BEFORE THE MARLBOROUGH SALMON FARM RELOCATION ADVISORY PANEL AT BLENHEIM

UNDER	the Resource Management Act 1991
IN THE MATTER	of Regulations under ss 360A and 360B of the Act
BETWEEN	**THE MINISTRY FOR PRIMARY INDUSTRIES**
	Applicant
AND	**MARLBOROUGH DISTRICT COUNCIL**

STATEMENT OF EVIDENCE OF DR. COLIN JOHN JOHNSTON
Dated this 22nd day of May 2017

GASCOIGNE WICKS
LAWYERS
BLENHEIM

Solicitor: Quentin A M Davies
Amanda L Hills

79 High Street
PO Box 2
BLENHEIM 7240
Tel: 03 578 4229
Fax: 03 578 4080

INTRODUCTION

1 My name is Colin Johnston. I am a veterinarian with 24 years' professional experience, of those I have worked with aquatic animals for 19 years. I hold full practising registration with the New Zealand Veterinary Council.

2 I qualified as a veterinarian (Bachelor of Veterinary Medicine and Surgery, BVMS) with Honours from Glasgow University in July 1993. I was awarded Membership of the Australian College of Veterinary Scientists (MACVSc) in Aquatic Medicine in 2003, by examination. I have acted as Membership Examiner for the Australian College of Veterinary Scientists in Aquatic Medicine in 2006. I have acted as Head Subject Examiner for the Australian College of Veterinary Scientists in Aquatic Medicine in 2012.

3 I am a Director and Principal of Brightwater Consulting Ltd. providing aquatic diagnostics, biosecurity advice and risk analysis to private aquaculture companies, research organisations and government departments in New Zealand and overseas. I am providing this expert evidence in this capacity.

4 I am also employed part-time as the Technical Director of Aquaculture New Zealand, although I am not providing this expert evidence in this capacity.

5 Brightwater Consulting Ltd. is also contracted to the Executive Officer position with Marlborough Shellfish Quality Programme Incorporated (MSQP) which provides all testing requirements to permit safe harvest of bivalve molluscan shellfish (from a human health perspective) across the top of the South Island. I am not presenting this expert evidence in this capacity either.

6 My previous employment in the area of aquatic animal health and biosecurity has included:

(a) Principal Adviser, Aquatic Animal Diseases for the Ministry for Primary Industries, New Zealand with responsibility to provide advice across all aspects of policy, surveillance, risk, diagnostics and trade rules;

(b) General Manager, Aquatic Resources, Aquaculture Directorate of the South Australian Government. One of the responsibilities I had was the management of the State aquatic animal health programme which included policy and legislative development.

(c) Veterinary Services Manager for Marine Harvest Scotland Ltd., where I had responsibility for the health and welfare of more than 40 million fish across more than 50 farm sites.

7 In aquatic animal health, disease and biosecurity matters, I have professionally represented:

(a) The State Government of South Australia at the National Aquatic Animal Health Technical Working Group (NAAH-TWG), the Aquatic Animal Health Committee (AAHC) and the Aquatic Consultative Committee on Emergency Animal Disease (AqCCEAD), all committees existing or having existed in the Federal Primary Industries Ministerial Committee structure of Australia.

(b) The Government of New Zealand on the Aquatic Animal Health Committee (AAHC) and the Aquatic Working Group of the Animal Health Quadrilaterals (covering Australia, Canada, New Zealand and United States of America).

(c) New Zealand on the mollusc diseases working group of the joint European Union, Canada, Australia and New Zealand Knowledge Based Bio Economy (KBBE) forum.

8 I lead, and have led, groups advising changes in aquatic animal zoosanitary trade rules for the Aquatic Animal Health Standards Commission of the World Organisation for Animal Health (OIE) in the following:

(a) Chair of the ad hoc working group on the Declaration of Freedom from Disease

(b) Chair of the ad hoc working group on the Safety of Commodities from Aquatic Animals

(c) Chair of the ad hoc working group on the Safe Treatment of Aquatic Animal Wastes and By-products; and

(d) Chair of the ad hoc working group on Pathogen Differentiation in Aquatic Animals.

9 I was appointed an Honorary Research Fellow at the University of Tasmania School of Aquaculture (2004 to 2007), and a visiting lecturer for year 2 and year 3 BSc(Aquaculture) students at Flinders University of South Australia (2003-2006). I have been author and co-author of 14 articles and peer reviewed papers on aquatic animal health, including matters related to risk analysis. I have acted as peer reviewer for numerous papers for the journals: Diseases of Aquatic Organisms, Transboundary and Emerging Diseases, Aquaculture and Journal of Fish Diseases.

10 I confirm that I have read and am familiar with section 7 of the Environment Court Practice Note 2014 which relates to expert witnesses. I agree to be bound by that Code of Conduct and confirm that I have not omitted to consider material facts known to me that might alter or detract from the opinions that I express in the following evidence. The evidence I give is within my expertise, save where the context indicates otherwise.

ENGAGEMENT & SCOPE OF EVIDENCE

11 The Advisory Panel, in their 4th minute, produced on the 12th May 2017, indicated that they had received information on four particular issues where it had subsequently formed the view that opportunity must be provided to the proponent of the proposal to provide a response.

12 I have been engaged by the New Zealand King Salmon Company Limited to provide expert opinion on the following, which covers issues within my area of expertise:

> "Assertions have been made that there have been major continued mortalities at both the Waihinau and Ruakaka farms, in particular over the years since 2014. It has been asserted that either such continued high mortality rates may not have been reported to the authorities or advised to Dr Diggles when he was preparing his report entitled Updated disease risk assessment report – relocation of salmon farms in Marlborough Sounds (7 September, 2016, Diggles). Alternatively, it has been asserted that that report did not properly record and identify the actual causes of continued high rates of mortalities, or address their significance in terms of sustainability of salmon farming in Pelorus Sound, or in terms of risk to other fauna."

BACKGROUND

13 I have read the report of Dr Diggles dated 7 September 2016, being an "Updated disease risk assessment report – relocation of salmon farms in Marlborough Sounds, New Zealand" based on the risk assessment presented to the Environmental Protection Authority (EPA) Board of Inquiry (BoI) on new salmon farms in the Marlborough Sounds. I note the inclusion of information on the New Zealand Rickettsia-like organism (NZ-RLO) in the updated report.

14 I have also read a selection of transcripts, presentations and submissions that refer to concerns over fish health and note that in my expert opinion many of

their conclusions are misguided. Although their desire for better fish health outcomes is one we all espouse.

15 I have, in preparing this evidence, spoken with the Ministry for Primary Industries (MPI) and was provided with written information indicating that the MPI had informed Dr Diggles of elevated mortalities, the discovery of the NZ-RLO and its presence on more than one farm site. I also had a professional conversation with Dr Diggles regarding elevated mortalities, the range of causes on the farms and the discovery of the NZ-RLO and *Tenacibaculum maritimum* (another bacterium) during his development of the updated risk assessment, and subsequently in preparing this evidence. As a result, I am satisfied that Dr Diggles was fully aware of elevated mortalities, the various causes of mortality and the presence of NZ-RLO on more than one farm site.

16 The mortalities over summer on low-flow sites, particularly the summer of 2014/15 and 2015/16 have been more than those which might be expected under ideal conditions. The drivers of the elevated mortality rates are however multifactorial and include enteritis (gut inflammation), upper gastrointestinal tract dysfunction (bloat), external skin damage from stinging organisms and late runting. All are exacerbated by generally poorer environmental conditions seen at low flow sites.

17 Ruakaka Bay farm over the summer of 2016/17 did not show significantly elevated mortalities above that expected in Chinook farming. Not only were environmental conditions more benign in terms of a lack of extended periods of warmer water, the company had also introduced an oxygen injection system which ensures a greater supply of oxygen (in terms of mg O_2 per hour) to the farm than that delivered purely by the relatively low flow seawater. A mitigation measure that may be mirrored by the placement of the same farm in a high flow site.

THE NEW ZEALAND RICKETTSIA-LIKE ORGANISM (NZ-RLO) AND ITS SIGNIFICANCE TO THE CONSIDERATION OF FISH HEALTH SUSTAINABILITY AND RISK TO OTHER FAUNA

18 In considering the relevance and importance of the NZ-RLO in terms of fish health and resultant biosecurity risk, the following are pertinent:

(a) The primary screening diagnostic test for NZ-RLO is a quantitative polymerase chain reaction (qPCR) test that detects nucleic acids from the NZ-RLO. This test is both highly sensitive and highly specific. As such, it can detect NZ-RLO at very low levels. The presence of NZ-RLO does not necessarily equate with clinical disease.

(b) There are good indications that NZ-RLO infection does not result in acute, severe clinical outbreaks of disease (epizootics).

(i) Molecular evidence of NZ-RLO is not found in all mortalities, i.e. it is not a necessary cause of mortality;

(ii) NZ-RLO has been isolated purely from skin lesions and not from the kidneys of fish, indicating that infection does not necessarily result in circulating infection (septicaemia);

(iii) NZ-RLO was found to be present in less than 50% of early skin lesions, indicating that it is not a necessary cause of skin lesions in the New Zealand presentation;

(iv) In the New Zealand presentation, very few pathognomonic lesions in the liver are noted in mortalities, indicating that pure, classical clinical infection resulting in mortality can rarely be confidently attributed to the organism;

(v) A genetically similar RLO was discovered in Tasmania in 2005[1]. A vaccine was subsequently developed, but has never been commercially used because the Tas-RLO did not produce enough clinical impact to warrant the use of the vaccine. The limited number of clinical cases occur only in the face of a concomitant stress factor.

(vi) Brosnahan et al. (2016)[2] state that the MPI considers that "NZ-RLO is not considered to be the primary cause of the mortalities in these fish".

19 Given these findings it is entirely appropriate to consider that any risk represented specifically by the presence of NZ-RLO is very low, and may be addressed by improving the environmental conditions. This is in complete agreement with the conclusions of Dr Diggles.

20 There is no justification either for linking the presence of the NZ-RLO in salmon farms to the occurrence of a rickettsia in scallops in the Marlborough Sounds.

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[1] Corbeil S, Hyatt AD, Crane MS (2005) "Characterisation of an emerging rickettsia-like organism in Tasmanian farmed Atlantic salmon *Salmo salar*", Diseases of Aquatic Organisms **64(1)**:37-44.
[2] Brosnahan C, Ha HJ, Booth K, McFadden AMJ & Jones JB (2016) "First report of a rickettsia-like organism from farmed Chinook salmon, *Oncorhynchus tshawytscha* (Walbaum), in New Zealand" New Zealand Journal of Marine and Freshwater Research. Accessed 18 May 2017; http://dx.doi.org/10.1080/00288330.2016.1242081

There are at least eight genus level lineages of Rickettsiaceae and 2 main clades just within the Rickettsia genus[3]. Rickettsiaceae and rickettsia-like organisms have been reported from 98% of scallops in the Marlborough Sounds and 81% from Coromandel waters in a survey in the year 2000[4]. Organisms from both the North Island and South Island were further characterised in 2002[5]. These organisms are ubiquitous in New Zealand scallops, present microscopically differently to NZ-RLO and are unequivocally different organisms to the NZ-RLO.

LONG TERM SUSTAINABILITY AND RISK TO OTHER FAUNA (GENERAL)

21 Dr Diggles indicates in his updated risk assessment that improving the farm environment by moving from low-flow sites to high-flow sites will have general benefits for fish health and reduce biosecurity risk to fauna external to the farms.

22 I reach the same conclusion as Dr Diggles, namely that in comparison to low-flow sites, a move to high-flow sites would result in improved fish health and biosecurity outcomes. I would like to explain why I reach that conclusion, and have set out my reasoning in Appendix A.

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Dr Colin John Johnston

[3] Castelli M, Sassera D, Petroni G (2016) "Biodiversity of 'Non-Model' Ricketsiales and Their Association with Aquatic Organisms", In: "Rickettsiales. Biology, Molecular Biology, Epidemiology and Vaccine Development." Ed. Thomas S, Springer International Publishing, Charm, Switzerland; pp59-90

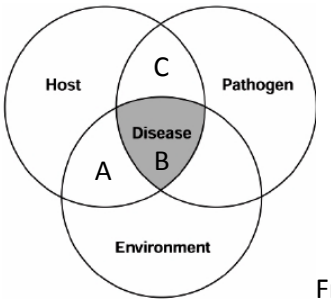
[4] Hine PM (2002) "Results of a survey on shellfish health in New Zealand in 2000", Surveillance **29(1)**, 3-7

[5] Hine PM, Diggles BK (2002) "Prokaryote infections in the New Zealand scallops *Pecten novaezelandiae* and *Chlamys delicatula*" Diseases of Aquatic Organisms **50**, 137-144

Appendix A:

THE IMPORTANCE OF ENVIRONMENT IN FISH HEALTH STATUS

23 It is important to remember that presence of pathogen does not mean presence of disease, nor does the mere exposure of a population or animal to a pathogen result in the successful establishment and spread of infection. What happens depends on a range of factors, generally broken down into 3 main groups; host factors, pathogen factors, environmental factors (diagram below)



From Sniezko, 1973[6]

24 It is possible that environmental factors may interact with host factors directly to result in purely environmental disease ("A"). The majority of expression or emergence of infectious disease in aquaculture is the result of the overlap of host, environment and pathogen ("B"). The greater the adverse environmental influence, the more likely that disease may result. It also works the other way; that by reducing adverse environmental influences, we can decrease the likelihood of both infection and disease.

25 High flow sites tend to have reduced adverse environmental influences compared to low flow sites, assuming that the flow rate at the higher flow sites are not excessive and result in exercise stress. It is my understanding that the current speeds at the newly identified high-flow sites lie within the envelope of those at existing higher-flow sites in Tory Channel, and that the fish have no issue in swimming at those speeds. The reasons for lower adverse environmental influences on higher flow sites as compared to lower flow sites are:

(a) Low flow areas are more prone to increased localised water temperatures and concomitant decreases in dissolved oxygen (DO) levels (warmer water holds less dissolved gas than cold water), plus

[6] Sniezko S (1973) "Recent advances in scientific knowledge and developments pertaining to diseases of fish", Advances in Veterinary Science and Comparative Medicine, **17**, 291-314.

more propensity for fluctuations of water temperature and DO. Rapid changes in environment are also stressful to fish;

(b) Higher flow sites will result in more stable water temperatures, more stable DO levels, less propensity for locally increased water temperatures;

(c) In addition, even at a given water temperature and therefore a given DO concentration, a high flow site will deliver a greater volume of water per hour to the fish population on a farm than a low flow site. As each litre of water contains a set amount of oxygen then more oxygen per hour will be delivered to the fish on a high flow site than on a low flow site. Somewhat akin to the oxygen injection discussed in paragraph 17, but delivered by nature.

26 Lesser adverse environmental influences result in better health and biosecurity outcomes as the fish are less stressed and have better immune and physiological status. Therefore, the probability of an infectious agent establishing and spreading through the farm population is reduced, simply because the adverse environmental influences are reduced.

27 We can also demonstrate using epidemiological principles what effect moving from lower flow to higher flow farm sites can have on the health status of the farms and biosecurity risk to other fauna presented by the farms.

EPIDEMIOLOGY – MODELLING PATHOGEN BEHAVIOUR

28 Does a farm site move from low-flow site to high-flow site result in a lower likelihood of a pathogen entering and spreading through the animal population on the farm?

29 The Basic Reproductive Rate (R_0) of a pathogen is a measure of what will happen in a population. It is in effect the number of subsequent susceptible hosts an infected animal directly infects. If R_0 is 1, then there is a steady state and infection neither dies away nor spreads. If R_0 is less than 1, then any infection that enters the population will actually disappear; it will not spread through the population. If R_0 is greater than 1, then the infection spreads. Logically therefore a downward pressure on the value of R_0 will result in better health and biosecurity outcomes – fewer (to zero) hosts infected and potentially shedding pathogens.

30 R_0 can be represented in epidemiological terms by the equation:

$$R_0 = \frac{\beta S}{r} \qquad [1]^7$$

β is a factor expressing the transmissibility of the pathogen between hosts;
S represents the number of susceptible hosts;
r indicates the rate of removal of the pathogen from the population.

31 Looking purely at flow rate; as we move from a low flow to a high flow scenario, we see that β is reduced as the transmissibility is diminished (shed pathogens spend less time within the farm – they are removed more rapidly). The value of β is further reduced in that fish are likely to be in a better immune/physiological status (all other factors being constant) and thus the transmissibility of the pathogen is further reduced. The value of r is also increased as fish in a better immune/physiological status are better able to respond to and eliminate the pathogen.

32 Therefore, if we decrease the numerator (βS) and increase the denominator (r) in equation [1] we exert downward pressure on the value of R_0.

33 Epidemiological models of pathogen behaviour therefore unequivocally indicate that the simple change of moving from low-flow to high-flow sites will result in better health outcomes, i.e. less likelihood of infection entering and spreading in any given population and thus less potential biosecurity risk to other fauna.

ASSESSING CHANGE IN POTENTIAL RISK TO OTHER FAUNA

34 Here we primarily consider wild fish. The risk to shellfish health from fish pathogens is negligible. Integrated multitrophic aquaculture systems, whereby fish and shellfish are raised alongside each other, show that fish and molluscs can co-exist. Where there is risk it is actually to the fish, from the molluscs which can act as parasite hosts or short term reservoirs of viruses.

35 In New Zealand waters the greatest risk to shellfish health comes from enzootic bacteria e.g. Vibrio species or mollusc parasites (Hine, 2002)[8].

36 Viruses affecting shellfish (i.e. causing pathology) are generally different to those causing disease in fish. For example, iridoviruses of shellfish lie in a

[7] Green DM (2010) "A strategic model for epidemic control in aquaculture" Veterinary Preventive Medicine, **94**, 119-127
[8] Hine PM (2002) Results of a survey on shellfish health in New Zealand in 2000. Surveillance. 29(1), 3-7

different taxonomic group to iridoviruses of fish (King et al., 2012)[9]. The closest relationship lies in the marine aquabirnavirus (MABV) group of the aquabirnavirus genus, which may have some weak pathogenicity under conditions of stress for the shellfish, however the MABV strains tend to be more risk to fish than molluscs in general (Renault, 2008)[10]. Aquabirnaviruses are enzootic in New Zealand waters and any presence in shellfish is most likely to have come from the wild.

37 We may also use epidemiological models of pathogen behaviour to estimate changes in potential risk to other fauna as a result of the relocation of farms from low-flow to high-flow sites.

38 In the aquatic environment spread of many pathogens is via density dependent transmission i.e. the more hosts in a given water volume, the more fish to fish interactions take place, each of which has a certain probability of being between an infected individual and a susceptible individual and each with a certain probability of successful transmission of a pathogen between the infected and susceptible.

39 In simple terms, farm populations tend to exist at higher densities (and probably higher population numbers) than wild fish populations. So, both β and S factors are higher for farmed stock than wild stock, thus, from equation [1], R_0 will tend to be higher for farmed stock than wild stock. In effect this means that the likelihood of establishment and spread of a pathogen in farmed stock is higher than for wild stock. It is easier for infection to pass from wild to farmed stock than vice versa. Risk is biased in the same direction.

40 Spread from farmed stock to wild stock may occur directly via host density dependent transmission where wild fish are inside the farm, whereupon the same factors we have previously considered that reduce risk on higher-flow sites still apply and for this transmission model the risk is lower on high-flow sites compared to low-flow sites.

41 Spread may also occur via water column mediated transmission. The modelling of this is much more complicated. The likelihood of successful entry, establishment and spread of a pathogen in a distant wild fish population is

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[9] King AMQ, Adams MJ, Carstens EB, Lefkowitz EJ (Eds.) (2012) Virus Taxonomy: Classification and nomenclature of viruses. Ninth report of the International Committee on Taxonomy of Viruses. International Union of Microbiological Societies Virology Division. Elsevier Academic Press, Oxford, UK

[10] Renault T (2008) Shellfish viruses. IFREMER, Manuscript Number 781, La Tremblade, France

dependent on the number of infected and shedding fish in the source farm population, the extent to which the pathogen is removed from the water column (by bacteria, small organisms, phages, ultraviolet radiation and absorption and filtration by non-target organisms) and the required infectious dose for the new host.

42 Whilst pathogens from a high flow site would be expected to travel to a new location faster they will arrive at a potential new host population in lower concentration per unit volume of water. This is because, all other factors being equal, for any fixed population that is infected and shedding there will be a fixed number of infective particles shed per time period. On a low flow site that represents a smaller volume of water having moved through the farm than a high flow site, and thus the relative concentration of pathogen per unit volume of water will be lower for high flow sites. This is significant because successful infection requires a specific concentration of pathogen (an infectious dose). Lower concentrations of a pathogen mean a lower likelihood of successful infection[11] as it is more likely that an infectious dose is not achieved.

43 So, whilst higher water flows may actually move an infectious particle further or faster, the positive effects on the health of the source farm and the dilution effects on the pathogen concentration have positive benefits for reducing risk to other fauna.

SUMMARY

44 In summary, if a desired outcome is the reduction of disease risk to both farmed and wild populations, and better health outcomes for the farms and the ecosystem, and if the choice is between farms remaining on low-flow sites or moving to high-flow sites, the answer is clear and unequivocal: We can expect better all-round outcomes at the high flow sites.

[11] Murray AG (2009) "Using simple models to review the application and implications of different approaches used to simulate transmission of pathogens among aquatic animals" Preventive Veterinary Medicine **88**, 167-177