Modelling origin and spread of Infectious Pancreatic Necrosis Virus in the Irish salmon farming industry: the role of inputs

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Abstract

Observed emergence of IPNV in farmed Irish salmon is simulated using a model originally developed to analyse the spread of the virus in Scotland [Murray, A.G. 2006a. A model of the spread of infectious pancreatic necrosis virus in Scottish salmon farms 1996-2003. Ecol. Model. 199, 64-72]. IPNV appears to have become established relatively recently in Ireland and the model is altered to explicitly simulate the origin of the spread of the virus. Input to freshwater farms was key to initiation of infection, but modelling suggests that endogenous spread was responsible for much of the subsequent increase in prevalence of IPNV. From the modelling, it is unlikely that direct imports accounted for most IPNV cases. If this is the case, cessation of imports, without a substantial improvement in biosecurity, would be likely to be of only limited effect in controlling IPNV. Marine IPNV prevalence appears to be insensitive to direct interventions in the marine environment (as in the Scottish model). A multi-element control strategy, targeting both endogenous spread and external input of infection and prioritising freshwater sites, but extending to marine sites, would probably now be required to eradicate IPNV from Ireland.

Introduction

Infectious Pancreatic Necrosis Virus (IPNV) can cause disease (IPN) in a range of farmed fish species, particularly salmonids (OIE 2003). In most affected species any mortality is of fry, but Atlantic salmon (*Salmo salar*) smolts can also suffer losses usually a few weeks after being moved to sea (Guy et al. 2006). The virus is also found in wild and escaped fish (Munro et al. 1976, Wallace et al. 2005), but disease is not reported in free-living fish. The virus has spread its range across most of the salmonid farming areas of the world (OIE 2003), and within Europe IPNV has shown a general tendency to spread from north to south (Roberts and Pearson 2005).

Ireland, at the southern limit of European salmon farming, has historically been considered to be free of IPNV; or at least infection has been very rare. However, reports of infection have increased in recent years and the first reported clinical outbreak of IPN in salmon occurred in 2003 (Ruane et al., 2007). The spread of IPNV was therefore analysed within the Irish salmon farming industry using a model that has been developed using data from the spread of IPNV among Scottish salmon farms (Murray 2006a). As the Irish industry is reliant on the importation of live fish/ova, the model was modified to simulate the origin of the epidemic by inclusion of the possible external input of new infection to the system.

2. Material and methods

2.1. Derivation of data

Under EU Directive 91/67/EEC all freshwater salmon facilities and marine smolts sites are inspected and tested for listed diseases each year. In Ireland, this testing also included screening for IPNV and data for the period 1994-2006 is shown in Table 1. Prevalence is the proportion of these samples that are positive, assuming sampling is not biased this approximates to the proportion of sites that are infected (Murray et al. 2003). Any sample containing at least one positive pool (based on tissue from 1 to 10 fish) was treated as a positive sample and therefore came from an infected farm. During the period 1994–2006 *ca.* 600 samples were taken from 55 freshwater sites (total number of fish 18,005) and *ca.* 320 samples were taken from 53 marine sites

(total number of fish 8,638). Virus isolation was carried out according to the method laid down in EC decision 2001/183/EC (Communities, 2001).

Irish salmon production rose through the 1990s; however, 2001 was a peak year and production was significantly reduced by 2006 (Browne & Deegan, 2006; Table 1). The spread of IPNV in Ireland (Table 2) occurred during this period of declining production – in contrast to the spread in Scotland during 1996-2003 which occurred concurrent with a doubling of salmon production (Smith, 2006).

Infection with IPNV appeared as isolated events in a few cases before 2001 in Ireland and the virus did not persist. Since 2002, 103 samples from freshwater Irish sites have yielded 9 IPNV positive results with prevalence tending to show year-on-year increase.

The proportion of marine sites that were infected (26%) was more than triple the freshwater prevalence, but it is likely that many of these would have been infected by smolts that were moved from Irish freshwater sites, rather than imported.

3. Model

The model used (Fig. 1) is based on that described by Murray (2006a). The farmed salmon population is divided into freshwater and marine components, within each of which the proportion of the population in susceptible (S) and infected (I) populations (Anderson and May 1979). Not all fish within an infected population are infected, but once a population is infected it is assumed to remain so until harvested.

Salmon are input to the model to freshwater as uninfected but susceptible fry where they may pick up infection by exposure to infected populations using the formula $b_f S_f I_f$ (Anderson and May 1979). After 1 year they are transferred to marine sites, these marine sites may receive smolts from multiple freshwater sites, and if any one is infected the receiving marine site is infected (Y). If the population is not infected it may pick up infection within the marine environment $b_m S_m I_m$. Here b is the transmission coefficient, reflecting all forms of infectious contact between sites within

the freshwater (b_f) or marine (b_m) environment, including movement of fish or equipment and natural transmission of virus by water movements or birds.

IPNV was already widespread in Scotland in 1996, the beginning of the period modelled by Murray (2006a), while the Irish data go back to a time when outbreaks were isolated. In these circumstances even occasional input of infected fish from a high prevalence population may be significant to a low prevalence population. The model is therefore extended to include the variables θ and ϕ to represent external inputs, or novel infection, into freshwater and marine farms respectively.

The proportion of marine sites receiving infected smolts Y is determined as:

$$X = I_f k/(1 + I_f(k-1))$$

$$Y = X + (1 - X)\phi$$

Where k is the number of freshwater sites used to source smolts. External inputs to marine sites that also receive infection from Irish freshwater sites do not affect prevalence of infection; Y, which includes inputs, is a modification of the variable X that was used in the Scottish model.

The model may be run in population-independent form, or it may be made population-dependent allowing for increased rates of contact between closer farms. As S and I are proportions, and so S + I = 1 in each environment (freshwater and marine), transmission rates must be multiplied by relative population for a given year m_y if this is to be applied (m_y = 1 for population-independent transmission). As IPNV emerged in Scotland in a period of increasing production, and in Ireland in a period of declining production, the population-independent model (m_y =1) seems more probable and so is used as default. As Irish production was falling, an incorrect assumption of independence would lead to over-estimation of future IPNV prevalence, while an incorrect assumption of population-dependence of transmission would lead to underestimation. It is therefore also safer to assume population-independence.

The model (Fig. 1) is thus

$$dS_f/dt = s\theta - m_y b_f S_f I_f - sS_f$$

$$dI_f/dt = s(1-\theta) + m_y b_f S_f I_f - sI_f$$

$$dS_m/dt = h(1 - Y) - m_y b_m S_m I_m - hS_m$$

$$dI_m/dt = hY + m_y b_m S_m I_m - hI_m$$

The parameters s and h are rates of turnover, the inverse of the time spent on freshwater and marine sites, respectively and m_y is the years population density factor described above.

4. Model parameter values

To run the model requires appropriate parameter values. These may be dependent on the structure of the industry, so the turnover parameters are s = 1 and h = 1/1.5 y^{-1}, as salmon are assumed to spend 1 year in freshwater and 18 month in seawater sites.

The number of sources of smolts used by marine sites is k, and different scenarios use k of 1-3. Scottish data suggested a k of 2 or 3 was appropriate although higher values did occur for a minority of sites (Raynard et al. 2005).

The relative population parameter m_y =1 for population-independent transmission and a different value relative to the initial year 1994. In the Scottish version of the model a logarithmic increase with time fitted observed production and hence population. However, Irish production peaked in 2001 and thereafter declined (Table 2). Projection may assume production continues to fall, stabilises or recovers. Population-dependent and -independent projections are very similar if the population stabilises. However, the default assumption is population-independence m_y = 1.

The values of the parameters b_f and b_m are systematically adjusted to optimise the fit of the modelled to the observed prevalences of infection. Optimal fit is found at the minimum sum of square differences between modelled and observed IPNV prevalence. In simulations without external inputs, $\phi = \theta = 0$, the initial IPNV prevalence value (I_{f0}, I_{m0}) are also adjusted to optimise the model's fit to observations. For simulation with inputs > 0 the initial prevalence is set to zero as these simulations aim to replicate the initial introduction of IPNV to the system.

As the freshwater component of the model is independent of the marine component, this freshwater IPNV prevalence can be fitted to observations first and thus only b_f and I_{f0} need be adjusted, allowing a systematic search through parameter space. This parameter space is 2D (b_f and I_{f0}) for scenarios with inputs, but only 1D (b_f) for scenarios without inputs. This is very different to the explosion of parameter combinations that exist in most, even quite simple, models and for which exhaustive exploration is not possible (Murray, 2001). When the freshwater component has been optimally fitted the marine component is fitted; for this the values of b_f and, if there are no inputs, I_{f0} are optimised.

Inputs of IPNV were not included in the Scottish model (Murray, 2006a), but it was noted that even a few cases of imported or vertically transmitted infection could be epidemiologically significant at low prevalence. Ireland imported considerable numbers of ova and fish and so potentially θ and $\phi > 0$. The range $\theta = 0.01$ to 0.08 is that from a single input to almost all observed IPNV being due to input, and so is explored using model experimentation. The upper part of the range appears unlikely as this would mean most cases were due to direct import, which would imply an absence of farm-to-farm spread.

Once the model has been fitted to the existing data, it can be projected to predict future development of IPNV's prevalence in Ireland. Scenarios are investigated whereby IPNV develops to steady-state with: no change in parameters; or where b_f or b_m are cut by 50%; or k is cut from 3 to 1. These scenarios are used to investigate possible control policies and are similar to those analysed for Scotland previously (Murray 2006a). Scenarios of cutting off input of infection are also included, simulated by setting ϕ or θ to zero. This control might be achieved by ceasing to take any imports from potentially infected sources, or by more effective screening to ensure imports are pathogen free.

5. Results and Discussion

5.1. Initiation of the IPNV outbreak in Ireland

IPNV has been detected in Ireland in most years since 1994 (Ruane et al. 2007), however, until about 2003 the number of cases was small (Fig. 2) and infection persisted for only short periods. In 2006 prevalence exceeded 56% of marine sites and 29% of freshwater sites, IPNV may thus be considered to have become endemic. This spread of infection is at the national level and does not imply that once a site becomes infected it remains infected, site level infection is likely to be transient (Murray 2006b).

In the absence of inputs ($\theta = \phi = 0$) the model can be fitted to observations by allowing a minimal initial prevalence and fitting transmission coefficients (Fig. 2), as was applied to modelling the Scottish epidemic (Murray 2006a). However, the initial 1994 values for prevalence of infection are vanishingly small (3×10^{-7} in freshwater and 9×10^{-10} for marine farms). The R_0 value is also large, again especially in freshwater (2.27 as opposed to 1.41 in Scotland; Murray 2006a). This implies that the Irish epidemic originated after 1994, indeed after 2001, because the very small earlier values are not meaningful in terms of numbers of infected farms. The high R_0 also suggests continuing input that allowed prevalence to increase faster than expected increase due to endogenous spread.

The high levels of spread required to explain the increase in IPNV in the absence of imports would result in high steady-state values were the epidemic to continue. Under this assumption prevalence is predicted to rapidly rise to around 55% of freshwater and 90% of marine sites by around 2008/9, whereupon it stabilises. The predicted freshwater prevalence for Ireland is higher than that predicted for Scotland, where only about 30% prevalence is predicted for population-independent modelling (Murray 2006a), although marine prevalence is similar.

These results indicate IPNV emerged recently in Atlantic salmon culture and we therefore consider scenarios with inputs whereby θ and $\phi > 0$. We restrict these runs to the period when IPNV prevalence was visibly increasing i.e. 2002-2006. Notably

this was a period during which Irish salmon production halved, in contrast to the period of emergence of IPNV in Scotland 1996-2003 when production doubled. Given these contrasting cases it seems unlikely that transmission is strongly dependent on population, and therefore population-independent is used for default.

5.2. Prevalence of IPNV in freshwater salmon farms

Freshwater prevalence can be analysed, in this model, independently of the marine IPNV prevalence and therefore we start with the freshwater farms. Prevalence in freshwater can be fitted assuming levels of input to θ = 0.005-0.08 of farms y^{-1} and R_0 values calculated. The value of R_0 falls as input rate increases and so fewer of the cases remain to be accounted for by endogenous spread. If input = 0.01 of sites y^{-1} R_0 = 2.06, while at 0.08 R_0 = 0.93.

The shapes of the simulated time series fit the observations best for low levels of input, with a minimum RMS error for input of θ = 0.005 y^{-1} (not shown). However, the RMS of the model's fit is dominated by 2004, when no IPNV was detected; values of θ up to 0.04 visually fit the observations reasonably well (Fig. 3). At least one event must have occurred during the period if IPNV was absent prior to 2000. Inputs >0.04 y^{-1} clearly under-estimate observed 2006 prevalence. These data do not give a clear optimal value for input, but a range of θ = 0.01-0.04 sites y^{-1}, i.e. 1 to 4 input events over the period 2002-6 are most consistent with observations.

This range of input values of θ = 0.01-0.04 y^{-1} is also supported by the R_0 values fitted against θ (Fig. 4). Freshwater R_0 values of 1.9 were estimated for Shetland and 1.4 for Scotland as a whole (Murray 2006a). If these values of R_0 applied in Ireland, i.e. biosecurity levels were similar in the two countries, they would fit inputs of approximately 0.013 or 0.04 y^{-1} respectively. If Irish biosecurity were better than in Scotland, then higher input rates might have applied; but lower inputs would apply if biosecurity were less good.

The steady-state freshwater IPNV prevalence calculated by the model is decreased at higher simulated inputs (Fig. 5). This counter-intuitive result occurs because the model is fitted to the observations and so estimates a lower rate of endogenous spread

when higher input is specified; if input is cut for any given calculated endogenous spread then the prevalence will decline. However, for inputs of $\theta = 0.03$ y^{-1} or less the effect of cutting off all inputs is <5% of sites (about 1 infected site), but if input >0.05 y^{-1} the effect of cutting off these inputs increases dramatically and by 0.08 y^{-1} cutting inputs alone would be enough to eventually (after decades) eradicate IPNV from freshwater sites. Conversely, if inputs are low then endogenous spread is dominant, and so improved biosecurity ($0.5b_f$) leads to a 30% drop in the proportion of sites that are infected, however if inputs are higher cutting b_f would be less effective. It is only for high-end estimates of θ that cutting off inputs is more effective than halving freshwater transmission b_f, although this is always necessary to achieve eradication.

5.3. Prevalence of IPNV in marine salmon farms

Modelled prevalence in the marine salmon farms is driven by movement of infected fish from freshwater farms, even when freshwater prevalence is relatively low. Higher assumed levels of external input into either marine or freshwater farms leads to lower estimates of R_0 because endogenous spread is not required (Fig. 6). If $k = 2$ instead of 3 the estimated value of R_0 is higher to compensate for reduced exposure to infected smolts. For $k = 3$ inputs of $\theta = 0.01$-0.02 y^{-1} into freshwater would be consistent with 1.45 R_0 obtained for Scotland if $k = 3$ (Murray 2006a). If $k = 2$ this 1.45 is only consistent with larger freshwater inputs (0.02 to >0.08) but if $R_0 = 1.75$, as obtained for Scotland for $k = 2$, this is consistent with low to moderate freshwater inputs (θ = <0.01-0.04). The optimal scenarios would appear to be those with 0.01-0.04 y^{-1} input into the freshwater farms. The model provides little constraint on marine inputs (to which results are very insensitive).

The freshwater input rates suggested by marine R_0 ($\theta = 0.01$-0.04 y^{-1}) are in line with those (0.01-0.04 y^{-1}) fitting observed prevalence time-series (Fig. 3), and the $\theta = 0.04$ y^{-1} suggested by freshwater R_0 (Fig. 4). All three indicate low to moderate input to freshwater, with significant endogenous spread. Of course higher freshwater inputs could apply if Irish biosecurity were more effective (lower R_0) than Scottish,

especially if k were low, and conversely very low inputs are implied if Ireland's freshwater R_0 was comparable to Shetland's.

The effect of policy changes on marine prevalence of IPNV is investigated for scenarios assuming changed policy from 2007 and running the model to steady-state over the pre-policy change range θ = 0.005 to 0.08 (Fig. 7). These policies are: existing control policies continued; cutting off freshwater inputs ($\theta = 0$), or marine inputs ($\phi = 0$), or cutting k from 3 to 1, or improved biosecurity leading to cutting b_f or b_m by 50%. These scenarios lead to almost identical results regardless of marine inputs, so results are shown for pre-policy change $\phi = 0.04$ y^{-1}. Results are sensitive to the level of freshwater inputs, so steady-state results are shown for the pre-policy change range $\theta = 0.005\text{-}0.08$ y^{-1}.

It is possible that Irish marine farms, being smaller, use fewer smolt sources than do Scottish farms, so k may be smaller. This would not affect freshwater modelling, but would imply either much higher rates of external input to marine sites or weaker biosecurity. If k = 2 is assumed (Fig. 8) this has only a marginal effect on the scenario predictions relative to k = 3 (Fig. 7), except that the effect of halving marine transmission (b_m) is increased and the effect of cutting k is reduced, so that these two policies result in closely comparable (5-10%) reductions in IPNV prevalence. The effect of cutting of freshwater inputs is less for high θ, post-cut prevalence = 0.36 as opposed to 0.18 when pre-cut $\theta = 0.08$. This is because b_m is larger when k = 2 so a given level of marine inputs ϕ sustains a higher level of marine infection when freshwater inputs are removed.

Marine steady state IPNV is most sensitive to the policy scenarios affecting freshwater prevalence, if b_f is halved or θ set to zero (Figs. 7 and 8). If pre-policy freshwater input rate $\theta < 0.04$ y^{-1} then cutting off these inputs is relatively ineffective, but if it were $\theta > 0.06$ y^{-1} then its removal has a big impact on marine prevalence of IPNV; this reflects the parameter's impact on freshwater prevalence (Fig. 5). There is a moderate sensitivity in marine IPNV prevalence to cutting k, the number of sources of smolts, if this is high. IPNV prevalence is not sensitive to direct control on marine activities (b_m) and is extremely insensitive to cutting off direct marine input (ϕ).

No single control policy is likely to be effective at controlling marine IPNV; control would have to be part of a multi-element strategy targeting inputs, and both marine and freshwater transmission. Only when very high levels of input are assumed is $R_0 < 1$ in both freshwater and marine environments (Fig 6), this means that it is unlikely IPNV could be eradicated simply by cutting off further inputs. However, except when very low inputs are assumed for both marine and freshwater environments, R_0 is <2, even if $k = 2$; if $k = 3$ this applies only when inputs are $<0.01\ y^{-1}$. This means that a policy that succeeded in halving transmission in both environments, when combined with the cutting off of further inputs, should eradicate IPNV from Irish salmon farms. However, such controls would be costly and might well not be cost-effective.

6. Conclusions

The model indicates that the current IPNV epidemic in Ireland probably began some time in 2001-2003 and was not related to earlier outbreaks that were detected but did not become established. It is possible that the current strain of IPNV arose spontaneously from local aquabirnaviruses, as appears to have happened with Australian aquabirnavirus (Crane et al. 2000), or was transferred with imported salmon, or other cultured fish or equipment. Such spread into Ireland might be regarded as part of a general north to south spread of IPNV across north-western Europe (Roberts and Pearson 2005); spread could be driven by physical spread of pathogens or of conditions appropriate for emergence of local pathogens.

Earlier outbreaks of IPNV infection did occur in Ireland. Eradication of freshwater outbreaks might have been due to good management, but with R_0 of 1.41 this could also be explained stochastically; there is a 24% chance this would have occurred by chance within one year given only one site was infected. However if marine $R_0 = 1.45$, and given 3 infected sites in 1995 and 1999, it is unlikely marine infection would be lost by chance within 2 years. It is possible biosecurity was extremely well enforced or that the virus was less virulent, and so little spread occurred between marine sites. Strong biosecurity to prevent viral spread between marine sites may be effective for marine IPNV control; but it is unlikely to be effective while the virus is present in freshwater sites.

The salmon production environments in Scotland and Ireland were very different. Production was far lower in Ireland (Table 1) than in Scotland, where a peak 170,000 tonnes y^{-1} was produced in 2003 (Smith, 2006). Irish marine production is localised in a few areas that are isolated from each other (Browne and Deegan, 2006), Scottish marine production occurred along most of the west coast and in western and northern offshore archipelagos (Smith, 2006). Irish production halved during the period of IPNV emergence of 2002-2006, while in Scotland production doubled over the main period of IPNV emergence, 1996-2003 (after this period Scottish production fell: Smith, 2006). The coastal environments are different, with the Irish coast containing more open and shallower bays, while the Scottish coastline is more fjordic and with more offshore islands. Irish waters are warmer than are the more northerly Scottish waters and day-length extremes are less. Application of the model to both countries shows its value to be more than local.

IPNV spread during conditions of rising salmon production in Scotland and declining production in Ireland and numbers of farms. This would tend to suggest that the increased incidence of IPNV is independent of population size, allowing a simplification to the model. Declines of production by 50% did not halt spread, and while further decline (under density dependence) might lead to such extremely low transmission that IPNV is eradicated, it is doubtful such an industry would be sustainable. Population-independent scenarios estimated slightly less onerous controls for eradication of IPNV from Scotland than were estimated assuming population-dependence (Murray 2006a); lower production in Scotland in 2004-2005 (Smith 2006) would also suggest a slightly easier than predicted control even under the population-dependent model.

The model results indicate great sensitivity of both freshwater and marine IPNV prevalence to processes in, and inputs, to freshwater sites. If inputs were large, i.e. affecting 8% or more of freshwater sites annually, then cutting them off would be sufficient to eradicate IPNV. However if inputs were more moderate, endogenous spread explains much of the current prevalence and cutting off inputs is predicted to be an ineffective method of eradication. Model fit to observation generally supports low to moderate levels of input; low levels of input might be due to rare mutation events. The model is rather insensitive to assumptions concerning the marine environment. The model is most sensitive to inputs and transmission in freshwater;

unfortunately, with relatively few cases of freshwater infection, the data used to constrain these processes are somewhat limited.

The model suggests that the most effective control on IPNV in Ireland is to control its spread in freshwater. Controls on marine transmission, numbers of sources of smolts and inputs on their own would have less effect on IPNV's prevalence, but improved controls on freshwater biosecurity, marine biosecurity and inputs would all be required for an effective eradication policy.

The model can be generalised to simulate other infectious diseases and it implies that repeated inputs are more significant for diseases that are constrained to low levels because of low rates of spread such as Bacterial Kidney Disease in the UK (Bruno, 2004), but are relatively unimportant for diseases that are spreading rapidly such as Infectious Salmon Anaemia in Norway before 1992 (Lyngstad et al., 2008).

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Table 1. Atlantic salmon production in Ireland from 2001 – 2005 (from Browne & Deegan, 2006)

Year	Production (tonnes)	Relative production
2001	23,312	100%
2002	21,423	91.9%
2003	16,347	70.1%
2004	14,067	60.3%
2005	13,764	59.0%

Table 2. Surveillance results for IPNV from Irish salmon farms 1994-2006.

Year	Freshwater sites		Marine Sites	
	Samples	Positives	Samples	Positives
1994	26	1	7	1
1995	30	0	22	3
1996	28	0	26	1
1997	36	0	23	0
1998	29	1	25	0
1999	25	0	20	3
2000	30	0	23	1
2001	26	0	23	0
2002	24	0	23	0
2003	22	1	21	2
2004	21	0	14	6
2005	19	3	17	6
2006	17	5	16	9

Figures

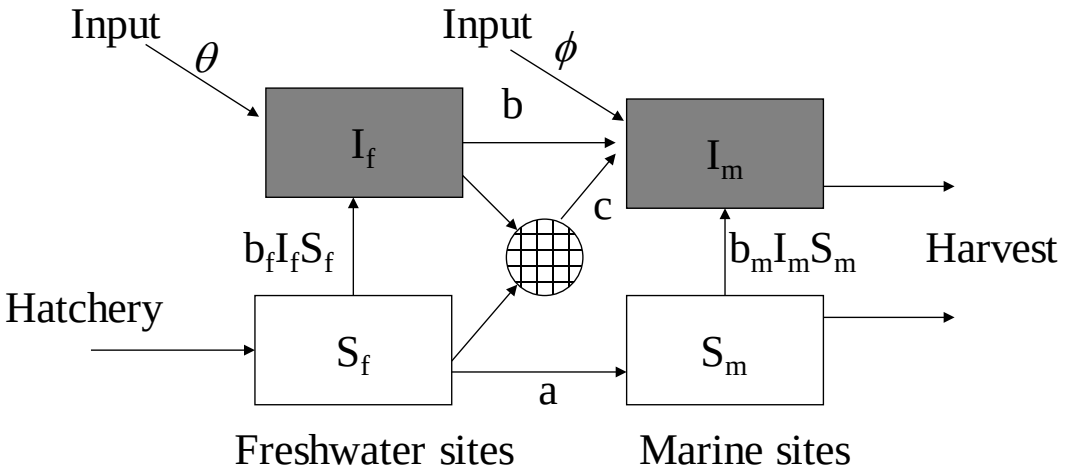

Fig. 1 - Model structure (after Murray 2006a). Salmon eggs are assumed initially uninfected, and may pick up infection in freshwater ($b_fI_fS_f$), after 1 year they are moved to seawater sites. These will be uninfected only if they receive smolts only from uninfected freshwater stocks (a); sites that receive only infected stocks (b) or a mixture of infected and uninfected stocks (c) will be infected. Uninfected sites may pick up infection ($b_mI_mS_m$), but infection is only lost when the stocks are harvested. The model is modified to include imports of infected stocks into freshwater (θ) or marine (ϕ) sites.

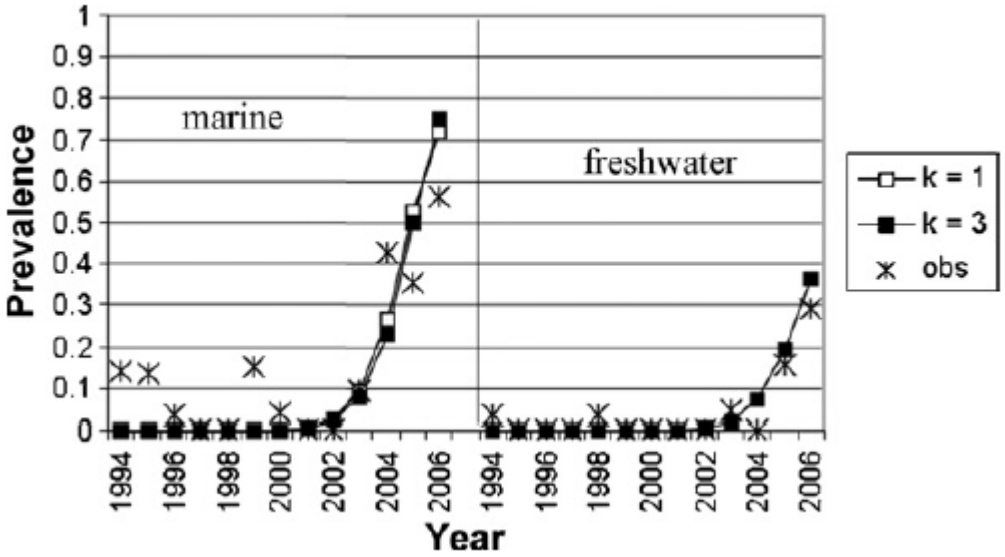

Fig. 2. Observed Irish IPNV prevalence (proportion of sites infected) and simulated prevalence time series without any inclusion of imports 1993-2006

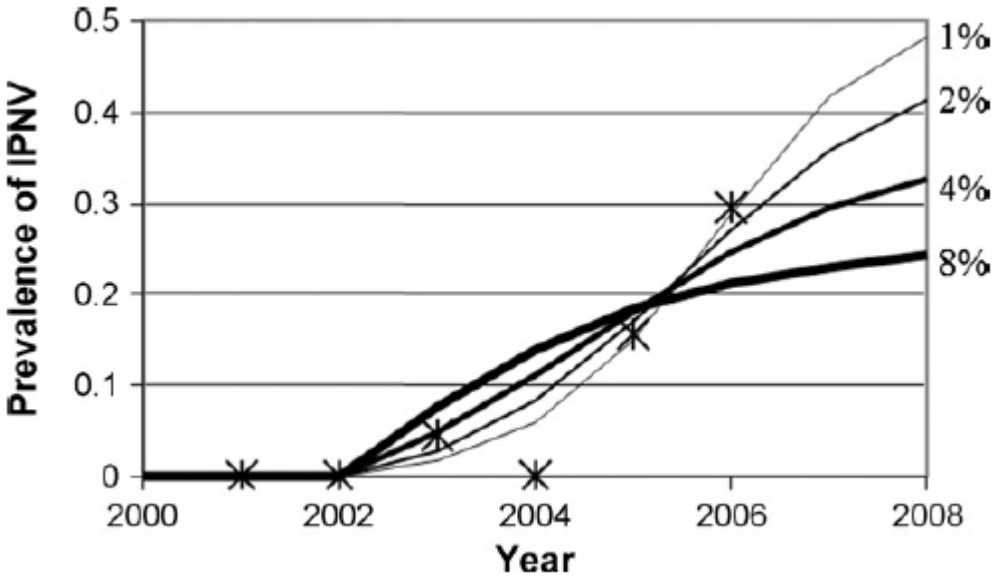

Fig 3. Observed and simulated Irish freshwater prevalence of IPNV (proportion of sites infected) time series assuming inputs θ of 0.01 (thinnest line), 0.02 y^{-1} (thin line), 0.04 y^{-1} (medium line) or 0.08 y^{-1} (thick line).

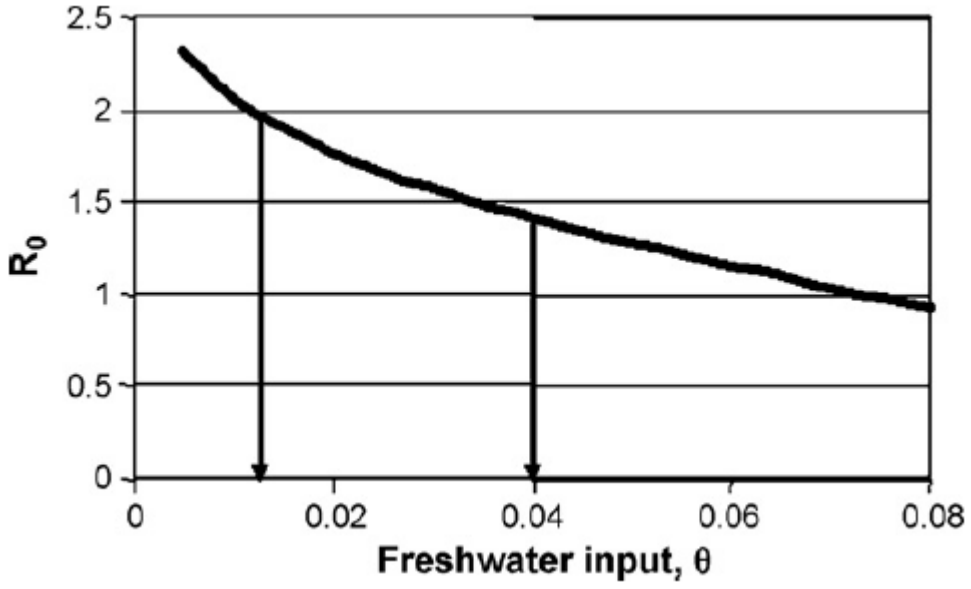

Fig 4. R_0 for freshwater versus assumed rate of inputs. Also shown is input level that generates an R_0 comparable to Scotland (1.45) as a whole and Shetland (1.9).

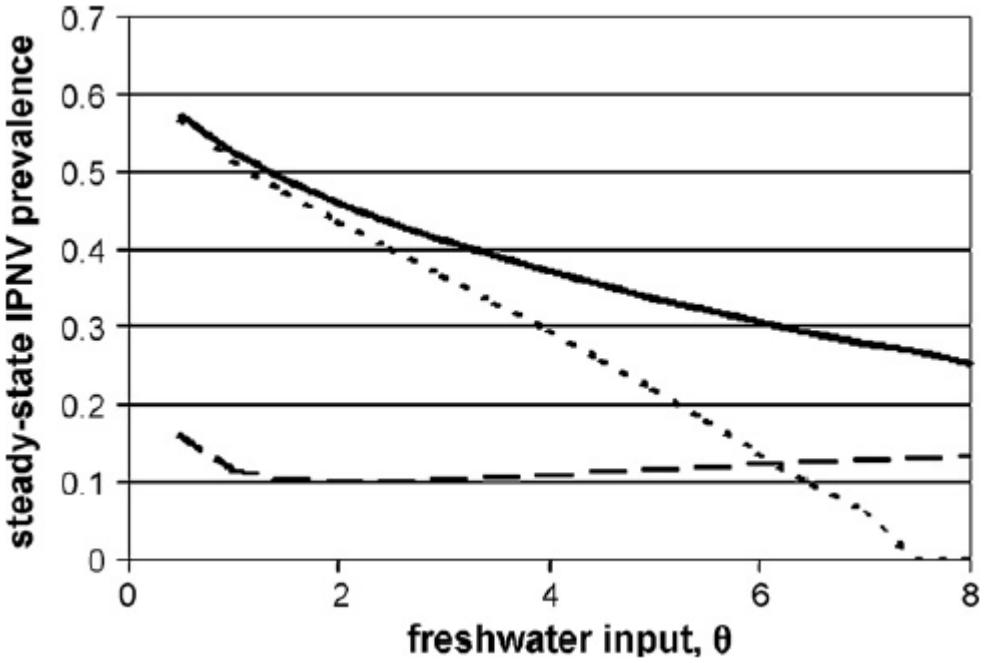

Fig 5. Steady state solution of freshwater IPNV prevalence (proportion of sites infected after 200 simulated years) assuming current epidemiological conditions (solid line) or that inputs cease (short dash line) or b_f is cut in half (long dash line).

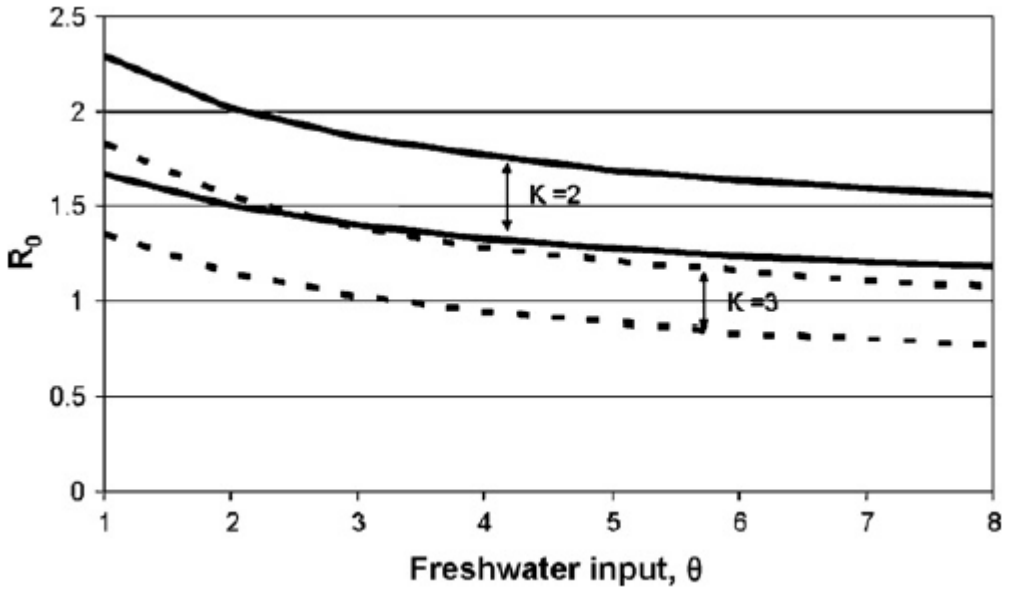

Fig 6. Estimated marine R_0 versus freshwater θ over the range 0.01 – 0.08. Solid lines are for k = 3 and dashed lines for k = 2, the upper line of each pair applies where θ = 0.01 and lower line applies θ = 0.08. An R_0 of 1.45 as estimated for Scotland with k = 3, would be consistent with θ of 0.01-0.02, for k =3 and θ = 0.02->0.08 for k

=2. An R_0 of 1.75 as estimated for Scotland for k =2 would be consistent with θ of <0.01 for k =3 and <0.01-0.04 for k =2.

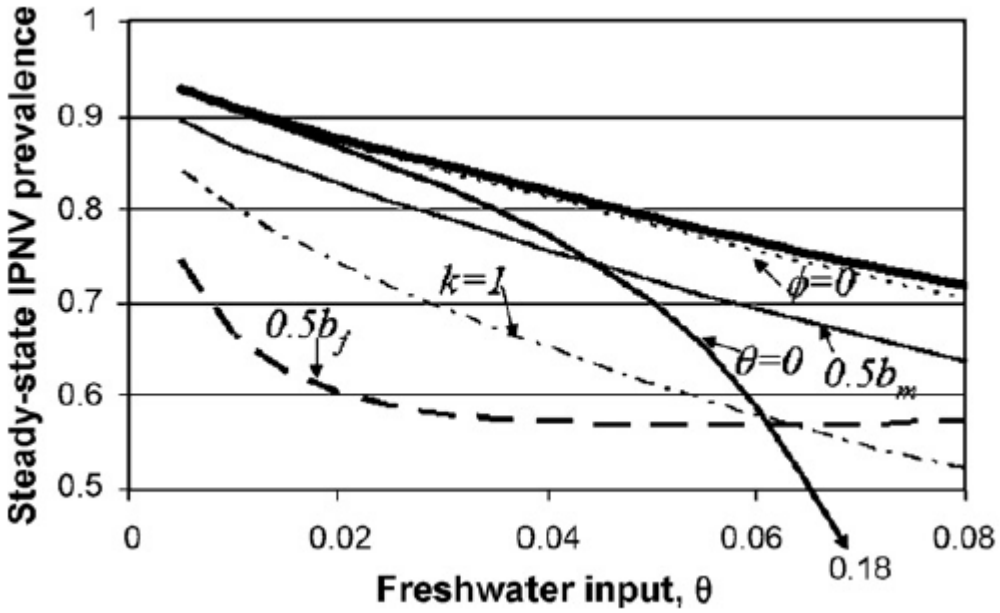

Fig 7. Steady state marine IPNV prevalence (proportion of sites infected after 200 simulated years) versus pre-policy change freshwater input θ , pre-policy change ϕ = 0.04. Policies are: current policy (thick line); or cessation of inputs to freshwater (θ =0, medium line, this collapses to 0.18 when cut from pre-policy change θ = 0.08); or marine sites (ϕ =0, thin dashed line that is hardly visible under current policy line, i.e. this has almost no effect); or improved biosecurity at freshwater (0.5b_f, thick dashed line); or marine (0.5b_m, thin solid line) sites; or reduced number of smolt sources k = *3 to* k =1 (dash dot line).

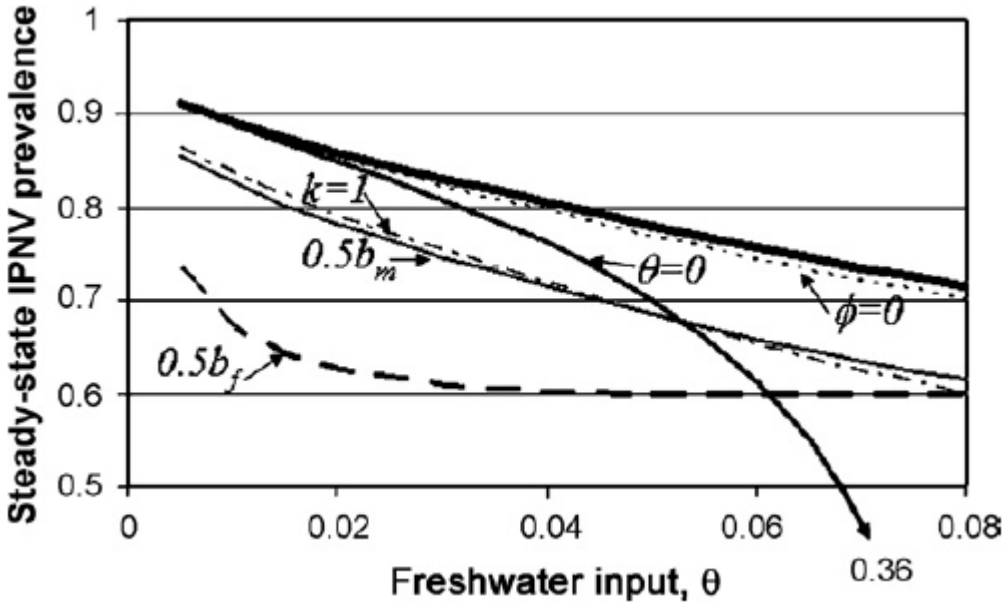

Fig 8. Steady state marine IPNV prevalence for k = 2 (proportion of sites infected after 200 simulated years) versus pre-policy change freshwater input θ, pre-policy change $\phi = 0.04$. Policies are as Fig 7 except smolt sources reduction is from k = 2 to k =1 (dash dot line).