

1 **Microplastic as a Vector for Chemicals in the Aquatic**
2 **Environment. Critical Review and Model-Supported Re-**
3 **interpretation of Empirical Studies.**

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29 **Abstract**

30 The hypothesis that ‘microplastic will transfer hazardous hydrophobic organic chemicals
31 (HOC) to marine animals’ has been central to the perceived hazard and risk of plastic in the
32 marine environment. The hypothesis is often cited and has gained momentum, turning it into
33 paradigm status. We provide a critical evaluation of the scientific literature regarding this
34 hypothesis. Using new calculations based on published studies, we explain the sometimes
35 contrasting views and unify them in one interpretive framework. One explanation for the
36 contrasting views among studies is that they test different hypotheses. When reframed in the
37 context of the above hypothesis, the available data become consistent. **We show that HOC**
38 **microplastic-water partitioning can be assumed to be at equilibrium for most microplastic**
39 **residing in the oceans.** We calculate the fraction of total HOC sorbed by plastics to be small
40 compared to that sorbed by other media in the ocean. We further demonstrate consistency
41 among (a) measured HOC transfer from microplastic to organisms in the laboratory, (b)
42 measured HOC desorption rates for polymers in artificial gut fluids (c) simulations by plastic-
43 inclusive bioaccumulation models and (d) HOC desorption rates for polymers inferred from
44 first principles. We conclude that overall the flux of HOCs bioaccumulated from natural prey
45 overwhelms the flux from ingested microplastic for most habitats, which implies that
46 microplastic ingestion is not likely to increase the exposure to and thus risks of HOCs in the
47 marine environment.

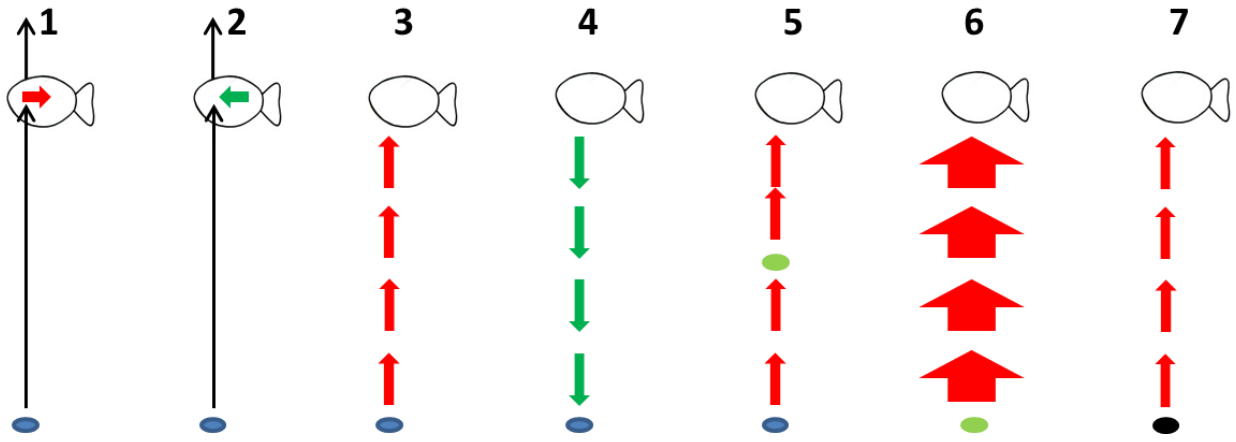
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49 Key words – bioaccumulation, microplastic, marine animals, marine debris, hydrophobic
50 organic chemicals

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52 TOC Art:

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56 **Introduction**

57 The contamination of the environment with plastic is considered highly undesirable for ethical
58 and esthetical reasons and is generally considered to be a major threat for the health of aquatic
59 ecosystems.¹ It has been demonstrated that numerous species ingest plastic debris or become
60 entangled by it.^{2,3,4,5} The physical damage to wildlife caused by larger forms of plastic (> 5
61 mm in size) is also well documented, although clear indications of harm at the level of
62 populations or communities have not been convincingly demonstrated. Still, the impact of
63 micro-plastics (< 5 mm) remains under investigation.^{6,7,8}

64 Because the research examining microplastics is still in its infancy, studies measuring their
65 fate and effects use widely different methods and approaches. As such, they provide
66 fragmentary information and often use high microplastic concentrations and/or other non-
67 environmentally relevant conditions to test for effects. Although this has resulted in several
68 recent publications demonstrating new evidence of adverse effects, the body of evidence
69 emphasises the need to harmonise methodologies and design tests using environmentally
70 relevant conditions.⁷ Addressing environmentally relevant conditions is a key prerequisite for
71 a scientifically sound assessment of the hazard and risks of microplastics in the environment,
72 which is key to really understand the possible risks of microplastics at the level of populations
73 or communities.

74 One potential hazard stems from the fact that plastic particles contain or efficiently absorb
75 hydrophobic organic chemicals (HOCs), which includes non-covalently bound additive
76 ingredients.^{9,10,11} This has sparked hypotheses (1) on how microplastic contributes to the
77 transport of HOCs, and (2) whether the ingestion of microplastic contributes to the
78 bioaccumulation of HOCs by marine animals.^{12,13,14,15,16,17,18} Both ideas have led to
79 considerable attention and debate. The ability of polymers to act as a source of HOCs to

80 organisms has been recognized for a long time as it is the essence of the passive dosing
81 approaches used in ecotoxicology.^{19,20} The past years this has been reconfirmed in the context
82 of marine plastic debris by a series of laboratory studies that used microplastic as the sole
83 source of HOCs.^{21,22,23} The studies in general support the above hypotheses, i.e. that
84 microplastics are a vector and source of HOCs to marine organisms.

85 However, the debate has formed from questions regarding whether plastic is a substantial
86 source of these toxic chemicals to aquatic organisms relative to other sources in the
87 environment. After all, given the low abundance of plastic particles relative to other media
88 present in the oceans, exposure to these HOCs via plastic is probably of less importance than
89 via natural pathways.^{7,24,25,26,27,28} Some recent papers argue that plastic is (potentially) an
90 important exposure route because the affinity of HOCs for plastic is high.^{12,21} This is likely
91 overruled when considering that the importance of plastic as a carrier is also dependent on the
92 abundance of plastic compared to that of other carrier media such as water, suspended organic
93 particulates or natural diet and prey items many of which have partition coefficients that are
94 similar to that of plastic.²⁹ Laboratory dietary exposures that demonstrate that plastic debris
95 can be a vector of HOCs to marine organisms have not truly tested hypotheses regarding the
96 relative importance of microplastics in comparison to other sources occurring in real
97 environments. Several recent quantitative assessments, modelling studies and reports have
98 concluded that the contribution of plastic to chemical fate and transport of HOCs in the
99 oceans, and to bioaccumulation of these chemicals by marine organisms is probably
100 small.^{24,25,26,28,30} This perceived dichotomy in the discussions of various published studies is
101 confusing and hampers progress towards scientific consensus regarding the actual risks of
102 microplastics. In turn, this may hamper policy development and the prioritization of research
103 needs and remediation measures.

104 The aim of this paper is to critically review and synthesize the literature concerning the role of
105 plastic as a carrier/vector of chemicals. This includes reviewing empirical and modelling
106 studies, as well as incorporating the lessons learned from the behaviour of sorbents other than
107 plastic (e.g. organic matter, black carbon, activated carbon, carbon nanomaterials, and passive
108 dosing or - sampling polymer materials) that share the same principles with respect to the
109 binding of HOCs. We also apply models to reinterpret laboratory studies in an attempt to
110 unify various study results into a unified interpretation framework.

111 To reach our objective, we first discuss the state of (non-)equilibrium of the binding of HOCs
112 to microplastics in the marine environment. Plastic particles can only constitute an ongoing
113 source of HOCs to the water or to aquatic organisms if the HOC concentration in these
114 particles is sufficiently high (i.e. higher than equilibrium) compared to that in the water or in
115 the organisms. This calls for a comparison of HOC (de-)sorption half-lives and of the
116 residence times and age of plastic particles in the oceans. As far as we know, no such
117 assessment has been performed yet. Second, we discuss the average distribution of HOCs
118 across media in the ocean, a distribution that can be assessed by accounting for (1) the relative
119 abundances of these media, and (2) the relative affinities of HOCs for these media.

120 Assessment of this distribution under environmentally realistic conditions is crucial in order
121 to understand the relative importance of plastic (compared to other media) in the transport of
122 HOCs or in the bioaccumulation of HOCs by marine organisms. A few earlier studies have
123 provided a similar distribution analysis,^{24,30} however, without taking into account all known
124 media, such as dissolved organic carbon, colloidal carbon, black carbon, detritus and
125 plankton. Third, we discuss recent studies that document transfer of HOCs from plastic to
126 marine organisms or vice versa, and provide novel model calibrations using data from these
127 studies. This provides a mechanistic interpretation of the results of these previous empirical
128 studies, unifies empirical and model-based approaches regarding the same research questions,

129 and allows for extrapolations to natural conditions in case these empirical studies were not
130 fully mimicking natural conditions. Furthermore, past literature is evaluated for
131 environmental relevance, i.e. extent of using realistic concentration ranges and completeness
132 with respect to covering processes that are known to occur in the field. Finally, we discuss the
133 implications of our analysis for risk assessment and suggest priorities for future research.

134 **The state of equilibrium of HOC partitioning in the oceans**

135 **Equilibrium sorption of HOCs to plastics.** HOCs are subject to partitioning across
136 environmental media such as water, sediment, biota, air, and since the 1950's: plastic.³¹ The
137 equilibrium partitioning coefficient for sorption to plastics K_{PL} [L/kg], is defined as:^{12,32}

$$138 \quad K_{PL} = C_{PL}/C_W \quad (1)$$

139 where C_{PL} [$\mu\text{g}/\text{kg}$] and C_W [$\mu\text{g}/\text{L}$] are the concentrations in plastic and water, respectively. Eq
140 1 is important to address the question whether HOCs are sorbed to microplastic or are
141 released by microplastic, because the spontaneous transfer of HOCs always occurs in such a
142 direction that the actual concentration ratio (C_{PL}/C_W) approaches the value of K_{PL} .¹⁵ For
143 instance, if $C_{PL}/C_W > K_{PL}$, then desorption from the plastic to water takes place and vice versa.
144 The time needed to reach equilibrium depends on the molecular properties of the HOC, the
145 properties of the seawater and the microplastic, **as well as on the volumes of these**
146 **compartments**. The kinetics of sorption to microplastic is beyond the scope of this review, but
147 it is obvious that sorption equilibrium may exist for microplastics that reside in the ocean for
148 already a long time, whereas equilibrium may not exist for microplastics that were released
149 recently. This means that two aspects need to be compared (1) the age distribution of
150 microplastics currently present in the oceans, and (2) the sorption equilibration times of
151 representative HOCs.

152 **Estimating the age distribution of microplastics in the oceans.** Annual world production
153 data for plastics are well known and show a gradual increase from 1.7 million tonnes in 1950
154 to about 299 million tonnes per year in 2013 (Figure S1, data from³¹). The curve can be
155 smoothed using a 2nd order polynomial (Figure S1), which accurately captures the trend but
156 averages some small fluctuations caused by stagnations in the world economy in the 1970's,
157 1980's and recently in 2007. It is commonly assumed that a more or less constant fraction of
158 the world plastic production ends up in the oceans.³³ The few million tonnes of plastic emitted
159 in the first years of production have an 'environmental age' of about 60 years, whereas the
160 production of 299 million tonnes in 2014 has by definition an age of less than a year. By
161 combining the annually emitted volumes per year with the age of these yearly volumes (i.e.
162 present year minus year of emission) an age distribution for the cumulative quantity of
163 emitted plastic can be calculated (Figure 1). Because a more or less constant fraction of the
164 yearly world production ends up in the oceans, Figure 1 also represents the expected age
165 distribution for **all plastic** in the oceans. The calculation (for 2015) shows that about 50% of
166 the plastic has been present in the oceans for more than 13 years, whereas 80% and 90% of all
167 the plastic is older than 4 and 2 year, respectively (Figure 1). If we use European production
168 data³¹ the age distribution is similar, however, it shows that 50% and 90% of the produced
169 plastics has been present in the European seas for more than 17 years and 3 years, respectively
170 (Figure 1). In reality, the oceans do not represent one uniform compartment.³⁴ However,
171 mixing within areas or in gyres can be considered more homogeneous. Furthermore, sources
172 and types of plastics do not substantially differ across the globe. Microplastics are considered
173 ubiquitous global contaminants, whereas transport and mixing causes the spreading of
174 microplastics in the oceans, with contamination of even very remote areas as a
175 result.^{34,35,36,37,38} Coastal areas may contain relatively 'young' plastic particles, yet these areas
176 also receive aged plastics from remote areas. Indeed beached plastic has been shown to also

177 come from remote sources. Remote areas like the Arctic³⁵ or deep sea sediments³⁹ are further
178 away from anthropogenic sources, implying that they may have a higher share of older
179 microplastics. We conclude that the age distribution of microplastic in a given area probably
180 does not show a strong spatial heterogeneity and that the age distribution as given in Figure 1
181 is roughly uniform across the different major oceanic regions.

182 **Comparing HOC equilibration times for microplastic with the age of plastic in the**
183 **oceans.** Several studies have addressed the sorption kinetics under realistic field conditions
184 and by using field-relevant types of microplastics. These studies generally reported desorption
185 half-lives of weeks to 1-2 year for many types of 0.5 – 5 mm sized microplastic for the most
186 hydrophobic classes of HOCs, like PCBs and PAHs^{40,41,42}, with shorter half-lives for the
187 smaller microplastics. Sorption of more hydrophilic compounds generally will be faster,
188 which means that the vast majority of HOCs will be at equilibrium after 2 years, whereas for
189 some very hydrophobic HOCs it may take longer to fully reach equilibrium, especially for the
190 larger macroplastic particles (i.e. > 5 mm). These sorption equilibration times can now be
191 compared to the ages of plastic in the oceans. It follows from the age distribution (Figure 1)
192 that 80 to 90 % of the plastic is older than 2 to 4 years and therefore will be at or close to
193 sorption equilibrium for all HOCs to be considered, including additives and plasticizers.
194 Virtually all plastic will be at equilibrium for the majority of the HOCs with half-lives in the
195 order of months. The remaining cases, i.e. the very hydrophobic HOCs sorbing to the fraction
196 of larger ‘young microplastic’ particles, are in an intermediate state with 50% of equilibrium
197 reached as the best estimated intermediate value. The above estimation most probably under-
198 predicts the magnitude of equilibrium for aged and free-floating microplastics. After all, the
199 above sorption half-lives have been assessed for microplastics in a rather pristine state, based
200 on exposures of up to a year at most. It is known that over longer time scales, embrittlement
201 and abrasion lead to the formation of much smaller particles, cracks and pores³⁷ which

202 increases the kinetics **rates** of sorption due to larger surface area and shorter intrapolymer
203 diffusion paths.^{32,42} **The slow fragmentation of plastic implies that generally the older plastic**
204 **also will be the smaller and thus more equilibrated microplastic, which (because of its smaller**
205 **size) also is likely to be bioavailable for a wider range of aquatic organisms.** Furthermore, part
206 of the aforementioned sorption half-lives were measured for microplastics enclosed in bags
207 with 1.3 and 10 mm mesh,⁴¹ which probably reduced hydrodynamic flow around the particles.
208 This means that the sorption kinetics **might** have been faster when the plastic particles would
209 have been floating freely. A recent paper measured concentrations of PAHs in both the plastic
210 and water phase in the North Pacific gyre and found that partitioning of PAHs between plastic
211 and water was virtually at equilibrium.⁴³ In summary, we conclude that with respect to
212 sorption of HOCs, sorption equilibrium is a valid general assumption for the majority of
213 microplastics currently present in the oceans.

214 **The multi-media distribution of chemicals in the oceans**

215 **The need for calculating the distribution of HOCs across environmental media.** Many
216 papers argue that plastic is highly abundant in the oceans and has very strong binding
217 properties for HOCs, such that plastic probably plays an important role in the transport and
218 transfer of HOCs in the oceans, and in the bioaccumulation of HOCs by marine
219 biota.^{12,13,17,21,22,23} This reasoning links the potential risk and harm of plastic to its presumed
220 role as a carrier of chemicals. This role, however, is also played by all other environmental
221 media such as water, air^{30,32}, and - for HOCs – other carbon-based media such as dissolved
222 organic carbon, organic colloids, black carbon and biota.^{24,32,44,45,46} Therefore, it is important
223 to know the relative quantity of chemicals sorbed by plastics in the oceans, compared to the
224 quantity held by these other media. Marine organisms ingest plastic together with regular prey
225 items, including the abundant detritus fraction, and phyto- and zooplankton species. Exposure

226 to HOCs by the ingestion of plastic may be substantial if the mass of HOC in the plastic is
227 sufficiently large compared to these other 'diet' components. This constitutes another reason
228 to assess the relative quantity of HOCs in plastics compared to other solid phases. This
229 relative quantity is governed by re-partitioning phenomena among environmental media,
230 including plastic. These media thus act as communicating vessels for chemical transfer.

231 **Plastics and other environmental media as communicating vessels.** The principles of
232 partitioning of HOCs across environmental media have been assessed extensively in
233 literature^{32,47,48}, including re-partitioning upon addition or removal of an environmental
234 sorbent or anthropogenic sorbents^{49,50} For instance, addition of sediment in a laboratory
235 bioaccumulation test system reduced chlorobenzene uptake by fish. This reduction could be
236 fully explained by the quantity of sediment added and the chlorobenzene K_p for the
237 sediment.⁵¹ Three decades ago, the effect of activated carbon addition on PCB
238 bioaccumulation by goldfish was tested in the laboratory, resulting in a 70.9–99.9% reduction
239 in PCB fish concentrations.⁵² Adding activated carbon as a remediation method for sediments
240 was also tested in the field, showing a 20-fold reduction in PCB bioaccumulation in golden
241 orfe.⁵³ Similarly, plastics have been studied as cleaning materials for the remediation of soils
242 and sediments.⁵⁴ Such applications require a sufficient quantity of sorbent material in order to
243 cause significant repartitioning and immobilization of HOCs. However, in other applications
244 plastics also have been applied in quantities that are deliberately low compared to those of
245 other environmental media. Polymers like LDPE, POM or silicone rubbers have been applied
246 as negligible depletion passive samplers^{9,55,56}, where the ability of plastic to reach an HOC
247 equilibrium state in weeks or months (see previous section) is used to infer freely dissolved
248 HOC concentrations in water.

249 These are just a few examples of a vast body of literature showing the reversibility of HOC
250 partitioning among media including polymers, where the direction of transfer is determined

251 by a concentration ratio being higher or lower than the equilibrium partition coefficient. Some
252 recent studies have reconfirmed these phenomena in the context of marine debris. For
253 instance, the addition of (clean) plastic to laboratory systems with a finite quantity of HOC
254 was observed to cause a decrease in the HOC concentration in the marine organisms being
255 tested.^{12,13,22} These authors, however, acknowledged that the abundance of plastic in the
256 marine environment would be too low to cause a similar cleaning effect *in situ*, which also is
257 likely for the opposite process. Below we discuss studies that have addressed this question
258 and provide an update of such calculations based on most recent data.

259 **The distribution of HOC across environmental media in the ocean.** The relative role of an
260 environmental compartment in the oceans in transport or transfer can be assessed by
261 calculating the quantity of HOC held by that compartment relative to the other
262 compartments.^{24,30,32} The mass of HOC in a medium is likely to be high if that medium is
263 abundant. Likewise, the mass of HOC in a medium is high if the affinity of the HOC as
264 quantified by the partition coefficient K_P is high for that medium. The total mass of a HOC in
265 a volume within a gyre, sea or ocean (Q_T) can be expressed as

$$266 \quad Q_T = C_W V_W + C_W \sum_{i=1}^n M_i K_{P,i} + C_W M_{PL} K_{PL} \quad (2)$$

267 The first term represents the HOC mass in the water with V_W and C_W being the volume of
268 **water** and the HOC concentration in water, respectively. The second term accounts for the
269 masses in 'n' non-aqueous compartments like dissolved organic carbon, organic colloids,
270 black carbon, detritus, phytoplankton and zooplankton (in this case $n=6$). Biota at higher
271 trophic levels could be included but, even though they may have higher levels of HOCs due to
272 biomagnification, are not accounted for here because of their negligible mass compared to
273 that of the other solid phases present.⁵⁷ In this term, M_i represents the mass of the
274 compartment in the ocean and $K_{P,i}$ the partition coefficient for sorption of the HOC to solid

275 phase 'i', based on the assumption of sorption equilibrium as motivated in the previous
276 section. Similarly, the last terms account for the mass of HOC present in plastic, with M_{PL} the
277 mass of plastic in the ocean compartment. In essence, the products $M \times K_P$ determine the
278 relevance of a phase. For instance, although the affinity of a HOC for plastic could be very
279 high, the role of plastic in transfer and accumulation would still be negligible overall, if the
280 abundance of plastic is low relative to the abundance of other media present. The fraction of
281 the mass of HOC in plastic can be expressed as a fraction of the total mass of HOC as (C_W
282 cancels out):

$$283 \quad M_{PL}K_{PL}/(V_W + \sum_{i=1}^n M_iK_{P,i} + M_{PL}K_{PL}) \quad (3)$$

284 Using this type of calculations, Zarfl and Matthies³⁰ calculated mass fluxes of PCBs, PBDEs
285 and perfluorooctanoic acid (PFOA) to the Arctic. Fluxes for transport of water, plastic and air,
286 were combined with concentrations of HOCs in these media (either estimated from
287 partitioning data, or measured), in order to obtain fluxes of the HOCs. They calculated that
288 the fluxes mediated by plastic were 4 to 6 orders of magnitude lower than those mediated by
289 oceanic currents and air. Similarly, Gouin et al²⁴ defined a representative coastal marine
290 ecosystem in which plastic was present. Using the abundances of media and a wide range of
291 HOC partitioning coefficients, they showed that sorption to polyethylene (PE) would occur
292 for a negligible <0.1 % of the mass of the chemicals. This means that the plastic abundance
293 currently present in coastal waters is insufficient to cause a meaningful redistribution of
294 HOCs from the oceanic environment to the plastic.

295 Recently, new data on the abundance of plastics suggest that there is currently 268,940 tonnes
296 of plastic floating in (all) oceans, of which 35,540 tonnes are < 4.75 mm microplastics.⁵⁸
297 Using these data we calculated the overall average distribution of HOCs across media in the
298 oceans. This update also included media that were thus far not accounted for, like organic
299 colloids, black carbon and zooplankton, next to the common phases water, dissolved organic

300 carbon (DOC) and phytoplankton. Several studies have highlighted the sources and
301 abundance of black carbon in the oceans.^{59,60,61,62} For the present calculations, abundances of
302 black carbon were estimated using data provided by Pohl *et al.*⁶², and for the other organic
303 carbon phases data were taken from Couwet (1978)⁶³ (Figure 2A). The abundances of these
304 phases range from 2.7×10^8 kg for plastic⁵⁸ to 1.4×10^{21} kg (or \sim L) for water⁶⁴ (Figure 2A).
305 This implies that an estimate of the average ‘whole ocean’ concentration of plastic would
306 equate to ~ 2 ng/L. We used one of the highest values for K_{PL} measured for a HOC on
307 microplastic; 10^7 L/kg.¹⁰ Several studies have shown that the suspended sediment partition
308 coefficients $K_{P,i}$ on an organic carbon basis are similar¹⁰ or higher²⁹ than those reported for
309 microplastic. Nevertheless, following Zarfl and Matthies,³⁰ the organic carbon partition
310 coefficients $K_{P,i}$ were set at a conservative value of $0.01 \times K_{PL}$, whereas for black carbon they
311 were set at a conservative $100 \times K_{PL}$.^{45,55,65} This implies that although the masses of plastic and
312 black carbon are estimated to be about equal, the mass of HOC sorbed to black carbon would
313 be about two orders of magnitude higher than to plastic in the ocean (Figure 2B). The
314 distribution calculated using these data and Eq 3 shows that most HOCs are present in the
315 water (Figure 2B, Table SI-1), which implies that oceanic water currents constitute the main
316 transport medium for HOCs across the ocean. Plastic binds 1.93×10^{-4} % of this model HOC
317 (Table SI-1), a percentage that will even be smaller for other combinations of HOC and
318 plastic because we used a worst-case calculation (i.e., highest possible K_P for plastic, low
319 estimates for K_P for the other carbon phases). So, although plastic concentrates HOCs by
320 factors up to 10^7 from the water, plastic is still irrelevant as a carrier phase because the mass
321 of water is about a factor of 10^{13} larger than that of plastic. Even if plastic concentrations
322 (locally) would be orders of magnitude higher than the ‘whole ocean’ average used in our
323 calculation, this would not change the large excess of other media compared to plastic.

324 Solid particles, however, may be more important for the vertical transport of HOCs because
325 of their tendency to settle. However, a similar calculation excluding the non-settling water and
326 DOC shows that colloids, detritus and black carbon nanoparticles would still dominate the
327 solid phase speciation, with only 2×10^{-2} % of HOC bound to plastic (Table SI-1). In
328 summary, these simple calculations confirm the results of earlier studies,^{24,30} showing that the
329 fraction of HOC held by plastic is negligible compared to that held by other media, which
330 implies that plastic-mediated transport is generally unimportant **in terms of HOC masses**. As
331 discussed in the previous section, most plastic particles containing additives at higher than
332 equilibrium concentrations will reach an equilibrium, given their long transport and residence
333 times in the oceans, rendering these calculations also applicable to these chemicals.

334 **The role of microplastic in bioaccumulation of HOCs to marine aquatic** 335 **organisms**

336 **Processes determining the relative importance of microplastic as a carrier of HOCs**
337 **under environmentally relevant conditions.** The mechanisms that explain the effect of
338 microplastic on bioaccumulation in marine organisms have been summarized in several recent
339 reviews,^{13,14,15,16,24,25,26,28,46} whereas numerous papers address these processes separately or in
340 detail. Here we give a brief summary of these isolated mechanisms that affect the role of
341 microplastic as a carrier of HOCs (Figure 3), and provide selected references supporting the
342 occurrence of these mechanisms:

- 343 1. Plastic being ingested leading to HOC transfer from the plastic to the organism
344 ('absorption').^{21,22,23,25,66}
- 345 2. Plastic being ingested leading to increased excretion of HOC from the organism
346 ('cleaning').^{24,25,28,67}

- 347 3. Plastic acting as a source of HOCs in the environment, which subsequently are
348 available for dermal uptake or uptake by the gills ('source').^{13,19,20,24,25}
- 349 4. Plastic accumulating HOCs from the seawater and organisms ('sink').^{12,22,24,25,54}
- 350 5. Desorption of HOCs from plastic followed by uptake by natural organic particles or
351 prey, followed by ingestion of prey ('indirect source - dietary').^{44,46,68}
- 352 6. Uptake of HOC by ingestion of regular (i.e. non-plastic) prey items ('dietary').^{44,46,68}
- 353 7. Uptake of HOC by dermal transfer or transfer across gills from other sources than the
354 plastic ('other source dermal').^{44,46,68}

355 Mechanism 1- 4 are common for all non-digestible sorbents and determine the net uptake flux
356 from the plastic. Studies that consider the mechanisms 1-4 can provide 'proof of principle' or
357 can provide mechanistically relevant knowledge regarding the uptake of HOCs from plastic.
358 Processes 1-4 are supported by several empirical and modelling studies as indicated.

359 Mechanism 2 is less well-recognized in the literature on plastic debris and may need further
360 experimental validation. However, the process is generally well known for animals and
361 humans as it is the essence of medical treatment after acute poisoning with HOCs.⁶⁹ Just like
362 plastic,⁶⁷ liquid paraffin,⁷⁰ olestra^{69,71} or activated carbon⁷² are non-digestible, non-absorbable
363 lipophilic polymeric phases, which have been shown to increase the excretion and decrease
364 the body burden of HOCs.²⁴ Processes 5 and 6 relate to natural pathways of HOC uptake by
365 organisms yet they are crucial to assess the relative importance of processes 1- 4 under
366 environmentally realistic conditions. Process 7 recognizes that HOCs originating from other
367 sources than plastic may be taken up from the water. A recent study for instance, showed that
368 plastic is an important yet not the only source of bisphenol A (BPA) from waste-handling
369 facilities, and that BPA in the leachate from the facilities was freely dissolved and not bound
370 to microplastics.⁷³ All of these studies have helped to further understand the isolated
371 processes. However, to answer the questions whether plastic is a relevant carrier of HOCs in

372 the natural environments, we have to evaluate *all* processes 1 – 7 occurring in realistic
373 environments. This also implies that studies that do not explicitly consider the processes
374 under 5-7, or that neglect the cleaning mechanisms related to plastic (2 and 4) are inherently
375 less informative for answering that question.

376 **Overview and critical evaluation of research approaches.** Studies that assessed the role of
377 plastic as a mechanism for bioaccumulation in the field have been reviewed recently.^{15,16} We
378 are aware of 13 studies (excluding seabirds) that somehow addressed the role of plastic in the
379 bioaccumulation of HOCs in the context of pollution with marine debris (Table SI-2). **All**
380 **these studies provide relevant information concerning this question**; however, they differ in
381 their research approach and in the extent to which they reflect environmental realism. An
382 overview of these studies that characterises the extent to which various processes and natural
383 conditions are accounted for, is provided as Supporting Information (Table SI-2). We divide
384 these studies in three main categories each having their specific merits and flaws, and discuss
385 these hereunder.

386 *Laboratory studies.* A first category of empirical studies has provided evidence for the
387 transfer of HOCs from plastic to biota under controlled laboratory conditions^{21,22,23,66,67,74} and
388 has been reviewed before.^{12,14,16} One study⁷⁴ was performed under environmentally relevant
389 conditions with all exposure pathways accounted for, and reported an increase in
390 accumulation of Σ PCBs in lugworms of 29%. This percentage, however, decreased at a
391 higher plastic dose and it was concluded that the effect could not be attributed to ingestion of
392 plastic.^{25,74} In several other studies, clean^{21,22,23} or relatively clean organisms⁶⁶ were exposed
393 to rather high quantities of HOC spiked plastics, which forces transfer of the HOCs to the
394 organism. Such experiments can be viewed as chemical bioaccumulation or toxicity tests
395 where plastic acts as vector for administering the contaminants to the test chemicals, invoking
396 effects of these chemicals once toxicity thresholds are exceeded. These non-equilibrium test

397 designs confirm earlier work showing that polymers will act as a source or carrier material for
398 HOCs towards media with lower than equilibrium fugacity. For instance, passive dosing is a
399 technique increasingly being used in ecotoxicology to control aqueous phase exposure
400 concentrations during toxicity tests.^{19,20} The main merit of these non-equilibrium set-ups is
401 that HOC transfer from plastic to biota is studied at a maximum possible HOC gradient
402 between plastic and organism, with a limited or negligible role of other uptake pathways like
403 uptake from water or food. This potentially allows the quantification of the parameters that
404 drive chemical transfer from the plastics in the gut, parameters that are urgently needed.^{24,25,28}
405 Because the aforementioned laboratory studies observed bioaccumulation or even adverse
406 effects of chemical exposure, they concluded that ingestion of plastic potentially constitutes a
407 hazard in the oceans. This reasoning, however, is not without problems. First, use of freshly
408 spiked or field-contaminated plastics in clean water leads to chemical desorption to the water,
409 leading to dermal uptake. Therefore, to confirm that the bioaccumulation was from *ingestion*,
410 zero aqueous phase concentrations need to be demonstrated, these data are, however, not
411 provided in these studies (e.g.^{21,22,23,66,74}). Second, the argument that ingested plastics will act
412 as a carrier is correct for chemicals that reside in the plastic at higher than equilibrium
413 fugacity (like additives), which however was shown to occur for only a minor fraction of the
414 plastics in the marine environment, based on desorption half-lives (see above). Third, for
415 chemicals that occur in plastic at lower than equilibrium fugacities, plastic would have an
416 opposite, i.e. beneficial effect. This effect would decrease the hazard due to plastic and
417 usually is not addressed in these studies. Fourth, some studies did not consider uptake from
418 natural exposure routes.^{21,22,23} In most environmentally realistic settings, the concentrations of
419 plastic would be far lower than those used in most of these studies. These studies therefore
420 underestimate the role of natural routes. For instance, the first mentioned experiment⁷⁴ was
421 performed under realistic conditions yet the authors could not clearly show that plastic acted

422 as a carrier for HOCs. Fifth, to confirm the hypothesis that plastic acts as a carrier of toxic
423 chemicals upon ingestion, ingestion of the plastic should be experimentally confirmed, which
424 however was not the case for some studies (Table SI-2).

425 *Model studies.* A second category of studies has applied models to interpret transfer from
426 plastic in a scenario analysis that includes all the other chemical uptake pathways (Figure 3)
427 as well.^{24,25,26,28,75,76} These studies thus provide an environmentally relevant quantitative
428 assessment of the relative importance of plastic as a carrier of chemicals and have been
429 reviewed recently.^{7,15} They combine empirically validated models for bioaccumulation from
430 regular prey with bioaccumulation from ingested plastic. Comparison of the HOC fluxes
431 bioaccumulated from ingested prey with those of ingested plastic, generally showed small to
432 negligible contributions of plastic to bioaccumulation by the various marine species like
433 lugworm, fish and seabirds.^{25,26,28,75,76} These models accounted for cleaning effects due to
434 plastic ingestion,^{24,25,26,28} were used for worst case scenarios setting the uptake from plastic at
435 maximum values (assuming 100% absorption),²⁸ or accounted for uncertainties in parameters
436 and input variables by using probabilistic approaches.²⁶ The main merit of these studies is that
437 they provide a mechanistic basis for understanding plastic-inclusive bioaccumulation, which
438 assists in data interpretation of empirical studies and experimental designs. Furthermore, they
439 allow for environmentally realistic scenario studies and extrapolations to low plastic
440 concentrations that occur in the environment, **or to higher concentrations in the future**. The
441 models that were applied are valid in terms of their agreement to first principles and
442 accordance with design criteria.⁷⁷ However, they can only provide indirect evidence, and lack
443 of validation against empirical data sometimes limits the credibility associated with these
444 modelling studies. We are aware of only two studies that compared model calculations with
445 empirical data,^{25,75} which implies that further validation is recommended (see next section).

446 *Field studies.* A third category of studies proposes to use the observed co-occurrences or
447 correlations among field data on plastic densities or chemical concentrations in plastic, with
448 chemical concentrations in organisms, as evidence supporting the hypothesis of plastic
449 transferring HOCs to organisms.^{38,78,79} The main merit of these field observations is that they
450 represent the ultimate reality of nature, which is the aim of this papers' research question. The
451 main challenge in observational field research, however, is proving causality because any
452 observed phenomenon can in theory be explained by many different mechanisms.⁸⁰ The
453 aforementioned correlations can be explained by several simultaneously acting processes, for
454 instance process 1 - 5 (Figure 3), or from any combination of these. The problem of multiple
455 causality through parallel uptake pathways means that it is difficult to unambiguously and
456 causally link bioaccumulation to ingestion of plastic alone. There is no reason to deny that
457 bioaccumulation of some HOCs can be linked to a high abundance of plastics that may act as
458 a source of these HOCs³⁸ (Figure 3, processes 3 and 5), however, the relative importance of
459 plastic *ingestion* is hard to disentangle. A final challenge associated with field studies is that if
460 statistical rigor is required, sufficient gradient in chemical concentrations, plastic abundance,
461 extent of plastic ingestion and mixing ratio with regular food is required. Such gradients are,
462 however, difficult to find on the scale of the oceans.³⁸

463 We conclude that three categories of studies have discussed the role and importance of
464 microplastic ingestion. They seem to reach different conclusions because they address
465 different hypotheses, different exposure scenarios and have different limitations based on the
466 type of study (i.e. modelling, laboratory or field experiment), which are not always clearly
467 discussed. In summary; laboratory studies that use high doses of only plastic tend to find an
468 effect of ingestion on HOC accumulation. Studies aiming at environmental realism (either
469 lab or model) by accounting for parallel uptake pathways tend to conclude that there is no (or
470 a negligible) effect. Field studies struggle with the problems of multiple causation, lack of

471 gradient and environmental variability, which limits their use to detect the contribution of
472 plastic ingestion to bioaccumulation.

473 **Unifying empirical studies, modelling studies, and theory.** To date, most model scenario
474 studies were prospective studies,^{24,25,26,28} with only two studies also comparing model
475 simulation results with empirical data.^{25,75} These scenario studies aimed at addressing
476 environmentally realistic settings by accounting for chemical bioaccumulation from water,
477 natural diet and ingested plastic. In this section we provide a synthesis of four categories of
478 information: (a) three published laboratory studies that provided evidence for transfer of
479 HOCs from microplastic to biota, (b) published model frameworks that include microplastic
480 as a component of the diet, (c) HOC release rate constants from microplastic measured under
481 gut mimicking conditions, and (d) theoretical estimations for these release rate constants
482 based on first principles. This way, the current model frameworks are further validated as they
483 are tested against published empirical data. The validity of the models was assessed based on
484 whether calibrated parameters align to independently measured parameter values, or whether
485 they agree to values that follow from first principles. In turn, inferences from empirical
486 studies that only addressed the release of HOCs from plastic in artificial gut fluids or that
487 applied plastic-only or plastic-dominated exposure conditions, can be and were extrapolated
488 to natural conditions by using the validated parameters. To that end, we parameterised a
489 previously published bioaccumulation model that includes plastic as one of the components in
490 the diet and that dynamically models HOC transfer in the gut²⁵ (provided as Supporting
491 Information, Table SI-3). The parameterisations and boundary conditions were set to match
492 the experimental designs and data from the studies by Browne et al²¹, Rochman et al⁶⁶, and
493 Chua et al.²² **The main optimization parameter is the rate constant for chemical desorption
494 from plastic in the organisms' gut^{15,25,27} (k_{IG} , d^{-1} , see previously published model provided as
495 Supporting Information), which also is the parameter providing the 'common currency' for**

496 the four categories of information mentioned above. The calibrated k_{IG} parameters were
497 compared (a) with ranges for this parameter that were determined experimentally by Teuten *et*
498 *al*¹² and Bakir *et al*,⁸¹ or recalculated from Tanaka *et al*⁸² (see Table SI-4) and (b) with
499 parameter values inferred from first principles (i.e. plastic particles sizes and HOC intra-
500 polymer diffusivities). For the definition of the previously published model and its
501 parameters, and for the current new calculations the reader is referred to the Supporting
502 Information.

503 *Comparison of k_{IG} values estimated from modelling studies, bioaccumulation studies,*
504 *desorption studies and first principles. Comparison of the magnitude of the rate constant for*
505 *desorption of HOCs from microplastics in the gut (k_{IG})* across different microplastic types and
506 sizes, organisms and chemicals, and obtained with different methods, has a range of about two
507 orders of magnitude (Table SI-5). The 10-90% inter quantile range $PR_{10-90\%}$ for the data in
508 Table SI-5, however, is only 0.3 – 9.8 d^{-1} with a median of 2.1 d^{-1} . For the separate categories
509 of studies the median values as well as the $PR_{10-90\%}$ are virtually identical (Figure 4). This
510 implies that there is a striking consistency among the data obtained for these microplastics of
511 different sizes and polymer types, chemicals and methodologies. The estimates from
512 bioaccumulation studies, laboratory desorption studies and first principles provide very
513 similar ranges for the ‘common currency’ desorption rate parameter k_{IG} . For PAH desorbing
514 from PE and PVC the range is only about 1 – 4 d^{-1} accounting for the bioaccumulation data
515 from Browne *et al*²¹ and Rochman *et al*⁶⁶, the direct desorption measurements of Teuten *et al*¹²
516 and Bakir *et al*⁸¹ as well as the theoretical values provided here (Table SI-5). The PCB k_{IG}
517 value estimated from the data provided by Rochman agrees well with values based on the
518 theoretical principles (Table SI-5).

519

520 **Summarizing Discussion**

521 **The role of microplastic in the transport of HOCs.** It has been shown that plastic is
522 ubiquitous in the marine environment.^{3,4,7} Still, on average the present mass of plastic is
523 negligible compared to that of other media that transfers HOCs across the oceans. We applied
524 a ‘back of the envelope’ calculation that showed that on average the fraction of HOCs sorbed
525 by plastic also is negligible compared to the fraction held by other media (Fig. 2). The
526 fraction held by plastic is so small that even if we would underestimate the abundance of
527 plastic by orders of magnitude, plastic still would be unimportant as a transfer pathway for
528 HOCs. The same reasoning holds with respect to other uncertainties. The calculations used
529 here were based on the assumption of equilibrium partitioning for the main portion of
530 microplastic in the oceans. The outcome reflects the current state of plastic pollution in the
531 oceans, which may be valid for several more years. However, the yearly production of plastic
532 has increased over the years and can be expected to further increase. This implies that the
533 fraction of ‘non-equilibrium plastic’ will increase, and thus that the non-equilibrium fraction
534 will become increasingly important. Non-equilibrium may increase the potential role of
535 plastic as a carrier for additives and plasticizers, and decrease its role as a carrier for POPs.
536 Still, given the abundance of other media that carry the same chemicals, a substantial role of
537 plastic in the transport of chemicals is not likely.

538 **The role of microplastic for the bioaccumulation of POPs, additives or plasticizers in the**
539 **marine environment.** The previous sections provided evidence that showed transfer of HOCs
540 from plastic to organisms or vice versa, dependent on the fugacity gradient that was used in
541 the various study designs. We provided a synthesis that showed that laboratory exposure
542 studies, model studies and field studies all align and can be interpreted with existing theory of
543 bioaccumulation and partitioning of chemicals to hydrophobic phases such as sediment
544 organic matter and plastics. We demonstrated that the uptake of HOCs from plastics by
545 marine biota can be explained from the principles of polymer diffusion. In turn, the diffusion

546 rates explain the release rates observed in the desorption experiments,^{12, 81} which in turn were
547 shown to be able to explain the observed bioaccumulation in experiments in which
548 uncontaminated test animals were fed contaminated plastic.^{21,22} We showed that experiments
549 or field studies that also account for uptake from natural pathways are inherently less sensitive
550 to detect an effect of plastic, which simply represents the situation in nature and is explained
551 from the theory presented here. Indeed, parameters estimated from such an experiment⁶⁶ were
552 consistent with those obtained from laboratory desorption experiments and theoretical
553 principles. The above model calibrations and agreement of its parameters with values
554 independently obtained from experiments, further supports the credibility of the models that
555 have been used to infer the role of plastic as a carrier of HOCs to marine biota under natural
556 conditions.^{24,25,28,75,76} Based on the synthesis we provided here, we suggest that the scientific
557 evidence is consistent, yet that the dichotomy in study outcomes is perceived and probably
558 reflects and is related to different exposure scenarios used in these different studies.

559 The central question we addressed is ‘to what extent plastic ingestion leads to increased
560 uptake of chemicals in nature’. Here we briefly reflect on the studies that specifically aimed at
561 analysing such conditions, taking the aforementioned credibility of validated modelling
562 approaches into account. Gouin et al²⁴ used a model proposed by Arnot and Gobas⁸³ and
563 modelled HOC uptake from a 10% plastic diet. Such a percentage probably is very high for
564 the vast majority of marine organisms living in diverse habitats,^{26,84} and instantaneous
565 equilibrium in the gut was assumed. This implies that a worst case was calculated, because
566 plastic might not fully equilibrate during gut passage. The biomagnification of HOCs was
567 calculated to decline up to 20% upon ingestion of plastic, for chemicals with a log K_{OW}
568 between 6.5 and 7.5.²⁴ In the laboratory, Rochman et al⁶⁶ exposed Medaka to a diet with 10%
569 plastic, and observed increased uptake of HOCs (Σ PAH) up to a factor 2.4. The 10% of
570 plastic in the diet as used in the studies by Gouin²⁴ and Rochman⁶⁶ is quite high compared to

571 conditions in many aquatic habitats²⁶ and thus can be considered to represent a (unrealistic)
572 worst case scenario. Besseling et al⁷⁴ investigated the effects of plastic ingestion on PCB
573 bioaccumulation by *A. marina*, taking all uptake pathways into account. A factor 1.3 increase
574 in bioaccumulation of \sum PCB was found, which however decreased with increasing plastic
575 concentration in the sediment. The increase was ascribed to physical effects of the plastic
576 ingestion and not to transfer of the chemicals from the plastic. Using the data from that study,
577 Koelmans et al²⁵ simulated the experiment and calculated a negligible contribution of plastic
578 in the transfer of PCBs compared to natural pathways. For an open ocean scenario, plastic was
579 calculated to attenuate biomagnification due to its fugacity being lower than that of biota
580 lipids. It was concluded that bioaccumulation due to ingestion by microplastic would be
581 negligible at plastic concentrations presently occurring in oceans sediments²⁵, a conclusion
582 that also was drawn for the modeled leaching of additives and subsequent uptake by
583 lugworms or cod.²⁶ In the previous section we showed that the parameters that were used in
584 these model studies align with values independently obtained from desorption studies, as well
585 as with values inferred from studies that only accounted for transfer from plastic. A fourth
586 model study was published recently by Bakir et al²⁸, who quantified the relative importance of
587 microplastics as a pathway for the transfer of HOCs to marine biota. First, the model was
588 validated using bioaccumulation data without plastic. Measured desorption rates (k_{IG}) from
589 plastics as in Table SI-5 were used and a plastic-inclusive model was implemented for
590 lugworm, fish and seabirds, with all uptake pathways accounted for. Their simulations
591 showed that plastic did not increase bioaccumulation for the lugworm, and decreased
592 bioaccumulation in fish and seabirds due to the ‘cleaning’ effect. However these differences
593 were marginal at the plastic concentration up to 5% dw of the diet. Only at a very high plastic
594 concentration of 50%, accumulation was predicted to increase (lugworm) or decrease (seabird
595 and fish), the latter decrease confirming the analyses by Gouin *et al.*²⁴ and Koelmans *et al.*²⁵

596 We argue that these empirical laboratory studies and model studies agree that up to realistic as
597 well as at very high concentrations of about 1 to 10% of plastic in the sediment or in the diet,
598 about a factor two change of bioaccumulation in either direction may occur. It has been
599 argued recently, that the unrealistic high microplastic exposure concentrations as used in
600 many studies do not provide any information on the current risks to marine ecosystems.⁸⁵
601 Instead, microplastic effect assessments should address more realistic i.e. lower and chronic
602 exposure conditions in sediments⁸⁵, as well as in the pelagic zone.⁸⁶ Under such more realistic
603 environmental conditions, organisms may simply ingest not enough microplastic particles
604 compared to natural prey, rendering the effect on bioaccumulation to be even below a 10-20%
605 difference in either direction.

606 As for field studies, we are not aware of reports that unambiguously quantify the quantity of
607 HOCs accumulated by marine aquatic organisms from ingested plastic, compared to natural
608 pathways. The variability in *in situ* bioaccumulation data has been analysed recently and was
609 shown to be between one to two orders of magnitude.⁶⁸ This implies that a factor of two
610 variation in either direction that can be seen under ideal conditions like in model studies or in
611 the laboratory, will be practically impossible to infer from field data. Effects of plastic
612 ingestion can be concluded to be smaller than the biological variability in bioaccumulation
613 data.⁶⁸ This implies that small effects of microplastic on bioaccumulation of HOCs can be
614 observed under artificial laboratory conditions, but in nature will be overwhelmed by natural
615 variability and by bioaccumulation from natural exposure routes.

616

617 **Implications for risk assessment**

618 Here we address the question whether microplastic ingestion leads to increased risks of
619 chemicals under realistic environmental conditions. The available data suggest that the effects

620 of microplastic ingestion on bioaccumulation probably stay within a factor of two, which is
621 within typical ranges of biological variability among individuals. For the majority of habitats,
622 bioaccumulation of HOCs from microplastic is probably overwhelmed by uptake via natural
623 pathways, a conclusion that also has been reached recently by the GESAMP WG40 working
624 group.⁷ It has been argued that such a carrier effect of microplastic probably also is of limited
625 importance for the risk assessment of HOCs,^{7,25} where assessment factors of 10 to 1000
626 usually are applied to account for variability and uncertainty in the effect assessment.
627 Furthermore, increased bioaccumulation or - magnification (including secondary poisoning)
628 only implies an increase in risk if the effect thresholds are exceeded, and such a formal risk
629 assessment to date has not been performed. To date, most studies aim at identifying a hazard
630 of microplastic ingestion by searching for potentially increased bioaccumulation. However, it
631 also has been argued¹⁵ that microplastic ingestion may increase bioaccumulation for some
632 chemicals in the mixture (additives, plasticizers) yet decrease the body burden of other
633 chemicals at the same time (POPs), in case these chemicals have opposing fugacity gradients
634 between plastic and biota lipids.^{24,25,28} For a balanced risk assessment aimed at protection of
635 populations or habitats, both effects should be considered, in relation to known effect
636 thresholds.

637 Our assessments used average and present oceanic conditions, which in reality will show
638 variation.³⁴ Given the high calculated factors between the current microplastic concentrations
639 and the microplastic concentrations required to cause an effect on chemical transport and
640 bioaccumulation, our assessment is rather robust with respect to such uncertainties. Still, risk
641 assessment should always consider local conditions where needed, and re-apply tests and
642 models for new cases. **The same holds for prospective assessments that have to consider**
643 **increased emissions of plastic.** Nanoplastic constitutes another uncertain factor, because the
644 abundance as well as the potential hazards of nanoplastic have not been addressed.^{18,87} **Our**

645 suggestion that the effects of ingestion of microplastic on bioaccumulation most probably is
646 limited for most marine habitats and therefore hard to confirm by field data does not imply
647 that plastics do not have deleterious effects on marine life.⁸⁸ To answer this ultimate question,
648 more environmentally relevant, long-term effect studies with various species are needed.

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652 to fish oil.

653 **Supporting Information Available**

654 Text, figures and tables addressing the plastic-inclusive bioaccumulation model description,
655 model parameters, model simulation results. Modeling kinetic rate constants for desorption to
656 artificial gut fluids. Calculation of the distribution of HOC across environmental media.
657 Overview of studies that addressed the role of plastic in bioaccumulation of HOCs.

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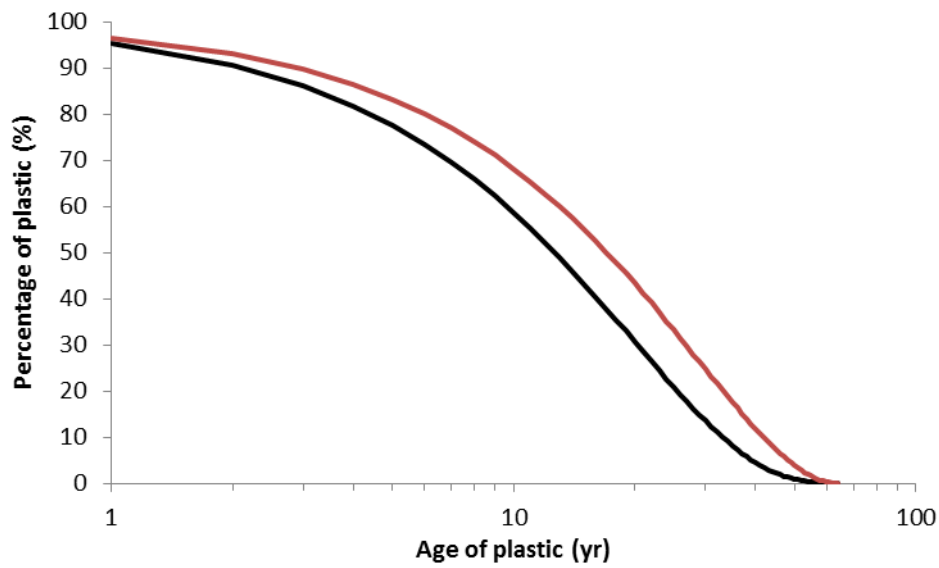
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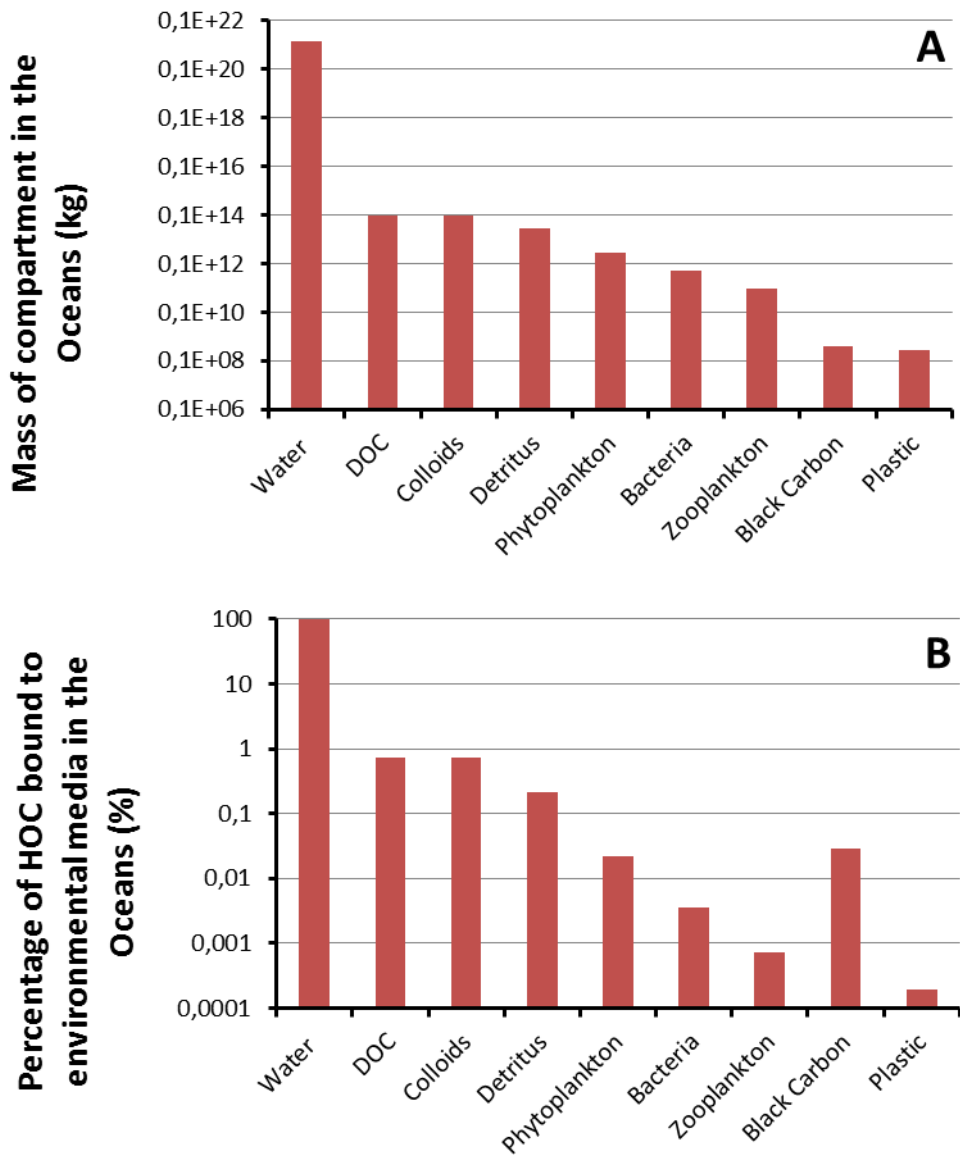
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664 **Figure 1.** Cumulative age distribution of plastic in the oceans based on world plastic
665 production data (—). For about 90% and 50% of the world-produced plastics, the residence
666 time at sea is more than 2 y and 13 y, respectively. The red curve (—) represents the
667 cumulative age distribution based on plastic production in Europe. Using the European
668 production data, the 90% and 50% cut offs relate to residence times at sea of more than 3 y
669 and 17 y, respectively.

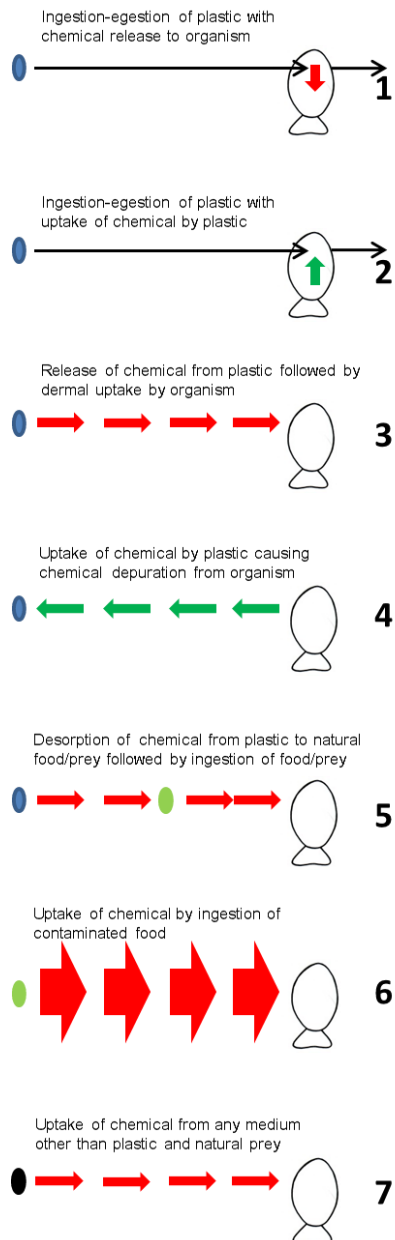
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676 **Figure 2.** Abundances of environmental media in the oceans (Panel **A**), and distribution of
 677 HOC across these environmental media calculated using Equation 2 and 3 (Panel **B**). Water
 678 dominates the HOC distribution holding 98.3% of HOC whereas plastic holds 0.0002 % of
 679 HOC (Panel **B**). The calculations use highest reported K_P values for plastic and low estimated
 680 K_P values for organic carbon phases.

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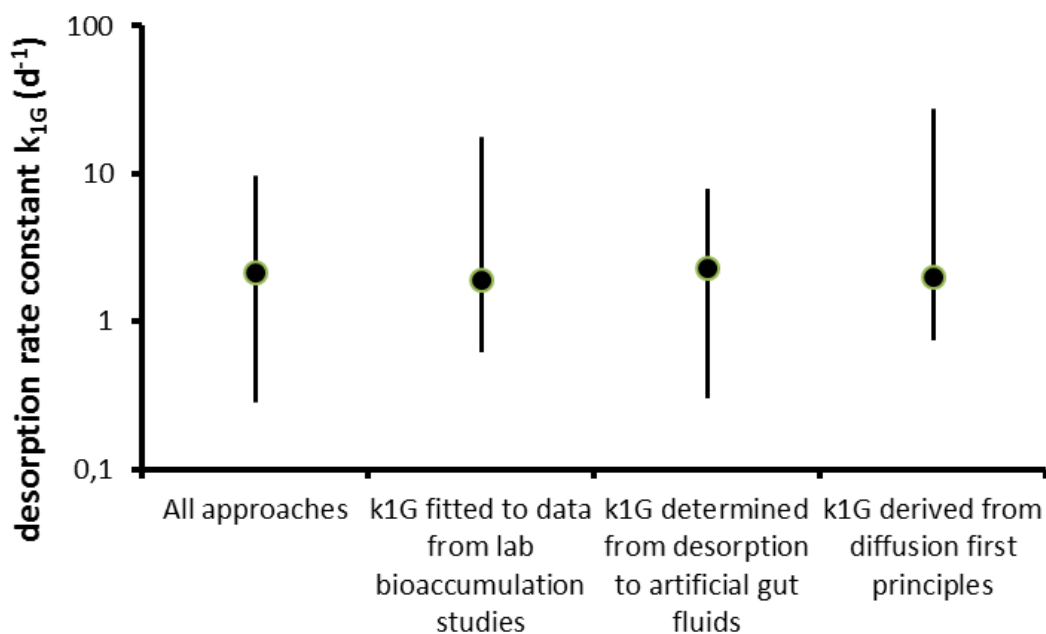
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685 **Figure 3.** Simultaneous processes affecting the relative importance of microplastics acting as
 686 a vector of HOC to aquatic organisms, i.e. fish. ●=MP. ●=Natural prey item. ●=Other
 687 source than MP. Black arrows (→) indicate transfer of MP. Red arrows (→) indicate HOC
 688 transfer to organism. Green arrows (←) indicate HOC transfer from organism. Arrow widths
 689 represent qualitative indication of relative importance of the pathway. Processes 1-5 involve a
 690 role of MP. Processes 5 and 6 involve natural uptake paths. Process 7 considers dermal
 691 exposure from other sources than MP. Per fish individual, processes occur simultaneously for
 692 same as well as different HOC, complicating interpretation of field data. Per fish individual,
 693 processes increasing or decreasing body burdens occur simultaneously for different HOC. Per
 694 HOC, uptake from natural path (6) plus non-MP source (7) generally overwhelms uptake
 695 from MP ingestion (1).

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707 **Figure 4.** Similarity in median value and range (10-90% inter quantile ranges; $PR_{10-90\%}$) of
708 the rate constants (k_{IG} , d^{-1}) for desorption of HOC from plastic in the gut of marine biota, as
709 calculated using data from all study approaches, laboratory bioaccumulation studies,
710 desorption studies using artificial gut fluids, and diffusion principles. Detailed calculation of
711 the ranges is provided as Supporting Information.

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