Models with delays

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Interactions are all due to exploitation

In this case, exploitation can describe parasitism or predation. Interactions can be mediated by exploitation in different scenarii: an increase in abundance at time t stimulates the growth of exploiters, which leads to negative effects later on. Conversely, when a predator is able to switch from one prey species to another, an apparent mutualis can emerge. In the case where parasitism is the dominant driver, we can assume the exploitation is species-specific and therefore competition only appears on the intraspecific interaction coefficient. In the case where predation is the most important process, we consider exploiters are generalist and act on all species.

In all cases, we define the interaction coefficient as a saturating function of density at time $t - k$ where k is the delay that can vary depending on the biology of considered exploiters. This saturating function can simply be taken from model II, whether it is for competition and mutualism.

$$
\begin{aligned}
\alpha_{t,ij} &= f(N_{t-k,j}) \\
\alpha_{t,ij} &= \frac{a_C}{h_{ij} + N_{t-k,j}}
\end{aligned}
$$

For all other interactions, for consistency reason, we keep the saturating version of the interactions.

Species-specific exploitation ("virus scenario")

The only interactions that are modified are the intraspecific interactions.

$$
N_{t+h,i} = \frac{\exp(r_i(T)) N_{t,i}}{1 + \sum_{j,j \neq i} \frac{a_C}{h_{ij} + N_{t,j}} N_{t,j} + \frac{a_C}{h_{ii} + N_{t-k,i}} N_{t,i}} - l N_{t,i}
$$

We can assume that k, the delay between the abundance of the competitor, is rather small as virus/fungi have a high growth rate, maybe a high transmission rate, and may react quicly to an increase in abundance.

Generalist exploitation ("zooplankton predation")

In this case, all interactions are modified and predators depend on the total density of species. For each time step, we define $N_{t,T} = \sum_i N_{t,i}$.

$$
N_{t+h,i} = \frac{\exp(r_i(T)) N_{t,i}}{1 + \sum_j \frac{a_C}{h_T + N_{t-k,T}} N_{t,j}} - l N_{t,i}
$$

We can assume a larger k (15 days?) as zooplankton is assumed to grow slower than parasites.

How to define h_T ? First, simplest proposition: $h_T = \sum_{i,j} h_{ij}$.

Loss rate is delayed

In this case, we emphasise on the loss rate. Interactions depend on processes different from exploitation: competition can emerge from scarce nutrients, mutualism, on the other hand, can either emerge from auxotrophy also beneficial to other species. Loss still depends partly on the predation and is therefore subject to the same type of transformation.

$$l_t = \frac{l_{max}}{h_l + N_{t-k,j}}$$

where l_{max} is the maximum loss rate (when N tends to infinity) while h_l is the abundance for which loss rate is half of its maximum value. For the sake of simplicity, we do not use a species-specific loss rate.

In this case,

$$N_{t+h,i} = \frac{\exp(r_i(T))N_{t,i}}{1 + \sum_j \frac{a_C}{h_{ij}+N_{t,j}}N_{t,j}} - l_t N_{t,i}$$

How to defined l_{max} and l_s? Finding a maximum and half-saturation mortality rate could first depend on the way l is determined. Unfortunately, Scranton & Vasseur (2016), from whom we use the value 15kg/(kg*year)=0.04, do not give a reference for their value. This is especially problematic as this value is in the lowest part of the range of values found in the literature, often even 10 times lower.

Parameter values Several studies have studied the primary production loss rate to virus and grazers. A rapid screening gives the following values: [0.02;0.11] in Li *et al.* (2000) in a model of the Strait of Georgia in the absence of zooplankton grazing (the maximum zooplankton grazing in this case is between 0.03 and 0.87, all rates being in day^{-1}); Chen *et al.* (2009) found 88% +/- 38%, sometimes reaching 225% of the total primary production, for a population made of up to 25% of microphytoplankton (the rest being nanophyto and picophyto, the last one representing 72% of the total chlorophyll-a - hard to really know the mortality of diatoms when the population is driven by Prochlorococcus and Synechococcus); in their review, Sarthou *et al.* (2005) give values between 45 and 110% only due to grazing, 0.13 potentially due to cell autolysis (in the absence of nutrients, or because of viral charge).

However, these values are taken either on the current primary production or the standing stock while we want to link mortality to a previous abundance. In the absence of supplementary informatio, a first proxy could be a 50% maximum loss rate (still in the lower part of the range), with h_l so that at very low density, the mortality rate is 0.04 (i.e., $h_l = \frac{l_{max}}{l}$ with l the initial value we had, 4%), $h_l = 12.5$.

References

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