Author Response

Thank your excellent feedback. Below, I will discuss how I would implement all of the suggested comments.

Reviewer Comment

- Section 2.2: Are there any equations for the parameterization schemes in the bioaccumulation of Hg in the model? Model equations are important for understanding the critical processes of the substance. Meanwhile, what are the critical parameters and coefficients for the critical processes in the model? The details of this model are not clarified in the method.
- Significantly, model performance should be evaluated against observations, which are deficient in this study. The literature Amptmeijer et al. (2025) is very important for this study. However, we cannot access to the paper because it is in preparation.

Author Response

My apologies. The publication of a key paper for this model was delayed. These details of this model are referenced in Amptmeijer et al. 2025, which is currently available with https://doi.org/10.5194/egusphere-2025-1486 I believe this is the main concern of the 3 points above. Here, the key equations and validation of both carbon fluxes and bioaccumulation are discussed in more detail. The model described and evaluated in Amptmeijer et al. 2025 is run with and without the bioaccumulation of Hg2+ and with and without the bioaccumulation of MMHg+ in consumers. I addition to this paper now beign available I would discuss in more detail exactly which equations we use.

Suggested edit

Used terminology: bioaccumulation, bioconcentration, biomagnification, and *in vivo* Hg methylation

Bioaccumulation in the marine environment refers to the total increase in pollutants in biota compared to that in the water. This can be quantified in nature by measuring the concentration of pollutants in both water and biota and estimating the difference. This is typically expressed as the bioaccumulation factor, BAF. For example, the bioaccumulation of $MMHg^+$ in organisms i can be calculated based on observations as:

$$\mathrm{BAF}_i^{MMHg^+} = \frac{C_i^{MMHg^+}}{C_w^{MMHg^+}} \tag{1}$$

In which,

$$\begin{aligned}
\mathrm{BAF}_i^{\mathrm{MMHg^+}} &= \text{The bioaccumulation factor of } \mathrm{MMHg^+} \text{ for organism } i\,[\mathrm{L \cdot kg^{-1}}] \\
C_i^{\mathrm{MMHg^+}} &= \text{The concentration of } \mathrm{MMHg^+} \text{ in organism } i\,[\text{ng Hg} \cdot \mathrm{kg^{-1}}] \\
C_w^{\mathrm{MMHg^+}} &= \text{The free concentration of } \mathrm{MMHg^+} \text{ in water}\,[\text{ng Hg} \cdot \mathrm{L^{-1}}]
\end{aligned}$$

Since the BAF can be based on field measurements, it is a commonly used metric to estimate the link between the concentrations of pollutants in seawater and those in biota. In this study, we are interested in separating the bioaccumulation into separate pathways: the direct uptake from the water (bioconcentration) and the increase in pollutants due to trophic interactions (biomagnification).

Bioconcentration, is the increase in the concentration of Hg in biota directly due to uptake from the water. Because the process of bioconcentration relies on the exchange of Hg between the dissolved phase and an organism, it depends on the surface area of the organic material that is in contact with the water. Due to this, small organisms, such as bacteria and phytoplankton, have a greater ability to bioconcentrate Hg (Mason et al., 1996; Pickhardt et al., 2006). However, the bioconcentration process is complicated and recent studies show that the bioconcentration of $MMHg^{+}$ is influenced by cell-dependent factors, such as the thickness of the phycosphere and the availability of transmembrane channels, while this is not the case for Hg^{2+} (Garcia-Arevalo et al., 2024). Bioconcentration is typically defined by the bioconcentration factor (BCF). The BCF for $MMHg^{+}$ in organism i, can for example be calculated as

$$\mathrm{BCF}_i^{Hg} = \frac{BC_i^{Hg}}{C_w^{Hg}} \tag{2}$$

In which,

$\mathrm{BCF}_i^{\mathrm{Hg}}$ = The bioconcentration factor of Hg for organism i $[\mathrm{L} \cdot \mathrm{kg}^{-1}]$
BC_i^{Hg} = The concentration of Hg in organism i due to direct bioconcentration $[\mathrm{ng\ Hg} \cdot \mathrm{kg}^{-1}]$
C_w^{Hg} = The free concentration of Hg in the water $[\mathrm{ng\ Hg} \cdot \mathrm{L}^{-1}]$

Here, Hg could either refer to Hg^{2+} or $MMHg^{+}$. Note that this defines the theoretical BCF. In nature it is typically impossible to directly measure the BCF, as it would be impossible to separate between $MMHg^{+}$ that is taken up directly from the water and $MMHg^{+}$ that is ingested via food. Bioconcentration is the most important step in bioaccumulation and phytoplankton can have a BCF of $MMHg^{+}$ between 2E4 L kg^{-1} and 6.4E6 L kg^{-1} (Gosnell & Mason, 2015).

Biomagnification is when $MMHg^{+}$ reaches higher concentrations at progressively higher trophic levels. The biomagnification factor, the fractional increase in $MMHg^{+}$ with each trophic level, is estimated to be 7.0 ± 4.9 (Harding et al., 2018; Lavoie et al., 2013). This means that in addition to the high increase in $MMHg^{+}$ in phytoplankton, there is a large increase in $MMHg^{+}$ at every consecutive trophic level. Many seafoods consist of high-trophic animals, such as cod, tuna, or marlin, which can have trophic levels between 4 and 4.8 (Nilsen et al., 2008; Sarà & Sarà, 2007). Biomagnification can increase the already high levels of $MMHg^{+}$ in phytoplankton by up to another factor $11.9^{4.8} \approx 145420$. This is typically defined by the biomagnification factor, BMF, which can be calculated assuming steady state for organism i, preying on organism j for $MMHg^{+}$ as:

$$\mathrm{BMF}_{i,j}^{Hg} = \frac{C_i^{Hg}}{C_j^{Hg}} \tag{3}$$

In which,

$\mathrm{BMF}_{ij}^{\mathrm{Hg}}$ = The biomagnification factor for trophic consumption of organism j by i [unitless]
C_j^{Hg} = The concentration of Hg in organism j $[\mathrm{ng\ Hg} \cdot \mathrm{kg}^{-1}]$
C_i^{Hg} = The concentration of Hg in organism i $[\mathrm{ng\ Hg} \cdot \mathrm{kg}^{-1}]$

The biomagnification factor of $MMHg^{+}$ is extremely high, Lavoie et al. (2013) estimates the diet-weighted average BMF for $MMHg^{+}$ as 8.1 ± 7.2 while it is only 4.7 ± 4.7 for Hg^{2+}. This combined with the higher toxicity of $MMHg^{+}$ is the reason why the bioaccumulation of $MMHg^{+}$ is of much higher concern than the bioaccumulation of Hg^{2+}.

In vivo methylation occurs when animals take other forms of Hg and transform it into $MMHg^+$ in organisms. Although the existence of this process has been demonstrated in specific organisms such as cuttlefish, it is poorly understood and only recently gaining attention (Gente et al., 2023). There is no direct evidence of in vivo methylation in the animals that we model, so it is not implemented in this model.
Overall the dominant pathway of the bioaccumulation of $MMHg^+$ is the bioconcentration of $MMHg^+$ in phytoplankton and consequent biomagnification. The important route is quantified by Wu et al. (2019) using a meta-analysis. They find that the concentration of MeHg at the base of the food web predicts 63% of the observed variability in high trophic level fish, while the remaining 37% is controlled by factors such as the dissolved organic matter content and oligotrophy.

Author Response

I would suggest to expand section 2.2 in the methods section (line 184) to explain the exact equations used in this paper to asses bioconcentration and bioaccumulation.

Suggested edit

The implementation of bioaccumulation is discussed and validated in more detail in Amptmeijer et al. (2025), but the core equations are discussesed here as well for clarity. The increase in bioconcentrated pollutant (Hg^{2+} or $MMHg^+$) per day for a functional group is calculated based on the biomass concentration of the group, the uptake rate, and the concentration of the pollutant, while it is reduced with a rate that is the sum of the release rate of the pollutant and the loss of biomass from group g, from both biologicall loss (respiration and mortality) and predation. The change in pollutant p due to bioaccumulation can then be calculated using the following equation:

$$\frac{dC_{g,p}^{BC}}{dt} = b_g \cdot C_p^{env} \cdot r_{g,p}^{bc} - C_{g,p}^{BC} \cdot (r_{g,p}^{rel} + r_g^{bl} + \sum_{z=1}^{n_z} r_{z,g}^{pred}) \tag{4}$$

$C_{g,p}^{BC}$ = Bioconcentrated pollutant p in group g [ng Hg m^{-3}]
b_g = Biomass of functional group g [mgC m^{-3}]
C_p^{env} = Environmental concentration of pollutant p [ng Hg m^{-3}]
$r_{g,p}^{bc}$ = Bioconcentration rate for group g and pollutant p [ng Hg mgC^{-1} d^{-1}]
$r_{g,p}^{rel}$ = Release rate of pollutant p from group g [d^{-1}]
r_g^{bl} = Biological loss rate for group g (mortality, respiration) [d^{-1}]
$r_{z,g}^{pred}$ = Predation rate by predator z on group g [d^{-1}]
n_z = Number of consumer groups feeding on group g
z = Index for consumer groups (predators) of g
t = Time [d]

While the change in pollutant p due to biomagnfication is also dependent on the predation and concentration of pollutants from both bioconcentration and biomagniciation in the prey. Additionally pollutant p is released via the turnover rate rather than the release rate as is the case for bioconcentration, the change in pollutant p due to biomagnification can then be calculated as follows:

$$\frac{dC^{BM}_{g,p}}{dt} = \sum_{s=1}^{n_s}(r^{pred}_{g,s} \cdot a_{s,p} \cdot (C^{BC}_{s,p} + C^{BM}_{s,p})) - C^{BM}_{g,p} \cdot (r^{to}_{p,g} + r^{bl}_{g} \sum_{z=1}^{n_z} r^{pred}_{z,g}) \quad (5)$$

So the total concentration of pollutant P in ng Hg m^{-3} is:

$$C_{(g,p)} = C^{BC}_{(g,p)} + C^{BM}_{(g,p)} \quad (6)$$

Since this tracks the pollutants per volume of water, the total bioaccumulation per biomass in ng Hg mgC^{-1} is then calculated as

$$C^{bg}_{(g,p)} = \frac{C_{(g,p)}}{b_g} \quad (7)$$

This is then converted to the bioaccumulation per dry weight based on an assumed ratio of carbon to dry weight of 0.2 for diatoms, 0.33 for flagellates and cyanobacteria, and 0.5 for zooplankton and fish based on Walve and Larsson (1999) and Sicko-Goad et al. (1984).

Author Response

In addition, I would provide the key take conclusion of the model evaluation of this paper.I would place this after section 2.2 so at line 196 in the manuscript.

Suggested edit

Performance of the GOTM-ECOSMO-MERCY model

The model is generally consistent with observational data and the previously validated 3D ECOSMO E2E model in terms of biomass. Minor exception are that the Chlorophyll-a concentration in the Gotland Deep matches the Northern instead of the Central Baltic Sea, and that the fish biomass in the Gotland Deep is overestimated by 7% compared to Thurow (1997). The model also predicts tHg content and Hg^{2+} and $MMHg^{+}$ levels in phytoplankton, zooplankton and fish 1 accurately, with MMHg+ bioaccumulation corresponding well with trophic interactions. A deviation is seen in the trophic level fish 2, which has a trophic level of 3.5–3.7 in the model, below the expected level for Atlantic Cod (4.0—4.2). Nonetheless, this level remains high making fish 2 representative of a high trophic level animals. The $MMHg^{+}$ bioaccumulation in fish 2 is consistent with the observed bioaccumulation for its trophic level. With the exceptions of the above discused minor exceptions, the model simulates biomass, Hg speciation and bioaccumulation in line with obsersations.

Reviewer Comment

The setup of the two scenarios is a sample. In my opinion, sensitivity analysis for critical parameters or uncertainty analysis of the results is needed. For the results and discussion, the illustrations are concise, and I cannot gain much in-depth discussion and thinking.

Author Response

I agree that the results of the paper are too limited, underexplored, and should be expanded upon in a sensitivity study. Additionally I would propse we plot the seasonality

of the interaction to create more depths in the analyses. I would propse to add to section 3.3 line 238 the following expansion of the results

Suggested edit

Seasonality of the difference in $MMHg^+$ bioaccumulation

The seasonality of the difference in $MMHg^+$ bioaccumulation caused the bioaccumulation of Hg^{2+} and the bioconcentration of $MMHg^+$ in consumers is shown in Fig. 1. For each calendar day (January 1st, January 2nd, etc.), the modeled daily values from each of the last 10 years of the simulations were averaged. The resulting time series represents an annual cycle of average daily conditions. From the producers functional groups only the diatoms are shown as the reaction is not group specific but rather caused by changes in dissolved Hg^{2+} and $MMHg^+$ which means the diffference caused for all phytoplankton groups was the same. This shows that, while the scale depends on the setup, there are interactions that consistently occur. In low trophic levels such as phytoplankotn and microzooplankton the bioaccumulation of Hg^{2+} causes a seasonal respone in the $MMHg^+$ bioaccumulation in phytoplankton which is consequently observable in low trophic level biota such as microzooplankton. While this reduction in $MMHg^+$ would compound into higher trophic levels, its effects in higher trophic level animals dwarves in comparison to the difference caused by incorporating the bioconcentration of $MMHg^+$ in consumers and it does not cause a difference larger than 3% in either fish 1 or fish 2 in any of the setups.

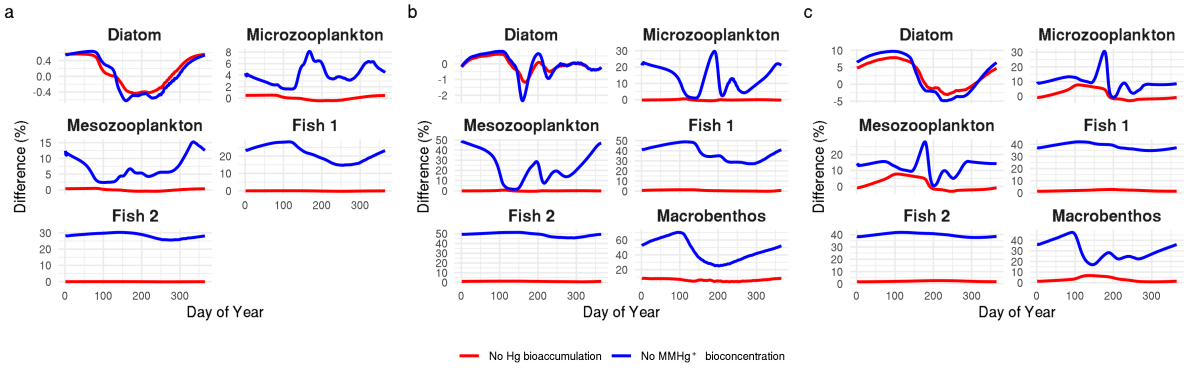

Figure 1: The seasonality of the difference in the bioaccumulation of $MMHg^+$ caused by the bioaccumulation of Hg^{2+} and the bioconcentration of MMHg in consumers for a) the Gotland Deep, b) the Northern North Sea and c) the Southern North Sea. In high trophic level such as fish 1 and fish 2 there is low seasonality and the effect of the bioconcentration of $MMHg^+$ in consumers is high while the effect of the bioaccumulation of Hg^+ is low. In low trophic levels, notably diatoms and microzooplankton there is strong seasonal component. The bioaccumulation of $MMHg^+$ is up to 5% lower in diatoms in the Southern North Sea if the bioaccumulation of Hg^{2+} is modeled in late summer when biomass is high. But the bioaccumulation of Hg^{2+} does not lead to a notable ($> 5\%$) difference at any moment in fish.

Author Response

I would propose that we do a sensitivity study for the first hypothesis, that bioconcentration in consumers is a significant driver of MeHg bioconcentration, by testing and visualizing the sensitivity of this process to both the bioconcentration rate of consumers

and of producers. I would run the model with 10 different bioconcentration factors between 0 (no biocentration) and 2.0 (double the estimated bioconcentration rate), for both consumers' and producers' bioconcentration. This would result in 20 different scenarios with varying bioconcentration rates to asses the sensitivity of importance of consumer level bioconcentration to the bioconcentration rates. I would suggest to add the following segment to form section 2.4 at line 209 in the methods as follows, in order to introduce the sensitivity study:

Suggested edit

In order to further investigate how bioconcentration in consumers affects bioaccumulation of $MMHg^+$, we performed a sensitivity analysis on the key drivers: the bioconcentration rate of consumers and the bioaccumulation rate of producers. To this extent, two sensitivity studies are performed. In the first sensitivity study, the bioconcentration rate in all consumers is multiplied by a scaling factor that is between 0.2 and 2.0 with 0.2 intervals. The effect of this on the bioaccumulation in fish 2 for the Gotland Deep is shown to visualize the impact. Then the relative contribution of bioconcentration in consumers on the bioaccumulation of $MMHg^+$ in fish 2 is shown for all three setups. For the second sensitivity study, the same approach is used but the bioconcentration rate of producers is multiplied by a scaling factor.

Author Response

And then present the results of this study as follow, by uncerting the part below at line 257 at section 3.6:

Suggested edit

Sensitivy of the consumer bioconcentration rate

The results of the first sensitivity study, in which the bioconcentration rate of consumers is altered, are shown in Fig. 2. Figure 2a illustrates that the $MMHg^+$ contribution from bioconcentration in consumers is linearly related to the consumer bioconcentration rate scaling factor. Thus, altering the bioconcentration rate by half or double yields the same relative effect on fish 2's $MMHg^+$ content from direct bioconcentration. Based on Table 1, we can see that in the Gotland Deep, the difference between the simulation with and without consumer bioconcentration is 0.0183 ng Hg mgC^{-1}. This means that picking a bioconcentration double the real rate would result in a 0.0183 ng Hg mgC^{-1} overestimation of $MMHg^+$ bioaccumulation in fish 2, while selecting bioconcentration rates half the true values would result in a reduction of 0.00915 ng Hg mgC^{-1}. However, the relative contribution of bioconcentration to total $MMHg^+$ bioaccumulation follows a non-linear pattern, as shown in Fig. 2b. This non-linearity occurs because the total $MMHg^+$ in fish 2 is influenced by both bioconcentration in consumers and bioconcentration in producers. When the consumer bioconcentration scaling factor is 0, bioconcentration in consumers makes no contribution to fish 2's $MMHg^+$ levels. Conversely, this contribution can never reach 100% because bioconcentration in producers and consequent biomagnification from lower trophic levels always contributes to the total $MMHg^+$ burden in fish. In the same way as in the results shown in Table 1, the relative importance of bioconcentration is consequently highest in the Northern North Sea, followed by the Southern North Sea and lowest in the Gotland Deep.

Sensitivy of the producer bioconcentration rate

The results of the second sensitivity study are shown in Fig. 3. Here, rather than the consumer bioconcentration rate, the producers' bioconcentration rates are multiplied by a scaling factor. Again, the effect of this scaling on the bioaccumulation in all trophic levels is visualised in Fig. 3a, and the effect of this scaling on the relative importance of consumer bioconcentration on $MMHg^{+}$ bioaccumulation is shown in Fig. 3b. If the bioconcentration scaling factor is 0, there is still $MMHg^{+}$ bioaccumulation in fish 2, both from direct bioconcentration and from bioconcentration in consumers and consequent biomagnification. The increase in fish 2 $MMHg^{+}$ per step of 0.2 in the scaling factor is 0.0083 ± 0.00030 ng Hg mg^{-1}. The relative contribution of consumer bioconcentration on $MMHg^{+}$ bioaccumulation is shown in Fig. 3b. An important note here is that while we scaled the bioconcentration factor of producers and consumers, $MMHg^{+}$ can also be bioaccumulated via the partitioning to dissolved organic matter (DOM) detritus and consequent biomagnification. This is especially important in the Northern North Sea. In the seasonally stratified water column, macrobenthos cannot feed directly off the phytoplankton bloom; thus, the dying and sinking of particles is an important flux that is consumed by the benthos. Benthos, in turn, is an important food source for fish 2. So scaling the producer bioconcentration rate has less effect in the Northern North Sea. In the Gotland Deep, the opposite is true; because the deep water is anoxic, there is no macrobenthos in the model. This means that the entire ecosystem is pelagic and detritus is less important than direct consumption of the phytoplankton bloom.

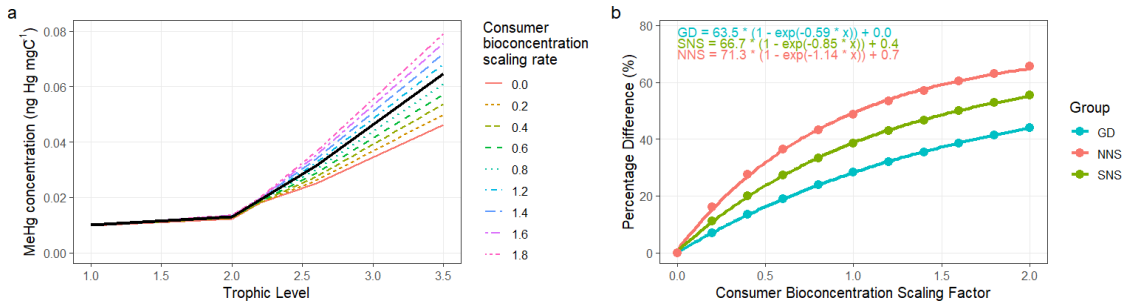

Figure 2: a) show the effect of the bioaccumulation of $MMHg^{+}$ per trophic level in the Gotland Deep. This shows an increase 0.0036 ± 0.00010 ng Hg mg^{-1} in fish 2 $MMHg^{+}$ bioaccumulation for every 0.2 step increase in the consumer bioconcentration scacling factor. 2b) shows the percentage difference due to bioaccumulation with different consumer bioconcentration scaling factors in all setups. GD refers to the Gotland Deep, NNS to the Northern North Sea and SNS to the Southern North Sea. When the consumers bioconcentration scaling factor is 0, the percentage difference due to bioconcentration is 0 %. As this increase the percentage increases. The relationship between the consumers bioconcentration factor and the percentage difference due to consumer bioconcentration is plotted assuming an saturating exponential relationship.

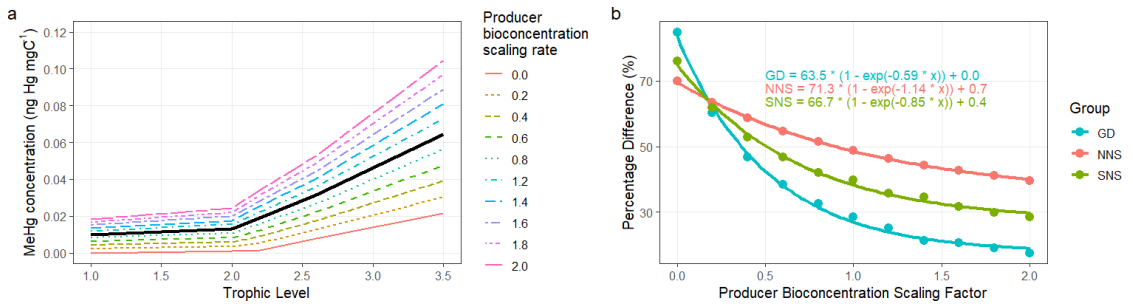

Figure 3: a) illustrates the influence of scaling the producers bioconcentration rate of $MMHg^+$ on the $MMHg^+$ bioaccumulation at each trophic level in the Gotland Deep. This shows an increase of 0.0084 ± 0.00032 ng Hg mg^{-1} in fish 2 $MMHg^+$ with every 0.2 increase in the producers scaling factor. 3b) highlights the difference due to consumer bioconcentration with different primary producers scalings factors across all setups. The relationship between producer bioconcentration scaling factor and the percentage difference in $MMHg^+$ bioaccumulation in fish 2 due to consumer bioconcentration is plotted using an expenontial decay function. This shows that in all cases the percentage difference is high when the producer bioconcentration factor is 0, and that this percentages decreases with an increasing scaling factor.

Author Response

Evaluating the total uncertainty in the conclusion would be beneficial. However, a challenge is that fully assessing this uncertainty is not feasible because it depends on factors like the size distribution of phyto- and zooplankton and the food web structure, which vary regionally and temporally, and are beyond the scope of the North and Baltic Sea model used in this study. To adresss this I suggest I estimate the upper and lower bounds of the importance of the bioconcentration of $MMHg^+$ based on field observations. to do this I would suggest to add the following part to the end of the Model Limitations section, on line 302.

Suggested edit

The results of our model represent just one possible outcome based on a regional setup representing the North and Baltic Seas, and the importance of bioconcentration can vary greatly depending on the bioconcentration factors of all species in the trophic chain. We can asses expected range of importance of consumer level bioconcentration by developing theoretical maximum and minimum values based on observational studies. We can estimate that direct bioconcentration in zooplankton may account for up to 50%, based on Lee and Fisher (2017), and similarly for mid-trophic level fish, based on Wang and Wong (2003).

We can use this to estimate the maximum expected contribution of consumer level bioconcntration on bioaccumulation by making two assumptions: (1) bioconcentration in both copepods and fish lies between 0 and 50% and is equal across all trophic levels, and (2) the food chain is linear, meaning that trophic level 3 feeds exclusively on trophic level 2, which feeds exclusively on trophic level 1. Under these assumptions, we can estimate the percentage of $MMHg^+$ in the diet of a given trophic level that originated from bioconcentration in primary producers as:

$$\mathrm{PBC}\%_n = (1 - \mathrm{BC})^{n-1} \times 100\% \tag{8}$$

where:

- PBC%$_n$ is the percentage of $MMHg^+$ in the diet of trophic level n that originates from bioconcentration at the primary producer level,
- BC is the fraction (0–1) of $MMHg^+$ at each trophic level originating from bioconcentration.

Although this is a simplification, it illustrates that a high bioconcentration estimate of 50% results in only 12.5% of $MMHg^+$ in the diet of a trophic level 4 fish originating from bioconcentration in primary producers, meaning that 87.5% originates from consumer-level processes. Even a low estimate of 10% still results in 27.1% of $MMHg^+$ in the diet of the same high-trophic-level fish originating from consumer-level bioconcentration.
The degree to which this interaction contributes to overall bioaccumulation depends on numerous additional factors that are not yet fully understood, including the size distribution of phytoplankton at the base of the food web, the trophic structure, consumer metabolic and respiration rates, and the assimilation efficiency of $MMHg^+$ from the diet. This complexity makes it difficult, if not impossible, to provide a definitive estimate of the importance of consumer-level bioconcentration and the uncertainity of the interaction. However, based on the bioconcentration rates provided in the current literature, we conclude that this process plays a key role the bioaccumulation of $MMHg^+$ in higher trophic levels.

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