1 Answers to revieuwer 1

Reviewer Comment

"Line 40-50. The authors mentioned the "bioconcentration", "bioaccumulation" and "biomagnification". For instance, the statement "bioconcentration is the most important step in bioaccumulation" lacks a clear distinction from biomagnification, risking confusion for readers unfamiliar with the terminology. What's the differences between bioaccumulation and biomagnification? In the subsequent manuscript, these two words were also used in confusion. The authors should explain and clarify them. "

Author Response

I agree that this distinction should be clarified better, especially since it is essential for understanding the paper. I would suggest that I expand the introduction by roughly half a page in which I explain in detail the differences between bioaccumulation, biomagnification and bioconcentration and give the equations that are commonly used for the bioaccumulation, biomagnification and bioconcentration factors. I would suggest to add the replace the section of line 40-62 by the updated text below:

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 Hg in organisms i can be calculated based on observations as:

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

In which,

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

Where Hg could either refer to Hg^{2+} or $MMHg^{+}$. 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

Than 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:

$$\frac{dC_{g,p}^{BM}}{dt} = \sum_{s=1}^{n_s} (r_{g,s}^{pred} \cdot a_{s,p} \cdot (C_{s,p}^{BC} + C_{s,p}^{BM})) - C_{g,p}^{BM} \cdot (r_{p,g}^{to} + r_g^{bl} \sum_{z=1}^{n_z} r_{z,g}^{pred}) \tag{5}$$

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

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

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

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

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.

Reviewer Comment

"Line 79-85. The second hypothesis is confused. This hypothesis lacks evidence and references, making it appears speculative."

Author Response

I agree that the second hypothesis lacked references. It is mostly supported by the work of Wu et al., 2019, so I would rewrite that as:

Suggested edit

The majority of $MMHg^+$ present in higher trophic levels is derived from their dietary intake (Lavoie et al., 2013). It is often assumed that $MMHg^+$ bioconcentration is not crucial for its bioaccumulation at higher trophic levels based on results such as those presented by Schartup et al., 2018, therefore it is, for example omitted from several Hg cycling and bioaccumulation models such as the model presentd by Rosati et al., 2022, or not incoroporated into higher trophic level as is the case in the model presented by Li et al., 2024. However, this assumption overlooks that bioconcentration occurs at all levels of the trophic hierarchy. For example, if microzooplankton and mesozooplankton acquire 5% of $MMHg^+$ through bioconcentration, mesozooplankton will have 5% less $MMHg^+$ from its diet, which consists of microzooplankton, and another 5% less due to absence of bioconcentration, leading to a total reduction of 10%. **The second hypothesis proposed here is that $MMHg^+$ bioconcentration in consumers significantly elevates $MMHg^+$ levels at higher trophic levels.** This concept has been previously suggested and studied by Wu et al., 2019. Their research found that the BCF in fish spans 3 to 7 orders of magnitude and greatly differs across studied sites, yet they did find a strong correlation between BCF and $MMHg^+$ concentration in fish. Thus, we are not the first to suggest that direct water uptake is a significant factor in $MMHg^+$ bioaccumulation, rather, this study extends this understanding by quantifying the role of bioconcentration in all consumers on $MMHg^+$ bioaccumulation in fish at higher trophic levels.

Author Response

Additionally, I think the first hypothesis can also be better phrased. It was added as by itself it is not a negative result that I believe is interesting enough to publish, but it could supplement this study. I would howeever propose 2 changes. I would suggest to refrase its introduction and to expand the results related to this as discussed below:

I would refrase the first hypothesis (line 72) as:

Suggested edit

While $MMHg^+$ is more concerning than Hg^{2+} at higher trophic levels, Hg^{2+} can form up to 98% of the bioaccumulated Hg in phytoplankton (Pickhardt & Fisher, 2007). This results in a large removal of Hg^{2+} during the phytoplankton bloom period (Soerensen et al., 2016). However it is demonstrated by Amptmeijer et al., 2025, which analyses the feedback of bioaccumulation on Hg cycling, that there is no change in average tHg and aqueous Hg caused by bioaccumulation, but that there is a seasonal variation in the aquatic tHg content due to bioaccumulation. This means that even if the average concentrations of tHg are not altered by bioaccumulation, there may still be an effect of Hg^{2+} bioaccumulation on $MMHg^+$ bioaccumulation as during the phytoplankton bloom tHg is reduced which could lead to a reduction of available $MMHg^+$ for bioaccumulation. It could be theorized that as the ecosystem reduces tHg during the phytoplankton bloom, it would reduce dissolved $MMHg^+$, as this is in active equilibrium with other Hg species and therefore reduce the availability of $MMHg^+$ for bioaccumulation. Based on this we propose our first hypothesis that **the bioaccumulation of Hg^{2+} can lower the bioaccumulation of $MMHg^+$ by removing Hg^{2+}, which in turn cannot be methylated and accumulated as $MMHg^+$.**

Author Response

As the first hypothesis is focussed mostly on the theory that seasonal bioaccumulation of Hg^{2+} could influence $MMHg^+$ bioaccumulation I suggest that we also expand the results by looking if there is no seasonal effect. For this I would suggest to add the following section before the hypothesis evaluation (line 249 in the preprint.)

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.

Author Response

Then I would also suggest to update the hypotheses evaluation (section 3.3 line 238) in the result section as follows

Suggested edit

Evaluation hypotheses 1; The effect of Hg^{2+} bioaccumulation on $MMHg^{+}$ bioaccumulation

Based on the results of the statistical analysis shown in Table 2, we can see that there is no significant difference (p = 0.99) caused by Hg^{2+} bioaccumulation on $MMHg^{+}$ bioaccumulation. Based on the Bayesian t-test, we estimate that the change is 1/0.35 = 2.86 times greater than the data, meaning there is a difference. We do note that the seasonal changes in the total Hg concentration due to bioaccumulation change the bioaccumulation of $MMHg^{+}$ at the base of the food web, and we can see this change in phytoplankton and low trophic level consumers, but it does not cause a notable (> 5%) change in $MMHg^{+}$ bioaccumulation in fish. Based on these results, we conclude that Hg^{2+} bioaccumulation does not play a major direct role in the bioaccumulation of $MMHg^{+}$. However, it should be noted that the bioaccumulation of Hg^{2+} can still play a role in the $MMHg^{+}$ content in biota by in vivo methylation. However, there is no data suggesting that this is a major pathway, so based on the current state of knowledge of $MMHg^{+}$ bioaccumulation, we conclude that the first hypothesis is incorrect and Hg^{2+} does not play a role in the bioaccumulation of $MMHg^{+}$ in coastal food webs.

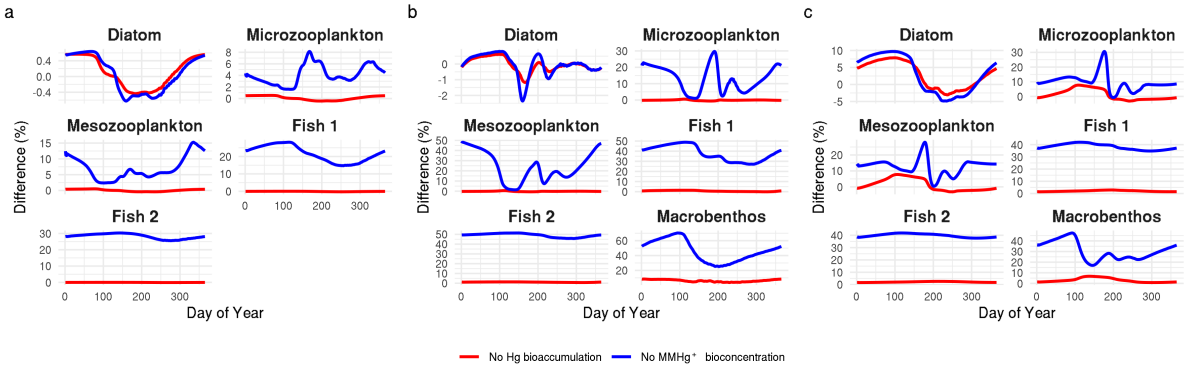

Figure 1: The seasonality of the difference in the bioaccumulation of $MMHg^{+}$ caused by the bioaccumulation of Hg^{2+} and the bioconcentration of MMHg in consumers for a) the Gotland Deep, b) the Northern North Sea and c) the Southern North Sea. In high trophic level such as fish 1 and fish 2 there 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.

Reviewer Comment

"Line 140: As mentioned, "Quantifying the importance of the bioaccumulation of Hg2+ and bioconcentration of MMHg+ in consumers on MMHg+ bioaccumulation". The authors should clarify whether prior models ignored multi-trophic bioconcentration, and then highlight the novelty of this work."

Author Response

Thank you for your excelent suggestions, I would suggest expanding the introduction by adding the following section to replace to short discussion on previous models at line 62. Then it would be more thoroughly revieuwed when the readers arrives at line 140.

Suggested edit

Current models

Multiple models have been developed to explain $MMHg^+$ bioaccumulation in marine ecosystems. Key examples include trophic transfer (Schartup et al., 2018), base-level accumulation (Zhang et al., 2020), planktonic bioaccumulation in the Mediterranean Sea (Rosati et al., 2022), MeHg dynamics on the Beaufort Shelf (Li et al., 2022), and speciation plus accumulation in the North and Baltic Seas (Bieser et al., 2023).
In all previous models, the bioconcentration of $MMHg^+$ is included because it is an essential driver. These models, however, do not include higher trophic level animals such as fish. It is concluded in Schartup et al., 2018 that the bioconcentration of $MMHg^+$ in zooplankton is not a major contributor and contributes less than 15% of total MeHg bioaccumulation. Consequently, in later models such as presented by Rosati et al., 2022 this interaction is not included because their model focuses on the base of the food web. The study performed by Li et al., 2022 includes the process of bioconcentration for invertebrates, but it is not included for vertebrates. This means that our model would be the first model to include bioconcentration at every trophic level.
The bioaccumulation of Hg^{2+} is much less studied and not incorporated in any of the above-mentioned models. This is because Hg^{2+} is much less toxic than $MMHg^+$ and therefore comparably understudied. While data is limited, this raises the speculative question if the link between the bioaccumulation Hg^{2+} and $MMHg^+$ is not underestimated as Hg^{2+} and $MMHg^+$ are in active equilibrium in the water.
The ECOSMO-MERCY coupled system, which is used by Bieser et al., 2023 and Amptmeijer et al., 2025 is the only coupled model that models the bioaccumulation of Hg^{2+} and $MMHg^+$ at higher trophic levels such as fish, while incorporating bioconcentration at every trophic level.

Reviewer Comment

"Line 226 Table 1. How to calculate the bioaccumulation and bioconcentration difference (%) ?

Author Response

Thank you for the noticing. It is cacluated as scenario/base case * 100%, but I will add this to the paper as this specification is indeed essential. I would suggest to add to section 3.2 to following clarification:

Suggested edit

The % bioconcentrated is calculated as Bioconcentrated (%) = $\frac{\text{Bioconcentrated}}{\text{Bioaccumulated}}$ and the difference (%) is calculated as Difference (%) = $\frac{\text{Scenario}}{\text{Default}}$. The values in red in the difference category indicate when the scenario causes a change larger than 10%. The

values are based on the last 10 years of the simulation and the top 20m of the water column, to create an average value that we can compare between the setups.

Author Response

And update the caption of Table 1 to also include this as follows:

Suggested edit

The bioaccumulated $MMHg^+$, the percentage of bioaccumulated $MMHg^+$ that originates from bioconcentraton, and the bioaccumulated $MMHg^+$ in the scenario without bioaccumulation of Hg^{2+} and the bioconcentration of $MMHg^+$ in consumers and the difference to the default scenario. The % bioconcentrated is calculated as Bioconcentrated (%) = $\frac{\text{Bioconcentrated}}{\text{Bioaccumulated}}$ and the difference (%) is calculated as Difference (%) = $\frac{\text{Scenario}}{\text{Default}}$.

Reviewer Comment

Line 316 "15% per trophic level". It is recommended to supplement sensitivity analyses.

Author Response

Thank you for this valuable suggestion. I would suggest to edd the following section to the methods section to introduce the sensitivity analyses:

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

The results of this senstitivity study I would present in the results as follows:

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.

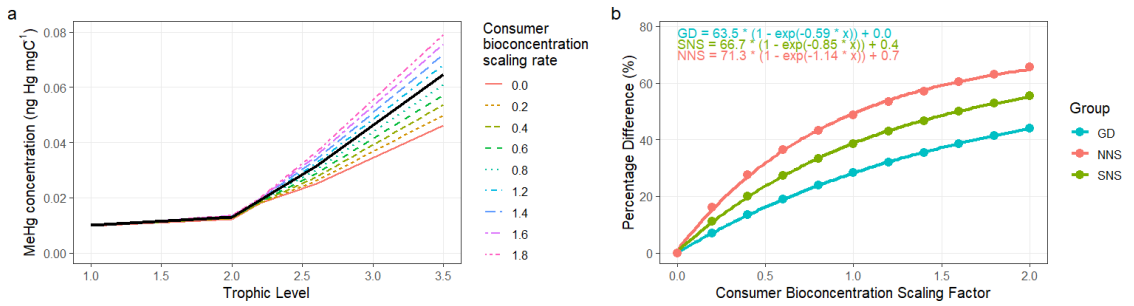

Figure 2: a) show the effect of the bioaccumulation of $MMHg^+$ per trophic level in the Gotland Deep. This shows an increase 0.0036 ± 0.00010 ng Hg mg^{-1} in fish 2 $MMHg^+$ bioaccumulation for every 0.2 step increase in the consumer bioconcentration scacling factor. 2b) shows the percentage difference 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.

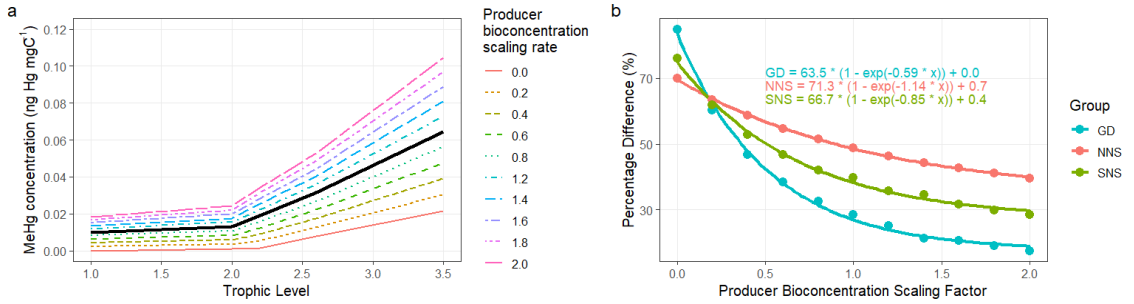

Figure 3: a) illustrates the influence of scaling the producers bioconcentration rate of $MMHg^+$ on the $MMHg^+$ bioaccumulation at each trophic level in the Gotland Deep. This shows an increase of 0.0084 ± 0.00032 ng Hg mg^{-1} in fish 2 $MMHg^+$ with every 0.2 increase in the producers scaling factor. 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

An additionaal component of the overal uncertainity is the uncertainty of the rest of the system, for example phytoplankton size distribution. Completley assesing this uncertainty is not really possible as our North and Baltic Seas setups cannot represent conditions that might be present in other areas. To adresss this I suggest I suggest I estimate the upper and lower bounds of the importance of the bioconcentration of $MMHg^+$ based on field observations and supplement the sensitivity analyses provided with this more general discussion arround the expected upper and lower bounds that are expected. 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:

- $\mathrm{PBC}\%_n$ 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.