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Claire Loiseau, Gabriele Sorci

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1 **Discussion Article**

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3 **Can microplastics facilitate the emergence of infectious diseases?**

4
5 Claire Loiseau ¹, Gabriele Sorci ²

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8 ¹ CIBIO-InBIO - Research Center in Biodiversity and Genetic Resources, InBIO
9 Associate Laboratory, Campus de Vairão, 7 Rua Padre Armando Quintas, 4485-661
10 Vairão, Portugal.

11
12 ² Biogéosciences, CNRS UMR 6282, Université de Bourgogne Franche-Comté, 6
13 Boulevard Gabriel, 21000, Dijon, France

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26 **Abstract**

27 Plastic pollution is a major environmental problem. Small plastic particles (called
28 microplastics) have been reported to have pernicious effects on human and wildlife
29 health, by altering physiological functions (e.g., immunity, metabolism) and interfering
30 with commensal microorganisms. However, in addition to these direct toxic effects, we
31 suggest that microplastic pollution might also exert deleterious effects, modifying (i)
32 the exposure to pathogens (e.g., multi-drug resistant bacteria) and (ii) the dynamics of
33 vector-borne diseases. Therefore, we argue that microplastics should be considered as
34 a ubiquitous environmental hazard, potentially promoting the (re)emergence of
35 infectious diseases. The implementation of multi- and interdisciplinary research
36 projects are crucial to properly evaluate if microplastic pollution should be added to the
37 current list of global health threats.

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51 **1. INTRODUCTION**

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53 Since the middle of the 19th century, plastic materials have revolutionized our daily
54 life, for better (e.g., in medical and digital sciences), and for worse (e.g., single-use
55 plastic items), quickly becoming a dreadful visible pollutant (Lavers and Bond, 2017).

56 In addition to large waste, plastic pollution also involves small (<5 mm) particles (e.g.,
57 microfibers, microbeads and particles produced during the degradation of large plastic
58 products), collectively called microplastics (Thompson et al., 2004). Microplastics are
59 everywhere: in oceans and deep-sea (Woodall et al., 2014), in terrestrial ecosystems
60 (Rilling and Lehmann, 2020), in the atmosphere (Fernando Amato-Lourenço et al.,
61 2020) and in organisms ranging from zooplankton (Cole et al., 2015) to vertebrates
62 (Carlin et al., 2020; Hou et al., 2021), including humans (Ragusa et al., 2021; Zhang et
63 al., 2021; Fig. 1).

64

65 The effect of microplastic ingestion on organismal health has been evaluated and now
66 there is evidence showing that microplastics can indeed exert deleterious effects on
67 several physiological and life history traits in both aquatic and terrestrial species (Blarer
68 and Burkhardt-Holm, 2016; Cole et al., 2015; Lo and Chan, 2018; Salerno et al., 2021).

69 A detailed and exhaustive description of the underlying mechanisms is well beyond the
70 scope of this discussion article; however, for instance, once ingested, microplastics can
71 induce the mechanical disruption of the intestinal barrier integrity, causing (i) an up-
72 regulated expression of genes related to oxidative stress, (ii) inflammatory infiltration
73 and production of pro-inflammatory cytokines such as TNF- α , IFN- γ and IL-6, or (iii)
74 the destruction of the structure of the intestinal mucosa (see Huang et al., 2021 for a
75 recent review). Other direct toxic effects arise from the release of additives, plasticizers,

76 trace elements and other chemicals that can provoke endocrine and inflammatory
77 disorders once microplastics are ingested (see recent reviews: Bradney et al., 2019;
78 Callaghan et al., 2021; Jiang et al., 2020; Yilmaz et al., 2020). That said, a proper
79 assessment of the potential deleterious effects of microplastic ingestion requires taking
80 into account environmentally-relevant concentrations of plastic particles (i.e., those to
81 which organisms are likely exposed in their natural environment during their lifespan).

82

83 Another important methodological issue when it comes to the assessment of the health
84 consequences of microplastic pollution is related to the use of virgin microplastics
85 *versus* aged particles, the reason being that microplastics released in the environment
86 are readily colonized by a diversity of microorganisms. More specifically, several
87 studies have reported that microplastics can carry different bacterial communities
88 depending on their types (Steinman et al., 2020) and have also different composition
89 and properties (antibiotic resistant bacteria and antibiotic resistance genes) compared
90 to bacteria found in the surrounding environment (Oberbeckmann et al., 2018; Wang et
91 al., 2021; Wu et al., 2019a; Zhang et al., 2019). Due to their persistence in the
92 environment, microplastics might therefore play a role in spreading potentially harmful
93 bacteria (both for humans and animals) across different environments (marine,
94 freshwater, terrestrial). This would represent an additional, indirect, impact of
95 microplastics on organismal health (Bowley et al., 2021; Prata et al., 2021). In addition,
96 there are several reasons to think that microplastics might also alter the capacity of
97 arthropod vectors to transmit a diversity of pathogens, through their effects on vector
98 microbiota and immunity, or through the effects on interacting species (predators,
99 competitors).

100

101 Despite the concern over the emergence and reemergence of infectious diseases at the
102 local and global scales (Morens and Fauci, 2013; Plowright et al., 2017), the possibility
103 that microplastic pollution might facilitate the spread of pathogenic microorganisms
104 and alter the dynamics of vector-borne diseases have been barely discussed and
105 investigated. In this discussion article, we would like to highlight the link between two
106 current global threats that are the increase in microplastic pollution and the acceleration
107 of disease emergence. There are numerous unknown indirect effects of microplastics
108 that need to be investigated to evaluate if and how microplastic pollution might promote
109 the emergence of novel pathogens, potentially affecting human, livestock and wildlife
110 health. Here, we indicate some research directions that might prove useful to assess the
111 risk that microplastic pollution might exert as promoter of infectious disease
112 (re)emergence. We focus on microplastics because of their ubiquitousness in the
113 environment and their small size, which allows them to be ingested by a large diversity
114 of aquatic and terrestrial organisms. We think they have the potential (i) to act as disease
115 vehicles into organisms more easily than large plastic waste and (ii) to provoke
116 physiological disorders into vectors of infectious diseases.

117

118 **2. MICROPLASTICS CAN ACT AS VECTORS OF ANTIBIOTIC** 119 **RESISTANT BACTERIA**

120

121 2.1. Increased exposure to antibiotic resistant bacteria

122 Virtually all human beings are today continuously exposed to plastic debris through
123 ingestion and inhalation (Zarus et al., 2021). A large amount of evidence has rapidly
124 accumulated showing that we ingest microplastics on a daily basis: several food items
125 and drinking water have been shown to be contaminated by microplastics (Zhang et al.,

126 2021). Similarly, plastic debris with a size $< 1\mu\text{m}$ are suspended in the air and can be
127 inhaled (Fernando Amato-Lourenço et al., 2020; Prata, 2018). Estimates indicate that
128 people living in highly urbanized areas inhale microplastic particles daily (Wright et
129 al., 2020) and this also applies to domestic indoor environments (Vianello et al., 2019).
130 The inhalation route has been corroborated by the finding of plastic particle
131 contamination in human lung biopsies (Fernando Amato-Lourenço et al., 2021).

132

133 Knowing all of this, an important parameter to take into consideration is that living
134 beings get exposed through ingestion and inhalation not only to the microplastics but
135 also to the microorganisms that live on them. The term “plastisphere” was coined by
136 Zettler and colleagues to describe the community of bacterial species that was found on
137 marine plastic debris in the North Atlantic subtropical gyre (Zettler et al., 2013). The
138 main result of their work, corroborated by several other studies, was that
139 microorganisms living on microplastics were not a subsample of the surrounding
140 environment, but rather showed a distinct diversity, composition and functional
141 properties (Amaral-Zettler et al., 2020; Mammo et al., 2020; Wang et al., 2020; Wu et
142 al., 2019a; Xue et al., 2020). Microplastics have several features that promote bacterial
143 colonization compared to the surrounding environment and natural substrates (Amaral-
144 Zettler et al., 2020; Wu et al., 2019a). In particular, bacteria can readily form a biofilm
145 on microplastics, which provides shelter and increases bacterial survival in the face of
146 environmental stressors (Lavery et al., 2020; Wang et al., 2020).

147 Antimicrobial resistance is one of the major health challenges that we will face in the
148 upcoming years (Cassini et al., 2019; Collignon et al., 2018). Several studies revealed
149 that microplastics selectively enrich antibiotic resistant bacteria and genes (ARBs and
150 ARGs; Liu et al., 2021; Sathicq et al., 2021; Wang et al., 2020). Evidence shows that

151 microplastics can harbor microbial communities carrying ARGs with abundances 100
152 to 5,000 times higher than planktonic bacteria (Zhang et al., 2020), although the process
153 of ARGs enrichment depends on the features of the microplastics and their ageing (Su
154 et al., 2021). Since biofilm formation favors horizontal gene transfer, microplastics can
155 become hotspots for ARGs because resistance genes carried by mobile elements can
156 spread more easily in the biofilm compared to planktonic bacterial communities (Arias-
157 Andres et al., 2018). Microplastics can also have affinity for antibiotics that are released
158 in the environment (Li et al., 2018) and for other chemical pollutants (Scott et al., 2021).
159 For example, antibiotics and trace metals can attach to microplastics and exert selection
160 pressures on bacteria living on plastic debris, and may also have a synergistic effect on
161 bacterial resistance due to co-selection (Imran et al., 2019; Yang et al., 2019; Fig. 2).
162 Such synergistic effects can result in strengthened selection on resistant strains, and
163 improve the resistome of microplastic-associated bacteria.

164 Although most studies have focused on microplastics in marine environments, the vast
165 majority of plastic particles come from land-based human activities (Xu et al., 2020).
166 Therefore, the distribution of microplastics can be seen as a mosaic of pollution
167 hotspots from which particles flow into other environments. Several hotspots of
168 microplastic pollution have been identified such as urban wastewater treatment plants
169 (WWTP) (Syranidou and Kalogerakis, 2022). These hotspots of microplastic pollution
170 are however, also hotspots for antibiotics, trace elements, ARBs and ARGs (Rizzo et
171 al., 2013; Zhang et al., 2022). In these environments, microplastics might then serve as
172 sinks for other pollutants and ARBs, and spread them in other environments via
173 leachate, landfill leakage, effluents of WWTPs, sewage sludge (Kelly et al., 2021).
174 Current estimates indicate that 25% of microplastics found in marine environments
175 come from WWTPs (Syranidou and Kalogerakis, 2022). However, although

176 microplastics can successfully contribute to spread ARBs (homogenizing the risk of
177 disease emergence), other environmental factors do contribute to the mosaic of disease
178 emergence risk, including proximity to pollution hotspots and human population
179 density. With this respect, urban WWTPs and landfills should be considered as
180 particularly risky sites, especially when in proximity of megalopolis. Urban
181 environments, highways and roads also produce large amounts of microplastics, such
182 as tire particles that are subsequently found in the soil (Sommer et al., 2018). As other
183 microplastic substrates, tire particles have been shown to harbor microbial communities
184 that differ from the surrounding soil. Bacteria colonizing tire particles also have
185 different sets of ARGs and, of more concern, the abundance of ARGs is enhanced when
186 tire particles are exposed to soils containing other pollutants such as heavy metals and
187 antibiotics (Ding et al., 2021).

188

189 2.2. Risks associated with increased exposome and resistome

190 Microplastics have been reported to carry pathogens of aquatic organisms (fish,
191 crustaceans, mollusks). Isolates of the fish pathogen *Aeromonas salmonicida*
192 expressing virulence factors related to the secretion systems Type II and IV, and several
193 toxins have been retrieved from plastic debris (Radisic et al., 2020; Viršek et al., 2017).
194 These isolates also carried antimicrobial resistance genes (lactamases and
195 chloramphenicol acetyltransferase), which raise the question of the potential impact
196 that microplastics might have as disease vectors in aquaculture (Dong et al., 2021). In
197 the same line, an incubation experiment showed that, compared to surrounding
198 seawater, mariculture-derived microplastics harbor (i) distinct bacterial communities as
199 the exposure time increases, (ii) a higher abundance of bacteria of the genera *Vibrio*,
200 *Pseudoalteromonas* and *Alteromonas* (Sun et al., 2020). The idea that microplastics

201 might be vehicles of pathogenic microorganisms in aquaculture systems is further
202 supported by recent evidence showing that oysters (*Oestra edulis*) have a higher uptake
203 rate of microplastics coated with bacteria compared to virgin particles. In addition,
204 oysters exposed to bacteria-coated microplastics have increased oxygen consumption
205 and respiration rate compared to oysters exposed to virgin microplastics (Fabra et al.,
206 2021). Therefore, benthic filter feeders are likely to be preferentially exposed to
207 bacteria-carrying microplastics and as such transfer them over the trophic chain, up to
208 humans.

209 Similarly, several human pathogens (including opportunistic ones) have been
210 discovered on plastic debris in different geographical regions and environments (Table
211 1). For instance, enteropathogenic strains of *Vibrio* and *Escherichia coli* have been
212 reported on microplastics sampled at the Guanabara Bay (Brazil) while water samples
213 had low bacterial titers (Silva et al., 2019). Hence, microplastics can substantially
214 contribute to spread such pathogens from areas with fecal bacteria contamination to
215 preserved zones. If these preserved zones involve recreational environments, such as
216 beaches and bathing waters, we might easily predict an enhanced human exposure to
217 pathogenic organisms conveyed by microplastics (Keswani et al., 2016).

218 We suggest that the combined effect of microplastic-associated exposome and
219 resistome might enhance the risk of emergence of novel bacterial pathogens. Although
220 increased exposure and increased antimicrobial resistance alone represent risk factors
221 for the emergence of bacterial diseases, microplastics might play the role of catalyzers
222 favoring the exposure to resistant strains. In addition to this, microplastics carrying
223 ARBs and ARGs might contribute to spread resistance genes over large distances, even
224 in areas with no antibiotic pollution and to homogenize globally the risk of emergence
225 of bacterial pathogens (Fig. 2). The example of polystyrene recovered in Antarctica,

226 harboring microorganisms able to resist multiple drugs, including cephalosporin and
227 quinolone (Laganà et al., 2019) should act as a serious red flag.

228 Although most of the evidence supporting the role of microplastics as vectors of
229 pathogens comes from aquatic environments, the possibility that particles suspended in
230 the air might also serve as vectors for pathogens, including airborne viruses, cannot be
231 discarded. For instance, it has been suggested that SARS-CoV-2 might have longer
232 survival on air suspended plastic particles than in the aerosol, contributing to the
233 transmission and the spread of the pathogen (Liu and Schauer, 2021). ARGs have
234 already been reported in airborne bacteria sampled in a WWTP in China, including the
235 opportunistic human pathogens *Comamonas* and *Moraxella* (Li et al., 2016). If these
236 airborne bacteria can use microplastics as aquatic microorganisms do, there might be a
237 risk of environmental contamination well beyond the surroundings of WWTPs.

238

239 2.3. Summary

240 Here, we argued that microplastic pollution is likely to pose a threat to human and
241 wildlife health, not only because of direct toxic effects due to the ingestion of plastic
242 particles, but also due to the exposure to pathogenic microorganisms carrying
243 antimicrobial resistance genes. Our argument is based on a series of well-established
244 observations showing that microplastics harbor communities of microorganisms that 1)
245 differ from those found on natural substrates or as planktonic forms, and 2) present a
246 greater abundance of ARBs and ARGs. From these two observations, we consider that
247 microplastics, by increasing the contact rate between hosts and pathogens, could
248 facilitate the emergence of multi-drug resistant bacterial pathogens and pose a threat to
249 humans, livestock and wildlife species. Actually, we might draw a parallel with the
250 recent emergence of bacterial pathogens associated with the use of medical devices

251 (such as prostheses and catheters). Bacteria can form biofilms on such devices (Pelling
252 et al., 2019; Stickler, 2008; Tande and Patel, 2014) as they do on microplastics and
253 several new pathogens are now associated with the use of urinary catheters (*Aerococcus*
254 spp., *Actinobaculum schaalii*, *Alloscardovia omnicolens*; Vouga and Greub, 2016). We
255 suggest that the potential health and economic costs associated with the emergence of
256 novel multi-drug resistant microorganisms is worth to stimulate more research in this
257 area, as to better quantify the risk associated with microplastic pollution.

258

259 **3. MICROPLASTICS CAN ALTER VECTOR-BORNE INFECTIOUS** 260 **DISEASE TRANSMISSION**

261

262 Emerging pathogens have been associated not only with antimicrobial resistance, but
263 also with the spread of arthropod vectors (Kilpatrick and Randolph, 2012). In fact, in
264 addition to parasites that are directly or environmentally transmitted, wildlife and
265 humans are exposed to multiple vector-borne pathogens, many of them being
266 considered as emerging or reemerging (e.g., Dengue, Zika, West Nile viruses; Jones et
267 al., 2008; Morens et al., 2004). Historically, vector control has relied on synthetic
268 insecticides and environmental management of breeding sites, but several novel vector
269 control tools are under development, such as the gene-drive technology or bacterial
270 infection of vectors (e.g., *Wolbachia*; Wilson et al., 2020). We suggest that microplastic
271 pollution, as a ubiquitous environmental hazard, should be taken into account in vector
272 control strategies since it could interfere either with life history traits of vectors and/or
273 their microbiota.

274

275 3.1 Alteration of vector life history traits

276 Many medically important insect-vectors or intermediate hosts of infectious diseases
277 (i.e., mosquitoes, black flies, snails) are freshwater organisms inhabiting lakes, rivers,
278 swamps or coastal ponds that can be heavily polluted with microplastics (Akindele et
279 al., 2020; Wagner and Lambert, 2018; Windsor et al., 2019;). Microplastics
280 contamination is not uniformly distributed around the globe and varies with freshwater
281 ecosystems. Although lakes are generally more contaminated than rivers, and riverine
282 and lacustrine sediments are more contaminated than the aquatic matrix (Cera
283 et al., 2020), microplastics in lotic ecosystems are more studied than in lentic systems.
284 In addition, invertebrates are largely understudied compared to vertebrates (i.e., fish;
285 Cera et al., 2021). Nonetheless, it has been shown that freshwater invertebrates have
286 the capacity to ingest microplastics, the quantity of uptake depending on their feeding
287 type and morphology (Scherer et al., 2017; Setälä et al., 2016). Interestingly,
288 experimental work found that adult mosquitoes do not avoid water contaminated with
289 microplastics when ovipositing, and that larvae do ingest microplastics that remain at
290 the adult stage with, however, no impact on their survival rate (Al-Jaibachi et al., 2018;
291 Al-Jaibachi et al., 2019; Cuthbert et al., 2019).

292 Depending on the vector species and their ecological niches, microplastics might have
293 variable influence on vector's life history traits and population dynamics. Even so, we
294 can expect various outcomes of the microplastic ingestion by the vectors, either direct
295 effects on development, growth or survival, or indirect effects on vectorial capacity
296 (Fig. 3). Vectorial capacity (VC) can be defined as a measurement of the efficiency of
297 vector-borne disease transmission (Garrett-Jones et al., 1964), corresponding to the
298 basic reproductive rate of a vector-borne pathogen. Vectorial capacity can be assessed
299 using the following equation:

300
$$VC = \frac{ma^2bp^N}{-\ln(p)}$$

301 where m refers to vector density, a to vector biting rate, b to vector competence (the
302 susceptibility of the vector to become infected and transmit the pathogen), p to the
303 probability of vector daily survival, and N to the pathogen extrinsic incubation period
304 (Cansado-Utrilla et al., 2021). Therefore, when vectors get easily infected, have high
305 survival rates, high density, and bite at high rate, they effectively transmit the disease.
306 We propose that microplastics have the potential to affect all these parameters (e.g.,
307 impairing vector survival, or decreasing extrinsic incubation rate).

308

309 The effect of microplastic pollution on vector-borne diseases can be nonetheless
310 particularly difficult to predict because of the complex ecological interactions that
311 operate at different temporal and spatial scales. Microplastics might decrease parasite
312 transmission if they have negative effects on vectors (reducing fecundity and/or
313 survival). It has been well documented, for example, that decreasing the survival of
314 adult *Anopheles* (the mosquitoes that transmit human malaria) reduced their vectorial
315 capacity (Brady et al., 2016). Negative impacts of microplastics have also been reported
316 on food intake, growth, reproduction or survival in freshwater invertebrates (Foley et
317 al., 2018). Therefore, snail-borne parasitic diseases, such as fascioliasis or
318 schistosomiasis, could be less prevalent if microplastic pollution increases aquatic snail
319 mortality and/or decreases cercariae release in water.

320

321 3.2. Modification of vector microbiota

322 Microplastics, and the associated ‘plastisphere’, might interact with the vector
323 microbiota and immune system and consequently increase the vectorial capacity.
324 Recent work has focused on the effects of microplastics on the microbiota (Fackelmann
325 et al., 2019) and the immune system in model organisms - in mice (Jin et al., 2019; Lu

326 et al., 2018) and zebra fish (Jin et al., 2018; Limonta et al., 2019; Qiao et al., 2019) -
327 and invertebrates (Ju et al., 2019; Zhu et al., 2018). For example, it has been shown that
328 ingestion of microplastics by dipteran larvae triggers the activation of phenoloxidase,
329 one of the effectors of innate immunity (Silva et al., 2021a), but also increases oxidative
330 damage (Silva et al., 2021b). Therefore, we might expect that insect vectors (such as
331 black flies transmitting nematodes causing river blindness, or mosquitoes transmitting
332 *Plasmodium* causing malaria) could have an altered vectorial capacity if the ingestion
333 of microplastics affects their physiology (e.g., the immune response) and/or the
334 microbiota. In fact, the microbiota has an important role in modulating the mosquito
335 immune response and can alter susceptibility to infection through either immune
336 priming or direct inhibition (Caragata et al., 2019). It has been shown that the removal
337 of the midgut bacteria through antibiotic treatment led to a greater susceptibility
338 of *Anopheles gambiae* to *Plasmodium falciparum* and of *Aedes aegypti* to dengue
339 virus infection (Dong et al., 2009; Xi et al., 2008). Bacteria can also influence mosquito
340 competence by impairing pathogen infection (Cirimotich et al., 2011a; Cirimotich et
341 al., 2011b). On the contrary, the presence of members of the Enterobacteriaceae family
342 in the mosquito midgut favors *Plasmodium* infection (Boissière et al., 2012), while
343 another recent study reported that *Aedes* mosquitoes harboring a *Serratia*
344 *marcescens* strain were more permissive to dengue virus infection than those free of *S.*
345 *marcescens* (Wu et al., 2019b). These few examples show the complexity of the
346 interactions between pathogens and microbiota that could affect mosquito vectorial
347 capacity. Considering that microplastics often lead to dysbiosis and alter immune
348 responses, we suggest that mosquitoes, or other arthropod vectors, ingesting
349 microplastics could see their susceptibility to pathogens decrease or increase,
350 depending on the bacteria and contaminants that microplastics carry (Fig. 3). Finally,

351 little is known regarding how the microbiome influences the extrinsic incubation period
352 (EIP; Cansado-Utrilla et al., 2021), but it has been shown that *Wolbachia* could increase
353 the EIP of the dengue virus in *Aedes aegypti* (Carrington et al., 2018). It cannot be
354 excluded that on the opposite, the plastosphere could shorten the EIP and have a positive
355 effect on pathogen transmission.

356

357 3.3. Changes in the trophic chain

358 We propose that microplastics might have even more subtle effects on the transmission
359 of vector-borne diseases, by altering the population dynamics of interacting species
360 (predator - prey, competing species). Even if microplastic pollution has no direct effect
361 on vector species, it may have one on natural enemies that play an essential role to
362 regulate the abundance of vectors, which is one of the factors, that influences the
363 vectorial capacity. Pathogen transmission could drastically increase if natural enemies
364 fail to regulate the vector proliferation (Fig. 3). For example, numerous aquatic insects
365 or amphibians are regulating mosquito larvae density either by direct predation (Quiroz-
366 Martínez and Rodríguez-Castro, 2007) or even by indirect stress caused by predators
367 (Bellamy and Alto, 2018). A study found that mosquito size, fecundity and longevity
368 decreased with indirect effects of predation, which would result in a significant decrease
369 of malaria transmission (Roux et al., 2015). On the opposite, if predators are negatively
370 affected by microplastic pollution, vectors would benefit from it and see their vectorial
371 capacity enhanced. In agreement with this hypothesis, a recent meta-analysis showed
372 that feeding activity of fish, such as the predatory performance or number of ingested
373 prey, was consistently impaired in the presence of microplastic pollution (Salerno et
374 al., 2021). Finally, human-driven alteration of top-down regulation of vector
375 populations has also been reported. An emblematic example is the building of a dam

376 on the Senegal River that was associated with an outbreak of schistosomiasis, due to
377 the interference with the migration of prawns, one of the main predators of the snails
378 that are the intermediate hosts of schistosome parasites. Reintroduction of prawns
379 resulted in reduced abundance of infected snails and reduced prevalence of
380 schistosomiasis (Sokolow et al., 2015).

381

382 Despite the manifold reasons to expect an effect of microplastic pollution on the
383 dynamics of infectious diseases, studies on the relationship between the exposure of
384 microplastics and risk of parasite infection in wildlife, livestock or humans are so to
385 speak inexistent. Only two correlative studies found positive association between
386 accumulation of microplastics and presence of parasites - in grey seals (Hernandez-
387 Milian et al., 2019) and fishes (Grazia Pennino et al., 2020), and only one experimental
388 study investigated the effects of microplastic ingestion in tadpoles and trematodes
389 (Buss et al., 2021). The latter study performed an experimental microplastic exposure
390 during one day (i.e., 24h) and found that individuals that ingested microplastics did not
391 show a significant increase in parasite infection risks. However, one can argue that the
392 time of exposure might not reflect the likely experience of wild organisms, which are
393 rather chronically exposed to this pollutant.

394

395 3.4. Summary

396 Here, we argued that microplastic pollution is likely to modify vector-borne disease
397 dynamics via two pathways, either by altering component of the vectorial capacity or
398 by modifying the interactions between species along the trophic chain.

399 First, our argument is based on studies showing that arthropods, either as larvae or
400 adults, are capable of ingesting microplastics, which are known to interfere with the

401 microbiota of organisms. Because infection dynamics of pathogens (viruses or
402 parasites) are regulated by the microbiota, we hypothesize that microplastics could
403 facilitate the emergence of vector-borne diseases by increasing the vectorial capacity.
404 Second, we believe that even if microplastics do not have a significant effect on the
405 vector microbiota and immunity, they might have undesirable consequences on
406 predators and/or competitors, which could fail to regulate vector populations.
407 To date, and to our knowledge, no study has investigated the impact of microplastics
408 on vectorial capacity, which is surprising given the possible health and economic
409 consequences. More research in this area should be performed to properly evaluate the
410 emergence risk of vector-borne diseases associated with microplastic pollution.

411

412 **4. RESEARCH DIRECTIONS**

413

414 We argued here that microplastic pollution might promote infectious disease emergence
415 through different pathways. Although there exists evidence supporting this view, we
416 realize that the link between microplastics and the dynamics of infectious diseases (in
417 particular vector-borne diseases) has been barely investigated. As such, we hope that
418 this work will contribute to stimulate further investigation to better understand how
419 microplastics may interfere with antimicrobial resistance and the (re)emergence of
420 vector-borne diseases.

421 Whatever the aspect of microplastic contamination we want to address, it is worth
422 reminding ourselves that whether in laboratory conditions or in the field,
423 environmentally realistic concentration of microplastics should be used (Lenz et al.,
424 2016), while avoiding primary and pristine microplastics that are not representative of
425 the contamination *in situ*. The age, shape, size and composition (additives and

426 contaminants) of microplastics as well as their bacterial communities are parameters
427 that should be better apprehended (Waldman and Rillig, 2020). We realize however
428 that identifying environmentally-relevant microplastic concentrations is already a
429 challenging task because it does not only depend on the presence of microplastics in
430 the environment but also on the actual exposure of organisms (spatially and
431 temporally), which is linked to ecological and physiological traits of the studied species
432 (e.g., feeding habits, lifespan). This suggests that environmentally-relevant
433 concentrations might even differ between species living in the same areas.

434 Our knowledge on the many-fold impacts of microplastic pollution on virtually all
435 levels of biological organizations (from gene expression to ecosystem functioning) is
436 rapidly expanding. Research work has been built on a variety of species, environments,
437 experimental contexts, and has provided indisputable evidence showing that
438 microplastics do represent a major environmental concern. However, as we learn more,
439 we think it is time to place greater emphasis on the possible role that microplastic
440 pollution might also play as promoter of infectious diseases emergence.

441 Taking this into account, we would like to provide some suggestions for future research.

442

443 *How does urbanization influence bacteria colonization on microplastics and infection*
444 *risk?*

445 We need to better understand how the process of microplastic colonization by
446 microorganisms changes along the gradient of anthropogenic disturbance. Heavily
447 polluted environments are hotspots for multi-drug resistant pathogens, however
448 microplastics have the potential to spread them at very large geographical scales. Long
449 term surveys of bacteria richness and diversity on microplastics should be performed
450 in soil and aquatic environment along gradient from cities and intensive agricultural

451 lands where the trace metals and antibiotics are most likely to occur, to wilderness areas
452 that present low human population density. This might allow us to draw risk maps,
453 providing a useful tool to guide decisions on where monitoring effort should be
454 prioritized.

455

456 *Can microplastics biofilm interfere and undermine mosquito control methods?*

457 Future work should focus on the possible effects of microplastics on vector control
458 strategies. *Wolbachia*-based strategy for controlling the transmission of dengue and
459 other arboviral infections has been largely investigated and field releases have already
460 occurred in different places (Kamtchum-Tatuene et al., 2017). However, microplastics
461 might be able to modify bacterial communities and/or their interactions, and potentially
462 wipe off the suppressive effects of *Wolbachia* on arboviroses. Biocontrol strategies
463 should definitely take into consideration this increasing environmental pollution.

464

465 *Does aquatic environment influence interaction between microplastics and population*
466 *dynamics of vectors?*

467 More effort should be put in the understanding of the environmental traits that might
468 act in synergy with microplastic pollution. For instance, microplastics present in
469 freshwater versus brackish water, or in small versus large body of water might affect
470 differently the population dynamics of vectors. This is not a trivial point since many
471 species of insect vectors or intermediate hosts have different ecological requirements
472 and habitat preferences. We should investigate which ecological and biogeographical
473 parameters are more likely to impact vector and intermediate host life history traits and
474 vectorial capacity in the context of chronic exposure to microplastics.

475

476 *Do microplastics change the population dynamics of vectors by reducing the*
477 *abundance and/or behavior of natural enemies?*

478 We advocate for a more holistic approach to the study of microplastics, that explores
479 the effects at the ecosystem level. Up to now, most of the studies have focused on the
480 direct effect of microplastic pollution on the species of interest. Nevertheless, the
481 abundance of vectors and intermediate hosts of human infectious diseases can
482 dramatically change if predators decline in number in polluted areas. We suggest that
483 future work should implement a more integrative approach to assess the effect of
484 microplastics on ecosystem functioning.

485

486

487

488

489 **5. CONCLUSIONS**

490

491 Microplastics are clearly not a classical pollutant, in the sense that they are ubiquitous
492 and highly persistent, which make them today an accepted indicator of the anthropocene
493 (Zalasiewicz et al., 2016). Evidence is rapidly accumulating showing that microplastics
494 can have pernicious health effects on organisms that ingest/inhale them due to their
495 direct toxicity. In this discussion piece, we draw the attention to an additional pernicious
496 effect of microplastic pollution. We argue that microplastics can facilitate the
497 emergence of infectious disease by increasing the exposure of bacterial pathogens to
498 human, livestock and wildlife and by altering the dynamics of vector-borne infections.
499 We suggest that if microplastics do play a role promoting the (re)emergence of
500 infectious diseases (both multi-drug resistant bacteria and vector-borne pathogens),

501 they will pose a more serious threat in terms of public health than contaminants
502 absorbed by microplastics (e.g., persistent organic pollutants; Amelia et al., 2021). Our
503 argument is based on two lines of reasoning: first, once an infectious disease has
504 emerged, the whole population of susceptible individuals is potentially at risk,
505 irrespective of whether they are exposed to the source of the emergence or not. The
506 COVID-19 pandemic perfectly illustrates this point. Although SARS-CoV-2 is
507 supposed to have initially infected humans exposed to animals carrying the virus, at a
508 very local scale, human-to-human transmission rapidly made the whole human
509 population at risk (including those not exposed to SARS-CoV-2 reservoirs). Obviously,
510 this argument does not apply to non-transmissible diseases induced by contaminants.
511 The second line of reasoning relies on the potential that microorganisms have to adapt
512 to novel environmental conditions through evolutionary changes. These evolutionary
513 changes might contribute to increase (or decrease) infectivity and virulence, depending
514 on the selective forces operating on the pathogens and the epidemiological context.
515 Again this provides a very different scenario compared to inert toxic compounds that
516 are not expected to become more toxic in response to natural selection.

517

518 We are convinced that interdisciplinary research projects are necessary to address all
519 these questions and to propose global maps of emergence risks of infectious diseases
520 associated with microplastic pollution, as it already exists with climate change. We are
521 aware that such investigations are difficult to launch because it has to bring together
522 experts from different biological fields, such as ecotoxicologists, microbiologists,
523 ecologists, chemists, epidemiologists and modeling scientists. Because the framework
524 that allow such holistic approach already exists into the 'One Health' concept (Zinsstag

525 et al., 2011), researchers should be encouraged to develop studies both in controlled
526 environments and *in situ* in order to anticipate future public health issues.

527

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533

534 **AUTHOR CONTRIBUTIONS**

535 C.L. and G.S. conceived and wrote the manuscript together. C.L. produced the paintings
536 in the figures.

537

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884

885 **Table 1.** A non-exhaustive list of bacterial species that can potentially cause human diseases (including opportunistic pathogens) found on
886 microplastics. We report the location and the environmental type to illustrate the diversity of sites and environments where microplastics have the
887 potential to carry pathogenic bacteria.

Class	Species	Location	Environmental type*	Reference
<i>Alpha Proteobacteria</i>	<i>Brevundimonas spp</i>	China (Beijing)	sewage	(Zhao et al., 2020)
<i>Beta Proteobacteria</i>	<i>Aquabacterium spp</i>	China (Beijing)	sewage	(Zhao et al., 2020)
	<i>Neisseria spp</i>	China (Beijing)	sewage	(Wang et al., 2021)
<i>Gamma Proteobacteria</i>	<i>Acinetobacter beijerinckii</i>	Norway (Øygarden)	intertidal zone	(Radisic et al., 2020)
	<i>Acinetobacter spp</i>	China (Guangzhou)	lake water	(Gong et al., 2019)
	<i>Acinetobacter spp</i>	China (Urumqi river)	river water	(Xue et al., 2020)
	<i>Aeromonas popoffii</i>	Norway (Øygarden)	intertidal zone	(Radisic et al. 2020)
	<i>Alteromonas spp</i>	China (Sungo Bay)	mariculture, shoreline	(Sun et al., 2020)
	<i>Enterobacter spp</i>	Ireland (Carlow)	river	(Murphy et al., 2020)
	<i>Escherichia coli</i>	UK (Scotland)	bathing beaches	(Rodrigues et al., 2019)
	<i>Escherichia coli</i>	Brazil (Guanabara Bay)	mangrove, agricultural, urban areas	(Silva et al., 2019)
	<i>Escherichia coli</i>	Ireland (Carlow)	river	(Murphy et al., 2020)
	<i>Legionella spp</i>	China (Beijing)	sewage	(Zhao et al., 2020)
	<i>Legionella spp</i>	China (Beijing)	sewage	(Wang et al., 2021)
	<i>Morganella morganii</i>	Norway (Øygarden)	intertidal zone	(Radisic et al., 2020)
	<i>Pseudoalteromonas spp</i>	China (Haihe estuary)	estuarine	(Li et al., 2019)
	<i>Pseudoalteromonas spp</i>	China (Sungo Bay)	mariculture, shoreline	(Sun et al., 2020)
	<i>Pseudomonas aeruginosa</i>	Texas (Gulf of Mexico)	coastal	(Sun et al., 2021)
	<i>Pseudomonas mendocina</i>	China (Haihe river)	river water	(Wu et al., 2019a)
	<i>Pseudomonas monteilii</i>	China (Haihe river)	river water	(Wu et al., 2019a)
	<i>Pseudomonas spp</i>	Singapore	coastline	(Curren et al., 2019)
	<i>Pseudomonas spp</i>	China (Urumqi river)	river water	(Xue et al., 2020)
	<i>Vibrio cholerae</i>	Brazil (Guanabara Bay)	mangrove, agricultural, urban areas	(Silva et al., 2019)
<i>Vibrio cholerae</i>	USA (Chesapeake Bay)	estuarine	(Lavery et al., 2020)	

	<i>Vibrio mimicus</i>	Brazil (Guanabara Bay)	mangrove, agricultural, urban areas	(Silva et al., 2019)
	<i>Vibrio parahaemolyticus</i>	North and Baltic sea	estuarine and coastal areas	(Kirstein et al., 2016)
	<i>Vibrio parahaemolyticus</i>	USA (Chesapeake Bay)	estuarine	(Lavery et al., 2020)
	<i>Vibrio vulnificus</i>	Brazil (Guanabara Bay)	mangrove, agricultural, urban areas	(Silva et al., 2019)
	<i>Vibrio vulnificus</i>	USA (Chesapeake Bay)	estuarine	(Lavery et al., 2020)
	<i>Vibrio spp</i>	Singapore	coastline	(Curren et al., 2019)
	<i>Vibrio spp</i>	UK (Scotland)	bathing beaches	(Rodrigues et al., 2019)
	<i>Vibrio spp</i>	China (Sungu Bay)	mariculture, shoreline	(Sun et al., 2020)
<i>Epsilon Proteobacteria</i>	<i>Arcobacter spp</i>	UK (Humber estuary)	estuarine	(Harrison et al., 2014)
	<i>Arcobacter spp</i>	USA (Chicago)	streams	(McCormick et al., 2016)
	<i>Arcobacter spp</i>	Singapore	coastline	(Curren et al., 2019)
	<i>Arcobacter spp</i>	Ireland (Carlow)	river	(Murphy et al., 2020)
	<i>Arcobacter spp</i>	China (Beijing)	sewage	(Wang et al., 2021)
	<i>Helicobacter spp</i>	Ireland (Carlow)	river	(Murphy et al., 2020)
<i>Actinobacteria</i>	<i>Corynebacterium spp</i>	China (Shanghai)	landfill leachate	(Su et al., 2021)
	<i>Mycobacterium spp</i>	China (Beijing)	sewage	(Zhao et al., 2020)
	<i>Mycobacterium spp</i>	China (Beijing)	sewage	(Wang et al., 2021)
	<i>Mycobacterium spp</i>	China (Henan province)	soil	(Yi et al., 2021)
<i>Clostridia</i>	<i>Clostridium perfringens</i>	Ireland (Carlow)	river	(Murphy et al., 2020)
	<i>Clostridium spp</i>	Singapore	coastline	(Curren et al., 2019)
	<i>Clostridium spp</i>	China (Guangzhou)	lake water	(Gong et al., 2019)
	<i>Clostridium spp</i>	China (Beijing)	sewage	(Wang et al., 2021)
<i>Flavobacteria</i>	<i>Chryseobacterium spp</i>	China (Guangzhou)	lake water	(Gong et al., 2019)

888 * Environmental type refers to both *in situ* sampling and microplastic exposure to environmental samples under experimental
889 conditions.

890 **Figure captions**

891

892 **Figure 1.**

893 Microplastics are pervasive pollutants found in virtually all environments (black
894 diamonds: 1- aquatic systems, such as oceans, rivers, ponds or swamps; 2- terrestrial
895 systems, such as agricultural land, meadows or forests). Sources of microplastic
896 pollution are also widespread, involving plastic emission from (asterisks): 1- cities,
897 villages, 2- roads, 3- sewage sludge, 4- atmospheric deposition. Particles smaller than
898 1µm can persist as suspended particles in the air. Environmental effects of microplastic
899 pollution involve a diversity of mechanisms. (A) and (B) refer to the possible
900 consequences of microplastic pollution on disease emergence in two specific
901 environments, as detailed in Figure 2 and 3, respectively.

902

903 **Figure 2.**

904 Bacteria along with fungi or algae can readily form a biofilm on microplastics (here
905 only bacteria are represented to not overload the figure). First, bacteria, both resistant
906 (red cells) and susceptible ones (blue cells) are attaching to the fragment of microplastic
907 (1). Binding of trace metals (2) and antibiotics (3) to microplastics can strengthen the
908 selection on resistance genes, resulting in increased exposure of humans to multi-drug
909 resistant bacteria. Biofilm i) confers protection from environmental stress and enhance
910 bacterial persistence, ii) favors horizontal gene transfer potentially promoting the
911 spread of resistant bacteria over susceptible ones.

912 The combination of enhanced bacterial persistence and the longevity of microplastics
913 makes them ideal vehicles of potentially pathogenic bacteria from polluted sites (e.g.,
914 wastewater treatment plants where microplastics, antibiotics, trace metals, multi-drug

915 resistant bacteria can be found at high concentration) to natural environments.

916

917 **Figure 3.**

918 Microplastics can exert direct and indirect effects on invertebrate vectors (e.g.,
919 mosquitoes) or intermediate species (e.g., snails) of human infectious diseases (e.g.,
920 malaria, dengue, schistosomiasis, river blindness). Direct toxicity of microplastics on
921 vector's life history traits might result in a reduction of disease transmission risk.
922 However, the disruption of vector homeostasis due to plastic debris and/or the
923 interference of the plastisphere with the vector's microbiota might also result in a better
924 vectorial capacity, and increase the risk of disease transmission. Even in the absence of
925 toxicity in vectors, microplastic pollution might affect their population dynamics
926 through changes in the abundance of interacting species (i.e., predators and
927 competitors), producing an increased risk of disease transmission.

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940 Figure 1

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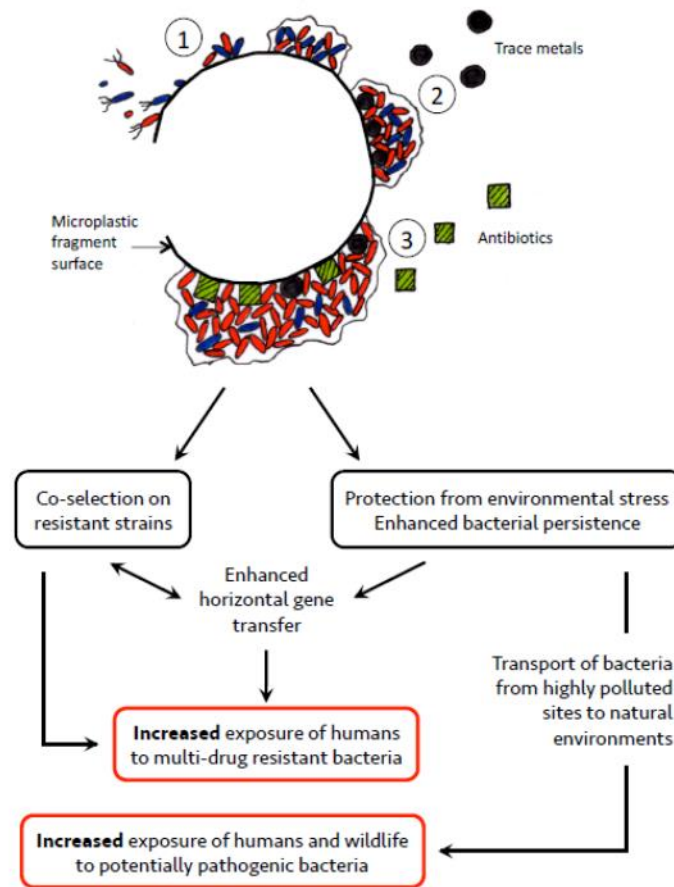
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953 Figure 2

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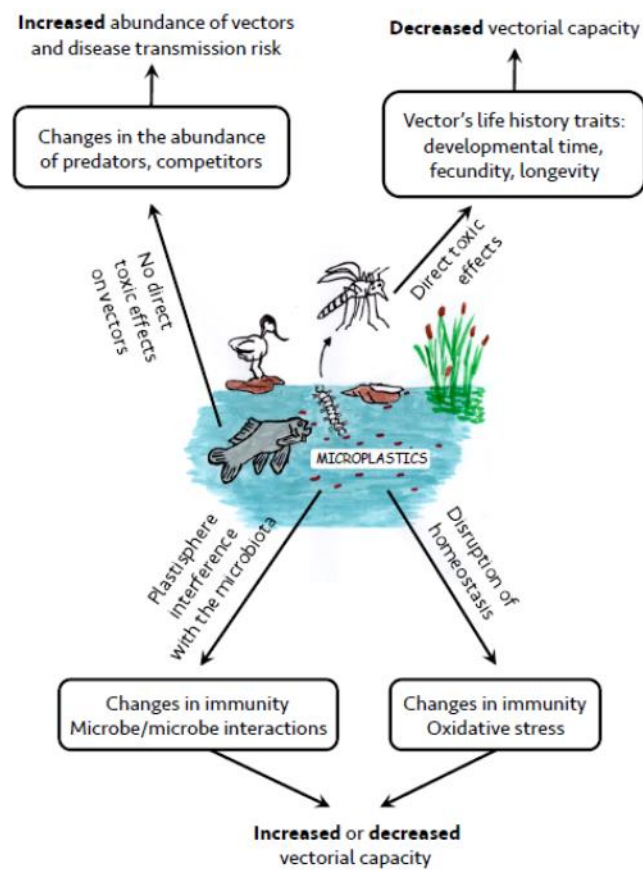
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963 Figure 3

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