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Response to Noakes' criticisms of Connors' statistical analysis

In his report to the Cohen Commission on salmon aquaculture Dr. Noakes (Noakes 2011) raises concerns about some of the analyses performed in my report for the Cohen Commission on relationships between salmon aquaculture and sockeye population dynamics (Connors 2011) stating:

“There are, however, several significant problems with his assumptions, methods, analyses, and conclusions.” (page 5)

Below I respond to these criticisms. Each criticism is in quotations and bold italics followed by my response. This document is not a general review of Dr. Noakes' report or of the analysis he performed; my comments are restricted to the specific criticisms he has of my analyses.

“Connors (2011), for instance, combined several variables in a pairwise fashion in a nonlinear model to test for relationships with Fraser River sockeye salmon production.” (page 5)

My analysis did not use non-linear models; instead my analysis fit linear models to a multi-population dataset of sockeye productivity in an information-theoretic context (Burnham and Anderson 2002). Specifically, linear models were constructed as mathematical representations of hypotheses for the decline in Fraser River sockeye salmon as identified by Peterman (2010), including salmon aquaculture production along juvenile sockeye marine migration routes. The likelihood of each hypothesis given the data was then computed and the relative support for each hypothesis was estimated based on the Akaike Information Criterion (AIC). AIC takes into account both the statistical goodness of fit of a model and the number of parameters estimated to achieve the degree of fit by imposing a penalty for the number of parameters in the model. AIC weights of each hypothesis were then computed as a measure of the probability that each hypothesis is the “best” among the set of hypotheses considered. To account for uncertainty in which model(s) best explained the data I calculated model-averaged estimates of the association between the parameters in each model and sockeye productivity. Finally, inference was based on the direction, magnitude and uncertainty in the model-averaged parameter estimates, and their predicted influence on sockeye productivity and mortality.

“Connors (2011) found no significant relationship between farmed salmon production and Fraser River sockeye salmon productivity, no significant relationship between pink salmon abundance in the North Pacific and Fraser River sockeye production but he did identify a significant negative relationship between winter sea surface temperature (SST) and Fraser River sockeye salmon productivity. Conducting multiple pair-wise comparisons increases the likelihood of spurious correlations...” (pages 5-6)

“Testing multiple pair-wise comparisons also greatly increases the likelihood of finding spurious correlations and wrongfully concluding there is an association between two or more variables when none exists.” (page 6)

“None of the combinations examined by Connors (2011, Table 6) were statistically significant (even before adjustments for multiple comparisons) so the discussion about possible links is simply unwarranted speculation.” (page 8)

My analysis took a multi-model inference approach based on the information-theoretic paradigm. This approach should not be confused with classical null hypothesis testing and associated correction of significance levels for multiple comparisons. Information-theoretic criteria such as the AIC used in my report are not a “test” in any sense, and there are no associated concepts such as test power or p-values or α -levels (Burnham and Anderson 2002). Instead inference was based on the direction, magnitude and uncertainty in the model-averaged parameter estimates, and their predicted influence on sockeye productivity and mortality, not on whether the confidence intervals of the multi-model averaged parameters capture zero. Doing so confuses two very different statistical paradigms. However, the large uncertainty around many of the parameter estimates should not be ignored which is why I state *“the large uncertainty in these estimated effects precludes drawing strong inference”*. (page 22 in Connors 2011)

“While Ruggerone and Nielsen (2004) may have been the motivation for Connors (2011) to only considering pink salmon in his analysis, it is much more reasonable to consider the relationship between sockeye, pink, and chum abundance in the North Pacific and Fraser River sockeye productivity. There is strong evidence that sockeye, pink, and chum salmon have a very high overlap at the trophic level and there is likely to be negative interactions among these species through competition (Johnson and Schindler 2008; Kaeriyama et al. 2004; Ruggerone and Nielsen 2004; Satterfield and Finney 2002).” (page 6)

The motivation for considering the influence of pink salmon abundance on sockeye productivity was (a) evidence for competition between pink and sockeye salmon in the open ocean and (b) because competition with pink salmon in the open ocean has been previously identified as a possible contributing factor to the decline in Fraser River sockeye productivity; there is a negative correlation between aggregate Fraser sockeye productivity and pink salmon abundance in the North Pacific (Peterman et al. 2010). Johnson and Schindler (2008), Kaeriyama et al. (2004), and Satterfield and Finney (2002) all show, using stable isotope analysis, that there is overlap in resource use among

pink, chum and sockeye salmon. However, these studies do not provide any evidence that this overlap in diet leads to reduced growth and/or survival of sockeye. Diet overlap is not evidence of competition, which requires a reduction in the fitness of one party in the presence of another (Krebs 2001). For pink salmon there is evidence of overlap in diet with sockeye salmon as well as for this resulting in reduced sockeye growth and survival (Ruggerone *et al.* 2005; Ruggerone and Nielson 2004; Ruggerone *et al.* 2003).

While it is entirely possible that competition with chum salmon as well as other sockeye salmon in the open ocean may contribute to variation in Fraser River sockeye salmon productivity, without clear evidence of this, and for the reasons outlined above, I chose to focus on pink salmon abundance.

“It does not make sense to consider pink salmon abundance only given the significant trophic overlap for the three species (sockeye, pink, and chum salmon) and particularly when no significant relationship was found between the abundance of pink salmon in the North Pacific and Fraser River sockeye salmon production when they were considered independently.” (page 6)

“The systematic combination of the factors into models to test for significant relationships is also troublesome particularly when no relationship was found between Fraser River sockeye salmon productivity and two of the time series (pink salmon abundance and farmed salmon production) when they were examined independently.” (page 6)

As previously stated, it is not appropriate to interpret the confidence intervals around the multi-model average parameter estimates as not “significant” when they capture zero. Doing so confuses the information-theoretic approach I took with statistical null hypothesis testing.

“While combining two time series in a model was done to capture any potential synergistic relationships, Connors (2011) does not present a compelling case for the comparisons he considered.” (page 7)

As stated on page 10 in Connors (2011) the comparisons considered were based on the variables independently identified by an expert panel for the Pacific Salmon Commission as probable to likely contributors to declines in Fraser River sockeye productivity. From the Pacific Salmon Commission report itself: “An Expert Advisory Panel was created, composed of 11 experienced researchers from Washington and British Columbia who are the authors of this report. As well, about 25 other experts were invited to attend the workshop to make presentations and to critically evaluate data and hypotheses about causes of the decline.” (page 3 in Peterman *et al.* 2010).

Combinations of these variables including interactions were considered because the expert advisory panel concluded: “...multiple hypothesized causal mechanisms are very likely to be operating simultaneously and their effects may be additive, multiplicative (i.e., synergistic), or may tend to offset one another's...” (page 4 in Peterman *et al.* 2010).

“... the implicit assumption in Connors’ (2011) analyses is that the magnitude or level of disease in farmed fish (specifically the ‘high risk’ diseases and/or the number of sea lice on farm fish) is proportional to farmed salmon production.” (page 7)

The hypothesis that the “magnitude or level of disease” on farmed fish is a function of total farmed fish abundance is an interesting one. However, nowhere in my report is it stated or assumed that the level (e.g., prevalence) of disease in farmed fish in general, or high risk diseases and/or sea lice in particular, is proportional to farmed salmon production. Instead I assume that pathogen transmission is a function of farmed salmon production and state: “Any pathogen transmission from farmed to wild salmon is likely to be mediated by both the abundance and spatial and temporal distribution of farmed salmon hosts.” (page 9 in Connors 2011)

I considered the abundance of farmed salmon hosts as a proxy for pathogen exposure because the abundance of infected hosts is a fundamental component of pathogen transmission (e.g., Anderson and May 1981). In its simplest form, if the abundance of uninfected hosts is S , and the abundance of infected hosts is I , it follows that the number of new infected hosts per unit area, per unit of time is βSI , where β is the transmission coefficient (i.e., how efficiently a pathogen is transmitted from an infected host to uninfected host). In other words the infection pressure faced by uninfected individuals is a function of the abundance of infected hosts. This relationship may be linear or non-linear and is likely to be mediated by many other factors. Farmed salmon production may be a relatively poor proxy for pathogen exposure to wild sockeye, but in the absence of data on the distribution of pathogens in farmed salmon that spans the entire sockeye time series it is the only source of information we have.

“Connors (2011) elected not to include data from the record 2010 return of Fraser River sockeye salmon in his analysis for a variety of reasons.” (page 7)

This statement might be misinterpreted to imply that I chose to not include the 2010 data. This is incorrect; I did not “elect” to exclude data from the record 2010 return. This data was not available at the time the report was prepared and I state in the report that the analyses should be repeated once the 2010 data are available (page 22 in Connors 2011).

Dr. Noakes presents the results of analyses that claim to include data up to and including the 2006 brood year (i.e., the 4 year olds that returned to the Fraser River in 2010). Korman (2011) is referenced as the source of the data, however, the Korman report and

supporting files prepared for the Cohen Commission and provided to myself and Dr. Noakes did not contain any information on the 2006 brood years (or the 2005 brood year for that matter) and so it is not clear where the 2005 and 2006 brood year data in Noakes (2011) came from.

An examination of Figure 1b and accompanying text in Noakes (2011) suggests that Dr. Noakes may have mistaken the productivity of Fraser sockeye from the 2003 and 2004 brood years as data on the 2005 and 2006 brood years:

“The negative $\log(R/S)$ for the 2005 brood year (Figure 1B) means that fewer salmon returned in 2009 (the progeny) than spawned in 2005 (the parent stock).” (page 2)

A handwritten signature in black ink, appearing to read 'B. Connors', with a long horizontal stroke extending to the right.

Brendan Connors
School of Resource and Environmental Management
Simon Fraser University

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