

## **Wikiprint Book**

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## 13 Major pitfalls in the application of EwE

EwE can produce misleading predictions about even the direction of impacts of policy proposals. Erroneous predictions usually result from bad estimates or errors of omission for a few key parameters, rather than 'diffuse' effects of uncertainties in all the input information. We warn EwE users to be particularly careful about the following problems that we have seen in various case studies.

### Incorrect assessments of predation impacts for prey that are rare in predator diets

It is easy to overlook a minor diet item in specifying diet composition for some predator. Unfortunately, while that prey type may not be important for the predator, it may represent a very large component of total mortality for the prey type. This is a particularly important problem in representation of mortality factors for juvenile fishes, which usually suffer high predation mortality rates but are often not major components of any particular predator's diet and are notoriously difficult to measure in diet studies (fast digestion rates, highly erratic and usually seasonal occurrence in predator diets).

Another way that 'minor' diet items can come to assume considerable importance is through 'cultivation-depensation' effects (Walters and Kitchell, 2001). Suppose for example that some small predatory fish is kept at low densities by another, larger predator, but the number of predation events needed to exert this control is small compared to the total prey consumption by the larger predator. It would be easy to miss this linkage entirely in formulating the initial Ecopath model. But then suppose the larger predator is fished down, 'releasing' the smaller predator to increase greatly in abundance. The smaller predator may then cause substantial decrease in juvenile survival rates of the larger predator, creating a 'delayed depensation' effect on the larger predator's recruitment. Possibly the larger predator was abundant in the first place at least partly because it was able to exert such control effects on predators/competitors of its own juveniles. Even if such 'perverse' trophic interactions are rare, they are certainly worth worrying about because they imply a risk that overfishing will result in delayed recovery or a persistent low equilibrium abundance for larger predators.

### Trophic mediation effects (indirect trophic effects)

We use the term 'mediation effect' for situations where the predation interaction between two biomass pools is impacted positively or negatively by abundance of a third biomass type. For example, predation rates on juvenile fishes by large piscivores may be much lower in situations where benthic algae, corals, or macroinvertebrates provide cover for the juveniles. Pelagic birds like albatrosses that feed on small fishes may depend on large piscivores to drive these small fishes to the surface where they are accessible to the birds. Some large piscivores may create enough predation risk for others to prevent those others from foraging on some prey types in some habitats.

When a mediation effect is in fact present but is not recognized in the Ecosim model development, it is not unlikely for the model to predict responses that are qualitatively incorrect. For example, fishing down tunas in a pelagic model is likely to result in predicted increases in abundance of forage fishes, and hence to predicted increases in abundance of pelagic birds. But in fact, reducing tuna abundance may have exactly the opposite effect, resulting in bird declines due to the baitfish spending less time at the surface when tuna are less abundant.

### Underestimates of predation vulnerabilities

Predation impacts can be limited in Ecosim by assuming low values of the exchange parameters ( $v$ 's) between behaviourally invulnerable and vulnerable prey 'states'. We call these exchange parameters 'vulnerabilities', and they are estimated by assuming ratios of maximum to Ecopath base estimates of prey mortality rates for each predator-prey linkage. That is, if  $M(o)_{ij} = C(o)_{ij}/B(o)_i$  is the base instantaneous natural mortality rate for prey type  $i$  caused by predator  $j$  base (Ecopath estimate) consumption rate  $C(o)_{ij}$  on prey base biomass  $B(o)_i$ , we assume that the maximum possible rate for very high predator  $j$  abundance would be  $v_{ij}B_i$  where  $v_{ij} = KM(o)_{ij}$ ,  $K > 1$ , represents the rate at which prey become vulnerable to predator  $j$ . By using a  $K$  near 1, i.e.  $v_{ij}$  only a little larger than  $M(o)_{ij}$ , Ecosim users can simulate the 'bottom up' control possibility that changes in predator abundances do not cause much change in prey mortality rates because these rates are limited by physiological or behavioural factors of the prey. The assumption that there are such limitations is supported by scattered observations where total mortality rates ( $Z$ ) were poorly correlated with changes in predator abundances.

Another way of saying that vulnerabilities of prey to predators are very limited is to say that predators are already eating almost every prey that does become vulnerable. If this is indeed true, then there is likely intense exploitation competition among predators for the prey that do become vulnerable, i.e. the number of vulnerable prey seen by each predator is severely limited by the number of other predators competing for those prey. This has potentially large implications for the dynamics of the predator: reductions in predator abundance may be accompanied by large increases in the densities of vulnerable prey available to each remaining predator. In such cases, Ecosim will predict a strong compensatory effect on the predator of reduced predator abundance (strong increases in food consumption rate and growth, or large decreases in predator foraging time with attendant decreases in mortality risk faced by the predator).

So the net effect of assuming low prey vulnerabilities is also to assume that predators should show strong compensatory responses to reduced abundance of conspecifics, which in simulations of increased fishing pressure means strong compensatory responses and hence lower risk of overfishing. An enthusiastic proponent of 'bottom up' control of trophic processes must therefore also be a strong proponent of the idea that it is hard to overfish. This is a very risky assumption.

### Non-additivity in predation rates due to shared foraging arenas

The default assumption in Ecosim is to treat each predation rate linkage as occurring in a unique 'foraging arena' defined by the behaviours of the specific prey and predator. In this formulation, elimination of one predator will result in a decrease in total prey mortality rate equal (at least initially) to the

Ecopath base estimate of that predator's component of the prey total mortality rate. This may be partly compensated by increases in mortality rate due to other predators if the prey increases in abundance and spends more time foraging in response to increased intraspecific competition, but in general this compensatory effect will not completely replace the initial mortality rate reduction.

But suppose this formulation is wrong, and in fact the mortality rate of the prey represents movement of the prey into behavioural or physiological states, (e.g., parasite loads) for which it is vulnerable to predators in general. In this case, removal of any one predator may simply result in the vulnerable prey individuals being taken just as fast, but by other predators. In this case, the total mortality rate of the prey will change much less than predicted by Ecosim.

For example, we recently used Ecosim to evaluate whether control of predatory sharks might help improve juvenile survival rates of monk seals off Hawaii. Sharks appear to be the proximate cause of many juvenile deaths, and it appears that juveniles are exposing themselves to much higher predation risk than normal due to decreases in prey abundance caused by a combination of lobster fishery and ocean productivity ('regime shift') effects. In this case, Ecosim predicts that shark control will at least temporarily improve monk seal juvenile survival rates. But if the real problem is not sharks, but rather that juvenile seals are spending more time exposed to predators in general, the Ecosim prediction about efficacy of control may be grossly optimistic: other predators may just take up the 'slack' after shark removal.

### **Temporal variation in species-specific habitat factors**

Attempting to fit Ecosim models to time series data has revealed some cases where an important species or biomass pool shows dramatic change that cannot be attributed to any known change in trophic relationships or harvesting. Then this dramatic but 'unpredictable' change appears to result in major trophic impact on the rest of the ecosystem. An example would be a planktivorous fish species that is important to piscivores in the system (so piscivores respond strongly to changes in its abundance), which shows high recruitment variation and occasional very strong year classes that support temporary piscivore increases. It is quite possible for such recruitment 'events' to be linked to very localized habitat factors that affect juvenile survival of the planktivore, so that each event results in a persistent cascade of abundance changes throughout the food web. Another example would be loss of specific spawning sites or habitat for one species, which causes it to decline despite favourable trophic conditions in terms of food supply and predation risk.

Ecosim can help us detect possible habitat problems, by revealing prediction 'anomalies' from biomass patterns expected under trophic and fishing effects alone. But there is also a risk of producing 'spurious' good fits to Ecosim, when Ecosim parameters are varied so as to explain as much of the biomass change as possible; that is, Ecosim may explain patterns as trophic/fishing effects that in fact have been due to habitat changes. This is a particular risk in situations where habitat change involves some fairly regular 'regime shifts' or cycles in habitat variables; Ecosim may well attribute cyclic biomass changes in such situations to predator-prey instabilities rather than environmental forcing.