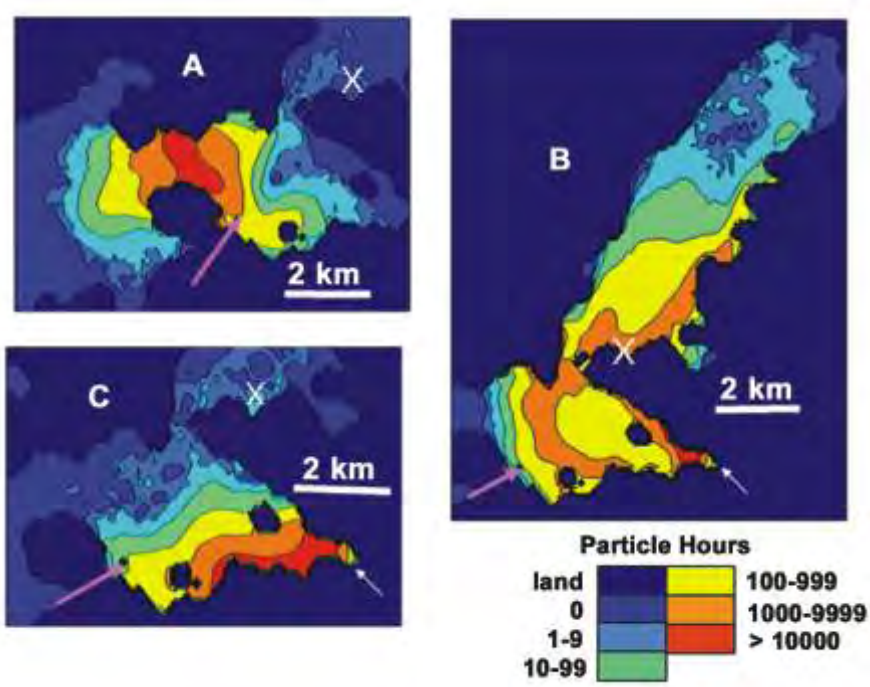
Figure 4
A salmon farm in Loch Shieldaig
(photo Michael Penston)

While sea lice larvae maintain themselves in the surface layer, they are otherwise treated as passive particles in the simulation. The larvae mature through two nauplii phases before becoming infectious copepodids. The two nauplii take a combined 4 days and copepodids survive a further week at 10°C; development time is temperature sensitive.[3] The particle-tracking model takes hydrodynamic model outputs and interpolates for the particles exact position, using inverse square weighting. Turbulent diffusion is also calculated based on local shear.

Particles are released over a double tidal cycle (25 hours) to ensure simulated dispersal is not sensitive to a specific state of the tide. Particles are tracked for 11 days of viability and the location of each particle is recorded hourly after the first 4 days. The final output from each simulation is the number of hours each grid square has been occupied by a particle (particle-hours) once that particle has become infectious. No attempt has been made to convert these relative model concentrations into absolute concentrations, as specific data on shedding from the farm has not been obtained for the times of the simulations and the aim of the modelling has been to identify areas and patterns of potential risk.

Some examples of preliminary model outputs are shown for selected periods of observed wind and tidal forcing (Fig. 5). From these results we see that concentrations of larval lice may be formed at locations distant from the salmon farm, in line with the expectations of the simple model. However, in this case the lice are trapped in coastal waters forming much more intense areas of concentration than in the relatively spread-out simple model (Fig. 3c). The location of these concentrations varies significantly with the wind direction. This may explain the difficulty several studies have had in linking lice on fish farms with lice on wild fish; any effect depends on not only lice production but also wind or other physical environmental factors. In our case the models, and field observations,[7] suggest winds in Loch Torridon may on occasion funnel lice to areas with sensitive sea trout populations. Conversely, Brooks[1] found sea lice from salmon farms in fjordic systems in British Columbia were likely to be washed out to sea by the time they matured. Any risk therefore depends on both biology and physics, and as both models continue to be developed specific areas identified as at risk may be reassessed.

Figure 5
Some examples of outputs from the Torridon model for sea lice copepodids under wind and tidal forcing observed in July 2000 (A), November 2001 (B), and April 2003 (C) winds. Units are log particle-hours per 100 m grid cell (a relative scale). Also shown are locations of lice source (pink arrow), location of a farm in upper Loch Torridon (white X) and of the mouth of the River Shieldaig (white arrow, not on A).



Conclusion

The areas at risk from pathogens transported by movement of water depend on both hydrodynamics and the biological properties of the pathogen. As a result, different pathogens re-

quire different models and different management strategies for their control. The simple tidal excursion model is shown to be adequate for describing ISAV dispersal in Scottish water and this was used successfully to devise strategies for ISA eradication from farmed salmon. Most spread occurred through movements of fish between sites and harvest visits by well boats.[6] Conversely, IPNV's dispersal is not well described by the tidal excursion distance and dispersal might potentially occur over 10s of kilometers under realistic net current velocities. IPNV's prevalence on Scottish salmon farms has been increasing rapidly over recent years[5] and part of the reason for this appears to be spread between marine sites, although other factors are involved. Sea lice dispersal is also not well described by either the tidal excursion model or the simple coupled model. In this case, more complex coupled modelling allows features such as local formation of high concentrations distant from sources to be simulated, in line with observations.

Acknowledgments

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References

1. Brooks KM. 2005. The effects of water temperature, salinity, and currents on the survival and distribution of the infective copepodid stage of sea lice (*Lepeophtheirus salmonis*) originating on Atlantic salmon farms in the Broughton Archipelago of British Columbia, Canada. *Rev. Fish. Sci.* 13:177-204
2. Brun E , Lyngstad T, Jansen P, Johnsen S, Sindre H, Modahl I, Jonassen CM. 2005. Epidemiological characterisation of infectious salmon anaemia outbreaks in Norway 2003 and 2004. In: *Abstracts of the European Association of Fish Pathologists 12th International Conference,* Copenhagen, O-002, p.7.
3. Johnson SC, Albright LJ. 1991. Development, growth, and survival of *Lepeophtheirus salmonis* (copepoda: caligidae) under laboratory conditions. *J. Mar. Biol. Assoc. UK* 71:425-463.
4. Murray AG, Gillibrand PA. 2006. Modelling Salmon Lice Dispersal in Loch Torridon, Scotland. *Mar. Poll. Bull.* (in press)
5. Murray AG, Busby CD, Bruno DW. 2003. Infectious pancreatic necrosis virus in Scottish Atlantic salmon farms, 1996-2001. *Emerg. Infect. Dis.* 9:455-460.
6. Murray AG, Smith RJ, Stagg RM. 2002. Shipping and the spread of Infectious Salmon Anemia in Scottish Aquaculture. *Emerg. Infect. Dis.* 8:1-5.
7. Penston MJ, McKibben, M, Hay DW, Gillibrand PA. 2004. Observations of sea lice larvae distributions in Loch Shieldaig, western Scotland. *Aquacul. Res.* 35:793-805.
8. Stagg RM. 2003. The eradication of an outbreak of clinical infectious salmon anemia from Scotland. In: *International Response to Infectious Salmon Anemia: Prevention, Control, and Eradication.* Proceedings of a symposium held in New Orleans, LA September 3-4 2002 (O Miller, RC Cipriano, eds.). US Dept. Agriculture, Animal and Plant Health Inspection Service; US Dept. Interior, US Geological Survey; US Dept. Commerce, National Marine Fisheries Service (Washington DC). Tech Bull. 1902:111-124.
9. Toranzo AE, Hetrick FM. 1982. Comparative stability of two salmonid viruses and poliviorus in fresh, estuarine and marine waters. *J. Fish Dis.* 5:223-23.

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Management of Infectious Salmon Anemia (ISA) in Norway

Jill Rolland

As the first nation to report the presence of infectious salmon anemia (ISA), Norway has the longest history of managing and regulating the disease. The elements of Norway's ISA management strategy are similar to those found in all countries combating the disease and include criteria for initiating disease investigations, criteria for declaring sites suspect or confirmed positive for ISA (and associated regulatory action), imposition of management zones, a compensation policy, and a vaccination policy. The importance of hydrography in the spread and maintenance of the disease in Norway is relatively less important than other risk factors, such as vectors and asymptomatic carriers. However, hydrography is one component used in delineating management zones. Currently Norway is in the process of reviewing its ISA management policy in light of current scientific knowledge of the disease.

Introduction

Infectious salmon anemia (ISA) is a significant disease of Atlantic salmon (*Salmo salar* L.) caused by an orthomyxovirus (ISAV). The disease was first identified in Norway in 1984[1] and subsequently has affected marine Atlantic salmon farming operations in other parts of Europe and North America.[2-7]

Despite ISA's emergence in the 1980s, it was not officially listed by the World Organization for Animal Health (OIE) as a reportable disease until 2003,[8] principally due to a lack of scientific and epizootic information. However, ISA became a European Union (EU) List 1 disease in 1991 under Council Directive 91/67/EEC.[9] Management of ISA is regulated within EU member nations by Directive 93/53/EEC[10] which requires eradication of a confirmed diseased population, surveillance, containment, and fallowing. Although Norway is not a member of the EU, trade agreements with the EU require that Norway follow the EU directives regarding ISA management. Management approaches to ISA in Norway, Canada, and the United States generally include the same components as the EU directive. However, within each country's regulations there is leeway to adjust for differences in local hydrography, ecosystems, and farming practices, all of which may affect transmission and rate of infection.

Regulatory Authorities for ISA Management

Multiple regulatory agencies are involved in aquatic animal diseases in Norway. These include the Norwegian Food Safety Authority and the Ministries of

Fisheries and Coastal Affairs, Agriculture, and Health. Veterinary authority lies within the Norwegian Food Safety Authority and this is the agency with primary responsibility for implementing Norway's ISA program. The Norwegian Food Safety Authority has published its ISA management protocols in a document entitled "Contingency Plan for Control of Infectious Salmon Anaemia (ISA) in Norway", dated August 13, 2004.[11] This paper outlines some of the major components of the Norwegian contingency plan, including initiation of disease investigations, regulatory action upon suspicion and confirmation of ISA, establishment of ISA management zones, indemnification for removal of infected stocks, and the ISA vaccination policy.

Initiation of Aquatic Animal Disease Investigation

Disease investigations are initiated when increased mortality, or suspicious or extended chronic disease problems, are observed in farmed salmon. For an official investigation to be initiated, specific criteria must be met. Increased mortality is one parameter that may initiate a disease investigation and is defined as a mortality rate of greater than 0.5‰ per cage per day for fish less than 0.5 kg in size, or as greater than 0.25‰ per cage per day for fish larger than 0.5 kg in size, or mortality rates close to these limits over a period of at least ten days. Other criteria prompting disease investigations include receipt of information regarding illegal transfer of fish from countries or areas known to be ISA positive, inconclusive laboratory results suggestive of possible ISAV infection, or suspicion that a farm has been otherwise exposed to the virus.

Regulatory Action in Suspect Cases of ISA

At least one of the following criteria must be met for a suspect ISA case to result in regulatory action: presence of postmortem findings consistent with ISA (by OIE standards), isolation and identification of ISA virus in cell culture from a single sample of any fish on the farm, reasonable evidence of the presence of ISAV in tissue or tissue material from two independent laboratory tests, a transfer of live salmonid fish when there are reasonable grounds to suspect that ISA was present at the time of transfer, or where investigations reveal other epidemiologic links to ISA-suspected farms or confirmed ISA-positive farms.

The regulatory action associated with being designated an ISA-suspect farm includes creating temporary management zones, restricting contact between farm sites, controlling well-boat traffic, and sharing information regarding the suspect site with stakeholders such as other farms, well-boat operators, processing plants, net-washing facilities, and feed companies. Within a temporary management zone there is increased supervision of activity and prohibition of movement into and out of the zone unless granted authority by the Norwegian Food Safety Authority. The actual extent of the zone may vary depending on the presumed risk of infection, as determined by the Norwegian Food Safety Authority.

To confirm a suspect ISA diagnosis, one of the following three sets of criteria must be met:

1. Observed clinical signs and postmortem findings consistent with ISA and the ISA virus detected by one or more of the following methods: isolation and identification of ISA virus in cell culture from at least one sample from any fish on the farm, detection of ISA virus by reverse-transcriptase-polymerase chain reaction (RT-PCR), or detection

of ISA virus in tissues or tissue preparations by specific antibodies against the ISA virus (e.g., immune fluorescence antibody technique (IFAT)).

2. Isolation and identification of ISA virus in cell culture in at least two samples from one or more fish at the farm tested on separate occasions.
3. Isolation and identification of ISA virus in cell culture from at least one sample from any fish on the farm with corroborating evidence of ISA virus in tissue preparations from any fish on the farm using either IFAT or RT-PCR.

Upon confirmation, official notice that depopulation is required must be provided to the farm by the Norwegian Food Safety Authority in a timely manner. The entire site must be depopulated within 80 working days following notification. Information regarding the confirmed diagnosis is shared with stakeholders. At this point an epidemiologic investigation will be initiated if it has previously not been undertaken. A control zone and a broader surveillance zone are created that surround the infected farm. Monthly inspections are undertaken by the Norwegian Food Safety Authority until all fish have been depopulated. Depopulation is followed by cleaning and disinfection of the site and an appropriate fallow period. Restocking is not allowed for a minimum of six months.

During the depopulation phase, all transportation by well-boat to slaughter facilities is controlled and may only be carried out by vessels specifically authorized to move infected fish. The transport route from the farm to the slaughter facility must be documented and approved. All ballast and transport water must be disinfected. Processing plant effluent and waste must be disinfected to ensure that viable virus is not released to the environment.

Salmon farm near Bergen
(photo by SMC Robinson)

Control Zone

The control zone is generally a circle with a 5-kilometer radius with the infected farm located at its center. The regulations stipulate that the zone will be established considering factors such as mortality, number of aquaculture facilities in the vicinity, boat traffic, topographical and hydrographical conditions, and other factors deemed important in the epidemiologic investigation.

In coastal areas, the control zone com-

prises an area with a radius of at least one tidal excursion, generally no less than 5 kilometers. Should ISA be detected in an inland area, the entire water catchment area of a watercourse is considered as the basis for the extent of the zone.

Surveillance Zone

The surveillance zone surrounds a control zone and should be established based on a survey of epidemiologic conditions including, but not limited to, mortality, topographical, and hydrographical limits. Surveillance zonation must also account for contact between farms within the control and surveillance zones. The extent of the surveillance zone will comprise all farms with overlapping tidal excursions to the ISA-positive farm, or an equivalent area based on hydrodynamic or epidemiologic data. The regulations stipulate that the surveillance zone should have a radius of 10 to 20 kilometers from ISA-positive farms. But in practice, this radius tends to be 10 km. In areas with multiple outbreaks, larger surveillance zones encompassing all outbreaks will be considered. Should ISA be detected in an inland area, a surveillance zone may be established around the control zone, if required, recognizing that geographical features often delineate zones in inland areas.

Lifting of Zone Restrictions

The control zone remains in place until all salmonid fish in the zone have been removed, cleaning and disinfection activities have been concluded, and a satisfactory fallow period has been completed. Zone restrictions are lifted after six months for ISA-positive farms, after three months for farms under suspicion of ISA (and suspicion is not ruled out prior to slaughter), and after two months for sites where ISA has not been confirmed. Surveillance zones remain in place for two years after the control zone has been lifted.

Vaccination

Vaccination is not an approved disease control method except under extenuating circumstances. Moreover, there are no ISA vaccines currently licensed in Norway. Should a vaccine become licensed in the future, under Norway's current regulations it would not be possible to repeal a surveillance zone until all vaccinated fish have been removed from the zone.

Compensation

Animals depopulated for disease control purposes are eligible for indemnification under the Norwegian Food Safety Authority's Act Relating to Food Safety and Plant and Animal Health § 22. The Act, however, does not include language for aquatic animals. Additionally, a second regulation, Regulation No. 509 dated 1991 (related to the prevention, control, and eradication of diseases in aquatic organisms) excludes provisions for indemnification.

Conclusions

In the United States and Canada, currents and tides are considered important epidemiologic factors in the transmission and maintenance of ISA. In Norway,

sites are spatially more distant from neighboring sites than in the US and Canada, and water movements are relatively less important in disease transmission than factors such as vectors and asymptomatic carriers.

As with all other nations challenged with this disease, Norway's management and regulation is an evolving process and as new scientific information becomes available there has to be the flexibility to incorporate the knowledge into management strategies. The current contingency plan for ISA in Norway was last revised in August 2004. The Norwegian Food Safety Authority is in the process of reviewing the latest scientific information regarding the epidemiology of the disease and considering how this information might be incorporated into an updated management strategy.

Acknowledgments

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References

1. Thorud K, Djupvik HO. 1988. Infectious salmon anaemia in Atlantic salmon *(Salmo salar* L.). *Bull. Eur. Assoc. Fish Pathol.* 8:109–111.
2. Rodger HD, Turnbull T, Muir F, Millar S, Richards RH. 1998. Infectious salmon anaemia (ISA) in the United Kingdom. *Bull. Eur. Assoc. Fish Pathol.* 18:115–116.
3. Stagg R, Bruno D, Cunningham C, Hastings T, Bricknell I. 1999. Focus on infectious salmon anaemia: epizootiology and pathology. *State Vet. J.* 9:1–5.
4. Mullins J, Groman D, Wadowska D. 1998. Infectious salmon anaemia in salt water Atlantic salmon *(Salmo salar* L.) in New Brunswick, Canada. *Bull. Eur. Assoc. Fish Pathol.* 18: 110–114.
5. Bouchard D, Keleher W, Opitz HM, Blake S, Edwards KC, Nicholson BL. 1999. Isolation of infectious salmon anemia virus (ISAV) from Atlantic salmon in New Brunswick, Canada. *Dis. Aquat. Org.* 35:131–137.
6. Lovely JE, Dannevig BH, Falk K, Hutchin L, MacKinnon AM, Melville KJ, Rimstad E, Griffiths SG. 1999. First identification of infectious salmon anaemia virus in North America with haemorrhagic kidney syndrome. *Dis. Aquat. Org.* 35:145–148.
7. Bouchard DA, Brockway K, Giray C, Keleher W, Merrill PL. 2001. First report of infectious salmon anaemia (ISA) in the United States. *Bull. Eur. Assoc. Fish Pathol.* 21:86–88.
8. Office International des Epizooties. 2003. *OIE Aquatic Animal Health Code.* 6th ed. Office International des Epizooties, Paris. 165 p.
9. Council Directive 91/67/EEC of 28 January 1991 concerning the animal health conditions governing the placing on the market of aquaculture animals and products. European Economic Community. 18 p.
10. Council Directive 93/53/EEC of June 1993 introducing minimum Community measures for the control of certain fish diseases. European Economic Community. 11 p.
11. Norwegian Food Safety Authority. 2004. *Contingency Plan for Control of Infectious Salmon Anaemia (ISA) in Norway.* Norwegian Food Safety Authority, Oslo. 37 p.

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Freshwater Finfish Disease—Hunting Pathogens In Ontario

Roselynn M. W. Stevenson

Disease problems encountered in freshwater fish culture in Ontario are flavobacterial skin and gill diseases, and sporadic furunculosis. Routine health screening of hatchery fish for Great Lakes Fishery Commission reporting purposes has found minimal levels of disease agents, raising questions about the extent of testing and the methods for testing that are required to demonstrate a pathogen is *not* present. The small sizes and sheer numbers of microorganisms ensures a world-wide distribution, so the relevant question about disease transmission may concern the roles of aquaculture and susceptible wild fish populations in selecting, amplifying, and spreading pathogenic microbes.

Introduction

In order to consider the best options for managing a disease outbreak in aquaculture, and evaluating the risk of the pathogen spreading in water movements, we need to know about the prior distribution of the pathogen in that aquatic environment. When a disease agent is considered to be endemic in an area, then the major questions pertain to disease outbreaks and the spread of a virulent pathogen strain from a point source, where infections have amplified the levels of the organism. Practical examples of this question in freshwater aquaculture in Ontario are rare, in part because cage-site aquaculture is not yet as intensive as on the Atlantic and Pacific coasts. But what are some of the issues of pathogen-spread in freshwater systems?

My perspective is that of a microbiologist, one using a range of culture and molecular techniques to detect pathogens in fish and in aquatic ecosystems. As such, I want to consider some of the practical questions of detecting disease agents that move around in freshwater ecosystems. More specifically, I want to consider the problems encountered in efforts to demonstrate that specific pathogens are *not* present in fish populations.

Freshwater Aquaculture in Ontario

Ontario aquaculture is predominantly rainbow trout culture, with an estimated 4,200 tonnes (9.25 million pounds) raised in 2003.[1] Freshwater cage sites in the Georgian Bay region, such as those in the North Channel by Manitoulin Island, now account for more than 75% of this production, while land-based operations (ponds and raceways) account for some 1,000 tonnes production. Currently, Ontario is examining and developing guidelines for selecting new cage culture sites and operating them in a sustainable manner. Models of water movement in the Great Lakes have generally been used to examine the spread of chemical pollutants, from industrial or other point sources, though recent research has attempted

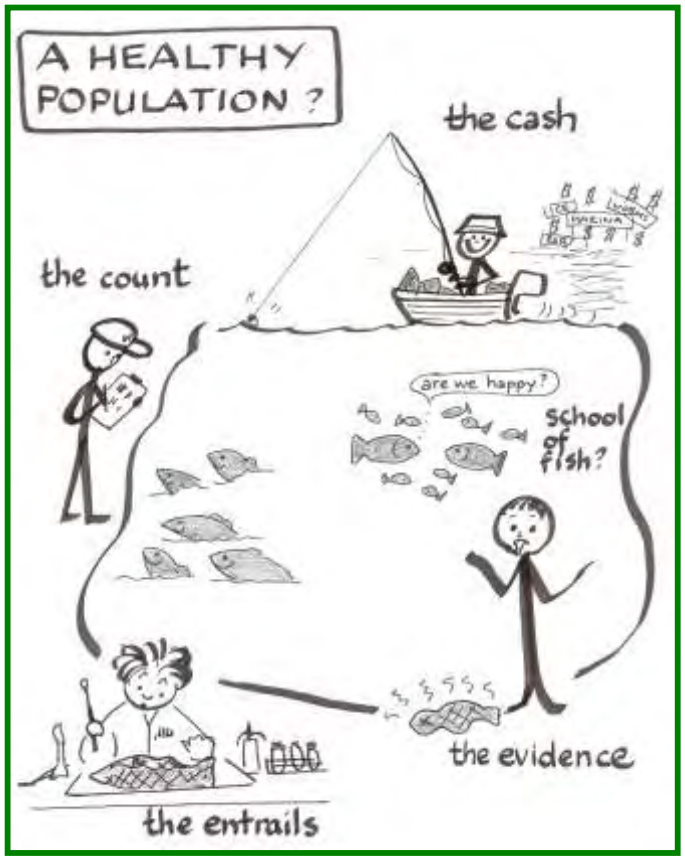
to assess the footprint of aquaculture cage sites and the impact of cage sediments on the local aquatic environment.

In 1997, Ontario legislation expanded the species that could be cultured to include some 40 species. However, production of tilapia and Arctic charr is relatively small, and brook trout, bass, and baitfish production is mainly for stocking and recreational fishing uses. Stocking for remediation and recreational programs accounts for another component of freshwater fish culture in Ontario. The Ministry of Natural Resources (OMNR) uses 10 hatcheries to raise lake trout, brook trout, as well as rainbow trout, and coho and chinook salmon, for stocking in the Great Lakes watershed. Community and angler groups also operate local hatcheries for re-stocking programs.

Fish Health in Ontario

Disease diagnosis for private sector aquaculture is done by the Ontario Veterinary College, Fish Pathology Laboratory at the University of Guelph, while fish health certification for Ontario producers involved in shipping fish inter-provincially is conducted through the Freshwater Institute (DFO) in Winnipeg. Health monitoring and infectious disease detection for the Ontario Ministry of Natural Resources (OMNR) hatcheries and for wild egg collections are carried out at the Fish Health Laboratory, College of Biological Science, University of Guelph, by a contractual agreement that began more than 30 years ago. Reports of disease or disease agents are used by OMNR for immediate management decisions, and are reported to the Fish Health Committee of the Great Lakes Fishery Commission (GLFC), in compliance with the GLFC fish disease control policy and model program.[2] This program is intended to reduce the risk of introducing or transferring serious disease agents into or within the Great Lakes basin. The Great Lakes Fish Health Committee (GLFHC) includes representatives of eight states, the Province of Ontario (OMNR), US Fish and Wildlife Service, Fisheries and Oceans Canada, and tribal agencies, which are concerned with the Great Lakes watershed.

Figure 1
Measuring the health of a freshwater fish population is a multifactorial exercise, involving economic returns, biological assessments, and public perceptions, as well as laboratory analysis of pathogens present in fish.



A healthy fish population?

The approach taken by the GLFHC to protect, assess and improve fish health is multidisciplinary, considering not only diseases and disease agents but also information reflecting general ecosystem health. Thus, potential health problems may be reflected in population audits, economic well-being of sports fisheries, reports of morbidity and mortality by the public, as well as monitoring and disease diagnosis activities (Fig. 1).

The list of diseases of concern that have been encountered in private aquaculture and OMNR operations may suggest Ontario microbiologists and fish pathologists lead a dull

life. Predominant as actual disease problems are the skin and gill diseases caused by *Flavobacterium psychrophilum* and *Flavobacterium branchiophilum*, which can be triggered by environmental and management conditions. While isolates of *Aeromonas salmonicida* ("typical form") are found on a recurring basis in some fish populations, cases of furunculosis disease are not common, perhaps because the predominant fish cultured is rainbow trout, a more resistant species. In the past, commercial aquaculture and the OMNR system have encountered problems with *Renibacterium salmoninarum* (bacterial kidney disease), *Yersinia ruckeri* (enteric redmouth disease) and infectious pancreatic necrosis virus (IPNV); however, these are isolated and rare occurrences. Viral isolates from hatchery fish or wild fish sampled during egg collections are rare, to the point that two isolations of an innocuous aquareovirus, in 1997 and 1999, were welcomed as a demonstration that laboratory tissue culture tests were effective at detecting naturally-occurring viruses. Occasional submissions of wild fish with abnormalities are received, including pike lymphomas, walleye with spores of *Heterosporis* in muscle tissue, and individual whitefish with furuncules.[3]

Detecting what's not there

Bruneau[4] and Thorburn[5] analyzed data collected by the FHL and the federal fish health certification program, and concluded that the apparent prevalence in fish populations of *A. salmonicida* and IPNV was too low to fit the statistical assumptions of using 60-fish lots for sampling. Thus, with the agreement of the GLFHC, OMNR hatcheries have moved to an annual testing of 252 fish from each hatchery as a means of increasing the efficiency of routine sampling. Good et al.[6] continued this analysis of data for *A. salmonicida* and *Y. ruckeri*, in order to identify factors, such as age and species, which would allow routine screening to be focused on high-risk fish lots. In hatcheries where these two pathogens have been detected, the first detection in a sampling year (April to March) was most often through submissions from fish lots with signs of disease, rather than from the routine screening program.[7] As a result, these authors questioned the value of continued routine testing of hatchery stocks as a means of monitoring for microbial fish diseases.

This view is of interest in considering the testing that would be required to establish that a geographic zone or watershed could be considered free of a particular disease or pathogen. The kind of evidence that might be presented would be: the

Outdoor circular tanks at the Alma Aquaculture Research Station, University of Guelph (David Bevan photo).

absence of clinical disease signs in susceptible fish species; the absence of positive test results for the specific agent in sick fish submissions; and the absence of positive test results for the agent in routine screening of apparently healthy fish.

This is essentially the evidence that is presented to indicate that whirling disease and *Myxobolus cerebralis* are absent from Ontario. The disease was first recognized in Pennsylvania in 1958 and relatively recently New York State hatcheries had disease problems and the infected fish were stocked into Lake Ontario. The Fish Health Laboratory at Guelph has tested fish from the Ontario Ministry of Natural Resources hatchery system, and wild fish used for egg collections, since before 1981. *M. cerebralis* has never been detected and there have been no reports from other sources that would suggest the disease occurs in aquaculture operations or wild populations. From 1981 to 1986, all fish in every OMNR hatchery lot were tested annually for *M. cerebralis*. With the recognition of the role of the oligochaete, *Tubifex tubifex,* in the life cycle, testing was reduced, focusing on wild fish populations from Lake Ontario during egg collections. No spores of *M. cerebralis* have been detected by microscopic examination of cartilage of fish heads, in samples prepared by the plankton centrifuge method.[8] However, the impact of consistently negative test outcomes don't so much "prove the negative" as lead to questions about whether enough fish were tested, and from the right species and places, and whether there might be a better test method.

What does a positive test mean?

But think about what happens when there *is* a positive test. First, the laboratory would be asked—hopefully—if perhaps they had made a mistake. Then the discussion would consider how significant one positive fish was, or maybe even three. Next, the test method would be considered—what it detects, at what level, with how much specificity—before talk moved on to "virulent *versus* avirulent strains".

This sequence is illustrated well by isolations of viral haemorrhagic septicemia virus (VHSV) from mortalities in freshwater drum (*Aplodinotus grunniens)* in the Bay of Quinte (eastern Lake Ontario) which occurred in April 2005. The FHL did not detect cytopathic effects on RTG-2 and CHSE cell lines, but histopath- ology suggested a viral involvement.[9] By September 2005, virus had been cultured on FHM cells at the Atlantic Veterinary Laboratory and the OIE reference laboratory had confirmed it was VHSV, "North American type". Olivier[10] describes a similar isolation of VHSV from a wild fish kill of mummichogs and three-spine sticklebacks in New Brunswick in

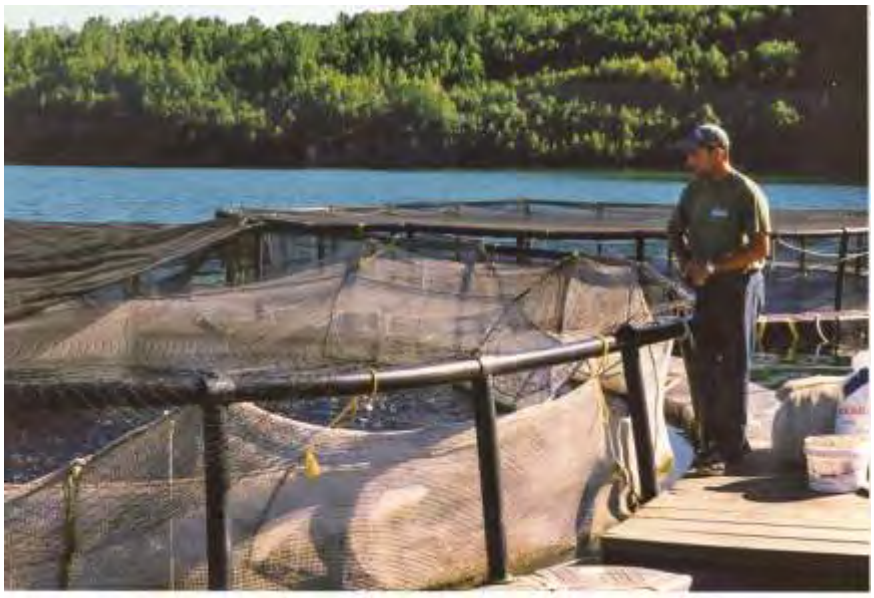
Hand feeding rainbow trout at North Wind Fisheries (David Bevan photo).

May 2000. The virus, described as "the non-pathogenic North American strain of VHS", was isolated on the cyprinid cell line, EPC, rather than on two salmonid cell lines.

So, do monitoring programs miss pathogens because of the choice of detection tests—such as the number of cell-lines used for virus screening? For both VHSV and *M. cerebralis*, we could embark on an extensive survey of fish and other environmental samples. We could use current knowledge of the biology of these agents to select the most likely fish species or geographic locations. We could test samples with molecular assays, such as PCR amplification, as well as conventional "Blue Book" methods. But notice that we still would be looking for presence of the disease agents—which *may* be virulent strains capable of inducing disease *if* they encountered susceptible fish species under conditions conducive to infection and pathogenesis. Just how many cell lines, tissue samples, enrichments, blind-passes, culture media, and molecular test methods are needed to screen fish populations? And how many different potential pathogens should the testing look for in a geographic area or watershed? Ultimately, the question is not only how much evidence is needed to show something is *not* present, but how many tests are really feasible and useful.

Dealing with a disease situation can make pathogen detection easier, not only because the disease signs may give clues to the agent involved, but also because the fish tissues carry much higher loads of the microbe. This is a practical consideration in routine health screening of fish, as the levels of a pathogen present in tissue of carrier fish may be well below the level needed to register as a positive test. This is an issue in discussing polymerase chain reaction (PCR) based tests, which have the reputation of detecting "one cell, dead or alive". However, the original tissue or sample material needs to have a substantial concentration of the pathogen to ensure a copy of the target gene is in the small sample volume being tested. In 1997, we used a PCR test to screen populations of freshwater coho and chinook salmon with a prior history of *R. salmoninarum*, the bacterial kidney disease agent.[11] Of 395 fish tested, 15.4% gave a positive PCR reaction for the gene encoding the major surface antigen of the bacterium, while the standard indirect fluorescent antibody staining reaction (IFAT) scored 2.5% of the fish positive. Thus, for any pathogen, the reported prevalence of positive fish in a population can be dramatically affected by the specific test applied. The reassuring aspect of this study was that, even using the PCR assay, most of the fish tested would be considered negative for *Renibacterium*.

Hand feeding rainbow trout at Moose Mountain Fisheries (David Bevan photo).



Distribution and Dispersal of Microbial Pathogens of Fish

Previously, we might have used VHSV as an example of a pathogen "exotic" to Ontario, to consider the fish health management decisions that

Prime sport-fishing locations for yellow perch are at the Georgian Bay cage-sites for rainbow trout, ensuring an aquaculture-wild fish interface (Amanda Mielke photos).

would need to be made in the case of an isolation or disease outbreak. This kind of discussion tends to focus on the likelihood of a point source introduction of a pathogen (or disease) from an outside source—involving ballast water, illegal imports, or baitfish movement.[12] These situations certainly arise, yet the isolation of VHS from freshwater drum in Ontario is consistent with Olivier's comment about the "widely held belief that most fish diseases originate in wild populations".[10] Occasional disease outbreaks observed in wild fish populations may be triggered by local environmental and physiological stresses, and an anomaly in a natural host-pathogen balance.

A "microbial world view" would argue that we should not be surprised to find pathogens to be widespread in aquatic environments. Fenchel[13] describes the distribution of microbes as cosmopolitan, because population sizes are enormous and the probability of unrestricted dispersal of small particles is high. Recent studies of global dispersal and distribution of bacteria[14]and microbial eukaryotes[15] suggest that the question is not whether a particular microbial pathogen is present in a location, but rather what it is doing there, and whether it is surviving and growing. A relevant question is whether aquaculture might act as a

magnet or magnifier for disease agents that exist either in the aquatic ecosystem or among wild fish. Increased water-contact between wild and cultured fish is apparent at cage sites in Georgian Bay, where the best yellow perch fishing is just outside the rainbow trout net-pens. Recognizing the ubiquitous presence of microbes, and the potential for local amplification of pathogen numbers by the presence of susceptible hosts, may be a good point to start thinking about how issues of transmission of pathogens and diseases in aquaculture might best be managed.

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References

1. Moccia RD, Bevan DJ. 2004. *Aquastats 2003: Ontario Aquacultural Production in 2003.* Available online at http://www.aps.uoguelph.ca/~aquacentre/aec/publications.
2. Meyer FP, Warren JW, Carey TG. 1983**.** *A Guide to Integrated Fish Health Management in the Great Lakes Basin.* Great Lakes Fishery Commission, Ann Arbor, MI, Spec. Pub. 83-2: 272 p.
3. Stephen Lord, Manager, Fish Health Laboratory, personal communication.
4. Bruneau NN, Thorburn MA, Wilson J, Meek A. 2001. Hatchery-level predictive values for infectious pancreatic necrosis virus and *Aeromonas salmonicida* in Ontario, Canada. *Prev. Vet. Med.* 48:129-141.
5. Thorburn MA. 1996. Apparent prevalence of fish pathogens in asymptomatic salmonid populations and its effect on misclassifying population infection status. *J. Aquat. Anim. Health* 8: 271-277.
6. Good CM, Thorburn MA, Stevenson RMW. 2001. Host factors associated with the detection of *Aeromonas salmonicida* and *Yersinia ruckeri* in Ontario, Canada government fish hatcheries. *Prev. Vet. Med.* 49:165-173.
7. Good CM, Thorburn MA, McEwen SA. 2001. Simulation modelling to evaluate the impact of eliminating routine bacterial screening from the Ontario provincial monitoring program. *Prev. Vet. Med.* 50:101-108.
8. MacConnell E. 2002. Whirling disease of salmonids. In: American Fisheries Society–Fish Health Section (AFS-RFS) blue book: *Suggested Procedures for the Detection and Identification of Certain Finfish and Shellfish Pathogens*, 2004 edition. AFS-FHS, Bethesda, Maryland.
9. Dr. John Lumsden, personal communication.
10. Olivier G. 2002. Disease interactions between wild and cultured fish—Perspectives from the American Northeast (Atlantic Provinces). *Bull. Eur. Ass. Fish Pathol.* 22:103-109.
11. Patric Senson, Fish Health Laboratory data.
12. Goodwin AE, Peterson JE, Meters TR, Money DJ. 2004. Transmission of exotic fish viruses: The relative risks of wild and cultured bait. *Fisheries* (AFS) 29(5):19-23.
13. Fenchel T. 2003. Biogeography for bacteria. *Science* 301:925-926.
14. Horner-Devine MC, Carney K, Bohnannan BJM. 2003. An ecological perspective of bacterial biodiversity (Review). *Proc. R. Soc. Lond.* B. 271:113-122.
15. Finlay BJ. 2002. Global dispersal of free-living microbial eukaryote species. *Science* 296: 1061-1063

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Summary of the Plenary Discussion on the Management Implications of the Role of Water Movement in Aquatic Animal Health

Moderators: Jamey Smith and Gilles Olivier

Rapporteur: Blythe Chang

A plenary discussion on the management implications of the role of water movement in aquatic animal health was held following the individual workshop presentations. The central question, and the major reason for holding this workshop, was: Does water movement (oceanography) play a role in aquatic animal health? The answer seems to be that it can, but its importance depends on:

- The distance between adjacent farms;
- The water circulation patterns in the area; and
- The nature of the disease agent released (quantity, quality, and timing), the ecology of the disease agent in the receiving environment, the nature of the disease agent received by a farm, and the susceptibility of the farmed animals to infection.

Gilles Olivier leading the discussion

Water movement can also be important for other issues that have a bearing on aquatic animal health, such as aquaculture site selection, environmental impacts of aquaculture operations, long-term planning of aquaculture in an area, and coastal zone management.

The role of water movement has already, to varying degrees, been included in aquaculture health management strategies. For example, in the adjacent waters of southwestern New Brunswick (SWNB) and eastern Maine (Bay of Fundy) there has been a process to establish and manage Bay Management Areas (BMAs) based in part on water movement. Recent efforts are employing water movement information with increased resolution and confidence, and may lead to a reduction in the number of BMAs for salmon farms. The intent is to provide a framework for fish health management, while also allowing farmers flexibility to sell their product during the best market conditions. The industry-authored Atlantic Canada Salmon Farming Sustainability Plan (New Brunswick perspective)[1] notes this as being key to future sustainability. Information on water movement has been an important consideration within this process. As noted in the presentation by Steve Ellis and Lori Gustafson, water movement does help explain the prevalence of ISA in this area. Knowledge of water movement will continue to play an important role in decisions regarding salmon farm sites in SWNB as part of the strategy to manage ISA.

"... water movement information is already being used in some areas for determining where farm sites should be located ..."

In British Columbia, oceanographic considerations are included in the farm siting process, especially in relation to predicted benthic impacts of farms. Direct application of water movement to fish health management will not likely occur until clear links are shown between water movement and disease spread among farms. In Norway, water movement is not a major consideration in farmed fish health management strategies. This is partly because Norwegian salmon farms are quite widely separated and partly because the currently used 5-km radius control zones seem to work adequately for the purposes of managing current diseases. While the potential role of water movement in disease spread in Norway is recognized, it is felt that other factors, such as wild fish, are more important.

In Scotland, management areas (MAs) for salmon farms are defined based on water movement (estimated tidal excursions). The boundaries between MAs should be placed between farms that have no overlaps of tidal excursions. Strict application of this guideline, however, can result in MAs that are too large to be managed as an entity. Water movement information can be used to help define smaller MAs, placing boundaries where there are "natural breaks" in the patterns of water circulation.

Some participants felt that in order to promote the use of water movement as a management tool in aquatic animal health, we need to clearly demonstrate how water movement affects the spread of disease (i.e. we need testable models that can show causal links). Other participants felt that we need to be proactive and that we can't always wait until we clearly demonstrate such links, especially when it has been shown that water movement does play a role in the spread of some diseases in some areas.

An important point to remember is that water movement is just one factor in the

spread of aquatic diseases. Other factors such as wild fish, transfers of cultured fish, and boat traffic, may be important and are likely to be more important in many situations. It was also noted that the issue of aquaculture health management is a subset of the larger issue of integrated coastal zone management (ICZM), and that water movement should have a role in ICZM planning.

Water movement information can be incorporated at different scales. At the site level, water movement can assist in determining the best arrangement of culture units (cages, longlines, etc.) and in the design of site-specific animal health management strategies (such as determining where depopulation of individual culture units is appropriate, versus site depopulation). As mentioned above, water movement information is already being used in some areas for determining where farm sites should be located; one of the considerations is the potential for disease spread among adjacent farms. On a larger scale, water movement information is being used to determine the boundaries of management areas and fish health control and surveillance zones.

The OIE (World Organization of Animal Health) concepts of zoning and compartmentalisation were discussed. An example given of the application of these concepts to aquaculture health management was the imposition of common fish health management practices, including controls on vessel traffic and biosecurity, to four adjacent BMAs within the SWNB salmon farming industry (Letete Passage, Back Bay, Lime Kiln Bay and Bliss Harbour). The justification for applying common fish health management practices in this larger area was mostly based on information on the amount of water exchange among farms in this area. It was recognized, however, that zones or compartments in the marine environment cannot be completely isolated since some potential disease vectors, such as wild fish, fishing vessels, and recreational boating, cannot be controlled. There is a need to quantify the various risks, to ensure that the most important ones are identified and addressed.

A fusion of the disciplines of oceanography and aquatic animal health is beginning to occur. In this workshop, we have seen some examples where standard oceanographic methods are being applied to aquatic animal health issues. A major shortcoming is the lack of information on the behavior of aquatic diseases, especially in field conditions. For example, we need more information on the shedding rates of disease agents from diseased or carrier animals, the survival rates of these agents in the water, the movement of the agents in the water, and the amount of a disease agent required to cause infection.

A general recommendation resulting from this plenary discussion was that water movement can and must be included in aquatic animal health management planning. However, the plenary group was unable to develop specific recommendations on how to implement this general recommendation. This was largely due to information gaps, which were discussed at the concluding plenary session of this workshop (see the next paper in this volume).

Reference

1. NBSGA (New Brunswick Salmon Growers' Association). 2005. Atlantic Canada Salmon Farming Sustainability Plan: New Brunswick. NBSGA, St. George, NB.

Summary of Plenary Discussion on Knowledge Gaps and Research Needs

Moderators: Nancy House and Nathalie Bruneau

Rapporteur: Nancy House

Background

The role of water movement and oceanography is gaining worldwide recognition as an important factor in the management of aquatic animal health. As other factors are considered in disease control, many aquatic disease specialists are questioning the effect water movement may play in disease recurrence. Our knowledge of how water movement affects the distribution and dissemination of disease is inadequate and for many diseases it is not known whether water movement influences the onset or severity of clinical disease. A further complicating factor in understanding the effect of water movement on disease is that the distribution and transmission of diseases can vary significantly between host species and in different aquatic ecosystems.

Models incorporating oceanographic data and specific disease parameters such as infectivity in water, vectors and intermediate hosts, viability and survival in water, decay rate, etc., can be used as management tools. However, for models to be accurate and valid for management decision-making, a specific model is needed for each disease.

Water movement can also be an important factor when delineating the boundaries of disease zones. Disease does not recognise political boundaries, and the creation of zones should incorporate geographical boundaries and water movements in marine waters and large inland water bodies (such as the Great Lakes). In addition, the careful location of cages and pens with respect to water movement may minimise the impact of upstream influences and enhance farm productivity.

As part of the workshop's plenary discussions, knowledge gaps and research needs were discussed in an effort to define areas where knowledge can be improved by incorporating water movements or oceanography factors into management decisions on aquatic animal health. This paper summarizes the brainstorming sessions of four plenary subgroups.

Knowledge Gaps

The key knowledge gaps identified in the plenary discussions were:

1. Role of water movement in exposing aquatic animals to pathogens

There is evidence that disease outbreaks occurring downstream may be related to disease outbreaks upstream. When outbreaks continue to occur re-

gardless of treatment of aquatic animals, disinfection of equipment, restrictions on movements of boats, etc. then it is likely that the pathogen is being transferred directly via water currents or indirectly by an intermediate or carrier host.

2. Water movement in areas where aquaculture occurs

Little is known about surface currents, sub-surface currents, or daily and seasonal exchanges of water in and around aquaculture areas.

3. Oceanographic data relevant to aquatic animal diseases and aquaculture sites

It is important to identify critical oceanographic factors, such as salinity, water temperature, water currents, tides, etc. that influence the transmission of specific diseases, and options for disease management for aquaculture sites and protection of wild resources.

4. Mechanisms or pathways involved in the epidemiology of the pathogen or disease

Mechanisms or pathways of transmission are known for some diseases. However, for the majority of serious infectious pathogens the vector, intermediate, or reservoir hosts are unknown, as are the life history stages that may be dormant outside their aquatic animal hosts.

Research Needs

1. Water movement

- Define the extent of the influence of water movement on dissemination and transmission of specific disease agents.
- Determine the hydrographic factors that most influence the spread or control of specific diseases.
- Improve the accuracy and inclusion of aquatic environmental data to increase confidence in disease control and epidemiological models.
- Refine disease control or epidemiological models by generating background data on a regular basis. This will allow models to be ground truthed (validated) and will refine their use for applicability across a broad range of hydrographic variables (including environmental extremes), especially where aquaculture occurs.

2. Biological components

- Although the biotic influences on some diseases are known, more research is required on pathogen hosts or vectors (identification of reservoir species) for many other diseases.
- Little is known about variables such as pathogen decay rate, host exposure time, behaviour in water, infectivity, viability, and survival (e.g., temperature, pH, salinity) for many diseases.

- Analysis of the influence of all the relevant biological and oceanographic factors is needed to determine the importance of each one in developing control or management programs for open-water disease outbreaks.
- More investigation is needed on the importance of 'pulses' in pathogen exposure versus long-term or low-density exposure.
- Seasonal dynamics associated with the apparent disappearance of pathogens and disease (below the sensitivity of screening technologies versus actual disappearance) need to be better understood. This requires more sensitive detection tools in parallel with analysis of seasonal environmental influences on pathogen infectivity and/or proliferation.
- Behaviour of organisms exposed to pathogens and possible changes in behaviour as a result of infection need to be evaluated against pathogen infection.
- Serological and genetic typing of pathogens (strain typing) is required.

3. Accurate model of disease transmission

A research project to develop an accurate model of disease transmission should include the following components:

- Select a pathogen for likeliness of success,
- Identify all hydrographic and biological factors,
- Rank factors in order of importance,
- Validate the model,
- Revise and increase accuracy of the model as knowledge improves.

Conclusion

Increasing interest in the relationship between water movement and aquatic animal disease has already led to preliminary collaborations between the aquaculture industry, aquatic disease specialists, and oceanographers. These collaborations have influenced disease management decisions that would previously have been based solely on host-pathogen interaction.

Further research on the relationship between aquatic diseases and water movement is recommended to allow the development of tools that can enhance current passive (circumvention) or chemo-therapeutant control methods for many diseases of concern. Disease transmission models that incorporate water movement could be used for disease prevention at individual farms or for farms within the same hydrographic area, as well as for the siting of aquaculture facilities. In addition, this knowledge is essential for accurate delineation of zones that are positive or negative for specific diseases for the national and trans-boundary aquatic animal health programs aimed at management of disease in both cultured and wild aquatic populations.